



AMERICAN ACADEMY™
OF OPHTHALMOLOGY

Academy
MOC Essentials®
Practicing
Ophthalmologists
Curriculum
2017–2019

Comprehensive Ophthalmology

Protecting Sight. Empowering Lives.™

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The Practicing Ophthalmologists Curriculum was developed by a group of dedicated ophthalmologists reflecting a diversity of background, training, practice type and geographic distribution.

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The Academy gratefully acknowledges the contributions of the American Association for Pediatric Ophthalmology and Strabismus.

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Background on Maintenance of Certification (MOC)

Developed according to standards established by the American Board of Medical Specialties (ABMS), the umbrella organization of 24 medical specialty boards, Maintenance of Certification (MOC) is designed as a series of requirements for practicing ophthalmologists to complete over a 10-year period. MOC is currently open to all Board Certified ophthalmologists on a voluntary basis; time-limited certificate holders (ophthalmologists who were Board Certified after July 1, 1992) are required to participate in this process. All medical specialties participate in a similar process.

The roles of the American Board of Ophthalmology (ABO) and the American Academy of Ophthalmology relative to MOC follow their respective missions.

- The mission of the American Board of Ophthalmology is to serve the public by improving the quality of ophthalmic practice through a process of certification and maintenance of certification that fosters excellence and encourages continual learning.
- The mission of the American Academy of Ophthalmology is to protect sight and empower lives by serving as an advocate for patients and the public, leading ophthalmic education, and advancing the profession of ophthalmology.

The role of the ABO in the MOC process is to evaluate and to certify. The role of the Academy in this process is to provide resources and to educate.

Organization of the POC

The Practicing Ophthalmologists Curriculum comprises 10 practice emphasis areas (PEA), plus Core Ophthalmic Knowledge.

- Core Ophthalmic Knowledge (a required segment for the ABO's MOC examinations.)
- Comprehensive Ophthalmology
- Cataract/Anterior Segment
- Cornea/External Disease
- Glaucoma
- Neuro-Ophthalmology and Orbit
- Oculoplastics and Orbit
- Pediatric Ophthalmology/Strabismus
- Refractive Management/Intervention
- Retina/Vitreous
- Uveitis

In addition to two practice emphasis areas of choice, every diplomate sitting for the DOCK examination will be tested on Core Ophthalmic Knowledge. The ABO defines Core Ophthalmic Knowledge as fundamental knowledge every practicing ophthalmologist should have regardless their practice focus.

Each PEA is categorized into topics presented in an outline format for easier reading and understanding. These outlines are based on a standard clinical diagnosis and treatment approach found in the Academy's Preferred Practice Patterns. For each topic, there are Additional Resources that may contain journal citations and reference to textbooks that may be helpful in preparing for MOC examinations.

Creation of the POC

The POC was developed by panels of Academy members who are practicing ophthalmologists in each of the ten practice emphasis areas. The panels reflect a diversity of background, training, practice type and geographic distribution. Additionally, all panel members are time-limited certificate holders actively participating in the MOC process.

The panels have reviewed the [ABO's content outlines](#) for the MOC examinations and developed and clinical review topics that they feel are most likely to appear on MOC examinations. These clinical topics also were reviewed by representatives from each subspecialty society.

Revision Process

The POC is revised every three years. The POC panels will consider new evidence in the peer-reviewed literature, as well as input from the subspecialty societies, and the Academy's Self-Assessment Committee, in revising and updating the POC.

Prior to a scheduled review the POC may be changed under the following circumstances:

- A Level I (highest level of scientific evidence) randomized controlled trial indicates a major new therapeutic strategy
- The FDA issues a drug/device warning
- Industry issues a warning

Comprehensive Ophthalmology

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Anterior polar cataracts

I. Describe the approach to establishing the diagnosis

A. Describe this disease and its etiology

1. Opacity of the anterior subcapsular cortex and capsule
2. Frequently autosomal dominant

B. List the pertinent elements of the history

1. Usually asymptomatic
2. Symptoms of glare

C. Describe pertinent clinical features

1. Often good vision
2. Central small opacity involving the anterior capsule
3. Frequently bilateral
4. Nonprogressive, usually

II. List the differential diagnosis

A. Penetrating capsular trauma

B. Traumatic stellate cataract

C. Anterior lenticonus

III. Describe patient management

A. Define medical therapy options

1. Eyeglasses or contact lenses

B. Define surgical therapy options

1. Phacoemulsification/ extracapsular cataract extraction (ECCE)
 - a. Capsulorrhexis may be challenging as the anterior capsule is often attached to the anterior polar cataract

IV. List the complications of treatment, their prevention and management

A. Complications of cataract surgery

1. Increased risk of radial capsular tear
 - a. Begin capsule tear away from polar cataract
 - b. Enlarge and encompass polar cataract if possible
 - c. Consider use of capsule staining

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.25-26.

3. AAO, Ophthalmology Monographs 30. A Compendium of Inherited Disorders and the Eye. 2006.

Posterior polar cataracts

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Opacity of the posterior subcapsular cortex and capsule
2. Familial autosomal dominant when bilateral; sporadic cases are usually unilateral
3. Slowly progressive

B. List the pertinent elements of the history

1. Nodal point location; more symptomatic than anterior polar cataract

C. Describe pertinent clinical features

1. Often relatively good vision
2. Central opacity involving the posterior capsule
3. Glare
4. Vision may be more impaired in bright light than in dim light

II. List the differential diagnosis

A. Posterior subcapsular cataract

B. Penetrating capsule trauma

C. Mittendorf dot

III. Describe patient management in terms of treatment and follow-up

A. Define medical therapy options

1. Mydriatic eyedrops as a temporizing agent
2. Eyeglasses and contact lenses for any refractive error

B. Define surgical therapy options

1. Phacoemulsification/extracapsular cataract extraction
2. No hydrodissection but hydrodelineation may be useful
3. Consider low flow, low vacuum surgery

IV. List the complications of treatment, their prevention and management

A. Complications of cataract surgery

1. Increased risk of posterior capsular tear with vitreous prolapse since capsular opacity may weaken the posterior capsule or hide a pre-existing capsular defect
2. Increased risk of loss of lens material into vitreous

V. Describe appropriate patient instructions

A. Risk and benefit of cataract surgery

B. Careful discussion of greater risks for capsule rupture and sequelae

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 26.

Ectopia lentis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Displacement of the lens
 - a. Congenital
 - b. Developmental
 - c. Acquired

B. Define the relevant aspects of epidemiology of this disease

1. Trauma is most common cause
2. Greater than 50% of patients with Marfan syndrome exhibit ectopia lentis

C. List the pertinent elements of the history

1. Decreased vision
2. Monocular diplopia
3. Glare
4. Poor near vision

D. Describe pertinent clinical features

1. Subluxed or total luxation of the lens
2. Phacodonesis
3. Marked lenticular astigmatism
4. Iridodonesis
5. Impaired accommodation
6. May dislocate into anterior chamber and cause pupillary block
7. Amblyopia

II. Define the risk factors and differential diagnosis

A. Traumatic

B. Non-traumatic

1. Primarily ocular
 - a. Pseudoexfoliation
 - b. Simple ectopia lentis
 - c. Ectopia lentis et pupillae
 - d. Aniridia
 - e. Congenital glaucoma
 - f. Chronic uveitis
2. Systemic
 - a. Marfan syndrome
 - b. Homocystinuria
 - c. Other rare systemic congenital syndromes

III. Describe patient management in terms of treatment and follow-up

A. Define medical therapy options

1. Eyeglasses for subluxed lenses
2. Aphakic contact lens in cases of significant subluxation or luxation

B. Define surgical therapy options

1. Intracapsular cataract extraction
2. Phacoemulsification/ extracapsular cataract extraction
 - a. Attend to any vitreous in anterior chamber
 - b. Stabilization of capsule with hooks
 - c. Capsular tension ring or segment with possible scleral fixation
3. Lensectomy/ pars plana vitrectomy

C. Rehabilitation following lens removal

1. IOL in bag - mild cases or aided by capsular tension ring
2. Iris fixated posterior or anterior IOL
3. Angle supported IOL
4. Sulcus sutured posterior chamber IOL
5. Contact lens or eyeglasses

IV. List the complications of treatment, their prevention and management

A. Complications of lens removal

1. Increased risk of vitreous loss
2. Increased risk of loss of lens material into vitreous
3. Increased risk of retinal detachment

V. Describe disease-related complications

A. Pupillary block glaucoma

B. Referral for work up of potential systemic complications of Marfan, homocystinuria, etc.

VI. Describe appropriate patient instructions

A. Genetic counseling in nontraumatic cases

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Por YM, Lavin MJ, Techniques of intraocular lens suspension in the absence of capsular/support. *Surv Ophthalmol.* 2005 Sept-Oct;50(5):429-62.

Hypermaturation cataract/morgagnian cataract

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Opacification of the cortical lens fibers
2. Swelling of the lens material creates an intumescent cataract
3. Hallmark of Morgagnian cataract is liquified cortex allowing the nucleus to move freely in the capsular bag
4. Degenerated cortical material leaks through wrinkled capsule

B. Define the relevant aspects of epidemiology of this disease/risk factors

1. Aging
2. Smoking
3. Trauma
4. Uveitis
5. Prolonged use of topical or systemic corticosteroids
6. Diabetes mellitus
7. Prior intraocular surgery
8. UV light exposure
9. Poor nutrition
10. Delay in treatment

C. List the pertinent elements of the history

1. Prior trauma, surgery, eye disease
2. Progressive protracted loss of vision
3. Photophobia and redness if phacolysis occurs

D. Describe pertinent clinical features

1. Poor fundus view
2. Loss of red reflex
3. May have phacolytic glaucoma or signs of anterior chamber inflammation
4. May have shallow anterior chamber due to lens swelling
5. Wrinkled anterior capsule
6. Calcium deposits in lens
7. Dense, white cortical material
8. Dense brown nucleus freely moving in capsular bag

E. Describe appropriate laboratory testing

1. B scan ultrasound to rule out other intraocular pathology
2. HgA1c

II. Describe the patient management in terms of treatment and follow-up

A. Define medical therapy options

1. Topical steroids to decrease inflammation

B. Define surgical therapy options

1. Phacoemulsification/extracapsular cataract extraction
 - a. Capsulorrhexis techniques
 - i. Capsule staining
 - ii. High viscosity viscoelastic
 - iii. Removal of liquid cortical material
 - iv. Additional viscoelastic to flatten anterior dome of capsule
2. Dense lens with high phaco energy gives consideration for manual expression of lens
 - a. Extracapsular cataract extraction
 - b. Small incision cataract surgery

III. List the complications of treatment, their prevention and management

A. Complications of surgery

1. Increased risk of capsular rupture with loss of lens into vitreous
2. Increased risk of vitreous loss
3. Increased risk of zonular dialysis

IV. Describe disease-related complications

A. Phacolytic or phacomorphic glaucoma

B. Exotropia

V. Describe appropriate patient instructions

A. Discuss benefits and risk of complex cataract surgery

B. Discuss risk of phacolytic and phacomorphic glaucoma with delayed surgery

C. Discuss risk of deprivational strabismus leading to diplopia after surgery. May require additional medical or surgical therapy to correct diplopia

D. Inability to evaluate posterior segment

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 15-16.

Cortical cataract/nuclear cataract/posterior subcapsular cataract

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Cortical cataract - opacification of the cortical lens fibers
2. Nuclear cataract - hardening and yellowing of the central lens fibers. Advanced nuclear sclerosis leads to dense brunescent nucleus
3. Posterior subcapsular cataract (PSC) - central opacification of the posterior cortical material with granular and plaque-like opacities

B. Define the relevant aspects of epidemiology/risk factors of these types of cataracts

1. Cortical cataract
 - a. Smoking
 - b. UV light exposure
 - c. Diabetes mellitus
 - d. Poor nutrition
 - e. Trauma
2. Nuclear sclerosis
 - a. Advanced age
 - b. History of smoking
 - c. Female gender
 - d. Family history
 - e. Lower education levels
 - f. History of Intraocular Surgery
 - g. High myopia
3. Posterior subcapsular (PSC)
 - a. Typically, younger, though may occur at any age
 - b. Prior intraocular surgery, especially vitrectomy
 - c. Use of corticosteroids (topical, inhaled or systemic)
 - d. History of intraocular inflammation
 - e. Diabetes
 - f. Trauma
 - g. History of periocular radiation
 - h. Alcoholism
 - i. Retinitis pigmentosa

C. List the pertinent elements of the history

1. Progressive loss of vision, more rapid with PSC and cortical cataracts
2. Glare

3. Monocular diplopia
4. Myopic shift and decreased color discrimination and contrast sensitivity with nuclear cataracts
5. Worsening of vision in bright lights and at near with PSC

D. Describe pertinent clinical features

1. Cortical cataract
 - a. Opacification of cortical lens fibers
 - b. May have water vacuoles in lens cortex and wedge-shaped whitish opacities extending from periphery of lens toward the center
 - c. May progress to form white intumescent cortical cataract
2. Nuclear sclerotic cataract
 - a. Central yellow to brown discoloration of the lens
 - b. Myopic shift
 - c. Relative shallowing of the anterior chamber
3. Posterior subcapsular cataract
 - a. Granular or plaque-like opacities on anterior aspect of posterior lens capsule, often central
 - b. Frequently fast-progressing
 - c. Decreased clarity of fundus details

II. Describe patient management in terms of treatment and follow-up

A. Define medical therapy options

1. Optimize eyeglasses/contact lens correction
2. Optimize lightening for reading
3. Follow-up examinations at appropriate intervals

B. Define surgical therapy options

1. Phacoemulsification/extracapsular cataract extraction
2. For cataracts sufficiently dense to obscure the intraoperative red reflex, capsule staining may improve ease and safety of capsulorrhexis

III. Describe appropriate patient instructions

A. Discuss benefits, risks and complications of cataract surgery

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Preferred Practice Pattern. Cataract in the Adult Eye. AAO. 2014.

Ophthalmic biometry

I. List the indications/contraindications

A. Indications

1. Calculation of intraocular lens (IOL) power required for surgical correction of refractive error
2. Evaluation of anisometropia

B. Contraindication

1. Open globe injury

II. Describe the pre-procedure/therapy evaluation

A. Refractive power of the cornea (keratometry)

1. Corneal topography

B. Axial length (biometry)

III. List the alternative techniques for biometry

A. Contact

1. A-scan

B. Non-contact

1. Immersion A-scan
2. Laser interferometry

IV. Describe technique to minimize errors

A. Describe appropriate character of good spikes and reproducibility

B. Comparison of two eyes

C. Comparison to refraction

D. Adjustment for phakia, aphakia, pseudophakia, silicone oil

V. List the complications of errors in measurement, their prevention and management

A. Errors in measurement

1. Keratometry inaccuracies
 - a. Contact lens wear
 - i. Out of CTL for a time, longer for RGPs
 - b. Prior keratorefractive surgery
 - i. Uncertainty about central corneal power
2. Axial length
 - a. Compression with A scan probe - shorten axial length
 - b. >0.3 mm difference in axial length (equates to approximately 1 diopter error in IOL calculation)
 - i. Repeat measurements

- ii. Independent technician
 - iii. Alternate biometry technique
- c. Poor fixation or failure to find the visual axis accurately
 - i. Staphylomas
 - i) Laser interferometry
 - ii. Mature cataract, dense PSC
 - i) Consider B-Scan

VI. IOL Formulas

- A. Various formulas employ keratometry readings, axial length and other physical characteristics of the eye to predict the refracting power of the eye with a specific power IOL
- B. Consider fourth-generation or modern theoretical formulas for eyes with especially long or short axial lengths

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2013-2014.
- 2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 152-162.
- 3. Hennessy MP, Franzco, Chan DG. 2003. Contact versus immersion biometry of axial length before cataract surgery. J Cataract Refract Surg. 29:2195-8.

Ophthalmic viscosurgical devices functions during surgery

I. Ophthalmic viscosurgical devices (OVDs) rheologic and physical properties

A. Elasticity

1. Tendency of an object to go back to its original size and form

B. Viscosity

1. Measure of the resistance of a solution to flow

C. Pseudoplasticity

1. Ability of the solution to transform under pressure from a gel to a more liquid substance

D. Cohesiveness/dispersiveness

1. Cohesive OVDs
 - a. The material tends to adhere to itself, not ocular tissue
 - b. Are able to maintain space or remain in place, and displace and stabilize tissues until subjected to turbulence from high flow of fluid through the chamber (high shear)
 - c. Tend to be easily aspirated and rapidly removed from the eye
 - d. Tend to flow out of the eye during phacoemulsification
 - e. May block the trabecular meshwork and cause the intraocular pressure (IOP) to rise
2. Dispersive OVDs
 - a. Tend to adhere to the surface of the tissue, instrument or implant
 - b. As there is little tendency for self-adherence, material is more likely to fracture than to aspirate in one bolus
 - c. Tend to remain in the eye adjacent to the corneal endothelium, providing protection during phacoemulsification
 - d. Can be used to move and isolate intraocular tissues
 - e. Poorly effective in maintaining space and sometimes difficult to remove
 - f. Reduced tendency for IOP elevation compared with cohesive OVDs
3. Other categories
 - a. "Viscoadaptive" and "viscous dispersive" used to describe OVDs with properties that overlap categories

II. Functions during surgery

A. Create and maintain space in the anterior segment

1. Maintain anterior chamber during incisions
2. Flatten anterior capsule during capsulorrhexis

B. Protect cells and tissues

C. Manipulation of tissues

1. Moving iris (repositing prolapsed iris, creating space in sulcus for instruments/implants)
2. Separating anterior or posterior synechiae
3. Viscomydriasis

4. Sequestering vitreous in posterior segment in cases of capsular or zonular disruption
5. Viscodissection of capsule during IOL exchange
6. Maintenance of capsule contour in cases of zonule laxity

D. Lubrication of tissues, instruments and implants

III. Complications

A. IOP increase

1. Due to incomplete removal of OVD
2. Treatment of known or suspected retention of OVD at time of surgery
 - a. Installation of long-acting intraocular miotics
 - b. Topical application of glaucoma medication
 - c. Short term oral carbonic anhydrase inhibitors
3. Treatment of elevated IOP postoperatively
 - a. Topical application of glaucoma medication
 - b. Short term oral carbonic anhydrase inhibitors
 - c. "Burping" the paracentesis at the slit lamp
 - d. Anterior chamber washout

B. Incision burn

1. Caused by occlusion of tubing
2. More likely with a dispersive OVD and brunescant cataract

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Bissen-Miyajima H. Ophthalmic viscosurgical devices. *Curr Opin Ophthalmol*. 2008;19(1):50-4.
3. Goldman JM, Karp CL. Adjunct devices for managing challenging cases in cataract surgery: capsular staining and ophthalmic viscosurgical devices. *Curr Opin Ophthalmol* 2007;18:52-7. Review.
4. Vajpayee RB, Verma K, Sinha R, et al. Comparative evaluation of efficacy and safety of ophthalmic viscosurgical devices in phacoemulsification [ISRCTN34957881]. *BMC Ophthalmol* 2005;5:17.

Extracapsular cataract extraction

I. List the indications/contraindications

A. Indications

1. High risk of complications with phacoemulsification in surgeon's judgment, e.g., weak zonules, shallow anterior chamber, brunescant lens, corneal endothelial dystrophy
2. Conversion to large incision extracapsular cataract extraction (ECCE) may be indicated if a significant portion of the nucleus is present following posterior capsular rupture or phacoemulsification unit malfunction

B. Contraindications = relative (disadvantages compared to phacoemulsification)

1. Combined trabeculectomy or presence of a prior bleb
2. Increased potential of suprachoroidal hemorrhage or of poor patient cooperation
3. Scleral thinning disorders such as scleritis and ectasia

II. Describe the instrumentation, anesthesia, and technique

A. Anesthesia

1. Regional injection
 - a. Peribulbar
 - b. Retrobulbar
 - c. Subtenon
2. General anesthesia under special circumstances

B. Instrumentation

1. Phacoemulsification machine not required
2. Option for manual or automated irrigation/aspiration (I/A) equipment

C. Technique

1. Speculum, +/- superior rectus bridle suture
2. Conjunctival peritomy followed by scleral cautery
3. Incision construction - typically placed superiorly
4. Inject ophthalmic viscosurgical device (OVD)
5. Capsulotomy
 - a. Can opener useful as small diameter continuous curvilinear capsulorhexis (CCC) may impede nucleus delivery
 - b. Capsulorhexis is an option, but must be of adequate diameter relative to nuclear size or make relaxing capsular incisions
6. Nucleus extraction
 - a. Bimanual expression
 - b. Vectus or lens loop extraction
7. Partial wound closure to allow chamber maintenance for irrigation/aspiration
8. Cortex removal
9. Inject OVD
10. Intraocular lens (IOL) placement
11. Wound closure/suturing

12. Conjunctival closure
13. Patch and shield

III. Advantages compared to phacoemulsification

- A. Potential for decreased intraoperative costs**
- B. Certain complications less likely (wound burn, endothelial trauma from excessive ultrasound time, tissue trauma from phacoemulsification tip)**

IV. Disadvantages compared to phacoemulsification

- A. Larger incision**
 1. More difficult to control anterior chamber depth
 2. Not self-sealing in case of intraoperative emergency (choroidal effusion/hemorrhage)
 3. Less forgiving of intraoperative external pressure (Valsalva, coughing, lid squeezing, speculum pressure, etc.)
 4. Conjunctival trauma
 - a. Disadvantage if bleb present or combined procedure needed
 - b. Less virgin conjunctiva available for future trabeculectomy
 5. Not appropriate for topical anesthesia
 6. Increased iris trauma leading to increased likelihood of intraoperative miosis or postoperative iris deformity
 7. Increased suture and incision-induced astigmatism - both early and late postoperatively
 8. Increased risk of incision complications (early and late)
 9. Need for greater physical restrictions postoperatively
 10. Potential prolonged postoperative refractive instability
 11. Long term refractive instability (astigmatism drift)
 12. Suture removal may be necessary
- B. Nucleus is not usually fragmented**
 1. Requires larger capsulorhexis
 - a. Often too large to overlap the optic x 360 degrees
 - b. Small diameter CCC may impede nucleus delivery
 2. More difficult with smaller pupils

V. Follow up care and instructions (differences compared to phacoemulsification)

- A. Globe protection may be required for a longer period of time**
 1. Protective shield advised during sleep
- B. Initial physical restrictions**
 1. Avoid Valsalva and dependent head position
 2. Avoid eye rubbing
- C. Suture-induced astigmatism**
 1. Tight sutures may need to be cut
 2. Refractive stability and final refraction delayed
 3. More frequent postoperative visits may be needed

D. Increased postoperative inflammation

1. Due to larger incision and perhaps greater iris trauma during surgery

E. Long-term wound-induced refractive instability

1. Longer term against -the-incision drift in astigmatism with large, superior incision
2. More frequent changes in refraction for several years

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Focal Points: Strategies for Complicated Lens Surgery, Module 8, 2005.

Ultrasound and cataract extraction

I. Nuclear emulsification - mechanism

A. Ultrasound - principles

1. Metal phacoemulsification (phaco) tip vibrates at high frequency
2. Motion may be longitudinal, torsional, elliptical or a combination
3. Vibration produces mechanical and cavitational effects at the tip which break apart lens tissue and create a repelling force
4. Ultrasound energy can damage ocular tissue (e.g. corneal endothelium) as a result of turbulence and frictional heat
5. Greater amounts of ultrasound power
 - a. Generate more tissue destruction and cutting ability
 - b. May be necessary for denser grade nuclei
 - c. Generate greater heat, increasing potential for wound burn
 - d. May cause endothelial cell trauma and corneal edema

II. Ultrasound power modulations

A. Refers to the ability to program the machine to vary the power delivery pattern

B. Continuous mode

1. When foot pedal is activated, the tip is constantly vibrating
2. Typically used for sculpting of the nucleus

C. Pulse mode

1. When foot pedal is activated, ultrasound automatically cycles on and off
2. Often used for evacuating nuclear quadrants and fragments

D. Burst mode

1. When the foot pedal is activated, a single burst of phaco energy is delivered
2. As pedal is further depressed, bursts occur more frequently
3. May be used to impale the nucleus during chopping

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Anterior capsulotomy - capsulorrhexis and can opener

Continuous curvilinear capsulorrhexis (CCC)

I. List the advantages

- A. Increases the resistance of the capsular bag to tearing during phacoemulsification
- B. Allows for optimal centration of the intraocular lens (IOL) in the capsular bag,
- C. CCC that overlaps the edge of the optic x 360 degrees decreases incidence of posterior capsular opacification and may reduce edge dysphotopsias
- D. After CCC, anterior capsule can support sulcus fixated IOL (with or without CCC-optic capture) if the posterior capsule is compromised

II. List the contraindications

- A. Cannot visualize the anterior capsule
- B. CCC may need to be abandoned in the setting of a tear has approaching the periphery or zonular fibers
- C. Capsular fibrosis
- D. Small diameter CCC may not allow delivery of a brunescent nucleus during extracapsular cataract extraction.

III. List the alternatives to this procedure

- A. Can opener capsulotomy
- B. Femtosecond laser capsulotomy

IV. Describe the instrumentation, anesthesia and technique

- A. Anterior chamber is kept adequately inflated with ophthalmic viscosurgical device (OVD) which also flattens the anterior lens curvature
- B. Anterior capsule incised with needle, cystotome, or forceps tips
- C. Anterior capsule is torn with either needle, cystotome, or capsule forceps in a continuous circular motion for 360°

V. List the complications of the procedure, their prevention and management

- A. Complications
 - 1. During the capsulorrhexis, the tear can extend to the periphery or zonules. At this point, consider converting CCC to can-opener capsulotomy
 - 2. During phaco, the CCC edge is torn or cut with a second instrument or with the phaco tip
 - 3. Performing phaco with a single radial tear in the CCC increases risk of a tear that "wraps around" into the posterior capsule
 - 4. CCCs with very small diameter
 - a. may cause posterior capsular block with hydrodissection and difficulty with cortical clean-up and IOL insertion

- b. can lead to postoperative anterior capsule fibrosis, and capsulophimosis which can reduce peripheral fundus visualization and visual acuity
- 5. Too large a CCC will eliminate the advantage of overlap of the IOL edge and may affect centration
- B. Prevention of complications (e.g. radial tear)**
 - 1. If visualization is poor, consider use of capsular dye
 - 2. Frequent refilling of OVD if the anterior chamber shallows
 - 3. Avoid overly large diameter
 - 4. Familiarity with capsule rescue technique
- C. Management of complications**
 - 1. Attempt to rescue a radial tear using above steps
 - 2. If radial tear is too peripheral, abandon the tear and consider additional relaxing incisions, tearing from the opposite direction, or converting to a can-opener capsulotomy
 - 3. If CCC diameter is too small
 - a. perform secondary enlargement after the IOL is implanted
 - b. consider doing relaxing incisions along the CCC edge

Can-opener capsulotomy

I. List the advantages

- A. Equalizes the tangential forces to each of the multiple cuts and decreases likelihood for "wrap around" tear into the posterior capsule**
- B. Easier to perform if visualization is poor**
- C. Easier to make a large diameter capsulotomy, compared to CCC**
- D. Allows for easier deliver of a large, brunescent nucleus during ECCE**

II. List the disadvantages

- A. Loss of the advantages of the CCC listed above**

III. Describe the instrumentation

- A. Cystotome, such as bent needle**
- B. OVD to inflate the anterior chamber and flatten the anterior surface of the lens**

IV. List the complications of the procedure

- A. Large hinged flap may result from incomplete capsulotomy**
 - 1. Inadvertent aspiration can tear into posterior capsule
 - 2. Incarceration in incision may not be recognized

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 41-42.
- 3. AAO, Focal Points: The Torn Posterior Capsule: Prevention, Recognition and Management, Module #4,

1999.

4. Little BC, Simth JH, Packer M. Little capsulorhexis tear-out rescue. J Cataract Refract Surg 2006;32:1420-2.

Hydrodissection and hydrodelineation

Hydrodissection

I. Purpose

- A. Permits rotation of the nucleus by severing cortical attachments to capsule
- B. Facilitates cortex removal

II. Describe the instrumentation and technique

- A. Syringe with hydrodissection cannula
- B. Can use balanced salt solution, non-preserved lidocaine, or ophthalmic viscosurgical device (OVD)
- C. Cannula tip is positioned beneath continuous curvilinear capsulorrhexis (CCC) edge
- D. A fluid wave is directed toward and along internal surface of the capsular bag
- E. Wave passes posteriorly behind the nucleus, causing slight forward bowing of the lens
- F. Nucleus is pressed downward to break capsulorrhexis-lenticular block and to propagate the fluid wave completely around the lens
- G. Lens may be rotated

III. List the complications of the procedure, their prevention and management

- A. Failure to loosen nucleus or epinucleus
 - 1. Greater forces imparted to capsular bag and zonule during attempted rotation resulting in zonular dehiscence or loss
 - 2. Increased risk of surgical complications if the nucleus does not rotate
- B. Intraoperative capsular block and "blowout" of posterior capsule
 - 1. Elevation of nucleus under the CCC edge can create intraoperative capsular block
 - 2. Continued irrigation results in trapped fluid between the lens and posterior capsule
 - 3. May tear the posterior capsule particularly with high risk patients (i.e.: posterior polar cataract, post vitrectomy, or trauma) where there is already a weakened or defective posterior capsule
 - 4. Gently push down on nucleus and lift anterior capsule edge with cannula to relieve block and release trapped fluid

Hydrodelineation

I. Purpose

- A. Separates epinucleus from the endonucleus
- B. Optional step that may be safer than hydrodissection in the presence of a capsular defect or posterior polar cataract
- C. Reduces overall size of the portion of nucleus that must be chopped or sculpted
- D. During removal of last nuclear fragments, epinuclear shell can stabilize posterior capsule and restrain it from trampolining toward the phaco tip

II. Describe the instrumentation and technique

- A. Same as hydrodissection instrumentation**
- B. During injection, tip is directed into the peripheral nucleus along a more internal tissue plane**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.42-43, 55, 194.

Management of astigmatism at the time of cataract surgery

I. List the indications and contraindications

A. Indications

1. Corneal astigmatism

B. Contraindications

1. Toric IOLs
 - a. Damage to capsular bag preventing good centration
 - b. Intention to wear a hard contact lens postoperatively
 - c. Excessive irregular astigmatism
2. Astigmatic keratotomy
 - a. Corneal pathology at the desired location of the incision
 - b. Large amounts of astigmatism may be better treated with toric IOLs

II. Describe the pre-procedure evaluation

A. Keratometry/topography

B. Assessment of corneal abnormalities

C. Accurate biometry and IOL calculation if implanting a toric IOL

III. List the alternatives to this procedure

- A. Standard monofocal IOL implantation with subsequent refractive surgery or spectacle/contact lens wear

IV. Describe the instrumentation, anesthesia and technique

A. Instrumentation

1. Toric IOLs
 - a. Corneal markers to identify the appropriate axis
 - b. Intraoperative aberrometry to confirm placement (optional)
2. Astigmatic keratotomy
 - a. Incisional keratotomy blade or femtosecond laser
 - b. Axis marking system

B. Anesthesia may be topical, intracameral, peribulbar, retrobulbar, or general

C. Technique

1. Patient data used to formulate plan using appropriate calculator/nomogram
2. Preoperative marking in upright position to avoid effects of cyclotorsion upon supination
3. Toric IOLs are rotated into proper position within the capsular bag
4. Relaxing arcuate incisions are typically placed in clear cornea just anterior to limbus

V. List the complications of the procedure, their prevention and management

A. Complications associated with toric IOLs

1. Movement of IOL may reduce efficacy
2. Rotation typically occurs early in the postoperative course and may be more common in myopic patients with large anterior segments
3. Incorrect marking may worsen preoperative astigmatism

B. Prevention and management of complications associated with toric IOLs

1. Careful preoperative measurements and corneal marking are essential
2. Complete removal of viscoelastic may lessen the likelihood of lens rotation
3. Lens rotation may be corrected by surgical repositioning
4. Residual astigmatism may be addressed by astigmatic keratotomy or use of glasses or contact lenses

C. Complications associated with astigmatic keratotomy

1. Unexpected refractive error
2. Incorrect placement of incisions
3. Perforation, gaping, melting at site of incision

D. Prevention and management of complications associated with astigmatic keratotomy

1. Careful preoperative measurements and corneal marking are essential
2. Perforations may require closure with suture

VI. Describe the follow-up care

A. Topical antibiotic and anti-inflammatory agents

B. Examination at one day, 2-4 weeks

C. Postoperative refraction at 2-6 weeks

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 68-75.
3. Titiyal JS, Khatik M, Sharma N, et al. Toric intraocular lens implantation versus astigmatic keratotomy to correct astigmatism during phacoemulsification. J Cataract Refract Surg. 2014 May;40(5):741-7.

Intraocular lens design

I. List the indications/contraindications

- A. Intraocular lenses (IOLs) are indicated for the surgical correction of aphakia.
- B. Most commonly used materials in IOL construction include acrylic, silicone, and polymethylmethacrylate (PMMA)
- C. Considerations in selection of IOLs include the following:
 - 1. Anterior chamber IOL (AC IOL) - fixation in the anterior chamber is considered when capsular support is inadequate for in-the-bag or ciliary sulcus fixation of the IOL
 - 2. Posterior chamber IOL (PC IOL) - fixation in the capsular bag is the best location for this type of IOL. Fixation in the ciliary sulcus, with or without suture, is a secondary option
 - 3. Availability of correct IOL power to achieve desired postoperative vision including consideration of spherical equivalent, reduction of astigmatism, and correction of presbyopia
 - 4. Compatibility of IOL design, intraocular location, and fixation method (capsular bag, ciliary sulcus, iris fixated, or anterior chamber)
 - 5. Compatibility of IOL material with other intraocular surgical materials used concurrently or in the future (e.g. silicone oil in the vitreous cavity)
 - 6. Requirement for incision size and availability of insertion system (injector system, forceps)
 - 7. Special ocular conditions necessitating adjunctive devices or unique designs (aniridia, iridectomy, ectopia lentis)
- D. IOL implantation may be contraindicated in the presence of uncontrolled active uveitis
- E. IOL implantation for infants undergoing cataract extraction is controversial

II. Describe the pre-procedure/therapy evaluation

- A. Comprehensive eye examination, keratometry, biometry, and IOL power calculations are needed prior to surgery to determine the correct power and type of lens needed
- B. Special attention should be given to concomitant ocular pathology such as corneal endothelial disease, glaucoma, uveitis, macular degeneration, and diabetic retinopathy. Primary location is usually a PC IOL in the capsular bag. Secondary locations are ciliary sulcus, with or without suture fixation, and anterior chamber
- C. Phacodonesis, especially with a history of trauma or the presence of pseudoexfoliation material, may indicate an issue with capsular support
- D. Assist the patient in setting reasonable expectations regarding the outcome of surgery

III. List the alternatives to this procedure/therapy

- A. Aphakia with contact lens (monocular) or eyeglass correction (binocular)

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.21, 94-100,112, 120-151.

Special cases: secondary intraocular lens implantation

I. List the indications/contraindications

A. Indications

1. When eyeglasses or contact lenses are unsatisfactory for the correction of aphakia

B. Contraindications

1. Same as for intraocular lens (IOL) implantation in general, i.e., active, uncontrolled uveitis
2. Consideration should be given to
 - a. Corneal health (endothelial cell count, pachymetry)
 - b. Presence of glaucoma
 - c. Ocular and systemic comorbidities

II. Describe the pre-procedure/therapy evaluation

A. Evaluation of anterior segment anatomy to determine best incision site and location for secondary IOL should include consideration of

1. Pre-existing astigmatism
2. Conjunctival scarring
3. Corneal vascularization
4. Sites of previous surgery (i.e.: functioning bleb)
5. Status of potential intraocular lens support:
 - a. Zonular support
 - b. Remaining anterior and posterior capsule
 - c. Presence of posterior capsulotomy
 - d. Presence of posterior synechiae
 - e. Iris anatomy
6. Gonioscopy looking at anterior chamber depth and presence of peripheral anterior synechia (PAS)
7. Presence of vitreous in the anterior chamber

III. List the alternatives to this procedure/therapy

- ### A. Aphakia with contact lens or eyeglass correction, laser vision correction for smaller refractive errors

IV. Describe the instrumentation, anesthesia and technique

- A. Anterior vitrectomy as needed
- B. Synechialysis as needed
- C. Use of ophthalmic viscosurgical device (OVD) to protect corneal endothelium
- D. Pupil dilated for posterior chamber implantation, constricted for anterior chamber implantation
- E. Variety of fixation techniques with non-absorbable suture and/or glue as necessary in special cases with

V. List the complications of the procedure/therapy, their prevention and management

A. Subluxation/dislocation

1. Prevention
 - a. Secure location, with suture if necessary
 - b. Proper placement of haptic foot plates in the angle for anterior chamber intraocular lenses (ACIOLs)
2. Management
 - a. Suture fixation (iris, sclera)
 - b. Repositioning IOL
 - c. IOL exchange

B. Uveitis/glaucoma/hyphema (UGH) syndrome

1. Prevention
 - a. One piece IOLs contraindicated for sulcus placement
 - b. Secure implantation of ACIOLs: early, rigid AC IOL designs were associated with precise sizing requirements
 - c. Modern flexible AC IOLs with open-loop design and footplates free of fixation holes can be used to achieve excellent results in selected cases
2. Management
 - a. Anti-inflammatory agents
 - b. Topical glaucoma medications
 - c. IOL repositioning or exchange

C. Pseudophakic bullous keratopathy (corneal decompensation)

1. Prevention
 - a. Minimizing surgical trauma with liberal use of viscoelastic and proper ACIOL orientation
2. Management
 - a. Penetrating keratoplasty
 - b. Lamellar endothelial keratoplasty

D. Cystoid macular edema

1. Prevention
 - a. Use of topical corticosteroid and non-steroidal anti-inflammatory drugs (NSAIDs) in the postoperative period may be protective
2. Management
 - a. Topical, sub-Tenon and intravitreal corticosteroids and topical NSAIDs
 - b. Intravitreal anti-VEGF agents

E. Endophthalmitis - incidence is greater than for primary cataract surgery

1. Prevention
 - a. Mitigation by maintenance of sterile technique
 - b. Watertight closure of incisions/ wounds

F. Vitreous hemorrhage

G. Hyphema

H. Retinal detachment

1. Mitigation by adequate anterior vitrectomy at time of surgery

I. Distortion of pupil with AC IOL

1. Prevention
 - a. Adjust AC IOL position until there is no traction on the iris
 - b. Use of a lens glide can be helpful
 - c. Maintenance of anterior chamber with OVD or infusion line

VI. Describe the follow-up care

A. Same as for IOL implantation in general

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Posterior chamber intraocular lens implantation

I. List the indications/contraindications

- A. Fixation in the capsular bag is recognized as the best option for location of a posterior chamber intraocular lens (PC IOL)**
- B. Fixation in the ciliary sulcus, with or without scleral or iris suture, represents a secondary option for posterior chamber placement**
- C. Anatomic consideration may preclude placement of an IOL in the posterior chamber, in which case, placement of an anterior chamber IOL or aphakia with contact lens or eyeglass correction remain alternatives**

II. Describe the pre-procedure/therapy evaluation

- A. A complete ophthalmic examination is required prior to implantation of an IOL**
- B. Biometry, keratometry and IOL power calculation is necessary to determine the correct power of the IOL to be implanted**
- C. Special consideration should be given to concomitant ocular comorbidities when selecting particular IOL types**

III. List the alternatives to this procedure/therapy

- A. Placement of an anterior chamber IOL or aphakia with contact lens or eyeglass correction**

IV. Describe the instrumentation, anesthesia and technique

- A. Forceps**
 - 1. Rigid, polymethylmethacrylate IOLs may be handled with fine, smooth forceps to aid in positioning
 - 2. Foldable IOLs may be inserted with specially designed forceps or injectors
- B. Technique**
 - 1. The incision size must be at least equal to the optic diameter for rigid IOLs, for foldable IOLs the incision size may be smaller
 - 2. The bag and anterior chamber are filled with ophthalmic viscosurgical device (OVD) and the lens inserted
 - 3. Each design requires nuances of technique, and it is the responsibility of the surgeon to learn the details of technique and requisite incision size for each lens

V. List the complications of the procedure/therapy, their prevention and management

- A. Tearing or stretching of the corneal incision may lead to poor wound sealing**
 - 1. Enlarging the incision to the appropriate size for insertion by any technique helps to preserve wound architecture and insure a stable chamber postoperatively
- B. Damage to the IOL optic or haptics, resulting in an unstable or optically inadequate IOL**
 - 1. Careful and meticulous handling of the IOL during the folding and loading into the injector system to prevent scratching of the optic
 - 2. If damage to the IOL occurs during insertion, the surgeon must be prepared to perform an immediate IOL exchange

C. Damage to intraocular structures (e.g., lens capsule, iris) can occur during insertion of an IOL

1. The surgeon must be prepared to address loss of adequate capsular support by suture fixation of the IOL or IOL exchange
2. The surgeon must be prepared to do vitreous clean up if the posterior capsule is ruptured causing vitreous prolapse

D. Hyphema secondary to surgical trauma

E. Improper placement of the posterior chamber IOL

1. One haptic in the bag and one in the sulcus
2. Avoid putting a one-piece lens in the sulcus

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, p.203-207.
3. Hollick EJ, Spalton DJ, Ursell PG, et al. The effect of polymethylmethacrylate, silicone, and polyacrylic intraocular lenses on posterior capsular opacification 3 years after cataract surgery. Ophthalmology. 1999;106:49-54.

Anterior chamber intraocular lens implantation

I. List the indications/contraindications

- A. Fixation in the anterior chamber angle is generally considered a choice when capsular support is inadequate for in-the-bag or sulcus fixation of a posterior chamber intraocular lens (PC-IOL)

II. Describe the pre-procedure/therapy evaluation

- A. A comprehensive eye exam is required prior to implantation of an anterior chamber intraocular lens (AC-IOL)
- B. Gonioscopy to determine angle anatomy and presence of peripheral anterior synechia (PAS) prior to planned AC IOL placement
- C. Consideration of pachymetry and endothelial cell count
- D. Biometry, keratometry, horizontal white to white diameter, and IOL power calculation may be helpful to determine the correct size and power of the AC-IOL to be implanted
- E. The larger incisions required by anterior chamber IOLs relative to foldable IOLs will need suture closure of the incision which may represent a source of postoperative astigmatism

III. List the alternatives to this procedure/therapy

- A. Contact lens
- B. Eyeglass correction
- C. PMMA or foldable 3 piece posterior chamber IOL in the sulcus with adequate capsular support, or fixated either to the iris or sclera

IV. Describe the instrumentation, anesthesia and technique

- A. The incision size must be at least equal to the optic diameter
- B. Adequate vitrectomy prior to lens placement
- C. An iridectomy is necessary to avoid pupillary block
- D. A lens glide may be used to facilitate insertion
- E. Maintenance of anterior chamber depth
- F. Miotic agent to constrict pupil prior to insertion
- G. Due to larger incision size, incision should be sutured at the end of the procedure

V. List the complications of the procedure/therapy, their prevention and management

- A. Rigid anterior chamber IOL designs are associated with precise sizing requirements and uveitis-glaucoma-hyphema (UGH) syndrome. However, modern flexible anterior chamber lenses with open-loop design can be used to achieve excellent results and carry a lower risk
- B. Other important complications include:
 - 1. Incorrect sizing (too large or small)
 - 2. Chronic eye pain or tenderness
 - 3. Distortion of pupil- Iris tuck or capture

4. Flipped orientation
5. Malposition of IOL
6. Pupillary block
7. Chronic iritis
8. Cystoid macular edema
9. Secondary glaucoma

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, p.201-203.
3. AAO, Ophthalmic Technology Assessment: Intraocular Lens Implantation in the Absence of Capsular Support, *Ophthalmology*, 2003, 110:840-859.

Presbyopic correcting intraocular lenses (IOLs)

I. List the indications and contraindications

A. Terms and definitions

1. Presbyopia-correcting intraocular lenses are designed to reduce or eliminate dependence on spectacles after cataract surgery, and may be divided into two broad categories
 - a. Multifocal IOLs
 - b. Accommodating IOLs

B. Indications

1. A presbyopia-correcting IOL is indicated for the visual correction of aphakia
2. Also intended to reduce or eliminate the need for optical correction for near, intermediate, and distance vision

C. Contraindications

1. Ocular or systemic conditions which may reduce the potential for excellent uncorrected visual acuity represent relative contraindications for presbyopia-correcting IOL implantation
2. Presbyopia-correcting IOLs may increase the potential for glare and halo

II. List the alternatives to this procedure

- A. Cataract surgery with a monofocal IOL with postoperative spectacle or contact lens use
- B. Cataract surgery with planned monovision

III. List the complications of the procedure, their prevention and management

- A. Residual refractive error can compromise the visual function achieved with presbyopia correcting IOLs
- B. Postoperative astigmatic refractive errors can be treated with limbal relaxing incisions or laser vision correction (LVC)
- C. Significant spherical errors can be treated with IOL exchange, IOL piggybacking, or LVC
- D. Patients may still require spectacles for some visual activities and should be counseled preoperatively regarding this possibility
- E. Patients with multifocal IOLs may have glare and halo with night driving. Depending on the IOL platform these symptoms may improve with pharmacologic manipulation of the pupil. In rare instances, patients may require IOL exchange if these symptoms are severe. Over time these symptoms may diminish due to neuroadaptation
- F. Observe for posterior capsule opacification. Patients with presbyopia-correcting IOLs can have reduced visual function especially at near, which may improve with Nd:YAG capsulotomy

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016
2. CMS Rulings, Department of Health and Human Services, Centers for Medicare & Medicaid Services. Ruling No. 05-01 May 3, 2005.
3. Mastering Refractive IOLs: The art and science. Edited by David F. Chang, 960 pages, 2008, Slack Incorporated.

Thermal injury or "phaco burns"

I. Describe the source of heat generation and its dissipation during phacoemulsification

A. Source

1. Frictional forces created by the ultrasonic tip vibration causes heating of the phaco tip or needle

B. Dissipation

1. Through cool fluid flow around the tip
2. through the cornea and ocular tissue

II. Describe intraoperative appearance of a thermal injury

A. Pre-burn - note appearance of non-aspirated lens emulsion ('lens milk')

B. Mild - corneal epithelium sloughs at incision site

C. Moderate - incision edges gape due to mild tissue shrinkage

D. Severe - whitened, friable and absent tissue with significant tissue shrinkage leading to full thickness defect

III. Describe what things might contribute to a thermal injury

A. Inadequate cooling fluid flow around phaco tip

1. Lack of irrigation
 - a. Tight incision compressing infusion sleeve
 - b. Low or empty bottle
 - c. Infusion tubing improperly set up or kinked
2. Lack of aspiration
 - a. Inadequate vacuum level
 - b. Obstruction by viscoelastic
 - c. Clogged tubing or tip

B. Prolonged, continuous use of ultrasound power

IV. Describe techniques, maneuvers or instrumentation that might reduce the chances of thermal injury during phacoemulsification

A. Test irrigation prior to insertion of phacoemulsification tip

B. Aspiration of some viscoelastic prior to engaging ultrasound power

C. Ensure incision size adequate for phacoemulsification tip

D. Consider adjusting phaco machine settings to minimize ultrasound power (pulse/burst modes, chop technique)

E. Consider continual or frequent external irrigation to incision site

V. Describe the intraoperative management of a phaco induced thermal injury

A. Mild - nothing if incision is still self-closing

B. Moderate - suture closure

- C. **Severe - suture plus patch graft of conjunctiva or sclera, partial thickness flap or relaxing incision.**
Cyanoacrylate glue may also be considered to seal wound

VI. Describe the postoperative follow up care

- A. **Evaluate for wound leak during early postoperative period**
- B. **If wound is leaking**
 - 1. Bandage soft contact lens
 - 2. Aqueous suppressants
 - 3. Wound revision or patch graft
 - 4. Corneal glue
- C. **Watch for postoperative induced high astigmatism**
 - 1. May regress gradually

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Detachment of Descemet membrane

I. Describe the etiology of this complication

- A. Edge of Descemet membrane may be caught at any incision site by an instrument, intraocular lens (IOL), or OVD which can result in a small or large detachment of Descemet membrane

II. Describe the intraoperative appearance of a torn or detached Descemet membrane

- A. May be torn but attached with a hinge and rolled-up free edge
- B. May be detached completely centrally but attached peripherally
- C. If completely detached, may appear as a free floating scroll of clear membrane, can appear similar to anterior capsule

III. Describe the effects of a detachment of Descemet membrane

- A. If small area involved, may have transient localized overlying corneal edema
- B. Larger area may have persistent epithelial and stromal edema until the area involved re-endothelializes over time
- C. Large Descemet membrane detachment or low endothelial cell density may result in chronic pseudophakic bullous keratopathy.

IV. Treatment options

- A. Observation - even large detachments may resolve with time
- B. Hypertonic saline drops
- C. Removal of small free floating scrolls
- D. Reattachment by air injection, expansile gas, or suture
- E. Keratoplasty (penetrating or endothelial) for persistent corneal edema

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.54-55.

Toxic anterior segment syndrome (TASS)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease (i.e. potential intraoperative sources of toxic substances)

1. Irrigating solutions and ophthalmic viscosurgical devices (OVDs)
 - a. Preservatives or additives (e.g., antibiotics, diluting medications)
2. Ophthalmic instrument contaminants
 - a. Detergent residues (ultrasonic, soaps, enzymatic cleaners)
 - b. Denatured OVD
3. Ocular medications
 - a. Incorrect drug concentration, pH, and osmolality
 - b. Preservative in medication solution (e.g., bisulfites in epinephrine)
4. Intraocular lenses (IOLs)
 - a. Polishing compounds
 - b. Cleaning and sterilizing compounds (e.g., chlorhexidine gluconate)

B. Define the relevant aspects of epidemiology of the disease

1. Patients undergoing cataract or anterior segment surgery often representing an endemic outbreak at a specific surgical center

C. List the pertinent clinical course

1. Typically occurs in the first 12-24 hours (vs 2-7 days for bacterial endophthalmitis)
2. Almost always limited to anterior segment
3. Improves with topical corticosteroids
4. Commonly presents with diffuse corneal edema
5. Blurry vision, eye pain, eye redness, photophobia

D. Describe pertinent clinical features

1. Intraoperative and postoperative corneal appearance after a toxic substance has been injected into the anterior chamber
 - a. Intraoperatively
 - i. Usually no effect
 - b. Postoperatively
 - i. Folds in Descemet membrane
 - ii. Corneal edema (limbus to limbus)
 - i) Versus central with peripheral sparing from surgical trauma
 - iii. Variable intraocular pressure (IOP) elevation due to trabecular meshwork damage
 - iv. Iris damage, possible pupil dysfunction
 - v. Cystoid macular edema
 - vi. Sterile anterior segment inflammation with possible hypopyon
 - vii. Fibrin formation in anterior chamber, on IOL, or iris
 - viii. Relative lack of vitreous cell or inflammation compared to anterior chamber

II. List the differential diagnosis

A. Causes of postoperative corneal edema

1. Mechanical trauma
2. Pre-existing endothelial compromise, e.g., Fuchs endothelial dystrophy
3. Excessive use of ultrasound energy
4. Descemet detachment

B. Infectious endophthalmitis

C. Uveitis flare-up

III. Describe patient management in terms of treatment and follow-up

A. Intraoperative and postoperative management for corneal damage from a toxic agent (Main treatment of TASS centers on prevention)

1. Intraoperative (Immediate recognition of toxic agent)
 - a. Irrigate anterior chamber with balanced salt solution to wash out all the toxic agent
2. Postoperatively managing from slight to progressively more severe inflammatory reaction
 - a. Have a low threshold for vitreous and/or anterior chamber culture with injection of antibiotics if infection is suspected
 - b. Mainstay is frequent topical corticosteroid drops (prednisolone acetate 1%)
 - i. Frequent follow-up necessary to gauge response
 - c. Lower IOP with aqueous suppressant drops if elevated
 - d. Progressive steroid use as indicated
 - i. Sub-Tenons injection of corticosteroid
 - ii. Intravitreal corticosteroid injection
 - iii. Systemic corticosteroids if needed to control inflammation

IV. List prevention and management

- A. Review of protocol for cleaning and sterilizing ophthalmic instruments
- B. Review of protocol for ordering medications and preparing medications
- C. Reusable instruments should be kept to a minimum

V. Describe disease-related complications

- A. Loss of vision
- B. Endothelial cell loss resulting in bullous keratopathy
- C. Secondary glaucoma due to PAS and trabecular meshwork damage
- D. Iris damage with fixed, dilated pupil
- E. Cystoid macular edema

VI. Describe appropriate patient instructions

- A. Frequent visits in the immediate postoperative period

1. Monitor inflammation, IOP, corneal edema, vision

B. Long-term management of cornea, IOP and vision

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.60.
3. Mamalis N, Edelhauser HF, Dawson DG.et al.Toxic Anterior Segment Syndrome Review/Update. J Cataract Refract Surg 2006; 32:324-333.

Retrobulbar hemorrhage

I. Describe the clinical features of a retrobulbar hemorrhage

- A. Increasing proptosis
- B. Lid ecchymoses
- C. Unable to displace globe posteriorly
- D. Intraocular pressure elevation
- E. Subconjunctival hemorrhage and/or bloody chemosis

II. Describe the mechanism of a retrobulbar hemorrhage and vision loss

- A. Puncture or tearing of orbital blood vessel
- B. Bleeding into an enclosed area (orbit) raises the orbital pressure which elevates intraocular pressure
- C. This compartment syndrome may restrict vascular supply to the optic nerve and globe, resulting in central retinal artery and/or vein occlusion, or optic nerve compression or ischemia
- D. Direct injury to optic nerve or compression of the nerve within the optic canal

III. Describe the adverse sequelae that can result from a retrobulbar hemorrhage

- A. Intraoperatively can cause loss of chamber and increased posterior pressure
- B. Loss of Vision
 - 1. Ischemic optic neuropathy or optic nerve compression
 - 2. Loss of vision from vascular injury (central retina artery occlusion, central retinal vein occlusion)

IV. Describe patient management in this situation

- A. If occurs preoperatively, cancel surgery unless hemorrhage is minor/limited
- B. If occurs intraoperatively, stop surgery and close incision temporarily if posterior pressure is progressive.
- C. Treatment aimed at rapidly lowering orbital and intraocular pressure
 - 1. Serial tonometry to measure success of treatment
 - 2. Digital massage if globe intact
 - 3. IV osmotic agents and aqueous suppressants
 - 4. lateral canthotomy and cantholysis
 - 5. Consider consultation with oculoplastic surgeon for orbital decompression if unable to lower intraocular pressure to acceptable range

V. Describe alternative anesthetic approaches to a retrobulbar injection

- A. Topical and intracameral anesthesia
- B. Sub-Tenon injection
- C. Peribulbar injection
- D. General anesthesia

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Suprachoroidal hemorrhage

- I. Describe the probable mechanism of a suprachoroidal hemorrhage**
 - A. Site of hemorrhage likely a bridging blood vessel which crosses the suprachoroidal space
 - B. Decompression or prolonged hypotony during or following intraocular surgery can lead to choroidal effusion, which can stretch and break bridging blood vessels
- II. Describe the risk factors associated with suprachoroidal hemorrhage**
 - A. Increased age
 - B. History of glaucoma
 - C. Axial length >25.8 mm
 - D. Prolonged hypotony during or following ocular surgery
 - E. Systemic hypertension
 - F. Arteriosclerotic heart disease
 - G. Elevated preoperative intraocular pressure (IOP) with rapid decompression
 - H. Drugs or conditions which affect coagulation (not true risk factor for hemorrhage, but hemorrhage will be worse in their presence)
 - I. Nanophthalmos
- III. Describe the intraoperative signs of a suprachoroidal hemorrhage**
 - A. Patient pain and agitation
 - B. Increased posterior pressure causing shallowing of anterior chamber
 - C. Firm eye to tactile pressure
 - D. Wound gape
 - E. Iris prolapse, spontaneous delivery of the lens, and expulsion of intraocular contents
 - F. Loss of red reflex or growing shadow appearing in red reflex
- IV. Describe the appropriate intraoperative management for suprachoroidal hemorrhage**
 - A. Immediate closure of wound
 1. Suture if not self-sealing
 2. Manual compression of incision while awaiting sutures
 - B. If can't close incision, consider posterior sclerotomy over site of shadow
 - C. Consider re-operation once intraocular pressure normalized
 - D. Consider referral to posterior segment surgeon
- V. Describe the surgical plan for a patient who is high risk for choroidal hemorrhage**
 - A. Aggressive IOP control
 - B. Aggressive systemic blood pressure control preoperatively and intraoperatively
 - C. Position patient so eye is higher than heart

- D. Small incision surgery**
- E. Pre-placed sutures if incision > 6 mm**
- F. Minimize operating time**
- G. Avoid hypotony and collapse of anterior chamber during surgery**
 - 1. Consider continuous anterior chamber infusion
- H. Discontinue anticoagulants preoperatively if approved by PCP**
- I. Discussion with patient during informed consent regarding elevated risk for suprachoroidal hemorrhage and sequelae**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Surgical trauma - hyphema

- I. List the anterior segment structures of a normal eye that might be sources for possible hemorrhage**
 - A. Iris pupillary margin vessels
 - B. Iris stromal vessels
 - C. Iris root major arterial vessels
 - D. Ciliary body vessels
 - E. Incision related vasculature (limbal vasculature)

- II. Describe the consequences that might arise from a hyphema created at surgery**
 - A. Decreased vision
 - B. Increased intraocular pressure (IOP) secondary to mechanical obstruction of trabecular meshwork by red blood cells
 - C. Corneal blood staining in the presence of high IOP
 - D. Chronic inflammation
 - E. Posterior synechiae
 - F. Diffusion of blood into vitreous

- III. Describe intraoperative options for management of bleeding**
 - A. Temporarily pressurize the globe
 - B. Tamponade with OVD or air injection into anterior chamber
 - C. Cautery if source of bleeding identified

- IV. Describe the postoperative management of a patient who had bleeding intraoperatively**
 - A. Close follow-up during the early postoperative period with highest risk of rebleeding, activity restrictions
 - B. Slit lamp biomicroscopic observation, record level/grade of hyphema
 - C. Monitor IOP
 - D. Iris immobilization with pharmacologic agents if needed
 - E. Avoid anti-coagulating agents short term
 - F. Investigate for coagulopathy
 - G. Consider sickle cell testing for at risk patients
 - H. If non-clearing, watch for possibility of late onset ghost cell glaucoma

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Microscope induced light toxicity

- I. Describe the nature of the injury that can be associated with the light microscope**
 - A. Photochemical damage to retina and retinal pigment epithelial layers from unfiltered blue and near ultraviolet radiation**

- II. Describe factors that contribute to the potential for damage and how each may be addressed**
 - A. Prolonged operating time**
 - 1. Reduce operating time
 - 2. Oblique lighting
 - 3. Use pupillary shield
 - B. High light intensity**
 - 1. Reduce light level to only that required for safe view
 - 2. Use a filter to exclude light below 515 nm

- III. Describe the patient complaint and workup**
 - A. Patients may have minimal to severe vision loss depending on location and severity of phototoxicity**
 - B. Paracentral or central scotoma or central vision distortion**
 - C. Retinal exam may not reveal any clinical abnormalities immediately**
 - D. Retina exam may reveal**
 - 1. Varying degrees of retina edema and/or pigmentary changes during early postoperative period, usually in a discrete oval pattern in or near macula
 - 2. Varying degrees of pigmentary mottling after early postoperative period

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Postoperative elevated intraocular pressure

I. List possible causal mechanisms for elevated intraocular pressure following cataract surgery

A. Decreased outflow facility

1. Obstruction by ophthalmic viscosurgical device (OVD)
2. Inflammation
3. Glaucoma suspects and glaucoma patients may be at higher risk

B. Pupillary block with an anterior chamber IOL (AC IOL) in the absence of peripheral iridectomy/iridotomy

C. Aqueous misdirection

D. Hyphema

II. Describe the approach to establishing the diagnosis of increased intraocular pressure

A. Patient symptoms

1. May have minimal to no symptoms
2. Pain in and around eye
3. Headache
4. Foggy vision
5. Nausea and vomiting

B. Examine cornea for corneal edema / "steamy" corneal appearance

C. Measure intraocular pressure (IOP)

III. Describe the management in terms of postoperative treatment and follow-up

A. Postoperative treatment

1. Short term ocular hypotensive agents
 - a. Topical or oral carbonic anhydrase inhibitors
 - b. Beta adrenergic antagonists
 - c. Alpha-2 adrenergic agonists
2. Laser peripheral iridotomy if pupillary block present
3. Side port decompression at the slit lamp

B. Postoperative followup

1. Vast majority resolve within days of surgery with medical management only

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2013-2014.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 60.

Intraoperative signs of posterior capsular rupture

I. List reasons why a posterior capsule might be ruptured intraoperatively

A. Operator error

1. Excessive hydrodissection with small capsulorrhexis and hard lens
2. Unrecovered errant continuous curvilinear capsulorrhexis to periphery which extends posteriorly
3. Broken by instrument

B. Defective capsule

1. Posterior lenticonus
2. Posterior polar cataract
3. Previous damage to posterior capsule from trauma or previous retinal surgery

C. Intumescent lens whose pressure extends initial capsulorrhexis tear

II. Intraoperative signs of capsule rupture

A. Anterior chamber (AC) deepens

B. Pupil appears to widen

C. Iris may appear concave

D. Lens particles may disappear posteriorly instead of circulating in AC

E. Lens nucleus may remain displaced/descend rather than returning to central position or it may move posteriorly

F. Lens fragments have poor followability

G. Vitreous may move anteriorly into AC and obstruct aspiration port of hand piece or entrap lens particles

III. Describe your management goals when faced with an open posterior capsule

A. Avoid anterior chamber collapse in an effort to prevent vitreous prolapse by filling with viscoelastic prior to removing handpiece

B. Carefully remove nucleus and particles of nucleus from the anterior segment

C. Carefully remove as much cortex as possible (performed with the anterior vitrector or after use of OVD)

D. Cut or reposit herniated vitreous

E. Insertion of intraocular lens (IOL)

1. Capsular bag if adequate support
2. If inadequate capsule support
 - a. In ciliary sulcus
 - i. Consider capture of IOL optic posterior to capsulorrhexis
 - b. With transscleral or trans-iris suture fixation
 - c. In anterior chamber
3. Adjust lens power

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.54, 97, 206-210.

Intraoperative management of dropped nucleus

- I. Describe pre-existing pathology or intraoperative events that might lead to a dropped nucleus**
 - A. Posterior polar cataract
 - B. Posterior lenticonus
 - C. Iatrogenic posterior capsule tear during vitreoretinal surgery or intravitreal injection
 - D. Zonular weakness
 - E. Broken posterior capsule intraoperatively
 - F. Prior trauma

- II. Describe the precautions you might take to mitigate complications or make posterior loss of the nucleus less likely in a patient who is predisposed toward having a problem with capsular support**
 - A. Have correct tools available; vitrector, lens loop, adequate viscoelastic
 - B. Consider local block and place incision where it can be enlarged if needed
 - C. Large anterior capsulorrhexis through which whole nucleus could be delivered if needed
 - D. Knowledge of a nuclear handling technique to
 1. Avoid multiple lens fragments
 2. Support and control entire nucleus without pressure or stresses on posterior capsule
 - E. Attempt to leave posterior epinuclear shell in place
 - F. Prolapse nucleus anteriorly before emulsification using manipulation or ophthalmic viscosurgical devices (OVD)
 - G. Do not over-inflate during hydrodissection to decrease irrigation pressure
 - H. Adjust fluidics to low flow settings
 - I. Consider use of intracapsular tension ring if zonular dehiscence is noted

- III. Describe consequences that could arise from nuclear material retained in the vitreous**
 - A. Increased inflammation
 - B. Cystoid macular edema
 - C. Elevated intraocular pressure (IOP)
 - D. Decreased vision and/or visual symptoms
 - E. Retinal detachment

- IV. Describe the various approaches to removal of a dropped nucleus**
 - A. Stop phacoemulsification while maintaining intraocular pressure
 - B. Instrument elevation of nucleus and particles through pupil if not too posterior
 - C. If not proficient with vitreoretinal surgical procedures, close and refer to vitreoretinal specialist after

removal of cortex and vitreous from anterior segment. Implant intraocular lens (IOL) when possible to accomplish safely and effectively

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, p.58.

Intraoperative management of iris prolapse

I. Describe possible consequences of iris prolapse

A. Intraoperative

1. Pupillary constriction
2. Increased atonicity and floppiness of iris
3. Patient discomfort

B. Postoperative

1. Segmental loss of iris tissue, loss of pupil function, aesthetic deformity, glare disability
2. Adhesions to anterior capsule, posterior capsule, pupil distortion
3. Possible iris incarceration in wound increasing risk of wound leak and endophthalmitis
4. CME

II. List causes of iris prolapse

A. Incorrect incision

1. Too posterior, near iris root
2. Too short

B. Excessive inflation of anterior chamber with viscoelastic

C. Floppy iris

1. After pupillary stretch
2. Use of alpha 1-adrenergic antagonists, e.g., tamsulosin (Flomax®)

D. Increased posterior pressure

E. Excessive fluid flow under iris

F. Poor dilation

III. Describe management options for iris prolapse

A. Adequate use of mydriatics pre-operatively

B. Reduce anterior chamber pressure

1. Remove excessive ophthalmic viscoelastic, through alternate incision if possible

C. Reposition iris with OVD or sweep prolapsed iris from alternate incision

D. Perform peripheral or sector iridectomy

E. Partially close incision if too large

F. Reduce irrigation flow

G. Move to alternate incision site after closing first incision

H. Insert iris restraining devices

I. Miotic injection at conclusion of surgery

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Management of intraoperative vitreous loss

I. List the potential adverse consequences associated with vitreous loss

- A. Retina tear/detachment
- B. Cystoid macular edema (CME)
- C. Endophthalmitis
- D. Retained lens material, creating glaucoma and inflammation
- E. Vitreous strands to incision
- F. Bullous keratopathy and endothelial decompensation
- G. Misshapen pupil
- H. Secondary glaucoma

II. Describe the intraoperative appearance of vitreous in the anterior segment

- A. Clear, cohesive, egg white-like material
- B. Displacement of other structures, iris, capsule, etc.
- C. Other structures may move when vitreous is contacted or moved
- D. Aspiration may seem less effective if vitreous entrapped in aspiration port
- E. May be more easily visualized with intracameral triamcinolone or air

III. Describe techniques for anterior vitrectomy; including instrumentation

- A. Limit amount of vitreous presented in the anterior segment by maintaining a pressure gradient with higher pressure in the anterior chamber
 - 1. Fill anterior chamber (AC) with an ophthalmic viscosurgical device (OVD) prior to removing irrigating instruments
 - 2. Reduce irrigation flow, avoiding anterior displacement of vitreous
 - 3. Maintain watertight incisions
- B. Manual
 - 1. Cellulose sponge and scissors at incision site externally for diagnosis
 - 2. Sweeping vitreous strands away from incision site using spatula through side port paracentesis
 - 3. Constrict pupil to check for residual strands of vitreous
- C. Automated vitrector - be knowledgeable of fluidics and mechanics
 - 1. Use infusion anteriorly and cut posteriorly to maintain pressure gradient
 - a. Limbal or a pars plana vitrectomy, depending upon the condition, training, and comfort of the surgeon
 - 2. Maintain watertight incisions, often suturing keratome incision and creating second sideport incision
 - 3. Sweep iris surface with spatula or cannula to find hidden strands lying on iris
 - 4. Constrict pupil to reveal strands of vitreous which may have been overlooked
 - 5. Don't cut under iris or into posterior vitreous cavity

6. Consider use of Preservative-Free intraocular triamcinolone to visualize vitreous strands

IV. Describe any variations in postoperative management or follow up you might employ

- A. Subconjunctival corticosteroid injection at procedure's conclusion**
- B. Indirect ophthalmoscopy for tears or detachments**
- C. Oral acetazolamide for IOP control if suspect retained viscoelastic**
- D. High dose topical corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs) for long duration to reduce chance of CME**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Abbasoglu OE, Hosal B, Tekeli O, et al. Risk factors for vitreous loss in cataract surgery. Eur J Ophthalmol. 2000;10:227-32.

Management of small pupil

- I. Describe why it is necessary for the surgeon to be able to manage pupil size intraoperatively**
 - A. Increases visualization of operative field**
 - B. Allows normal sized capsulorrhexis**
 - C. Easier cortical removal**
 - D. Reduces complications**
 - 1. Capsule rupture and vitreous loss
 - 2. Damage to iris

- II. List what preoperative factors or conditions which might result in a small pupil**
 - A. Pseudoexfoliation**
 - B. Diabetes mellitus**
 - C. Chronic miotic therapy**
 - D. Synechia from previous inflammation**
 - E. Alpha 1-adrenergic antagonists (e.g., tamsulosin, (Flomax ®))**

- III. List the options for pupil enlargement, describing the technique and complications of each**
 - A. Pre-operative pharmacologic**
 - 1. 10% phenylephrine
 - 2. NSAID drops
 - B. Instill additional OVD**
 - 1. Elevated postoperative intraocular pressure if retained
 - C. Intraocular irrigation of preservative-free pupil dilation agents, e.g. epinephrine**
 - 1. TASS if prepared incorrectly
 - D. Stretching, one handed or two handed (contraindicated in IFIS)**
 - 1. Iris sphincter tears, bleeding
 - 2. Postoperative mydriasis
 - E. Multiple sphincterotomies**
 - 1. Postoperative mydriasis
 - F. Lysis / stripping of iris / pupillary adhesions**
 - 1. Mild bleeding
 - 2. Loss of pigmented pupillary collar
 - G. Mechanical hooks / pupil rings**
 - 1. Iris sphincter tears, bleeding

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Postoperative shallow or flat anterior chamber

I. Establishing the diagnosis

A. Etiology

1. Wound leak
2. Pupillary block
3. Aqueous misdirection (ciliary block glaucoma)
4. Suprachoroidal hemorrhage
5. Suprachoroidal effusion
6. Capsular block syndrome

B. Pertinent history

1. Postoperative eye trauma (including eye rubbing)
2. Ocular pain
3. Decreased vision
4. Redness
5. Tearing

C. Clinical features

1. Wound leak and choroidal effusion are associated with low intraocular pressure (IOP)
2. Shallow anterior chamber associated with normal or high IOP can be the result of pupillary block, aqueous misdirection, suprachoroidal hemorrhage or capsular block syndrome

D. Testing and evaluation

1. B scan ultrasound can demonstrate choroidal effusion or suprachoroidal hemorrhage
2. Anterior segment imaging (ultrasound biomicroscopy or anterior segment ocular coherence tomography) can show pupillary or capsular block or evidence of aqueous misdirection
3. Seidel test for wound leak

II. Risk factors

A. Pupillary block

1. Uveitis with posterior synechiae
2. Anterior chamber lens without patent peripheral iridotomy
3. Forward displacement of sulcus intraocular lens (IOL)
4. Incorrect orientation of IOL with angulated haptics leading to forward vaulting of optic
5. Iridovitreal synechiae

B. Suprachoroidal hemorrhage

1. Advanced age
2. Poorly controlled hypertension
3. Concomitant glaucoma procedure

C. Wound leak/choroidal effusion

1. Poorly constructed wound
2. Poor wound closure
3. Phacoemulsification burn
4. Eye rubbing or trauma

III. Describe patient management in terms of treatment and follow-up

A. Medical therapy options

1. Cycloplegia (atropine) to decrease risk of choroidal effusion
2. If high IOP, aqueous suppression
3. If low IOP with wound leak, pressure patch or bandage contact lens

B. Describe surgical therapy options

1. Laser or surgical peripheral iridotomy for pupillary block (then permanent treatment of underlying etiology)
2. Disruption of anterior hyaloid face with neodymium yttrium-aluminum-garnet (Nd: YAG) laser surgery or vitrectomy for aqueous misdirection
3. Drainage of choroidal effusion or hemorrhage if non-resolving and associated with persistent flat anterior chamber
4. If wound leak present, repair wound with suture or tissue adhesive

IV. Describe disease-related complications

- A. Peripheral anterior synechiae**
- B. Corneal decompensation**
- C. Visual loss**
- D. Optic nerve damage with sustained elevated IOP**
- E. Endophthalmitis with wound leak**

V. Describe appropriate patient instructions

- A. Proper medication use**
- B. Eye shield**
- C. Avoid eye rubbing**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Kaplowitz K, Yung E, Flynn R, Tsai JC. Current concepts in the treatment of vitreous block, also known as aqueous misdirection. *Surv Ophthalmol.* 2015 May-Jun;60(3):229-241.

Corneal complications of phacoemulsification and intraocular lenses

I. Describe the approach to establishing the diagnosis

A. Etiology

1. Acute postoperative endothelial dysfunction/corneal edema due to mechanical trauma, corneal incision burns, prolonged intraocular irrigation, inflammation, retained nuclear particles, elevated intraocular pressure (IOP) or introduction of toxic substances (TASS-Toxic Anterior Segment Syndrome)
2. Late endothelial dysfunction due to retained nuclear particles in the angle
3. Vitreocorneal adherence and persistent corneal edema after complicated extracapsular cataract extraction or phacoemulsification
4. Descemet membrane detachment
5. Suboptimal lens choice (Iris clipped or closed loop ACIOLs) or poorly positioned IOLs

B. Epidemiology

1. More common in patients with underlying corneal endothelial dysfunction such as Fuchs dystrophy

C. Pertinent history

1. Blurred, "foggy" vision worse in the morning than evening
2. If corneal edema significant with associated bullous keratopathy, symptoms include foreign body sensation, epiphora

D. Clinical features

1. Descemet folds
2. Corneal clouding
3. Microcystic edema
4. Subepithelial bullae

II. Define the risk factors

A. Fuchs dystrophy

B. Complicated cataract surgery with prolonged surgical time or vitreous loss

C. Phacoemulsification techniques with less control of dispersed ultrasound energy

D. Closed-loop or rigid anterior chamber IOL

E. Phacoemulsification of brunescent cataract with prolonged emulsification time

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Frequent topical corticosteroids and, topical hyperosmotic agents. Corneal edema generally resolves within 4-6 weeks.
2. Aqueous suppressants for elevated intraocular pressure
3. Bandage (therapeutic) contact lens if bullae are symptomatic

B. Describe surgical therapy options

1. Penetrating keratoplasty or endothelial keratoplasty if edema is not resolving and patient is symptomatic after appropriate waiting period after surgery (several months)
2. Removal of retained lens fragments

IV. List the complications of treatment, their prevention and management

- A. Complications of corticosteroid drops**
- B. Complications of bandage contact lens**
- C. Complications of keratoplasty**

V. Describe appropriate patient instructions

- A. Patients should be advised that resolution of corneal edema may be prolonged in the context of denser cataracts or Fuchs endothelial dystrophy**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Focal Points: Evaluation of Impaired Visual Acuity Following Cataract Surgery, Module #6, 1996, p.5-7.
3. AAO, Focal Points: Phacoemulsification, Module #6, 1994, p.10.

Wound leak or filtering bleb (complication of cataract surgery)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology

1. Wound leak with filtration of aqueous through the wound

B. List the pertinent elements of the history

1. May be asymptomatic
2. Possible symptoms: irritation, excessive tearing, blurred vision, contact lens intolerance, pain

C. Describe pertinent clinical features

1. Depending on amount of wound leak, anterior chamber may be shallow or fully formed
2. Hypotony
3. Wound will be seen to leak after application of fluorescein
4. If a scleral wound is buried under conjunctiva, an inadvertent filtering bleb may form
5. Chronic wound leaks are associated with fistula formation and possible epithelial downgrowth

II. Define the risk factors

A. Poorly constructed wound

B. Poor intraoperative wound closure

C. Poor patient wound healing

D. Vitreous or iris incarceration

E. Phacoemulsification burn

F. Eye rubbing

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Most leaks resolve with medical management
 - a. Pressure patching or use of bandage soft contact lens
 - b. Suppression of aqueous production (with carbonic anhydrase inhibitors or beta-adrenergic antagonists)
 - c. Stimulation of wound healing by decreasing topical corticosteroids and nonsteroidal antiinflammatory drugs

B. Describe surgical therapy options

1. If there is a significant wound leak with shallow or flat anterior chamber, obvious wound separation, iris prolapse, or no improvement with medical management within 24-48 hours, the cataract wound should be revised.
 - a. The wound should be sutured closed
 - b. Consider use of tissue adhesive in selected cases
2. Techniques to eliminate inadvertent bleb formation vary considerably and consist of procedures to enhance inflammation in the wound and seal the leak by cicatrization of the bleb

IV. List the complications of treatment, their prevention and management

- A. Complication of decreasing corticosteroids: increased anterior chamber inflammation
- B. Complication of wound revision: infection, induced astigmatism (prevented by avoiding tight suture closure)
- C. Complication of bleb cicatrization: conjunctival buttonhole

V. Describe disease-related complications

- A. Infection
- B. Hypotony maculopathy
- C. Prolapse of uveal tissue through wound
- D. Astigmatism
- E. Contact lens intolerance
- F. Corneal dellen
- G. Choroidal effusion
- H. Blurred vision

VI. Describe appropriate patient instructions

- A. Proper medication use
- B. Eye shield, especially at bed time
- C. Avoidance of eye rubbing
- D. Minimal physical exertion

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Postoperative inflammation after cataract surgery

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Low-virulence bacterial pathogens: *Propionibacterium acnes*, *Staphylococcus epidermidis*
2. Retained lens material
3. Under-treatment of surgical trauma
4. Mechanical trauma from an intraocular lens (IOL) due to lens design or incorrect placement
5. Exposure to contaminated instruments/fluids intraoperatively or IOL can cause toxic anterior segment syndrome (TASS)
6. Uveitis: initial presentation of endogenous or exacerbation of preexisting

B. List the pertinent elements of the history

1. Endophthalmitis with low-virulence bacterial pathogens: patients may have few if any early symptoms. Light sensitivity, variable ocular redness, pain, and visual compromise develop weeks to months after surgery (delayed endophthalmitis)
2. Significant retained lens material after complicated surgery: patients typically have immediate postoperative pain, redness, and diminished vision
3. TASS: inflammation may commence within hours of surgery

C. Describe pertinent clinical features

1. In delayed endophthalmitis, infection begins as low-grade inflammation which is transiently responsive to corticosteroids, but returns or persists when the medication is tapered
 - a. Later, granulomatous keratic precipitates may appear on the corneal endothelium and the IOL surface
 - b. White plaques are commonly found on the capsular bag in cases of *Propionibacterium acnes*
2. In cases with retained lens material, granulomatous uveitis is present often with increased intraocular pressure (IOP) and corneal decompensation

D. Describe appropriate testing for establishing the diagnosis

1. If any suspicion of endophthalmitis, aqueous and vitreous cultures should be taken
2. If capsular plaques are present, an attempt should be made to culture this material
3. Gonioscopy and/or ultrasound biomicroscopy, to detect lens fragments in the angle/sulcus and confirm IOL malposition

II. Define the risk factors

- A. Patients with history of uveitis
- B. Complicated cataract surgery (e.g., vitreous loss)
- C. Improper cleaning/sterilization of instruments

III. List the differential diagnosis

- A. Delayed endophthalmitis
- B. Uveitis due to other etiologies

- C. Retained lens fragments
- D. TASS
- E. Uveitis-Glaucoma-Hyphema (UGH) syndrome secondary to IOL design or poor position

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. If infection not suspected and no significant retained lens material: topical corticosteroids, non-steroidal anti-inflammatory drugs and cycloplegics, ocular IOP lowering drops if needed
2. Sub-Tenons corticosteroids

B. Describe surgical therapy options

1. If infection suspected
 - a. intravitreal/intracapsular injection of vancomycin for treatment of *Propionibacterium acnes*
 - b. Removal of IOL and lens capsule may be required
2. Retained lens fragments causing significant inflammation should be removed
3. Intraocular lenses causing chronic inflammation should be explanted, repositioned, or fixated

V. Describe disease-related complications

- A. Corneal decompensation
- B. Cystoid macular edema
- C. Inflammatory deposits on IOL
- D. Accelerated posterior capsule opacification
- E. Inflammatory (uveitic) glaucoma

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Holland SP, Morck DW, Lee TL. Update on toxic anterior segment syndrome. *Curr Opin Ophthalmol* 2007;18:4-8.

Vitreous incarceration in wound

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Migration of vitreous through the pupil with adherence to the wound
2. Usually occurs in setting of posterior capsule rupture, but may also occur with an intact capsule in the setting of zonular dehiscence

B. Describe pertinent clinical features

1. Vitreous strand seen on slit lamp exam extending to main wound or paracentesis site
2. Pupil often peaked

II. Define the risk factors

A. Incomplete removal of vitreous from the anterior segment during surgery

B. Posterior capsule tear

C. Zonular dialysis

III. Treatment

A. Observation

B. Describe medical therapy options

1. Corticosteroid and non-steroidal anti-inflammatory drug (NSAID) drops for secondary cystoid macular edema (CME)/inflammation

C. Describe surgical therapy options

1. Nd: YAG laser for lysis of fine vitreous strands
2. Anterior or posterior vitrectomy
 - a. If considerable vitreous is incarcerated in the wound
 - b. If there is associated CME or uveitis unresponsive to medical or laser surgical therapy.

IV. Describe disease-related complications

A. Chronic inflammation

B. CME

C. Cosmetic pupil deformity

D. Glare symptoms

E. Endophthalmitis

F. Retinal tear or detachment

G. Corneal decompensation

V. Describe appropriate patient instructions

A. Follow-up if vision loss or eye pain commences

B. Discuss symptoms of retinal detachment

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 62.

Induced astigmatism (complication of cataract surgery)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology

1. Cataract incision (longer and more anterior incisions induce astigmatism)
2. Tight sutures causing steepening in that meridian
3. Corneal wound burn
4. Incorrect axis of limbal relaxing incision performed at time of cataract surgery
5. Tilted implant due to malposition or compromised zonules/capsular bag
6. Incorrect axis of toric intraocular lens (IOL)

B. List the pertinent elements of the history

1. Blurred vision unless optical correction in place
2. Toric lens malposition may be noted immediately, or the lens may rotate later in the postoperative period

C. Describe pertinent clinical features

1. Steep cylindrical axis points to the tightest suture
2. Corneal striae may emanate from tight sutures

D. Testing

1. Slit lamp exam
 - a. Evaluate lens position including toric IOL axis alignment
 - b. Inspect cornea for evidence of tight sutures or wound burn
2. Keratometry
3. Topography
4. Refraction

II. Define the risk factors

A. Tight sutures or wound burn

B. Wound gape if suture tension is unevenly distributed

C. Inattention to precise toric IOL alignment and placement of limbal relaxing incisions; careful preoperative planning and upright preoperative marking to identify cyclotorsion are essential

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Eyeglasses
2. Contact lenses

B. Describe surgical therapy options

1. Suture lysis
2. Astigmatic keratotomy

3. Limbal relaxing incisions
4. Photorefractive keratectomy (PRK), laser in situ keratomileusis (LASIK)
5. Repair of a wound dehiscence
6. Reposition tilted IOL or rotate misaligned toric lens

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Focal Points: Phacoemulsification, Module #6, 1994, p.9.

Posterior capsule opacification

I. Describe the approach to establishing the diagnosis

A. Etiology

1. Proliferation of viable lens epithelial cells across the posterior capsule with secondary capsular wrinkling and opacification

B. Epidemiology

1. Most common complication of modern cataract surgery
2. Incidence varies with patient age, lens material, and edge design

C. List the pertinent elements of the history

1. Decreased vision
2. Glare

D. Describe pertinent clinical features

1. Posterior capsule wrinkles and opacification
2. Opacification either due to fibrosis, cortical pearls or a combination

II. Define the risk factors

A. Incomplete cortical clean-up

B. History of intraocular inflammation

C. Younger age

D. Longer postoperative interval

E. Intraocular lens (IOL) in sulcus

F. Continuous curvilinear capsulorrhexis diameter larger than optic

G. Lens material and design

III. Describe patient management in terms of treatment and follow-up

A. Describe surgical therapy options

1. Observation
2. Neodymium yttrium-aluminum-garnet (Nd:YAG) laser posterior capsulotomy

IV. List the complications of treatment, their prevention and management (See Neodymium yttrium-aluminum-garnet laser posterior capsulotomy)

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p.77, 80, 90, 95, 104-110.
3. Dewey S. Posterior capsule opacification. Curr Opin Ophthalmol 2006;17:45-53.
4. Schaumberg DA, Dana MR, Christen WG, et al. A systematic overview of the incidence of posterior capsule opacification. Ophthalmology. 1998;105:1213-1221.

Anterior capsule fibrosis and phimosis

I. Describe the approach to establishing the diagnosis

A. Etiology

1. Anterior capsule opacification (fibrosis) and/or contraction (phimosis)

B. List the pertinent elements of the history

1. May be asymptomatic
2. Decreased vision when capsular phimosis results in intraocular lens (IOL) decentration, tilt, or extends into visual axis
3. Glare
4. Sensation of peripheral cloud or haze
5. Refractive shift with change in effective lens position

C. Describe pertinent clinical features

1. Anterior capsular opacification and variable amount of capsular contraction
2. Zonular traction and potential weakening
3. IOL subluxation or tilt
4. Poor visibility of the peripheral retina

II. Define the risk factors

A. Small capsulorrhexis

B. Abnormal or asymmetric zonular support

C. Pseudoexfoliation

III. Describe patient management in terms of treatment and follow-up

A. Describe surgical therapy options

1. Relaxing incisions in the anterior capsule may be created radially with a neodymium yttrium-aluminum-garnet (Nd: YAG) laser

IV. List the complications of treatment, their prevention and management

A. Damage to intraocular lens (pitting)

1. Anterior focus of laser beam

B. Elevated intraocular pressure (IOP)

1. Pre- and post-treatment with a IOP lowering drop
2. Consider IOP check in those at risk
3. If the IOP is elevated, appropriate glaucoma medication should be used until IOP spike resolves

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Neodymium yttrium-aluminum-garnet laser posterior capsulotomy

I. List the indications/ contraindications

A. Indications

1. Visually symptomatic posterior capsule opacification (PCO)
2. To enhance view of fundus
3. Posterior capsular distension syndrome

B. Contraindications

1. Lens implant exchange likely
2. Unstable intraocular lens (IOL)
3. Untreated acute retinal tear
4. Uveitis, including inflammation associated with *P. acnes* or other low virulence organism

II. Describe the pre-procedure evaluation

- ### A. Comprehensive eye examination to determine that PCO is the cause of reduced vision

III. List the alternatives to this procedure

- ### A. Surgical capsulotomy with needle/knife
- ### B. Surgical polishing of capsule

IV. Describe the instrumentation, anesthesia, and technique

A. Instrumentation - Nd: YAG laser/slit lamp apparatus

B. Anesthesia - topical anesthesia

C. Technique

1. Pharmacologic pupil dilation
2. Optional pretreatment with IOP lowering drop
3. Patient comfortably seated and positioned at slit lamp/laser; head may need to be stabilized
4. If desired, contact lens applied
5. Posterior defocus of laser beam (if preferred)
6. Application of laser to capsule
7. Application of appropriate IOP lowering medication for intraocular pressure (IOP) elevation prophylaxis
8. Measure postoperative IOP in high risk individuals (e.g., patients with glaucoma)

V. List the complications of the procedure and prevention and management

A. Retinal tear/detachment

1. Pretreatment examination of peripheral retina

2. Treatment of preexisting retinal breaks
- B. Prolapsed vitreous into the anterior chamber**
 1. Apply laser so the capsulotomy does not extend beyond the optic
- C. Transient elevation of IOP**
 1. Pre- and posttreatment with IOP lowering drops
- D. Diabetic retinopathy**
 1. Pretreatment retina exam
 2. Careful post-treatment exam for progression of retinopathy or appearance of neovascularization of the iris
- E. Uveitis/cystoid macular edema can be induced by liberation of retained lens material**
 1. Requires frequent observation and treatment with topical corticosteroids and IOP lowering medication
- F. Damage to IOLs**
 1. Laser damage may induce pitting of lens optic with potential for reduced visual function
 2. May be prevented by posterior defocus of laser beam and with the aid of a contact lens
- G. Dislocation of the IOL**

VI. Describe the follow-up care

- A. Patients should be made aware of symptoms related to retinal tear or detachment and encouraged to report such changes immediately**
- B. In cases where retained lens material has been liberated, follow-up is necessary to evaluate inflammation, macular edema and/or elevation of IOP**
- C. Consider post-operative topical anti-inflammatory drug, especially in high-risk patients**

VII. Describe appropriate patient instructions

- A. Patients can return to full activities immediately following laser treatment**
- B. Patients must be encouraged to report any reduction in vision, alteration in field of vision, and onset of light flashes followed by "floaters"**
- C. Patients should be made aware that small "floaters" are commonly noted transiently following laser capsulotomy**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Preferred Practice Pattern, Cataract in the Adult Eye, 2006.

Intraocular lens decentration and dislocation

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Asymmetric haptic placement with one haptic in the capsular bag and the other in the sulcus
2. Posterior capsular tear
3. Amputated or damaged haptic
4. Broken intraocular lens (IOL) fixation suture
5. Eccentric, excessively large, or torn capsulorrhexis
6. Insufficient zonular support
7. Irregular fibrosis of the capsular bag
8. Implantation of IOL with insufficient haptic length in ciliary sulcus

B. Define the relevant aspects of epidemiology of the disease

1. More common in patients with weak zonular fibers due to pseudoexfoliation syndrome or trauma
2. May be associated with axial myopia and a large anterior segment
3. Potentially more symptomatic in patients with multifocal lens implants

C. List the pertinent elements of the history

1. May be asymptomatic
2. May complain of severe glare, diplopia (monocular) and reduced vision
3. If the IOL dislocates posteriorly, patients will note sudden blurred vision

D. Describe pertinent clinical features

1. Refractive error associated with change in effective lens position
2. In symptomatic patients, the edge of the IOL is typically seen within the undilated pupil
3. In cases of late dislocation, the entire IOL-capsule complex may be displaced into the vitreous

II. Define the risk factors

- A. Pseudoexfoliation syndrome or any condition with progressive loss or weakening of zonule
- B. History of trauma or posterior segment surgery
- C. Complicated intraoperative course with loss of zonules or capsular rupture
- D. Poorly dilating pupil with uncertain placement of IOL haptics during surgery

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Use of pilocarpine or brimonidine to keep pupil constricted so edge of IOL is no longer in visual axis

B. Describe surgical therapy options

1. Surgical correction may be more difficult after YAG capsulotomy
2. Place both haptics in capsular bag if one is in the sulcus

3. If zonular fibers are intact, consider reposition of IOL into ciliary sulcus
 - a. One-piece acrylic lenses should not be placed in the sulcus due to the potential for iris chafing and inflammation
4. If zonule is compromised, iris or scleral fixation may be used to secure and center the implant
5. The IOL may be removed and replaced with either an anterior chamber intraocular lens (ACIOL), a sclerally fixated posterior chamber intraocular lens (PCIOL) or an iris-fixated IOL

IV. List the complications of treatment, their prevention and management.

- A. The IOL may fall into the vitreous cavity during attempt at lens repositioning. This would require referral to a vitreoretinal specialist
- B. Late suture breakage may require further surgical management

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 100-101.
3. AAO, Focal Points: Managing Retained Lens Fragments and Dislocated Posterior Chamber IOLS after Cataract Surgery, Module #7, 1996, p. 4-9.

Incorrect intraocular lens power

I. Describe the approach to establishing the diagnosis

A. Etiology

1. Preoperative factors
 - a. Incorrect biometry (axial length measurement or keratometry readings)
 - b. Improper lens power calculation due to use of inaccurate data or imperfect predictive power of formula
 - c. Prior refractive surgery
 - d. Manufacturing defects or mislabeled intraocular lens (IOL), rare
2. Intraoperative causes
 - a. Use of incorrect implant
 - b. Intraocular lens (IOL) inversion or improper placement in sulcus
3. Postoperative conditions
 - a. Capsular block syndrome
 - b. Shallow anterior chamber
 - c. Instability or change in position of IOL

B. List the pertinent elements of the history

1. Prior refractive surgery
2. Prior glaucoma surgery
3. Pseudoexfoliation and zonular laxity

C. Describe appropriate testing for establishing the diagnosis

1. Verify that the power of the inserted IOL was the intended power
2. Recheck the axial length and the keratometry postoperatively
3. Dilated exam to check for capsular block and IOL positioning issues

II. Define the risk factors

- A. Simultaneous keratoplasty or retinal detachment repair
- B. Prior refractive surgery
- C. Staphyloma or extremely short/long eyes
- D. Dense cataracts with unreliable measurements
- E. Failure of surgeon to confirm proper IOL selection in operating room

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Eyeglasses
2. Contact lenses

B. Describe surgical therapy options

1. IOL exchange

2. Piggyback IOL
3. Keratorefractive surgery
4. Nd:YAG laser anterior or posterior capsulotomy for capsule block

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Patient Safety Bulletin #2. Minimizing Wrong IOL Placement. <http://www.aao.org/patient-safety-statement/recommendations-of-american-academy-ophthalmology->

Patients with diabetes mellitus and cataract surgery

I. Describe how the high-risk characteristic or comorbidity affects the surgical condition

- A. Diabetes mellitus hastens cataract development**
- B. Neovascularization of the iris and associated complications**
 - 1. Poorly dilating pupil
 - 2. Neovascularization of the angle
 - 3. Posterior synechiae development
 - 4. Spontaneous hyphema and/or bleeding during cataract surgery
- C. Diabetic macular edema changes preoperative axial length measurements for IOL**

II. List the surgical complications for which the patient is at increased risk

- A. Neovascular glaucoma with hyphema**
- B. Worsening of retinopathy with vitreous hemorrhage**
- C. Clinically significant macular edema**
- D. Impaired corneal epithelialization**

III. List steps that can be taken to reduce the operative risks of cataract surgery

- A. Pre-operative blood sugar control**
- B. Careful preoperative slit-lamp biomicroscopic examination including gonioscopy (when indicated) to detect iris neovascularization**
- C. Consider pre-operative evaluation by retinal specialist for treatment of diabetic retinopathy with laser or injections or combined pars plana vitrectomy with endolaser**
- D. Topical steroids and nonsteroidal anti-inflammatory drug agents (NSAIDs) preoperatively and postoperatively as needed**
- E. Silicone intraocular lenses should be avoided in diabetic eyes at high risk for subsequent vitreous surgery in which silicone oil might be injected**
- F. Pre-operative optical coherence tomography (OCT), fluorescein angiography, or both should be considered for clarification of macular pathology**

IV. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results

- A. Accelerate the progression of diabetic retinopathy**
- B. Trigger the development of rubeosis, particularly in the setting of significant retinal nonperfusion and posterior capsule rupture**
- C. Increased posterior capsule opacification**

V. Is the follow up care different than for routine surgery?

- A. The follow-up interval is dictated by the severity of the diabetic retinopathy**

B. Aggressive treatment of postoperative inflammation to reduce the risk of diabetic macular edema

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Murtha T, Cavallerano J. The management of diabetic eye disease in the setting of cataract surgery. *Curr Opin Ophthalmol* 2007;18:13-8. Review.
3. Takamura Y, Kubo E, Akagi Y. Analysis of the effect of intravitreal bevacizumab injection on diabetic macular edema after cataract surgery. *Ophthalmology*. 2009 Jun;116(6):1151-7. Epub 2009 Apr 19.
4. Lanzagorta-Aresti A, Palacios-Pozo E, Menezo Rozalen JL, Navea-Tejerina A. Prevention of vision loss after cataract surgery in diabetic macular edema with intravitreal bevacizumab: a pilot study. *Retina*. 2009 Apr;29(4):530-5.
5. Cheema RA, Al-Mubarak MM, Amin YM, Cheema MA. Role of combined cataract surgery and intravitreal bevacizumab injection in preventing progression of diabetic retinopathy: prospective randomized study. *J Cataract Refract Surg*. 2009 Jan;35(1):18-25.
6. Hartnett ME, Tinkham N, Paynter L, Geisen P, Rosenberg P, Koch G, Cohen KL. Aqueous vascular endothelial growth factor as a predictor of macular thickening following cataract surgery in patients with diabetes mellitus. *Am J Ophthalmol*. 2009 Dec;148(6):895-901.e1. Epub 2009 Oct 17.

Compromised endothelium and cataract surgery

I. List the surgical complications for which the patient is at increased risk

- A. Compromised visualization during surgery
- B. Prolonged postoperative corneal edema
- C. Pseudophakic bullous keratopathy (PBK)

II. List steps that can be taken to reduce the operative risks

- A. Minimize the amount of phacoemulsification energy
- B. Protect the corneal endothelium
 - 1. Pachymetry and endothelial cell counts may be used to help define risk of PBK
 - 2. Use of dispersive or visco adaptive OVD
 - 3. Reapplication of OVD during surgery
 - 4. Consider use of scleral tunnel to minimize Descemet membrane trauma
- C. Poor multifocal IOL candidates
 - 1. Decreased contrast sensitivity
 - 2. Emmetropia less likely after PK or endothelial keratoplasty

III. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results

- A. Potential for long-term PBK despite uncomplicated cataract surgery
- B. Some patients may achieve a satisfactory post op outcome even in the presence of early corneal edema preoperatively
- C. Consider mild myopic refractive target if anticipating additional corneal procedure (endothelial keratoplasty)

IV. Are the patient instructions different (post-op care, vision rehabilitation)?

- A. Corneal recovery may be three months or more after cataract surgery before an endothelial keratoplasty is entertained

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO Focal Points: Strategies for Complicated Cataract Surgery, Module #9, 2005.
- 3. Storr-Paulsen A, Nørregaard JC, Farik G, Tårnhøj J. The influence of viscoelastic substances on the corneal endothelial cell population during cataract surgery: a prospective study of cohesive and dispersive viscoelastics. Acta Ophthalmol Scand. 2007 Mar;85(2):183-7.

Cataract surgery following refractive surgery

I. List the surgical complications for which the patient is at increased risk

- A. Dissatisfaction with the refractive results of cataract surgery
- B. Difficulty calculating intraocular lens implant power
 - 1. True corneal power difficult to assess
- C. Dehiscence of refractive keratotomy incisions
- D. Transient refractive shifts in RK patients after surgery which may last weeks with wide fluctuations
- E. Edema in flap interface
- F. Increased risk of suboptimal visual quality with use of multifocal IOLs

II. List steps that can be taken to reduce the operative risks

- A. Counsel all patients about the increased likelihood of a lens power calculation error, possible need for additional surgery, and diurnal fluctuations (RK patients)
- B. Take steps to improve the accuracy of lens power calculations
- C. Avoid cutting into or across radial and arcuate keratotomy incisions, if possible, to avoid dehiscence. Consider scleral tunnel incision if radial incisions of RK are too close together to avoid
- D. Avoid crossing the interface of a laser in-situ keratomileusis (LASIK) flap with the keratome incision
- E. Evaluate corneal shape using topography, including higher order aberrations especially if considering multifocal IOL

III. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results

- A. For eyes with a history of incisional keratotomy, it may take weeks to several months to obtain refractive stability
- B. If a lens exchange is necessary, it should be performed only after refractive stability has been achieved, typically at one month or more
- C. Post-myopic refractive surgery eyes may have long axial lengths
 - 1. Advise these patients
 - a. Increased risk of retinal hole, tear, or detachment in the pseudophakic state
 - b. Discuss early warning signs of RD and need for prompt evaluation

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 159-161.
- 3. AAO, Focal Points: IOL Power Calculation in Patients with Prior Corneal Refractive Surgery, Module #6, 2013.

Surgery of dense nuclear cataracts

I. List the surgical complications for which the patient is at increased risk

- A. Thermal and mechanical to cornea
- B. Increased phacoemulsification time resulting in an increased risk of postoperative corneal edema
- C. Mechanical and thermal injury to iris
- D. Posterior capsule rupture and vitreous loss
- E. Dropped nucleus

II. List steps that can be taken to reduce the operative risks

- A. Capsule staining with trypan blue for improved visualization of rhexis
- B. Chop techniques to minimize zonular stress during surgery
- C. Minimize the amount of phacoemulsification energy expended
 - 1. Femtosecond laser softening
 - 2. Pulse and Burst phaco settings
 - 3. Torsional US energy with longitudinal bursts
- D. Generous use of dispersive or highly retentive OVD
- E. Modified angled (Kelman) phaco tip
- F. Consider extracapsular cataract extraction or small incision cataract surgery instead of phacoemulsification

III. Is the follow up care different than for routine surgery?

- A. Prolonged corneal recovery and increased post-op inflammation may require additional postoperative visits
- B. Corneal edema may or may not resolve

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Surgery of white cataracts

I. List the surgical complications for which the patient is at increased risk

- A. Completion of capsulorrhexis**
 - 1. Visualization
 - 2. Increased intra-lenticular pressure leading to a sudden radial extension of the anterior capsulotomy
- B. Posterior capsule rupture and vitreous loss**
- C. Dropped nucleus**

II. List steps that can be taken to reduce the operative risks

- A. Enlarge pupil**
- B. Stain the anterior capsule with dye**
- C. Flatten the dome of the anterior capsule with adequate ophthalmic viscosurgical device (OVD) before performing the capsule puncture**
- D. Make a small initial opening in the anterior capsule to remove liquid cortex, then add additional OVD if necessary prior to completing the capsulorrhexis**
- E. Prevention of anterior chamber shallowing during rhexis with careful creation of corneal incision**
- F. Make small, controlled rhexis initially, enlarge prior to nucleus disassembly**

III. Unknown visual prognosis

- A. B-scan ultrasound pre-operatively to verify anatomy does not ensure good visual outcome**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Cataract surgery following a glaucoma filtering procedure

I. Describe how the high-risk characteristic or comorbidity affects the surgical condition

A. Glaucoma filtration surgery may

1. Accelerate cataract development
2. Cause the development of anterior or posterior synechiae
3. Be associated with a large iridectomy
4. Be associated an intraocular drainage tube
5. Be associated with weak zonules

B. Eyes with glaucoma often dilate poorly

1. Previous miotic therapy
2. Pseudoexfoliation syndrome
3. Neovascularization of the iris
4. Posterior synechiae

C. Presence of filtration bleb or drainage tube may require alternate location for surgical incisions

II. List the surgical complications for which the patient is at increased risk

A. Increased filtration through the bleb or drainage device during surgery

B. Variable IOP in the week postoperatively

C. Decreased filtration or bleb failure following surgery and long-term loss of intraocular pressure control

D. Zonular damage

E. Damage to integrity of filtration bleb

1. Bleb leak
2. Increased risk of endophthalmitis

III. List steps that can be taken to reduce the operative risks

A. Assure adequate pupil dilation

B. Avoid making the cataract incision near the glaucoma filter or drainage device.

C. Clear cornea incision to avoid incising the conjunctiva and Tenon capsule

D. Minimize postoperative inflammation by appropriate use of anti-inflammatory agents

1. Often longer than in a standard procedure
2. Reduce chance of bleb fibrosis

E. Avoid additional zonular stress during surgery

F. Suture corneal wound postoperatively

G. Avoid over-pressurizing the eye at the conclusion of the case to prevent bleb rupture

IV. List the implications of the high-risk characteristic or comorbidity on the long-term surgical

results

A. The long-term visual results determined primarily by the course of glaucoma progression

1. Most glaucoma filters and Setons lose effectiveness over time
2. IOP must be monitored on regular basis

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.

Pseudoexfoliation (exfoliation syndrome) and cataract surgery

I. Nature of condition

- A. **Age-related disease causing deposition of fibrillar amyloid-like material throughout tissues of the body**
- B. **Ocular deposition**
 - 1. Lens capsule
 - 2. Iris
 - 3. Ciliary body/zonules
 - 4. Endothelium simulating KP
- C. **Demographics**
 - 1. Patients tend to be 60 years or older
 - 2. Geographic clustering suggests a hereditary pattern.
 - a. Scandinavians
 - b. Eastern Europeans
 - c. Russians
 - d. Ethiopians
- D. **Pertinent clinical features**
 - 1. Concentric deposition of fibrillar material on anterior lens capsule
 - a. Rings noted on the lens capsule
 - b. Best viewed with dilated pupil
 - 2. Transillumination defect and fibrillar material at the pupillary margin
 - 3. Open angle with brown clumps of fibrillar material on trabecular meshwork or near Schwalbe line
 - 4. Flakes of fibrillar material on corneal endothelium
 - 5. Evidence of zonular weakness
 - a. Phaco or iridodonesis
 - b. Lens subluxation or even luxation

II. List the surgical complications for which the patient is at increased risk

- A. **Intraoperative miosis**
- B. **Zonular laxity or instability**
- C. **Floppy iris and iris prolapse**
- D. **Vitreous loss**
- E. **Anterior capsulorrhexis contraction or phimosis**
- F. **IOL tilt and decentration**
- G. **Postoperative intraocular inflammation due to changes in blood aqueous barrier**
- H. **Corneal decompensation**
- I. **Increased intraocular pressure during the immediate postoperative period**

III. List steps that can be taken to reduce the operative risks

- A. Take appropriate steps to assure an adequate pupil size for safe cataract surgery**
 - 1. Mechanical stretch, sphincterotomies
 - 2. Devices such as hooks, rings
 - 3. Viscomydriasis
 - 4. Intraoperative epinephrine
- B. Avoid excessive stress to the zonules during surgery, i.e. chop technique**
- C. Consider capsular tension ring implantation if mild to moderate zonular laxity is present**
- D. Consider sulcus fixation, scleral fixation, iris fixation, capsular tension ring, or anterior chamber IOL implantation if moderate to severe zonular laxity is present**
- E. Avoid an overly small capsulorrhexis, which may increase the chances of phimosis**
- F. Consider intraoperative carbachol/acetylcholine or post-op topical antihypertensive drops/oral meds to control immediate IOP spikes**

IV. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results

- A. Moderate to severe zonular laxity may produce symptomatic pseudophakodonesis**
- B. Lens and/or capsular bag decentration may occur early or late after surgery**
- C. Intraocular pressure (IOP) control may improve after cataract surgery in eyes with pseudoexfoliation**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 11, 57, 85, 111.
- 3. AAO, Focal Points: Strategies for Complicated Lens Surgery, Module #8, 2005.
- 4. Shingleton B, Crandall A, Ahmed I. Pseudoexfoliation and the cataract surgeon: preoperative, intraoperative, and postoperative issues related to intraocular pressure, cataract, and intraocular lenses. J Cataract Refract Surg. 2009;35(6):1101-20.

Small eyes and cataract surgery

- I. List the surgical complications for which the patient is at increased risk**
 - A. Endothelial trauma
 - B. Iris trauma and prolapse
 - C. Intraocular lens (IOL) implant power calculations
 - D. Intraoperative suprachoroidal effusion

- II. List steps that can be taken to reduce the operative risks**
 - A. Use highly dispersive ophthalmic viscosurgical device (OVD) carefully to maintain adequate space
 - B. Preoperative IV mannitol to shrink vitreous volume and increase anterior chamber working space
 - C. Maintain adequate infusion bottle height throughout surgery to prevent hypotony
 - D. Careful construction of corneal entry site for the cataract incision to avoid the iris root
 - E. Calculate lens implant power using a latest generation power formula
 - F. Select IOL with flexible haptics and shorter overall length based on size of capsular bag/anterior segment
 - G. Consider dry PPV to create space in extremely shallow anterior chamber
 - H. If uncomfortable with the surgical techniques in nanophthalmic eyes, consider referral

- III. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results**
 - A. Intraoperative iris trauma may cause glare symptoms or polyopia and require repair
 - B. Persistent corneal edema which may require additional surgery
 - C. Sequelae of choroidal effusion
 - D. Errors in lens power calculation error may necessitate an IOL exchange or implantation of a secondary piggyback lens
 1. Piggyback should be placed in the sulcus

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Wladis EJ, Gerwirth MB, Guo S. Cataract Surgery in the small adult eye. *Surv Ophthalmol* 2006; 51; 153-61.
3. AAO, Focal Points: Strategies for Complicated Cataract Surgery, Module #9, 2005.

High myopia and cataract surgery

I. List the surgical complications for which the patient is at increased risk

- A. Difficulty measuring axial length and calculating intraocular lens (IOL) power, especially if posterior staphyloma is present
- B. Perforation of the globe during the retrobulbar or peribulbar block
- C. Errant capsulorrhexis
- D. Excessive movement of the iris-lens diaphragm causing an deep anterior chamber
- E. Greater patient discomfort if surgery is performed under topical anesthesia due to iris movements and reverse pupillary block
- F. Damage to iris or capsule from dynamic anterior chamber

II. List the postoperative complications for which the patient is at increased risk

- A. Retinal detachment
 - 1. Young male patients without PVD especially high risk
- B. An IOL implanted in the ciliary sulcus is more likely than a lens placed in the capsular bag to be unstable or decenter
 - 1. Large size of ciliary sulcus relative to haptic diameter of IOL
 - 2. Toric IOL rotation in large capsular bag

III. List steps that can be taken to reduce the operative risks

- A. Preoperative
 - 1. Carefully examine peripheral retina to ensure retinal integrity and consider treatment of pathology that might predispose to retinal detachment
- B. Intraoperative
 - 1. Reduce irrigating bottle height to avoid overinflated anterior chamber
 - 2. Avoid incision leakage
 - 3. Ensure adequate, but avoid overpressurization of AC with OVD during capsulorrhexis construction
 - 4. Avoid repeated collapse of AC when exiting the eye to avoid distortion of the vitreoretinal interface
 - 5. Use spatula or cannula to elevate pupil margin, providing path for irrigation fluid thus breaking the 'reverse pupil block' which can cause an excessively deep AC
 - 6. Avoid silicone IOL
 - 7. Attempt optic capture if haptics placed in the sulcus

IV. List steps that can be taken to reduce the postoperative complications

- A. Careful indirect ophthalmoscopy to check for retinal breaks
- B. Give the patient specific information on the signs and symptoms of retinal detachment symptoms (flashes, floaters, shadow)

V. List the implications of the high-risk characteristic or comorbidity on the long-term surgical results

- A. Unaided visual acuity may be suboptimal if there is an IOL power calculation error (most likely to occur in the setting of staphylomata)**
- B. Posterior capsulotomy may increase risk of retinal detachment**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. AAO, Focal Points: Strategies for Complicated Cataract Surgery, Module #9, 2005.

Uveitis and cataract surgery

I. Describe how the high-risk characteristic or comorbidity affects the surgical condition

- A. Inflammation accelerates cataract development
- B. Topical and systemic steroid use accelerates cataract development
- C. Uveitis causes glaucoma, potentially complicating surgery
- D. Uveitis reduces endothelial cell count

II. List the surgical and postoperative complications for which the patient is at increased risk

A. Surgical

- 1. Small pupil with posterior synechiae requiring pupil/iris manipulation
- 2. Weakened zonule possibly leading to zonular dehiscence or IOL dislocation
- 3. Hyphema

B. Postoperative

- 1. Intraocular inflammation aggravated
 - a. Postoperative corneal edema
 - b. Cystoid macular edema
 - c. Synechiae leading to iris bombe, glaucoma, and pupil capture behind part of IOL optic
 - d. IOL removal necessary due to effect of inflammation, with cyclitic membrane, unresponsive low grade inflammation, hypotony, and maculopathy
- 2. Posterior capsular opacification
- 3. Elevated IOP
- 4. Corneal and scleral melt
- 5. Phthisis bulbi

III. List steps that can be taken to reduce the operative risks

A. Control intraocular inflammation months before surgery

- 1. Topical antiinflammatory medications
- 2. Consider systemic immunosuppressives

B. Intraoperative techniques

- 1. Break synechiae
- 2. Capsular dye
- 3. Capsular tension ring
- 4. Removal all cortex
- 5. In children, consider posterior capsulotomy and anterior vitrectomy

C. Maintain control of inflammation after surgery

- 1. Increased number of postoperative visits

IV. List the implications of the high-risk characteristic or comorbidity on the long-term surgical

results

- A. Increased risk of cystoid macular edema (CME)**
- B. Increased risk of inflammation**
- C. Increased risk of elevated IOP**
- D. Increased risk of optic capture and pupillary block in eyes that form synechiae**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. AAO, Cataract Surgery and Intraocular Lenses, 2nd edition, 2001, p. 58.
3. AAO, Focal Points: Strategies for Complicated Cataract Surgery, , Module #9, 2005.

Capsular tension rings

I. Definition

- A. A small ring or partial ring that is inserted into the capsular bag in order to distribute tension on the zonular fibers away from areas of weakness towards areas of zonular strength

II. List the indications and contraindications

A. Indications

1. FDA approved for cases of weak or partially absent zonular fibers
2. Used in cataract surgery or secondary intraocular lens placement in cases of known or suspected zonule defects
3. Special rings exist including rings with eyelets for scleral fixation, partial segment rings, and rings with indentations which allow easier removal of cortical material

B. Contraindications

1. Capsule defect

III. Describe the pre-procedure evaluation

- A. Clock hours of zonular weakness
- B. Presence of phacodonesis
- C. Presence of vitreous prolapse
- D. Identification of risk factors such as pseudoexfoliation, Marfan syndrome, trauma history, etc.

IV. List the alternatives to the procedure

- A. Iris or scleral fixation of intraocular lens (IOL)
- B. Anterior chamber IOL placement
- C. Aphakia with spectacle or contact lens correction

V. List the complications of the procedure, their prevention and management

A. Intra-operative

1. Extension of zonular dialysis or capsular tears
2. Dislocated ring
3. Inadvertent sulcus placement

B. Postoperative

1. Late decentration or dislocation of the IOL/ring/capsular bag complex

C. Prevention

1. Continuous curvilinear capsulorhexis
2. Safety suture secured through one eyelet of ring to allow retrieval
3. Possible suture fixation to sclera

VI. Describe the follow up care

- A. Monitor intraocular pressure**
- B. Retinal evaluation for detachment and cystoid macular edema**
- C. Look for IOL/ring/capsule tilt and decentration**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
2. Hasane K, Butler M, Ahmed II. Capsular tension rings and related devices: current concepts. *Curr Opin Ophthalmol* 2006 17(1):31-41
3. Cionni RJ, Osher RH, Marques DM, et al. Modified capsular tension ring for patients with congenital loss of zonular support. *J Cataract Refract Surg* 2003;29:1668-1673.
4. Menapace R, Findl O, Georgopoulos M, et al. The capsular tension ring; designs, application, and techniques. *J Cataract Refract Surg* 2000;26:898-912.

Cataract surgery in special situations (trauma)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Contusive ocular trauma
2. Penetrating ocular injury
3. Other causes
 - a. Radiation
 - b. Electrical shock
 - c. Chemical injury
 - d. Surgical trauma

B. List the pertinent elements of the history

1. Recent or distant trauma
2. History of exposure to known causative agents
3. Previous intraocular surgery

C. Describe pertinent clinical features

1. Focal or diffuse fluffy white cortical cataract develops shortly after penetrating ocular injury with capsule rupture
 - a. Risk of lens particle or phacoantigenic glaucoma
 - b. Consider retained intraocular foreign body
2. Subcapsular, cortical cataract and nuclear cataracts may develop weeks to years after ocular injury without lens capsule rupture
3. Zonule damage may result in vitreous prolapse, phacodonesis, lens subluxation or complete luxation of a traumatized lens
4. Iris damage is often present
 - a. Iris sphincter tears after contusive injury
 - b. Iridodialysis
 - c. Traumatic mydriasis
 - d. Focal transillumination defect
 - i. Should prompt workup for retained intraocular foreign body

D. Describe appropriate work-up

1. Slit lamp examination
 - a. Gonioscopy to check for angle recession or retained foreign body
2. Ultrasound of the posterior segment if the posterior segment can not be visualized

II. Define the risk factors

A. Male gender

B. High-risk occupations

C. Contact sports

- D. Motor vehicle accidents
- E. Domestic violence

III. Surgical timing

- A. Intraocular inflammation, hemorrhage and pressure should be controlled prior to surgical intervention if possible

IV. List the surgical challenges for which the patient is at increased risk

- A. Difficulties with capsulorhexis
- B. Zonular weakness
- C. Vitreous prolapse
- D. Iris damage
- E. IOL placement
 - 1. Capsular support sufficient
 - a. Capsular bag/Sulcus fixation
 - 2. Capsular support insufficient
 - a. Iris/scleral fixation
 - b. ACIOL
 - c. Aphakic contact lens

V. List the implications of the high-risk characteristics on the long-term results

- A. Visual outcome typically determined by extent of traumatic injuries eg. Retinal damage
- B. IOL decentration or dislocation
- C. Traumatic glaucoma

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 11: Lens and Cataract, 2015-2016.
- 2. Mian SI, Azar DT, Colby K. Management of traumatic cataracts. *Int Ophthalmol Clin* 2002 Summer;42(3):23-31.
- 3. Dannenberg AL, Parver LM, Brechner RJ, Khoo L. Penetration eye injuries in the workplace. The National Eye Trauma System Registry. *Arch Ophthalmol* 1992 Jun;110(6):843-8.

Neurotrophic keratopathy

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Dysfunction of the ophthalmic (first) division of the trigeminal (fifth) cranial nerve
 - a. Trauma, including surgical injury (e.g., trigeminal rhizotomy) and eye surgery
 - b. Tumor
 - i. Compressive mass effect
 - ii. Neuroleptic spread from skin cancer via cavernous sinus
 - c. Herpes zoster ophthalmicus
 - d. Herpes simplex virus keratitis
2. Topical ophthalmic medications
 - a. Chronic topical anesthetic use/abuse
 - b. Beta-adrenergic antagonists
3. Central nervous system compromise of trigeminal nerve function (rare)

B. List the pertinent elements of the history

1. Past medical history
2. Past ocular history
3. Family history

C. Describe pertinent clinical features

1. Decreased corneal sensation
2. Decreased tearing
3. Punctate epithelial erosions
4. Non-healing epithelial defects, sterile ulceration
5. Central, inferior paracentral cornea most often involved
6. HSV keratitis
 - a. Punctate epithelial erosions
 - i. Fluorescein staining
 - ii. Occasional vortex pattern
 - b. Chronic epithelial regeneration lines
 - c. Trophic ulcers
 - i. Thickened, rolled, gray edges
 - ii. Minimal rose bengal staining of edges
 - iii. Intense fluorescein staining/pooling in base
7. Herpes zoster ophthalmicus

D. Describe appropriate laboratory testing for establishing the diagnosis

1. Neuroimaging for suspected tumor, cerebrovascular accident, demyelinating disease if indicated

II. List the differential diagnosis

- A. Aqueous tear deficiency
- B. Exposure keratopathy
- C. Toxicity of topical medications/preservatives (toxic ulcerative keratopathy)
- D. Factitious keratopathy

III. Describe patient management in terms of treatment and follow-up

- A. Describe medical therapy options
 - 1. Treatment of underlying disease
 - 2. Tear supplementation with frequent preservative-free lubricants
 - 3. Reduce evaporative tear loss
 - a. Goggles, moisture shields
 - b. Humidifiers
 - 4. Treatment of concomitant dry eye
- B. Describe surgical therapy options
 - 1. Punctal occlusion
 - 2. Lateral or medial tarsorrhaphy
 - 3. Correction of eyelid abnormalities, lagophthalmos

IV. List the complications of treatment, their prevention and management

- A. Topical lubricants
 - 1. Epithelial toxicity from preservatives/use preservative-free
- B. Surgery (See Punctal occlusion) (See Tarsorrhaphy)

V. Describe disease-related complications

- A. Corneal scarring
- B. Infectious keratitis
- C. Sterile corneal ulceration
- D. Corneal thinning, perforation

VI. Describe appropriate patient instructions

- A. Proper administration of topical medications
- B. Proper frequency and timing for use of topical lubricants
- C. Emphasize need for follow up and adherence to treatment plan
- D. Consider referral for systemic workup

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
- 2. AAO, Focal Points: Neurotrophic Keratitis, Module #2, 2003.

Fungal keratitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Filamentary fungi
 - a. *Fusarium*
 - b. *Aspergillus*
2. Yeast
 - a. *Candida*

B. List the pertinent elements of the history

1. History of trauma with vegetable matter, soft contact wear
2. More insidious onset than with bacterial keratitis
3. Fewer inflammatory signs and symptoms initially compared to bacterial keratitis
4. High index of suspicion helpful

C. Describe pertinent clinical features

1. Gray-white, dry-appearing infiltrate with delicate filamentous or feathery, edge
2. Multifocal or satellite infiltrates may be present
3. Endothelial plaque
4. Hypopyon
5. Focal dense, creamy suppuration that may resemble keratitis induced by gram-positive bacteria
6. Occasional invasion of the iris tissue and posterior chamber
7. May result in a dense fibrinoid response in the anterior chamber
8. May be associated with secondary glaucoma

D. Describe appropriate testing and evaluation for establishing the diagnosis

1. Corneal scraping for smears and cultures
2. High index of suspicion for ulcers that fail to respond to treatment
3. Referral to corneal specialist encouraged if index of suspicion is high

II. Define the risk factors

- A. Trauma to the cornea with plant material commonly associated with filamentary fungal keratitis
- B. Agricultural work and contact lens wear
- C. Topical corticosteroid therapy
- D. Compromised immune status and ocular surface commonly associated with *Candida* keratitis

III. List the differential diagnosis

- A. Bacterial keratitis
- B. *Acanthamoeba* keratitis
- C. Mycobacterial keratitis
- D. Herpes simplex virus keratitis

IV. Describe disease-related complications

- A. Fungal endophthalmitis**
- B. Corneal perforation**
- C. Residual corneal scarring and vascularization**
- D. Glaucoma**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Bron AJ, Seal DV, Hay J. Ocular Infection: Investigation and Treatment in Practice. St Louis: Mosby; 1998.
3. O'Brien TP. Therapy of ocular fungal infections. *Ophthalmol Clin North Am.* 1999;12:33-50.
4. O'Day DM. Fungal keratitis. In: Leibowitz HM, Waring GO III, eds. *Corneal Disorders: Clinical Diagnosis and Management*. 2nd ed. Philadelphia: Saunders; 1998:711-718.
5. Prajna NV, John RK, Nirmalan PK, et al. A randomised clinical trial comparing 2% econazole and 5% natamycin for the treatment of fungal keratitis. *Br J Ophthalmol.* 2003;87:1235-7.

Vernal keratoconjunctivitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Immune-mediated atopic eye disease of children
2. Hypersensitivity reaction affecting predisposed individuals during childhood until early adolescence

B. Define the relevant aspects of epidemiology of the disease

1. More prevalent in countries with dry, hot climates
2. Young males before 10 years of age more commonly affected
 - a. Self-limited, typically lasting 2 to 10 years

C. List the pertinent elements of the history

1. History of other atopic manifestation
 - a. Eczema or asthma in three quarters of patients
 - b. Family history of atopy
2. Bilateral and symmetrical
3. Seasonal exacerbation
4. Symptoms
 - a. Itching (most specific)
 - b. Photophobia
 - c. Tearing
 - d. Hyperemia
 - e. Discharge

D. Describe pertinent clinical features

1. Conjunctival
 - a. Papillae
 - b. Ropy mucous (associated with tarsal papillae)
 - c. Horner-Trantas dots
2. Corneal (more common in tarsal form)
 - a. Punctate epithelial keratitis
 - b. Shield ulcer

E. Describe appropriate testing and evaluation for establishing the diagnosis

1. Diagnosis most commonly made clinically without need for laboratory testing

II. Define the risk factors

A. Family history (genetic predisposition)

B. Hot dry climate

C. Young males (but the disease also occurs in females)

III. List the differential diagnosis

A. Other forms of allergic conjunctivitis

1. Atopic keratoconjunctivitis
2. Seasonal allergic conjunctivitis
3. Perennial allergic conjunctivitis
4. Giant papillary conjunctivitis

B. Infectious conjunctivitis

1. Bacterial
2. Viral

C. Toxic keratoconjunctivitis

D. Infectious keratitis

IV. Describe patient management in terms of treatment and follow-up

A. Avoidance of known allergens

1. Minimize exposure to nonspecific triggering factors (sun, dust, salt water, wind)

B. Medical therapy options

1. Supportive
 - a. Cool compresses
 - b. Artificial tears (to dilute allergen)
2. Topical
 - a. Mast cell stabilizer/antihistamine
 - i. Mast cell stabilizer
 - ii. Antihistamines
 - iii. Combined
3. Anti-inflammatory
 - a. Topical nonsteroidal antiinflammatory drug (NSAID)
 - b. Topical corticosteroids (lower intraocular penetration formulations effective)
 - c. Topical cyclosporine
4. Systemic
 - a. Oral antihistamine
 - b. Oral NSAID
 - c. Rare case of sight-threatening disease may require immunosuppressant doses of steroids or systemic immunomodulators

V. List the complications of treatment, their prevention and management

A. Topical mast cell stabilizers

1. May cause irritation and toxic reactions (replace or discontinue)

B. Topical antihistamine

1. Headache, burning, dry eyes, dry mouth, somnolence (replace or discontinue)

C. Topical corticosteroids

1. May induce cataract, glaucoma and infection

D. Topical cyclosporine

1. May cause irritation and burning

E. Systemic antihistamine

1. Somnolence, dry mouth, anorexia, or constipation

VI. Describe disease-related complications

A. Superior tarsal papillae

1. Pseudoptosis
2. Conjunctival scarring with entropion and trichiasis, or corneal scarring

B. Limbal papillae

1. Corneal scarring, neovascularization, secondary infection

C. Shield ulcers

1. Corneal scarring, secondary infection

VII. Describe appropriate patient instructions

A. Avoidance of known allergens

1. Avoid exposure to nonspecific triggering factors (sun, dust, salt water, wind)

B. Compliance with treatment

C. Explanation of the natural history of the disease and its potential vision threatening complications

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Anwar MS. The role of aspirin in vernal keratoconjunctivitis. J Coll Physicians Surg Pak 2003;13:178-9.
3. Tabbara KF, al-Kharashi SA. Efficacy of nedocromil 2% versus fluorometholone 0.1%: a randomised, double masked trial comparing the effects on severe vernal keratoconjunctivitis. Br J Ophthalmol 1999;83:180-4.
4. el Hennawi M. A double blind placebo controlled group comparative study of ophthalmic sodium cromoglycate and nedocromil sodium in the treatment of vernal keratoconjunctivitis. Br J Ophthalmol 1994;78:365-9.
5. Sharma A, Gupta R, Ram J, et al. Topical ketorolac 0.5% solution for the treatment of vernal keratoconjunctivitis. Indian J Ophthalmol 1997;45:177-80.
6. Bleik JH, Tabbara KF. Topical cyclosporine in vernal keratoconjunctivitis. Ophthalmology 1991;98:1679-84.
7. Kilic A, Gurler B. Topical 2% cyclosporine A in preservative-free artificial tears for the treatment of vernal keratoconjunctivitis. Can J Ophthalmol 2006;41:693-8.
8. Sacchetti M, Lambiase A, Mantelli F et al. Tailored approach to the treatment of vernal keratoconjunctivitis. Ophthalmology 2010;117(7):1294-1299.

Atopic keratoconjunctivitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Individuals with other, non-ocular manifestations of atopy: hay fever, rhinitis, asthma, atopic dermatitis and eczema
2. Immunoglobulin (IgE) E mediated hypersensitivity reaction
3. Depressed systemic cell-mediated immunity
 - a. Increased incidence of bilateral and recalcitrant herpes simplex virus keratitis

B. List the pertinent elements of the history

1. Non-ocular atopic disorders
2. Severe ocular itching
3. Stringy ocular discharge
4. Perennial symptoms with exacerbations and remissions

C. Describe the pertinent clinical features

1. Symptoms
 - a. Itching
 - b. Tearing
 - c. Burning
 - d. Photophobia
 - e. Decreased vision
2. Signs
 - a. Lids
 - i. Thickening (lichenification of facial skin-
 - ii. Crusting
 - iii. Edema
 - iv. Fissures
 - v. Ptosis
 - vi. Blepharitis
 - b. Conjunctiva
 - i. Small, medium or giant papillae
 - ii. Hyperemia
 - iii. "Milky" edema of tarsal conjunctiva
 - iv. Stringy discharge
 - v. Cicatrization and symblepharon in advanced disease
 - c. Cornea
 - i. Punctate epitheliopathy
 - ii. Persistent epithelial defect
 - iii. Shield ulcer
 - iv. Micropannus

- v. Subepithelial scarring
 - vi. Increased incidence of keratoconus and bilateral, recalcitrant herpes simplex virus keratitis
- d. Lens
 - i. Anterior or posterior subcapsular cataracts

II. Define the risk factors

- A. Presence of atopic disease**
- B. Exposure to environmental precipitants**

III. List the differential diagnosis

- A. Vernal keratoconjunctivitis**
 - 1. Seasonal
 - 2. Less corneal neovascularization than atopic keratoconjunctivitis
 - 3. Less symblepharon formation and cataracts than atopic keratoconjunctivitis
- B. Allergic conjunctivitis**
- C. Contact lens-associated giant papillary conjunctivitis**
- D. Infectious conjunctivitis**
 - 1. Viral
 - 2. Bacterial
- E. Rosacea-associated blepharokeratoconjunctivitis**
- F. Medication-associated toxic conjunctivitis**

IV. Describe patient management in terms of treatment and follow-up

- A. Describe medical therapy options**
 - 1. Topical therapy
 - a. Antihistamines
 - b. Corticosteroids
 - c. Cyclosporine/tacrolimus
 - 2. Oral therapy
 - a. Corticosteroids
 - b. Immunosuppressants
 - i. Cyclosporine
 - ii. Methotrexate
 - 3. Systemic antihistamines

V. List the complications of treatment

- A. Complications of topical corticosteroid treatment**
 - 1. Cataracts
 - 2. Glaucoma
 - 3. Predisposition to corneal infections

VI. Describe disease-related complications

A. Conjunctiva

1. Cicatrization
2. Symblepharon

B. Cornea

1. Vascularization
2. Scarring
3. Ulceration

C. Lens (cataract)

VII. Describe appropriate patient instructions

A. Avoid environmental precipitants

B. Stop eye rubbing

C. Compliance with suggested medical therapy important for successful treatment and reducing therapy-associated complications

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Abelson MB, Udell IJ. Allergic and toxic reactions. In: Albert DM, Jakobiec FA, eds. Principles and Practice of Ophthalmology. Vol 2. 2nd ed. Philadelphia: Saunders, 2000:781-803.
3. AAO, Preferred Practice Patterns Committee, Cornea and External Disease Panel. Conjunctivitis Preferred Practice Pattern, 2013.

Thygeson superficial punctate keratitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disorder

1. Unknown

B. Describe the relevant epidemiology of the disorder

1. Infrequent
2. Affects children to older adults

C. List the pertinent elements of the history

1. Intermittent photophobia
2. Tearing
3. Mild blurring of vision
4. Burning, foreign body sensation
5. Usually no conjunctivitis
6. Spontaneously resolves but recurrent
7. Usually bilateral, but may start unilateral
8. Untreated lasts for weeks to 1-2 months

D. Describe pertinent clinical features

1. Scattered clumps of fine epithelial lesions which are round, oval, or stellate
2. Lesions are slightly elevated and may have mild punctate staining over them and subepithelial infiltrates beneath them
3. Variable number and location
4. Minimal or no conjunctival reaction

II. List the differential diagnosis

- A. Staphylococcal toxic keratitis
- B. Rosacea
- C. Herpes simplex virus (HSV) keratitis
- D. Dry eye
- E. Molluscum contagiosum
- F. Epidemic keratoconjunctivitis

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Topical corticosteroids
 - a. Usually exquisitely sensitive to low dose steroids tapered over several days
2. Topical cyclosporine tapered over many months may be of benefit

3. Bandage soft contact lenses provide temporary relief of symptoms and may lead to temporary resolution of the lesions

IV. List the complications of treatment, their prevention and management

- A. **Corticosteroid toxicity and steroid dependence**
 1. Using the lowest dose for the shortest time that is effective is important
- B. **Corneal scarring generally is not seen with this disorder although anterior stromal haze may occur but resolves over time**

V. Describe disease-related complications

- A. **Persistent or recurrent discomfort**
- B. **Blurred vision**
- C. **Steroid dependence complications of glaucoma or cataract**

VI. Describe appropriate patient instructions

- A. **Use the lowest amount of corticosteroids for the shortest time necessary to relieve symptoms**
- B. **Seek ophthalmic care if symptoms persist despite treatment**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Schwab IR, Thygeson P. Thygeson superficial punctate keratopathy. in: Pepose JS, Holland GN, Wilhelmus KR, eds. Ocular Infection and Immunity. St. Louis: Mosby; 1996:403-407.

Marginal corneal infiltrates associated with blepharoconjunctivitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Toxic/immune response to staphylococcal antigens from lid margin organisms

B. List the pertinent elements of the history

1. Acute onset
2. Photophobia
3. May have history of pre-existing blepharitis, lid crusting, chalazia, but not essential
4. Often history of prior episodes

C. Describe pertinent clinical features

1. Usually round infiltrate in anterior stroma peripheral cornea
2. Relative clear zone between lesion and the limbus
3. Conjunctival injection, diffuse or localized
4. Often lid margin changes of staphylococcal blepharitis

D. Describe appropriate laboratory testing for establishing the diagnosis

1. None needed unless failure of response to appropriate treatment

II. Define risk factors

- A. Staphylococcal blepharitis

III. List the differential diagnosis

- A. Microbial keratitis- bacterial, herpes simplex virus (HSV)
- B. Contact lens associated marginal corneal infiltrates
- C. Peripheral ulcerative keratitis of rheumatoid and other autoimmune disease
- D. Phlyctenulosis
- E. Rosacea keratitis
- F. Atopic keratoconjunctivitis

IV. Describe patient management in terms of treatment and follow-up

A. Define medical therapy options

1. Therapy of blepharitis with warm compresses, lid scrubs, topical antibiotic ointment to lid margins
2. Topical corticosteroids or corticosteroid/antibiotic combination
3. Oral tetracyclines or erythromycin may be considered

V. List the complications of treatment, their prevention and management

- A. Exacerbation of active microbial keratitis if inaccurate diagnosis. Culture of lids, conjunctiva or cornea may be considered if diagnosis is uncertain
- B. Corticosteroid induced elevation of intraocular pressure. Limit duration of treatment with corticosteroids. Usually there is a rapid response and they may be tapered after 5 to 7 days

VI. Describe disease-related complications

- A. Peripheral corneal scarring, occasionally thinning, very rare perforation

VII. Describe appropriate patient instructions

- A. Warm compress to the lids (long-term, daily therapy)
- B. Lid scrubs
- C. Antibiotic ointment to lid margins after lid hygiene
- D. Consider long-term use of oral tetracyclines as a prophylactic measure
- E. Advise patients to seek care if develop redness or pain in eye
- F. Patients should be advised not to self-treat with topical corticosteroids
- G. Patients should be aware that problem may recur

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.

Ulcerative keratitis: corneal epithelial defect and stromal inflammation, without or with stromal ulceration

I. Describe the approach to establishing the diagnosis

A. Describe the disease

1. Epithelial defect
2. Stromal inflammation from immune response
3. Possible stromal ulceration

B. List the pertinent elements of the history

1. Contact lens wear
2. Ocular trauma
 - a. Foreign body
 - b. Exposure to fluids (water and chemicals)
3. Prior ocular surgery, including keratorefractive procedure
4. Prior ocular disease
 - a. Recurrent erosion
 - b. Herpes simplex virus (HSV) keratitis
 - c. Varicella zoster virus (VZV) keratitis
5. Recent topical ophthalmic medications
 - a. Anesthetics
 - b. Antibacterials
 - c. Antivirals
 - d. Corticosteroids
 - e. Nonsteroidal anti-inflammatory drugs (NSAIDS)
 - f. BAK preserved drops- frequent use
6. Systemic disease
 - a. Diabetes mellitus
 - b. Connective tissue disorder
 - c. Immunosuppression
7. Nutritional status, including alcohol use
8. Recent exposure to others with "red eye"

C. Describe appropriate testing and evaluation for establishing the diagnosis

1. Infectious ulcerative keratitis
2. Immune-mediated ulcerative keratitis
 - a. Serological tests

II. Define the risk factors

A. Viral epithelial keratitis

1. Adenovirus keratoconjunctivitis
 - a. Exposure to infected individual or contaminated fomite
2. HSV epithelial keratitis
 - a. Environmental triggers such as sun exposure, recent illness, or recent ocular surgery
3. VZV epithelial keratitis
 - a. Increasing age
 - b. Immunosuppression
 - c. Malignancy

B. Suppurative microbial keratitis

1. Contact lens use (especially extended-wear use)
2. Ocular trauma (corneal foreign bodies, chemical and thermal injuries)
3. Previous ocular and eyelid surgery
4. Loose sutures
5. Previous corneal surgery (including refractive surgery and penetrating keratoplasty)
6. Medication toxicity (medicamentosa)
7. Immunosuppression (systemic and local)
8. Anesthetic abuse or neurotrophic cornea (from recurrent herpes, surgery, neurologic disease)
9. Ocular surface disease (trichiasis, exposure/lid abnormalities, tear film abnormalities)
10. Adjacent infections (blepharitis, conjunctivitis, dacryocystitis, canaliculitis)

C. Nonsuppurative keratitis

1. Ocular surface disorders
 - a. Neurotrophic keratitis
 - b. Recurrent erosion
 - c. Bullous keratopathy
 - d. Keratoconjunctivitis sicca
2. Systemic conditions
 - a. Connective tissue disorders
 - b. Stevens-Johnson syndrome
 - c. Mucous membrane pemphigoid
 - d. Atopic dermatitis/blepharoconjunctivitis
 - e. Vitamin A deficiency

III. List the differential diagnosis

A. Ulcerative keratitis (corneal epithelial defect and stromal inflammation)

1. Punctate and dendritic epithelial keratitis and epithelial erosions with stromal infiltrate
 - a. Viral infections
 - i. Adenovirus keratoconjunctivitis
 - ii. HSV epithelial keratitis
 - iii. VZV epithelial keratitis
2. Suppurative or necrotizing ulcerative keratitis

- a. Microbial infections
 - i. Bacterial keratitis
 - ii. Fungal keratitis
 - iii. Acanthamebic keratitis
- b. Herpetic keratitis (persistent corneal epithelial defect with necrotizing herpes simplex virus stromal keratitis)
- c. Zoster-associated neurotrophic keratopathy
- 3. Nonsuppurative ulcerative keratitis
 - a. Marginal corneal infiltrate associated with blepharoconjunctivitis
 - b. Sterile infiltrate due to contact lens wear or corneal trauma
 - c. Peripheral ulcerative keratitis associated with systemic immune-mediated disease
 - d. Mooren ulcer
- B. Corneal epithelial defect without stromal inflammation**
- C. Nonulcerative keratitis: stromal inflammation without epithelial defect**
 - 1. Phlyctenulosis and rosacea keratitis
 - 2. Vernal and atopic keratoconjunctivitis
 - 3. Various forms of nonulcerative keratitis (HSV stromal keratitis, VZV stromal keratitis, Epstein-Barr virus keratitis, interstitial keratitis)
- D. Nonulcerative opacity: stromal opacification with corneal thinning but intact corneal epithelium**
 - 1. Terrien marginal corneal degeneration
 - 2. Senile furrow degeneration
 - 3. Postinfectious and posttraumatic corneal scarring

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.

Peripheral ulcerative keratitis associated with systemic immune-mediated diseases

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Vasculitis and immune complex deposition

B. Describe the relevant epidemiology of this disease

1. 50% have underlying systemic connective tissue disease
 - a. Rheumatoid arthritis (most common)
 - b. Granulomatosis with polyangiitis (formerly Wegener's)
 - c. Systemic lupus erythematosus (SLE)

C. List the pertinent elements of the history

1. Review of systems looking for connective tissue disease
2. Ocular symptoms vary based on the underlying etiology
 - a. Foreign body sensation and photophobia
 - b. Tearing
 - c. Pain may or may not be present.

D. Describe pertinent clinical features

1. Peripheral epithelial defect, infiltration of the corneal stroma, and thinning

II. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Treat systemic disorder and other comorbidities
 - a. Consider referral to specialist
2. Maintain adequate lubrication of the ocular surface
3. Promote corneal re-epithelialization
4. Caution using topical steroids which may accelerate cornea melting

B. Describe surgical therapy options

1. May require surgical treatment

III. List the complications of treatment, their prevention and management

A. Systemic side effects of corticosteroids, cytotoxic and immunosuppressive agents

B. Surgical complications

1. Graft rejection
2. Infection
3. Recurrent corneal ulceration and perforation

IV. Describe disease-related complications

A. Disease related complications specific to underlying disease

B. Ocular complications include

1. Uveitis
2. Dry eye
3. Cataract
4. Retinal vasculitis
5. Conjunctivitis
6. Scleritis
7. Eyelid involvement
8. Corneal melt/perforation
9. Loss of vision

V. Describe appropriate patient instructions

A. These patients must be managed in concert with the appropriate medical specialist

1. Patients must understand the importance of maintaining relationship with rheumatologist or other internist.

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Virasch VV, Brasington RD, Lubniewski AJ. Corneal Disease in Rheumatoid Arthritis. In Cornea. ed. Krachmer, Mannis, Holland, 3rd Ed. 2011, Elsevier inc.
3. Sugar J, Cortina MS. Corneal Disease Associated with nonrheumatoid Collagen-Vascular Disease, In Cornea. ed. Krachmer, Mannis, Holland, 3rd Ed. 2011, Elsevier inc.

Primary acquired melanosis of the conjunctiva

I. Describe the approach to establishing the diagnosis

A. Describe pertinent clinical features

1. Flat, brown lesion of the conjunctival epithelium
2. Unilateral
3. Single or multiple
4. Irregular margins
5. Signs of malignant transformation to melanoma
 - a. Enlargement
 - b. Nodularity
 - c. Increased vascularity or inflammation
6. Rare pigmentary variations
 - a. No visible pigmentation (amelanotic)
 - b. Eyelid margin pigmentation
 - c. Corneal epithelium may have fine pigmentation

B. Describe appropriate laboratory testing for establishing the diagnosis

1. Periodic follow up with photographic documentation as indicated
2. Biopsy with histopathological examination to determine malignant potential (atypia vs no atypia is a pathologic determination)
 - a. Large or progressive lesion of bulbar conjunctiva
 - b. Darkly pigmented lesion of palpebral conjunctiva, fornix, or caruncle
 - c. Multiple pigmented lesions

II. Define the risk factors

A. European ancestry

III. List the differential diagnosis

A. Congenital pigmented lesions of the conjunctiva or episclera

1. Benign epithelial melanosis of conjunctiva (complexion-associated conjunctival pigmentation)
2. Congenital conjunctival nevus
3. Ocular melanosis (melanosis oculi) and oculodermal melanocytosis (nevus of Ota)
4. Scleral pigmentation around perforating nerves

B. Acquired pigmentation of the conjunctiva

1. Acquired conjunctival nevus
2. Secondary acquired melanosis of conjunctiva
3. Melanoma of conjunctiva (See Melanoma of the conjunctiva)

IV. Describe patient management in terms of treatment

- A. Referral to external disease specialist or ocular oncologist may be most appropriate for serial examination with photography and/or excisional biopsy with cryotherapy**
- B. Higher risk of mortality with incomplete excision without cryotherapy**

V. List the complications of treatment, their prevention and management

- A. Incomplete excision**
 - 1. Risk of malignant transformation to conjunctival melanoma if atypia present
 - 2. Follow-up to check for local recurrence or spread to preauricular or other regional lymph nodes

VI. Describe disease-related complications

- A. Progression to melanoma of the conjunctiva**
 - 1. Higher melanoma risk if histopathological features associated with malignancy (atypia) are present

VII. Describe appropriate patient instructions

- A. Need for follow up**
- B. Awareness of possible recurrence**
- C. Awareness of possible transformation to malignant melanoma**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
- 2. Shields J, Shields C, Mashayekhi A, et al. Primary Acquired Melanosis of the Conjunctiva: Experience with 311 Eyes. *trans Am Ophthalmol Soc.* 2007 Dec; 105: 61-72.

Melanoma of the conjunctiva

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Malignancy of conjunctival melanocytes
2. Arises from either primary acquired melanosis of the conjunctiva, preexisting conjunctival nevus or *de novo*

B. List the pertinent elements of the history

1. Duration of lesion
2. Course of lesion, including changes of pigmentation, size and inflammation (comparison with old photographs, if available)
3. Bleeding from lesion
4. Melanoma affecting skin or uvea
5. Previous biopsy

C. Describe pertinent clinical features

1. Location of lesion (palpebral versus bulbar conjunctiva) and unilaterality
2. Size, thickness, number and nodularity of lesion(s)
3. Check whether lesion is attached to the underlying sclera or is freely movable
4. Vascularity (feeder vessel(s)) of lesion
5. Palpation of ipsilateral preauricular, submandibular, and cervical lymph nodes
6. Iris and deep corneal neovascularization may indicate intraocular extension
7. Rule out uveal melanoma with dilated fundus examination, transillumination or ultrasonography
8. Examine skin or recommend dermatology consult to look for cutaneous melanoma

D. Describe appropriate laboratory testing for establishing the diagnosis

1. Excisional biopsy for suspicious lesion with cryotherapy to reduce mortality risk
2. Medical or oncology consultation
3. Consider genetic testing

II. Define the risk factors

A. Risk factors for lesion

1. Middle-age or elderly
2. White race or light-skinned ethnicity
3. Primary acquired melanosis of the conjunctiva or acquired nevus of conjunctiva

III. List the differential diagnosis

A. Conjunctival nevus

B. Primary acquired melanosis

C. Pigmented epithelial tumor, including squamous cell carcinoma

IV. Describe appropriate patient instructions

- A. Knowledge of possible recurrence, local invasion, metastases, loss of vision, and death**
- B. Need for long-term follow up**
- C. Genetic testing**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
2. Shields CL. Conjunctival melanoma: risk factors for recurrence, exenteration, metastasis, and death in 150 consecutive patients. *Trans Am Ophthalmol Soc* 2000; 98:471-492.

Anterior basement membrane dystrophy (map-dot-fingerprint corneal dystrophy or Cogan microcystic corneal epithelial dystrophy)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Majority of cases likely spontaneous, but autosomal dominant inheritance reported

B. Define the relevant aspects and epidemiology of this disease

1. Increased incidence with age
2. Female > male
3. Recurrent corneal erosions may occur

C. List the pertinent elements of the history

1. Occasional decline in best corrected visual acuity (BCVA)
2. Pain upon awakening (secondary to corneal erosions)
3. Photophobia
4. Tearing
5. History of recurrent erosions
 - a. Pain
 - b. Tearing
 - c. Foreign body sensation
6. May be asymptomatic

D. Describe pertinent clinical features

1. Map lines
2. Dots or microcysts
3. Fingerprint lines
4. Erosion (epithelial)
5. May affect keratometry readings and accuracy of IOL calculations

E. Describe appropriate testing for establishing the diagnosis

1. Slit lamp biomicroscopic exam with retroillumination

II. Define the risk factors

- A. Older age
- B. Family history
- C. Female gender

III. List the differential diagnosis

- A. Meesmann dystrophy**
- B. Reis-Buckler dystrophy**
- C. Corneal abrasion (acute traumatic or recurrent)**

IV. Describe patient management in terms of treatment and follow-up

A. Describe the medical therapy options

- 1. 5% Sodium chloride solution and/or ointment (for ointment use, the most important aspect is to use it prophylactically at bedtime)
- 2. Artificial tears and ointment
- 3. Pressure patch or bandage contact lens for recurrent erosion

B. Describe the surgical therapy options

- 1. Epithelial debridement with or without diamond burr polishing
- 2. Anterior stromal puncture in non-visual axis locations
- 3. Phototherapeutic keratectomy (PTK)

V. List the complications of treatment, their prevention and management

- A. Scar formation with anterior stromal puncture or PTK**

VI. Describe disease-related complications

- A. Irregular astigmatism and loss of BCVA**
- B. Recurrent erosions**
- C. Infection**
- D. Persistent epithelial defect**
- E. Hyperopic shift after PTK**
- F. Refractive surprise in PTK patients following cataract surgery with IOL implantation**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.

Corneal edema

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Decreased corneal endothelial pump and/or barrier function
 - a. Trauma
 - b. Infection
 - c. Immune-mediated
 - d. Hypoxia
 - e. Toxicity of topical or intraocular pharmacologic agents
 - f. Endothelial dystrophies and dysgeneses
2. Disrupted corneal epithelial barrier function
3. Increased intraocular pressure (IOP)

II. Define the risk factors

- A. Increased IOP
- B. Intraocular inflammation
- C. Intraocular surgery

III. List the differential diagnosis

- A. Corneal stromal scarring - prior trauma, infection, inflammation
- B. Corneal stromal inflammation, infiltrates
- C. Congenital corneal opacification

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Endothelial dysfunction
 - a. Topical hyperosmotic agents - 2% or 5% NaCl ointment at bedtime, drops during day
 - b. Lowering IOP may reduce edema
 - c. Use of hair dryer on cool setting upon awakening
2. Treatment of intraocular inflammation if present

B. Surgical options

1. Endothelial keratoplasty
2. Penetrating keratoplasty if stroma scarring present

V. Describe disease-related complications

- A. Secondary infection, melting due to loss of epithelial barrier
- B. Corneal scarring

VI. Describe appropriate patient instructions

- A. Proper use of topical medications**
- B. Proper use of hair dryer to reduce corneal edema**
- C. When to seek further care**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
- 2. AAO, Corneal Dystrophies & Degenerations: A Molecular Genetics Approach, 2003, p.68-69,74,79,83.

Punctal occlusion

I. List the indications/contraindications

A. Indications

1. Treat ocular surface disorders associated with abnormal tear film
 - a. Keratoconjunctivitis sicca
 - b. Exposure keratopathy
 - c. Neurotrophic keratopathy
 - d. Contact lens intolerance related to tear insufficiency
2. Prolong retention and reduce systemic absorption of topically administered drugs

II. Describe the pre-procedure evaluation

- A. Slit-lamp biomicroscopic examination with vital dye staining
- B. Schirmer testing

III. List the alternatives to this procedure

- A. Topical therapy: lubricants and/or cyclosporine
- B. Increase humidity: room humidifier, moisture shields
- C. Tarsorrhaphy
- D. Bandage contact lens

IV. Describe the instrumentation, anesthesia and technique

A. Plug

1. Reversible punctal plug
 - a. Silicone or dissolvable collagen or polymer
 - b. Topical anesthesia
 - i. Insert plugs into punctum with package inserter or forceps
2. Permanent intracanalicular plug
 - a. Topical anesthesia
 - i. Insert plugs into canaliculus with forceps

B. Surgical

1. Local and/or topical anesthesia
2. Cauterize punctum with high-temperature handheld battery cautery or radiofrequency unit

V. List the complications of this procedure, their prevention and management

A. Epiphora

1. Prevention - reversible procedure prior to permanent occlusion, occlude 1 punctum at a time in each eye
2. Treatment - removal of plug

- B. Loss of plug**
 - 1. Treatment - larger plug or cauterize
- C. Recanalization of obliterated punctum**
 - 1. Prevention - avoidance of laser to occlude punctum
- D. Inflammation of lid or corneal irritation**
 - 1. Treatment - removal of plug
- E. Canaliculitis or dacryocystitis**
 - 1. Risk associated with intracanalicular plug placement or displaced punctal plug

VI. Describe the follow-up care

- A. Slit-lamp biomicroscopic evaluation several weeks after procedure**

VII. Describe appropriate patient instructions (post-op care, vision rehabilitation)

- A. Avoid rubbing inner canthus (if silicone plug with exposed heads)**
- B. Instructions on continued lubrication use**
- C. Describe expectations for improved comfort, visual acuity**
- D. Instructions for contact lens use if appropriate**
- E. Return immediately if any signs of infection, redness, pain or discharge**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
- 2. Ophthalmic Technology Assessment: Punctal Occlusion for the Dry Eye, Ophthalmic Technology Assessment Committee Cornea Panel, American Academy of Ophthalmology, *Ophthalmology*, September 1997, Vol 104, 1521-1524.
- 3. Smart Plug Study Group. Management of Complications after Insertion of the SmartPlug Punctal Plug. *Ophthalmology*, October 2006, Vol 113, 1859-1862.

Tarsorrhaphy

I. List the indications/contraindications

A. Indications

1. Severe keratopathy, persistent epithelial defect, or corneal thinning resulting from
 - a. Neurogenic exposure keratopathy (Cranial Nerve (CN) VII palsy)
 - b. Neurotrophic corneal ulceration (CN V deficit, herpes simplex and herpes zoster keratitis)
 - c. Severe keratitis sicca
 - d. Progressive corneal thinning or descemetocoe (e.g., peripheral ulcerative keratitis due to rheumatoid arthritis)
 - e. Limbal stem cell deficiency (e.g. Stevens-Johnson syndrome and chemical burns)
 - f. Eyelid abnormalities (e.g. trauma, previous eyelid surgery, cicatricial or restrictive eyelid disease, ectropion and floppy eyelid syndrome)
 - g. Exposure (thyroid eye disease (thyroid related orbitopathy), orbital tumors)

B. Contraindications

1. Active microbial (bacterial or fungal) keratitis (although tarsorrhaphy may be necessary in some cases after the infection is controlled)

II. Describe the pre procedure evaluation

- A. Visual acuity testing to document vision
- B. Slit-lamp biomicroscopic examination documenting keratopathy location and examining palpebral conjunctiva for foreign bodies or keratinization
- C. External examination to document eyelid abnormalities (e.g. lagophthalmos, eyelid retraction, and proptosis)
- D. Assessment of corneal sensitivity
- E. Determination of what type of tarsorrhaphy is necessary (permanent vs. temporary) and extent of tarsorrhaphy (lateral vs. medial vs. central vs. total)

III. List the alternatives to the procedure

- A. Frequent lubrication with ointments and artificial tears
- B. Bandage contact lens
- C. Amniotic membrane grafting
- D. Temporary eyelid taping
- E. Botulinum toxin injection to induce a temporary ptosis
- F. Moisture chambers
- G. Other eyelid surgery as indicated to correct ectropion, lagophthalmos, or eyelid retraction, including tarsal strip procedure and gold eyelid weights

IV. Describe the technique

A. Temporary suture

1. Local anesthesia

2. Place suture through upper and lower lids. Placing over bolster may prolong retention

B. Temporary glue

1. Topical anesthesia
2. Manually oppose upper and lower eyelids with slight eversion and apply cyanoacrylate glue to lid margin and lashes

C. Permanent

1. Local anesthesia
2. De-epithelialize portion of eyelid margin for adherence
3. Place vertical incision in upper and lower tarsus
4. Place absorbable sutures in horizontal mattress fashion joining upper and lower lid tarsal groves

V. List the complications of the procedure, their prevention, and management

A. Temporary

1. Premature loosening of sutures
2. Suture infection (prevention: antibiotic ointment)

B. Permanent

1. Tarsorrhaphy dehiscence (prevention: leave sutures for longer or use nonabsorbable sutures)
2. Wound infection (prevention: antibiotic prophylaxis)
3. Trichiasis or eyelid abnormalities after tarsorrhaphy is severed (prevention: meticulous technique when performing tarsorrhaphy)
4. Corneal epithelial defects or corneal ulceration from loose or inappropriately placed sutures or from misdirected eyelashes resulting from the procedure (prevention: removal of loose sutures, epilation of eyelashes)

VI. Describe follow up care

A. Regular follow-up to monitor corneal and tarsorrhaphy status

VII. Describe appropriate patient instructions

- A. Instructions on the use of antibiotic ointment to the eyelids following tarsorrhaphy**
- B. Instruction on the use of lubricants and/or topical antibiotics, depending on the underlying problem**
- C. Instruction on importance of regular follow-up**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.

Pterygium excision

I. List the indications

A. Indications

1. Decreased vision
 - a. Lesion encroaching on the visual axis
 - b. Regular or irregular astigmatism
 - c. Disrupted tear film
2. Visual side effects such as glare and halos around lights or difficulty driving at night
3. Restriction of motility causing diplopia
4. Persistent/recurrent discomfort and/or inflammation
5. Cosmesis
6. Interference with contact lens wear
7. To rule out malignant process if suspicious features are present

II. Describe the pre-procedure evaluation

- A. Refraction
- B. Assessment of tear film
- C. Motility evaluation
- D. Slit-lamp biomicroscopy
- E. Consider corneal topography to evaluate induced astigmatism

III. List the alternatives to this procedure

- A. Observation
- B. Topical lubricants and anti-inflammatory agents to reduce discomfort and erythema

IV. Describe the instrumentation, anesthesia and technique

A. Instrumentation

1. Microsurgery with standard anterior segment instruments
2. A diamond burr may be of benefit in smoothing a rough corneal surface after lesion removal

B. Anesthesia

1. Topical and/or subconjunctival anesthetic may be sufficient in some cases
2. Peribulbar anesthetic injection helpful to reduce blepharospasm

C. Technique

1. The pterygium can be resected by incising the body of the lesion and dissecting it at the limbus or by the corneal portion of the lesion first
 - a. Dissection should remove subconjunctival fibrovascular tissue while sparing as much of the conjunctiva as possible.
 - b. Options for repair of the defect left in the conjunctiva

- i. Free conjunctival autograft
 - ii. Sliding conjunctival pedicle flap
- c. Amniotic membrane can be used instead of conjunctiva although recurrence is more likely
- 2. MMC applied to scleral bed can be considered in high risk cases
 - a. Local beta irradiation has been used but risk late scleral necrosis
 - b. The MMC should be irrigated off thoroughly
 - c. Postoperative use of MMC drops may carry higher complications of scleral melt

V. List the complications of the therapy

- A. Recurrence- most frequent**
 - 1. 50% for bare sclera techniques - not recommended
 - 2. 5-20% or more with conjunctival flaps and grafts
- B. Pyogenic granuloma formation**
- C. Infection and necrosis of the corneoscleral bed**
- D. Diplopia, strabismus**
- E. Dellen associated with swollen or excessively thick conjunctival or amniotic membrane graft at limbus**
- F. Corneal and/or scleral melting with MMC or radiation**

VI. Describe the follow-up care and patient instructions

- A. Topical corticosteroid-antibiotic combinations frequently initially and tapered over several weeks to months**
- B. Follow-up visits allow suture removal as grafted tissue becomes adherent**
- C. Observation for recurrence**
- D. Antibiotics can be discontinued once epithelial integrity has been re-established; topical steroids are often continued for a few months to reduce the risk of recurrence.**
- E. Ocular lubrication**
- F. Protection of the eye from UV exposure with sunglasses**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.
- 2. Hirst LW. The treatment of pterygium. *Surv Ophthalmol* 2003;48:145-180.
- 3. Mutlu FM, Sobaci G, Tatar T, et al. A comparative study of recurrent pterygium surgery: limbal conjunctival autograft transplantation versus mitomycin C with conjunctival flap. *Ophthalmology* 1999;106:817-21.
- 4. Chen PP, Ariyasu RG, Kaza V, et al. A randomized trial comparing mitomycin C and conjunctival autograft after excision of primary pterygium. *Am J Ophthalmol* 1995 Aug;120:151-60.
- 5. Frucht-Pery J, Sigalos CS, Ilisar M. Intraoperative application of topical mitomycin C for pterygium surgery. *Ophthalmology* 1996 Apr;103:674-7.
- 6. Kucukerdonmez C, Akova YA, Altinors DD. Vascularization is more delayed in amniotic membrane graft than conjunctival autograft after pterygium excision. *Am J Ophthalmol* 2007;143:245-249.
- 7. Luanratanakorn P, Ratanapakorn T, Suwan-Apichon O, et al. Randomised controlled study of conjunctival autograft versus amniotic membrane graft in pterygium excision. *Br J Ophthalmol* 2006;90:1476-80.
- 8. Prabhasawat P, Tesavibul N, Leelapatranura K, et al. Efficacy of subconjunctival 5-fluorouracil and triamcinolone injection in impending recurrent pterygium. *Ophthalmology* 2006;113:1102-9.
- 9. Frucht-Pery J, Raiskup F, Ilisar M, et al. Conjunctival autografting combined with low-dose mitomycin C for prevention of primary pterygium recurrence. *Am J Ophthalmol* 2006;141:1044-1050.

Intraocular pressure and aqueous humor dynamics

I. Tonometry: clinical measurement of intraocular pressure (IOP)

A. Indications and contraindications

1. Indications
 - a. Measurement of IOP for screening and monitoring of glaucoma
2. Relative contraindications
 - a. Corneal laceration
 - b. Flat anterior chamber

B. Pre-procedure evaluation

1. Slit lamp biomicroscopy to evaluate cornea for abnormalities that may affect IOP measurement accuracy
2. Pachymetry to measure corneal thickness
3. Assess patient for things that might affect tonometry accuracy
 - a. Abdominal or thoracic obesity
 - b. Tight collar or necktie
 - c. High astigmatism
 - d. Breath holding or Valsalva
 - e. Contact lens
 - f. Extraocular muscles acting on restricted globe
 - g. Lid squeezing
 - h. Narrow intrapalpebral fissure

C. Different techniques to measure IOP

1. Goldmann tonometry
 - a. Measures IOP by applanating (flattening) the cornea
 - b. Most widely used in clinical practice and research
 - c. Corneal and scleral properties can affect measurement
 - d. Requires periodic calibration
2. Tono-Pen: handheld tonometer that contains a strain gauge and produces an electrical signal as the tip applanates a very small area of the cornea
 - a. Helpful in patients with nystagmus and corneal irregularity
 - b. Underestimates in eyes with higher IOP and overestimates in eyes with lower IOP compared with Goldman
3. Digital palpation
 - a. May be useful in uncooperative patients
 - b. With keratoprosthesis, useful to compare with other eye
 - c. Can be used intraoperatively

D. Tonometry instrumentation and technique

1. Goldmann tonometry
 - a. The patient is instructed to relax, keep the eye still and lids open and avoid breath-holding

- b. Applanation is defined as when the inside edges of the prism-split circular meniscus just touch at the midpoint of their pulsations
 - c. Tonometer tip is cleaned with antiseptic solution
 - 2. Tono-Pen tonometry
 - a. Can be used with the patient supine or upright
 - b. Tonometer tip is covered with rubber cover
 - c. Tonometer tip is touched to the central cornea repeatedly until sufficient measurements are taken by the instrument and the average read from the digital display
 - d. Tonometer tip cover is discarded
- E. Complications of the procedure, their prevention and management**
 - 1. Complication of contact tonometry
 - a. Corneal abrasion
 - i. Prevent by slow careful applanation and encouraging patient to maintain steady head and eye position.
 - ii. Most applanation induced abrasions heal overnight without treatment.
 - b. Antiseptic toxicity to the epithelium
 - i. Tonometer tip should be allow to fully dry between patients
 - c. Potential for transmission of infection
- F. Considerations in interpretation of this procedure**
 - 1. Central corneal thickness may affect IOP measurements by Goldmann and Tono-Pen
 - a. Pachymetry measurements should be used to help interpret, but not to "correct" IOP measurements. In general, Goldmann IOP measurements in eyes with greater central corneal thickness (> 585 microns) should be considered falsely high, and those with thinner central corneal thicknesses (< 500 microns) should be considered falsely low
 - b. CCT should be re-measured after corneal surgery
 - 2. Other corneal properties can affect measurement
 - a. Corneal edema predisposes to falsely low IOP measurements
 - b. Corneal stiffness (i.e. scarring) predisposes to falsely high IOP measurements
 - c. Corneal astigmatism may require prism adjustment
 - 3. A single IOP measurement is only a random sample of a dynamic picture. IOP fluctuates throughout the day and night
 - 4. Technician or clinician influences on IOP measurement
 - a. Pressure from fingers holding lids may be transmitted to globe and elevate IOP
 - b. Excess fluorescein (thick mires) may cause overestimation of IOP
 - c. Inadequate fluorescein (thin mires) may cause underestimation of IOP
 - d. Improper vertical alignment of mires may cause overestimation of IOP
 - e. Repeated applanation tonometry reduces IOP readings

II. Aqueous Humor Dynamics and IOP

A. Route

- 1. Trabecular Outflow (predominant route)
 - a. Ciliary body→
 - b. Posterior chamber→
 - c. Pupil→

- d. Anterior chamber →
 - e. Trabecular meshwork →
 - f. Schlemm canal →
 - g. Collector channels→
 - h. Venous system
2. Uveoscleral Pathway
- a. Ciliary body→
 - b. Posterior chamber→
 - c. Pupil→
 - d. Anterior chamber →
 - e. Ciliary muscle →
 - f. Supraciliary and suprachoroidal space →
 - g. Intact sclera
- B. Aqueous humor formation**
- 1. Site of production is the ciliary processes
 - 2. The majority of aqueous humor formation is independent of IOP
- C. Functions of aqueous**
- 1. Maintain intraocular pressure
 - 2. Provide nutrition and remove metabolic products
- D. Aqueous characteristics**
- 1. Very low protein level compared with plasma
- E. Rate of aqueous formation**
- 1. Varies diurnally and decreases during sleep
 - 2. Decreases with
 - a. Inflammation
 - b. Carotid occlusive disease
 - c. Certain systemic drugs
 - i. Anesthetics
 - ii. Systemic IOP lowering agents
- F. Aqueous humor outflow**
- 1. Facility of outflow varies greatly in normals
 - 2. Decreased in glaucoma
- G. IOP level varies directly with the rate of aqueous formation and episcleral venous pressure and inversely with outflow facility**
- H. Trabecular outflow**
- 1. Majority of outflow is through trabecular meshwork with the juxtacanalicular tissue the major site of outflow resistance
 - 2. Pressure dependent outflow
- I. Uveoscleral outflow**
- 1. Predominantly via CB to supraciliary and suprachoroidal space
 - 2. Pressure independent outflow
 - 3. Increased by prostaglandins analogues

J. Episcleral venous pressure

1. Same as central venous pressure about 8-10 mmHg
2. Increases with
 - a. Disease of orbit
 - b. AV shunts

K. IOP distribution

1. Mean IOP 15.5 mmHg
2. Non-gaussian distribution skewed toward higher IOPs

L. Factors influencing IOP

1. Time of day
2. Breath holding
3. Systemic and topical medications
4. Higher with increased fluid intake
5. Lower with ETOH/cannabis intake
6. Higher reclined than vertical
7. Increases with age
8. Higher in first degree relatives of POAG patients

M. Diurnal variation

1. IOP varies 2-6mmHg over 24hr period
2. Diurnal fluctuation >10mmHg suggestive of glaucoma
3. Peak IOP usually during early morning hours
4. Systemic hypotension during sleep → decreased optic nerve perfusion → optic nerve damage

Additional Resources

1. AAO Basic and Clinic Science Course, Section 10 Glaucoma, 2015-2016.
2. Allingham, RR et al. Shields Textbook of Glaucoma 6th Edition 2011
3. Tonometry in Adults and Children. Eisenberg, et al. Ophthalmology 1998; 105:1173-1181.
4. Positive Correlation between Tono-Pen Intraocular Pressure and Central Corneal Thickness. Dohadwala et al. Ophthalmology 1998; 105 1849-1854.
5. Intraocular Pressure difference in Goldmann Applanation tonometry vs Perkins Hand-held Applanation tonometry in Overweight Patients. Gonzaga dos Santos et al. Ophthalmology 105; 2260-2263.
6. Sources of Error with Use of Goldmann-type Tonometers. Whitacre et al. Survey of Ophthalmology 1993; 38(1):1-30.

Post-traumatic or angle recession glaucoma

I. Describe the approach to establishing the diagnosis

A. List the pertinent elements of the history

1. History of blunt ocular trauma, usually with hyphema
2. Traumatic event may occur months to years prior to development of glaucoma
3. Glaucoma occurs in 7-9% of those with angle recession after trauma

B. Describe pertinent clinical features

1. Elevated intraocular pressure (IOP)
2. Optic nerve and visual field findings consistent with glaucoma
3. Gonioscopy reveals
 - a. Broad angle recess with widened ciliary body face (compare to opposite eye because some eyes may have very wide angles but are normal)
 - b. Absent or torn iris processes
 - c. Localized peripheral anterior synechia (PAS) may be present at the border of the recession

II. Define the risk factors

- A. History of blunt ocular trauma
- B. Angle recession of 180 degrees or more
- C. Young men, with greater prevalence among African American men than white men
- D. Up to 50% of fellow eyes may develop increased IOP suggesting a possible predisposition to glaucoma

III. List the most common or critical entities in the differential diagnosis

- A. Primary open angle glaucoma
- B. Secondary glaucomas which alter the appearance of the angle
- C. Contusion angle deformities (i.e., cyclodialysis cleft which may look similar but has a different clinical course)
- D. Normal anomalous appearing angles with wide ciliary body face

IV. Describe appropriate patient instructions

- A. Regular IOP checks for the rest of life at least once a year (follow Preferred Practice Guidelines from AAO for glaucoma suspects)
- B. Heightened risk of increased IOP in fellow eyes

Additional Resources

1. AAO, Basic and Clinical Science Course: Section 10: Glaucoma 2015-2016.
2. AAO, Glaucoma Medical Therapy: Principles and Management, 1999, p.204.
3. AAO, Preferred Practice Pattern: Primary Open-Angle Glaucoma Suspect. October 2015.

Subacute angle-closure glaucoma

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Specific anatomic configuration of the anterior segment and the anterior chamber angle
2. Relative pupillary block with appositional closure of a portion of the angle

B. Define the relevant aspects of epidemiology of this disease

1. Race - frequency is greatest among Alaskan & Greenland Inuit and Asians; less common in African-Americans
2. Sex - women affected 3 to 4 times as often as men
3. Age - increases with age, peaking between 55 and 70 years, then declining
4. Hyperopia

C. List the pertinent elements of the history

1. Frequently there are no identifiable symptoms
2. Brief episodes of blurred vision, haloes, and mild ocular pain
3. Symptoms tend to recur under provoking circumstances
4. Resolves spontaneously after cessation of inciting activity or with sleep
5. History of angle-closure glaucoma in family
6. Patient may complain of frequent headaches described as migraines or sinus

D. Describe pertinent clinical features

1. Peripheral angle narrowing
2. Lens tends to be situated more anteriorly; the lens may be large
3. Gonioscopy may reveal varying degree of iris convexity (determined by degree of pupillary block) and angle narrowing; on indentation, peripheral anterior synechiae (PAS) may be seen
4. Varying degree of optic nerve cupping, often asymmetric

E. Describe appropriate laboratory testing for establishing the diagnosis

1. Visual field and other assessment of optic nerve function
2. Consider anterior segment imaging to evaluate axial length, anterior chamber depth, lens thickness, and iridotrabecular apposition

II. List the differential diagnosis

A. Plateau iris syndrome

B. Intermittent secondary angle-closure related to instability of lens

C. Acute angle-closure

D. Glaucoma associated with uveitis

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. If IOP is elevated, glaucoma medications may be used to lower pressure
2. Consider low dose pilocarpine

B. Describe surgical therapy options

1. Laser peripheral iridotomy
2. Argon laser iridoplasty
3. Surgical iridectomy (in patients unable to undergo laser iridotomy)
4. Surgical iridectomy associated with glaucoma filtration surgery
5. Lens extraction

IV. List the complications of treatment, their prevention and management

- A. Post-laser surgery IOP elevation
- B. Post-laser surgery persistent inflammation
- C. Closure of iridotomy: repeat laser or surgical iridectomy
- D. Dysphotopsia and monocular diplopia (rare)

V. Describe disease-related complications

- A. Glaucomatous disc damage and visual field loss
- B. Acute angle-closure glaucoma
- C. Chronic angle-closure glaucoma with progressive PAS formation

VI. Describe appropriate patient instructions and follow-up

- A. Symptoms of acute angle-closure glaucoma attack and need for immediate attention
- B. Need for regular follow-up examinations
- C. Possibility of residual glaucoma following laser treatment which may require further medication and/or surgery
- D. Evaluate other eye for risk of angle-closure glaucoma and treat as appropriate
- E. Recommend evaluation of family members

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Ritch R, Lowe RF. Angle-closure glaucoma: clinical types. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas. 2nd ed. St. Louis, MO: Mosby; 1996: 827-829.
3. AAO, Glaucoma Medical Therapy: Principles and Management, 1999, p.193-4,196.

Chronic angle-closure glaucoma

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Peripheral iris bowing (anterior bowing)
 - a. Prolonged apposition or repeated subacute attacks lead to gradual peripheral anterior synechia (PAS) formation
 - b. PAS begin as pinpoint synechiae reaching to the mid trabecular meshwork and then gradually expand in width
 - c. PAS "creep" anteriorly

B. Define the relevant aspects of epidemiology of this disease

1. Frequency is highest among Alaskan and Greenland Inuits and among other Asian groups
2. Higher frequency among women
3. Typically associated with hyperopia
4. "Creeping" angle-closure is relatively uncommon in whites
5. Black and Asian patients with angle closure tend to have creeping angle closure
6. Higher prevalence with a family history of angle closure

C. List the pertinent elements of the history

1. Possible prior history of acute angle-closure glaucoma
2. Possible prior history of subacute angle-closure glaucoma with characteristic findings of intermittent head or brow ache associated with blurred vision and/or haloes around lights.

D. Describe pertinent clinical features

1. Elevated intraocular pressure (IOP)
2. PAS on gonioscopy
3. Clinical course resembles that of open-angle glaucoma
 - a. Modest, variable elevation of IOP
 - b. Progressive cupping of the optic nerve head
 - c. Glaucomatous visual field (VF) loss
 - d. Lack of symptoms
4. Some eyes may eventually develop an acute attack of angle-closure glaucoma with pupillary block
5. Glaukomflecken and/or sector iris atrophy may indicate previous attacks of angle-closure glaucoma

II. Define the risk factors

- A. Hyperopia
- B. Short axial length and/or shallow anterior chamber
- C. Family history of angle-closure glaucoma
- D. Inuit or Asian ethnic groups
- E. Older age
- F. Female gender

III. List the differential diagnosis

- A. Neovascular glaucoma
- B. Iridocorneal endothelial syndrome (particularly Chandler syndrome)
- C. Inflammatory glaucoma
- D. Phacomorphic glaucoma
- E. Ciliary body swelling, cysts, masses
- F. Aqueous misdirection
- G. Posterior segment tumors
- H. Scleral buckling procedures with secondary angle closure
- I. Plateau iris
- J. Ciliochoroidal effusions (PRP, CRVO, nanophthalmos, medication-induced)

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

- 1. Miotics (parasympathomimetic agents) - however, higher strength miotics and prolonged use may exacerbate the condition by aggravating pupillary block
- 2. Beta-adrenergic antagonists
- 3. Alpha-adrenergic agonists (sympathomimetics)
- 4. Carbonic anhydrase inhibitors
- 5. Prostaglandin analogues

B. Describe the surgical therapy options

- 1. Laser peripheral iridotomy -- this may eliminate any element of pupillary block but may not lower IOP
- 2. Surgical iridectomy if laser surgery not possible
- 3. Surgical filtering procedures
- 4. Laser iridoplasty
- 5. Cataract extraction may be indicated if there is a phacomorphic component and could possibly be more effective than laser or surgical peripheral iridotomy in preventing further extension of peripheral anterior synechiae

V. List the complications of treatment, their prevention and management

- A. Post-laser surgery IOP elevation
- B. Post-laser surgery persistent inflammation
- C. Post-laser surgery cataract
- D. Dysphotopsia and monocular diplopia (rare)

VI. Describe disease-related complications

- A. End stage glaucoma with severe visual field loss and eventual loss of central vision
- B. IOP generally rises slowly or is intermittently elevated, so pain or visual symptoms are unusual

VII. Describe appropriate patient instructions

- A. Instruct patient about the need for regular follow up exams as there may not be obvious symptoms

- B. Patients need to realize that laser iridotomy will probably not cure disease and additional treatment will almost certainly be warranted**
- C. Patients should be informed of the chronic nature of the disease and the need for lifelong follow-up**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Murphy RM, Bakir B, O'Brien C, et al. Drug-induced Bilateral Secondary Angle-Closure Glaucoma: A Literature Synthesis. J Glaucoma. 2016 Feb; 25(2):e99-e105.
3. Ritch R, Lowe RF. Angle-closure glaucoma: Clinical types. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas, 2nd ed. St. Louis, MO: 1996: 825-827.
4. AAO, Glaucoma Medical Therapy: Principles and Management, 1999, p.198-199.

Secondary angle-closure glaucoma

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Secondary angle closure due to iris membranes
 - a. Neovascularization of the iris
 - b. Epithelial downgrowth or fibrous ingrowth
 - c. Iridocorneal endothelial syndrome (ICE)
2. Secondary pupillary block from
 - a. Uveitis
 - b. Lens related disorders e.g. subluxed crystalline lens, spherophakia, and malpositioned intraocular lenses (IOLs)
3. Retinal conditions
 - a. Ciliary body edema after panretinal photocoagulation, central retinal vein occlusion, placement of scleral buckle
 - b. Intraocular gas with forward shift of lens-iris diaphragm
 - c. Choroidal detachment (serous or hemorrhagic)
 - d. Intraocular tumors
 - e. Medication-induced: sulfonamide derivatives such as topiramate

B. List the pertinent elements of the history

1. History of ocular surgery and/or trauma
2. History of uveitis
3. History of diabetes, central retinal vein occlusion, carotid occlusive disease
4. Medication history
5. Pain
6. Decreased vision

C. Describe the pertinent clinical features

1. Shallow or flat anterior chamber (AC) both central and peripheral
2. Anterior displacement of lens, IOL or vitreous face

D. Describe appropriate testing and evaluation for establishing the diagnosis

1. Anterior segment exam with gonioscopy
2. Indirect ophthalmoscopy
3. B-scan ultrasound

II. Define the risk factors

- A. Diabetes, systemic hypertension, retinal vascular occlusion, carotid occlusive disease, age
- B. Eyes with history of previous intraocular surgery (especially trabeculectomy)
- C. Previous trauma or other lens-related disorders

III. List the differential diagnosis

- A. **Pupillary block glaucoma**
- B. **Aqueous misdirection**

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Treatment of elevated IOP with medical hypotensive therapy
2. Treatment of anatomical aspect of disease
 - a. Cessation of sulfonamide derivative if identified as the cause of secondary angle-closure glaucoma
 - b. Cycloplegics to break pupillary block following uveitis, following retinal procedures, or in ciliochoroidal effusions to deepen anterior chamber and prevent synechial angle closure.

B. Describe surgical therapy options

1. Laser peripheral iridotomy to treat pupillary block for some forms of secondary angle-closure glaucoma
2. Pan retinal photocoagulation and anti-VEGF therapy in neovascular glaucoma
3. Referral to glaucoma specialist as drainage devices may be needed to control IOP in many forms of secondary angle-closure glaucoma

V. Describe disease-related complications

- A. **Corneal decompensation**
- B. **Cataract**
- C. **Peripheral anterior synechiae (this defines the disease)**
- D. **Complications from glaucoma surgery**
- E. **Chronic IOP elevation with subsequent required medical or surgical therapy**
- F. **Retinal detachment (most often associated with "kissing" hemorrhagic choroidal detachments)**
- G. **Metastatic disease associated with intraocular tumors**

VI. Describe appropriate patient instructions

- A. **Close postoperative follow-up required with care to avoid strain, trauma**
- B. **These patients must be managed in concert with the appropriate medical subspecialist**
 1. Patients must understand the importance of maintaining these relationships

Additional Resources

1. AAO, Basic and Clinical Sciences Course. Section 10: Glaucoma, 2015-2016.
2. Shields MB. Textbook of Glaucoma. 6th edition. Baltimore: Williams & Wilkins, 2010.
3. Ritch R, Shields MB, Krupin T. The Glaucomas. The C.V. Mosby Company
4. Ritch R, Shields MB. The Secondary Glaucomas. The C.V. Mosby Company
5. AAO, Ophthalmology Monographs 4. Glaucoma Surgery: Principles and Techniques, 2nd ed. 1998.
6. Epstein DL, Allingham RR, Schuman JS, eds. *Chandler and Grant's Glaucoma*. 4th ed. Baltimore: Williams & Wilkins; 1997.
7. Stamper RL, Lieberman MF, Drake MV, eds. *Becker-Shaffer's Diagnosis and Therapy of the Glaucomas*. 8th ed. St Louis: Mosby; 2009.
8. AAO, Focal Points: Gonioscopy in the Management of Glaucoma, Module #3, 2006.

Aqueous misdirection (malignant glaucoma, ciliary block glaucoma)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of aqueous misdirection

1. Usually follows ocular surgery
2. There is anterior rotation of the ciliary body postoperatively (rarely this rotation can occur spontaneously)
3. Posterior misdirection of aqueous humor into the vitreous cavity causes shallowing of the peripheral and central anterior chamber with elevation of intraocular pressure (IOP)

B. Define the relative epidemiologic aspects of this disease

1. A rare event that occurs most frequently following glaucoma surgery in patients with angle closure
2. Fellow eye is at increased risk of developing aqueous misdirection

C. List the pertinent elements of the history

1. Pain
2. Photophobia
3. Decreased vision
4. Red eye

D. Describe pertinent clinical features

1. Uniformly shallow or flat anterior chamber
2. Anterior displacement of lens, intraocular lens (IOL), or vitreous face
3. Anteriorly rotated ciliary processes
4. Normal or elevated IOP
5. Shallow anterior chamber in presence of patent iridotomy/iridectomy

E. Describe appropriate testing and evaluation for establishing the diagnosis

1. Ultrasound
 - a. B-scan to rule out ciliary body effusion or suprachoroidal hemorrhage
 - b. UBM may be used to show anterior rotation of the ciliary processes

II. Define the risk factors

- A. Eye with acute or chronic angle closure
- B. Persistent shallowing of the anterior chamber in the perioperative period
- C. Small eye

III. List the differential diagnosis

- A. Acute suprachoroidal hemorrhage
- B. Pupillary block glaucoma
- C. Choroidal effusion, which can also shallow the anterior chamber, but IOP is low in this condition

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Treat aqueous misdirection
 - a. Intensive cycloplegic therapy
 - b. Stop/avoid parasympathomimetic agents (misdirection may be aggravated by parasympathomimetic therapy)
2. Treat elevated IOP
3. Medical therapy is successful in approximately 50% of cases
 - a. Many days of therapy may be necessary to reverse the acute problem
 - b. Indefinite therapy may be necessary to prevent recurrence

B. Describe surgical therapy options (laser surgery and incisional)

1. Neodymium yttrium-aluminum-garnet (Nd: YAG) laser surgery disruption of anterior vitreous face in aphakic or pseudophakic eyes
 - a. Placement of hole in anterior hyaloid peripheral to the IOL may be more effective than placement in other locations
 - b. If a PI is already present, lysis of the anterior hyaloid is possible through the PI
2. Pars plana vitrectomy, necessitating referral to a retina specialist
3. Zonulo-hyaloido-vitrectomy (anterior hyaloid vitrectomy performed via clear corneal incision through peripheral iridectomy in pseudophake)

V. List the complications of treatment, their prevention and management

A. Intensive cycloplegic therapy

1. Dilated pupil
2. Blurred vision

B. Complications of glaucoma medical therapy

C. Complications of glaucoma laser surgery and incisional surgery

1. Persistently elevated IOP
2. Recurrence of malignant glaucoma

VI. Describe disease-related complications

A. Decreased vision

1. Chronic IOP elevation with subsequent optic disc damage and visual field loss
2. Cataract

B. Chronic IOP elevation requiring medical or surgical therapy

C. May recur after pars plana vitrectomy especially in phakic eyes because of difficulty in removing anterior vitreous

VII. Describe appropriate patient instructions

- A. May need to maintain long-term low dose of cycloplegic therapy to avoid recurrence (taper as possible)
- B. Follow recommended IOP-lowering regimen (medical or surgical) to prevent permanent vision loss
- C. Continued management and follow-up essential, especially in light of risk to fellow eye

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Shields MB. Textbook of Glaucoma. 4th edition. Baltimore: Williams & Wilkins; 1998.
3. Ruben S, Tsai J, Hitchings R. Malignant glaucoma and its management. *Br J Ophthalmol*. 1997;81:163-167.
4. Quigley HA, Friedman DS, Congdon NG. Possible mechanisms of primary angle-closure glaucoma and malignant glaucoma. *J Glaucoma* 2003; 12: 167-80.

Laser trabeculoplasty

I. List the indications and contraindications

A. Indications

1. Primary open-angle glaucoma with uncontrolled intraocular pressure (IOP)
 - a. Primary therapy
 - b. Adjuvant therapy when medical therapy is inadequate or not tolerated
2. Secondary open-angle glaucoma with uncontrolled IOP (primary or adjuvant therapy)
 - a. Pseudoexfoliation glaucoma
 - b. Pigmentary glaucoma
 - c. Steroid-induced glaucoma
3. Inadequate IOP control following incisional surgical therapy of open-angle glaucoma
4. Ocular hypertension with uncontrolled IOP in selected cases
5. Selective laser trabeculoplasty (SLT) after previous laser trabeculoplasty

B. Contraindications

1. Absolute contraindications
 - a. Acute angle-closure glaucoma
 - b. No visible angle structures, with or without angle-closure glaucoma
 - c. Traumatic glaucoma with angle recession
 - d. Uveitic and neovascular glaucoma with or without angle closure
2. Relative contraindications
 - a. Developmental glaucoma
 - b. Failed previous laser trabeculoplasty
 - c. Less than 90 degrees of open angle

II. Describe the pre-procedural evaluation

- A. Gonioscopy
- B. Assess trabecular pigmentation
- C. Measure intraocular pressure
- D. Review other treatment options with the patient

III. List the alternatives to this treatment or therapy

- A. Medication
- B. Incisional surgery

IV. Describe the instrumentation and technique

- A. Laser surgery
 1. Frequency-doubled neodymium Nd:YAG laser surgery (selective laser trabeculoplasty)

2. Argon laser trabeculoplasty
3. Diode laser trabeculoplasty

V. List the complications of the procedure, their prevention and management

- A. Elevated IOP in the immediate post-laser surgery period
- B. No reduction, or an elevation, in IOP in the late postoperative period

VI. Describe appropriate patient instructions

- A. During the immediate post-laser surgery period
 1. Use topical anti-inflammatory agent in addition to any other glaucoma medications
 2. Resume normal activities
 3. Call for redness, pain or change in vision

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. AAO, Glaucoma Surgery, 1998, p.179-187.
3. AAO, Atlas: Color Atlas of Gonioscopy, 2000, p. 81, 83, 113, 114, 115.
4. Ophthalmic Technology Assessment: Laser Trabeculoplasty for Primary Open-Angle Glaucoma, Ophthalmic Technology Assessment Committee Glaucoma Panel, American Academy of Ophthalmology, *Ophthalmology*, October 1996, Vol 103, 1706-1712.

Incisional filtering surgery for open angle glaucoma

I. List the indications/contraindications

A. Indications

1. Elevated IOP on maximally tolerated medical therapy and laser trabeculoplasty (if an alternative) with a significant risk of optic nerve damage or vein occlusion
2. Poor tolerance of or compliance with medications

B. Contraindications

1. Blind eye
2. Extensive conjunctival scarring
3. Extremely thin sclera
4. Poor visual potential
5. Need for contact lenses for visual rehabilitation (relative contraindication)

II. List the alternatives to this procedure

A. Continue medical therapy with risk of progressive vision loss

B. Laser procedures

1. Laser trabeculoplasty
2. Ciliary body cyclophotocoagulation

C. Other procedures

1. Implantable shunt device
2. Nonpenetrating filtering surgery
3. Angle surgery

III. Describe instrumentation, anesthesia, and technique

A. Knowledge of both internal and external anatomy of limbal area

B. Anesthesia

1. Peribulbar / retrobulbar anesthesia
2. Topical / intracameral / Sub-Tenons anesthesia
3. General anesthesia

C. Use of anti-fibrotic agents

1. Greater success and lower IOP following surgery
 - a. 5-Fluorouracil
 - b. Mitomycin C
2. Risk for failure or complication
 - a. Active anterior segment neovascularization (rubeosis iridis)
 - b. Active iritis (uveitic glaucoma)

- c. Younger patients (< 50 years of age)
- d. Patients of African descent
- e. Aphakia
- f. Previous failure of filtering surgery
- g. Episcleral fibrosis is most common cause for filtration failure and anti-fibrotic agents inhibit cellular proliferation and fibrosis
- h. Chronic topical glaucoma medication

D. Trabeculectomy technique

- 1. Exposure with corneal traction suture or superior rectus bridle suture
- 2. Limbus-based vs. fornix-based conjunctival flap
- 3. Partial thickness scleral flap
- 4. Application of antimetabolite to episcleral surface for desired amount of time, if planned
- 5. Copious irrigation of antimetabolite from surgical field
- 6. Paracentesis
- 7. Sclerectomy
- 8. Consider peripheral iridectomy
- 9. Flap closure: adequate to guard against postoperative hypotony
 - a. Permanent sutures
 - b. Releasable sutures
- 10. Check for fluid flow through fistula by reforming AC with BSS via paracentesis
- 11. Possible excision of Tenon's capsule
- 12. Closure of Tenon's capsule and conjunctiva

IV. List the complications of this procedure, their prevention and management

A. Intraoperative / early perioperative

- 1. Conjunctival tear / buttonhole
- 2. Scleral flap complications
- 3. Intraocular hemorrhage
- 4. Vitreous presentation

B. Early postoperative

- 1. Infection
- 2. Hypotony
- 3. Flat anterior chamber
- 4. Aqueous misdirection
- 5. Hyphema
- 6. Transient IOP elevation
- 7. Choroidal effusion
- 8. Suprachoroidal hemorrhage
- 9. Loss of vision

C. Late postoperative

- 1. Leakage, failure, or migration of filtering bleb

2. Cataract
3. Persistent uveitis
4. Blebitis
5. Endophthalmitis
6. Symptomatic bleb/dellen formation
7. Hypotony
8. Aggravation of dry eye
9. Cystoid macular edema
10. Hypotony maculopathy
11. Induced astigmatism
12. Peripheral anterior synechiae

D. Prevention and management of complications

1. Meticulous surgical technique
2. Use minimum needed dosage and duration of anti-fibrotic agents intraoperatively
3. Close postoperative follow-up care

V. Describe the follow-up care

A. Postoperative follow-up

1. Monitoring of IOP and bleb
2. Evaluation of anterior chamber depth
3. Evaluation for possible choroidal detachment
4. Follow-up of other eye's glaucoma status

B. Postoperative regimen

1. Topical corticosteroids
2. Topical antibiotics
3. Eye shield at bed time
4. Topical cycloplegic agents
5. Possible sub-Tenon's or systemic corticosteroids

C. Postoperative IOP elevation

1. Bleb massage by the patient, or by the physician at the slit lamp.
2. Pull releasable suture
3. Laser suture lysis
4. Modulation of healing response
 - a. Discontinue topical corticosteroids
 - b. Aqueous suppressant to decrease flow through bleb leak
 - c. Subconjunctival injections of 5-fluorouracil
5. Needling of a failing or encapsulated bleb with subconjunctival injection of antimetabolites

D. Postoperative hypotony

1. Intracameral injection of ophthalmic viscosurgical device (OVD) (viscoelastic)
2. Bleb compression (e.g. pressure patch, bandage contact lens, etc.)
3. Autologous blood injection

4. Transconjunctival scleral flap sutures
5. Compression suture
6. Surgical bleb revision

VI. Describe appropriate patient instructions

- A. A written instruction sheet detailing what to look for and do at first signs of an infection and what activities to avoid**
- B. Need for frequent postoperative visits**
- C. Adherence to postoperative regimen**
- D. Careful attention to ocular status (e.g. pain, vision, redness, etc.)**
- E. In postoperative period, avoid heavy physical activity**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. AAO, Glaucoma Surgery: 1998, p.19-63.
3. AGIS Investigators. The Advanced Glaucoma Intervention Study (AGIS): 11. Risk factors for failure of trabeculectomy and argon laser trabeculoplasty. Am J Ophthalmol. 2002;134:481-98.

Combined cataract and filtration surgery

I. List the indications

A. Indications

1. Uncontrolled glaucoma in the setting of visual compromise due to cataract
2. Cataract requiring extraction in a patient with advanced visual field loss where a transient increase in postoperative intraocular pressure (IOP) could cause further damage
3. Cataract requiring extraction in a glaucoma patient requiring multiple medications to control IOP or in whom medical therapy is poorly tolerated

II. Describe the pre-procedure evaluation

A. Preoperative examination

1. Visual acuity, refraction, pin hole vision, glare testing and possibly Potential Acuity Meter to determine extent of visual impairment from cataract
2. Slit-lamp biomicroscopic examination
 - a. To evaluate severity of cataract and to look for evidence of conditions that might lead to complications, i.e. exfoliation, phacodonesis, other signs of trauma
 - b. To evaluate conditions that might make glaucoma surgery more likely to fail, i.e., uveitis, neovascularization, signs of previous surgery or scarred conjunctiva
3. IOP
4. Dilated fundus examination
5. Visual field examination
6. Gonioscopy to evaluate angle for neovascularization and presence of peripheral anterior synechiae

B. Preoperative history

1. Determine severity of visual symptoms from cataract
2. Determine how well patient is tolerating glaucoma medications
3. Investigate patient's general health to determine how well they will be able to tolerate a longer combined surgery versus two separate procedures
4. Consider discontinuing certain medications preoperatively
 - a. Parasympathomimetic agents
 - b. Agents that inhibit clot formation, in consultation with the patient's primary care physician

III. List the alternatives to this procedure

- A. Separate cataract and glaucoma surgery
- B. Combined cataract and nonpenetrating procedures
- C. Combined cataract and angle surgery
- D. Combined cataract and tube shunt procedures
- E. Combined cataract and endoscopic ciliary body photocoagulation
- F. Cataract surgery alone
- G. Laser trabeculoplasty

IV. Describe the instrumentation and technique

A. (See Incisional filtering surgery for open angle glaucoma)

1. Standard cataract extraction instrumentation (phacoemulsification preferred over extracapsular cataract extraction, when possible)
2. Clear corneal approach preferred for maintenance of conjunctival integrity

V. List the complications of this procedure, their prevention, and management

A. (See Incisional filtering surgery for open angle glaucoma)

VI. Describe the follow up care

- A. Increased inflammation may require additional anti-inflammatory medications and/or additional injections of antimetabolites**
- B. Laser suture lysis vs. releasable sutures, as needed**
- C. Reinstitution of glaucoma medications may be needed for high IOP**
- D. Important to monitor appearance of bleb for leaks or signs of bleb failure**

VII. Describe appropriate patient instructions (post-op care, vision rehabilitation)

- A. Avoid heavy lifting, bending or straining for several weeks**
- B. Stress compliance with medication regimen for optimal outcome**
- C. Stress compliance with frequent post-operative visits**
- D. Underscore that visual rehabilitation will be slower than with routine cataract surgery and that it will be longer before a stable refraction is reached**
- E. Review possible need for additional outpatient manipulations in the perioperative period such as laser suture lysis, releasable sutures, and repair of wound leaks**
- F. Give patient an instruction sheet on the care of a filtered eye and signs/symptoms of infection**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Jampel HD, Friedman DS, Lubomski LH, Kempen JH, Quigley H, Congdon N, Levkovitch-Vergin H, Robinson KA, Bass EB. Effect of technique on intraocular pressure after combined cataract and glaucoma surgery: An evidence-based review. *Ophthalmology* 2002;109:2215-24.
3. AAO, Glaucoma Surgery, 1998, pp. 67-83.
4. Verges C, Cazal J, Lavin C. Surgical strategies in patients with cataract and glaucoma. *Curr Opin Ophthalmol* 2005;16:44-52.
5. Friedman DS, Jampel HD, Lubowski LH et al. Surgical strategies for coexisting glaucoma and cataract: an evidence-based update. *Ophthalmology* 2002; 109 (10), 1902-13.
6. AAO. Focal Points: Update on Glaucoma Surgery, Module #6, 2012.
7. Friedman DS, Jampel HD, Lubomski LH, et al. Surgical strategies for coexisting glaucoma and cataract: an evidence-based update. *Ophthalmology*. 2002; 109: 1902-13.

Aqueous shunt surgery

I. List the indications/contraindications

A. Indications

1. Traditionally reserved for difficult glaucoma cases where conventional filtering surgery has failed or has a high likelihood to fail
2. Indications are broadening and may vary among surgeons
 - a. Previous failed incisional filtering surgery
 - b. Prior intraocular surgery, including cataract surgery
 - c. Significant superior conjunctival scarring
 - d. Neovascular glaucoma
 - e. Prior corneal transplant with glaucoma
 - f. Iridocorneal endothelial syndrome
 - g. Congenital glaucoma where anatomy precludes standard surgery
 - h. Trauma
 - i. Chronic uveitis with glaucoma
 - j. Patients who need to wear contact lenses

B. Relative contraindications

1. Anterior chamber placement in eyes with endothelial dysfunction or shallow anterior chamber
2. Intraocular tumors
3. Patients unable to comply with postoperative medications and follow-up

II. List the alternatives to the procedure

- A. Incisional filtering surgery
- B. Angle surgery
- C. Cyclodestruction

III. List complications of the procedure, their prevention and management

A. Hypotony with flat anterior chamber

1. Risk reduced by using valved device or temporary occlusion of non-valved device
2. Reformation of anterior chamber with ophthalmic viscosurgical device (OVD) (viscoelastic) should be performed
3. Early: may be due to leakage around tube entry into anterior chamber; usually transient
4. Late: may require surgical revision

B. Tube-cornea touch

1. Surgeon may avoid by proper placement of the tube

C. Tube occlusion

1. Causes
 - a. Iris
 - b. Vitreous

- c. Blood or fibrin
- 2. May be cleared with Nd:YAG laser surgery or surgical intervention
- D. Tube or plate exposure or erosion**
 - 1. Best prevented with proper positioning and closure technique
 - 2. May require surgical revision or explantation
- E. Tube migration**
- F. Valve malfunction**
- G. Failure by excessive bleb fibrosis**
 - 1. May consider surgical revision or replacement
- H. Motility disturbance**
 - 1. May cause persistent diplopia
 - 2. Management options include observation, prism, muscle surgery, or removal of the shunt.

I. Infection of external wound or endophthalmitis

- 1. Rare; usually accompanied by exposure of tube or plate
- 2. Intensive antibiotic treatment, surgical revision of exposure site or explantation of hardware may be necessary

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
- 2. Minckler DS, Francis BA, Hodapp, et al. Aqueous shunts in glaucoma: A report by the American Academy of Ophthalmology. *Ophthalmology* 2008;115:1089-98.

Laser iridotomy for angle closure

I. List the indications/contraindications

A. Indications

1. Pupillary block
 - a. Any attack of acute angle-closure glaucoma
 - b. Documented history of primary angle-closure glaucoma (acute, subacute, intermittent, chronic)
 - c. Anatomically narrow angle (pupil block, plateau iris) determined to be at risk for angle-closure glaucoma
 - d. Phacomorphic glaucoma
 - e. Aphakic/pseudophakic pupil block
 - f. Silicone oil block (do inferior peripheral iridotomy (PI) since oil rises to the top of the angle)
 - g. Iris bombe

II. Describe the pre-procedure evaluation

- A. Question patient regarding symptoms of subacute / acute angle closure
- B. Slit-lamp biomicroscopic evaluation of anterior chamber depth and to check for iris and angle neovascularization
- C. Gonioscopy
- D. IOP

III. List alternatives to this procedure

- A. Surgical iridectomy
- B. Daily parasympathomimetic use to pharmacologically constrict pupil and pull iris away from angle
 1. There is some controversy to this treatment since miotic therapy actually has potential to anteriorly displace lens-iris diaphragm and increase pupillary block
- C. Cataract surgery if there is a phacomorphic component

IV. List the complications of this procedure, their prevention and management

A. Intraoperative / perioperative

1. Blurred vision
 - a. Usually transient; caused by methylcellulose, corneal surface irregularities, blood, pigment dispersion
2. Hemorrhage at iridotomy site
 - a. Occurs with neodymium yttrium-aluminum-garnet (Nd:YAG), can be minimized with argon laser pretreatment to coagulate blood vessels
 - b. May interfere with visibility during treatment
 - i. Compress eye with iridotomy lens to raise intraocular pressure (IOP) temporarily and provide tamponade to slow a steady stream of blood
 - c. Usually self-limited; occasionally results in small hyphema. Often worse in patients on anticoagulants

3. Pigment dispersion
 - a. Occurs to some extent in every patient
 - b. In thick, heavily pigmented iris, may interfere with visibility during procedure
4. Corneal injury
 - a. Causes transient epithelial / stromal whitening (argon) or focal stromal disruption (Nd:YAG)
 - i. Can interfere with delivery of laser energy
 - ii. Resolves in days-weeks
 - b. More common if anterior chamber very shallow
5. Corneal epithelial irregularity / abrasion
 - a. Can be minimized with careful application and removal of iridotomy lens, and post-treatment lubrication with artificial tears
6. IOP spike
 - a. Usually occurs within 1 hour of treatment
 - b. Minimized by perioperative use of alpha agonist or other topical glaucoma medications

B. Post-operative

1. Blurred vision due to corneal injury, blood, pigment dispersion
 - a. Usually resolves within 24-48 hours
2. Inflammation
 - a. Due to breakdown of blood-aqueous barrier
 - b. Alleviated by postoperative use of topical corticosteroids and/or non-steroidal anti-inflammatory drugs (NSAID)
 - c. Usually resolves within one to three weeks
3. Glare / visual disturbance
 - a. More common with large opening that is adjacent to the lid margin and may result from prismatic effect
 - b. Symptoms often lessen with time
4. Focal lens opacity
 - a. More common with high energy levels and iridotomy not peripheral enough
 - b. Due to focal disruption of lens capsule
 - c. Non-progressive
5. Closure of iridotomy
 - a. Can occur within days/weeks due to blood and pigment dispersion and require touchup once eye is quiet
 - b. Closure 6-12 weeks post treatment due to proliferation of pigment epithelium; more common after argon peripheral iridotomy (PI) and occurs in up to 1/3 of patients
 - c. Closure common in neovascular glaucoma and uveitis due to inflammatory membranes or pigment dispersion. Frequent touchup may be required

V. Describe the follow-up care

- A. Topical corticosteroids/NSAIDs for at least 4 days post-procedure**
- B. Intraocular pressure check 30 to 90 minutes after treatment**
- C. Follow-up visit 1-3 weeks after treatment**
 1. Repeat gonioscopy post-treatment to confirm less narrowing of angle

2. Confirm patency of iridotomy
3. Evaluate anterior chamber reaction
4. Dilated retinal exam, if not done previously due to narrow angle

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Ritch R, Lieberman J. Laser iridotomy and peripheral iridoplasty. In: Ritch R, Shields MB, Krupin T, eds. The Glaucomas, 2nd ed. St. Louis, MO; 1996: 1549-1573..
3. AAO, Glaucoma Surgery: 1998, p.168-173.
4. Fleck BW, Wright E, Fairley EA. A randomized prospective comparison of operative peripheral iridectomy and Nd:YAG laser iridotomy treatment of acute angle closure glaucoma: 3 year visual acuity and intraocular pressure control outcome. Br. J Ophthalmol 1997 81:884-8.
5. Ritch R, Shields MB, Krupin T, eds. The Glaucomas. 2nd ed. St Louis: Mosby; 1996.
6. Shields MB. Textbook of Glaucoma. 4th ed. Baltimore: Williams & Wilkins; 1997:490-496.

Ocular hypotony

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Ocular trauma
 - a. Ruptured globe
 - b. Cyclodialysis cleft
2. Postsurgical
 - a. Wound leak after intraocular surgery
 - b. Overfiltration after glaucoma surgery
3. Aqueous underproduction
 - a. Chronic inflammation
 - b. Ciliary body destruction
 - c. Overuse of pressure lowering meds
4. Rhegmatogenous retinal detachment
5. Ocular ischemia

B. List the pertinent elements of the history

1. Ocular trauma
2. Ocular surgery
3. Pain, variable/blurred vision, conjunctival injection and photophobia

C. Describe the pertinent clinical features

1. General
 - a. Blurred vision
 - b. Low intraocular pressure (IOP)
 - c. Corneal folds
 - d. Flat or Shallow anterior chamber
 - e. Retinal or chorioretinal folds in the macula
 - f. Cystoid macular edema (CME)
 - g. Choroidal detachments/hemorrhage

D. Describe the appropriate testing and evaluation for establishing the diagnosis

1. Ocular ultrasound to evaluate choroidal detachments and retinal status
2. Computed tomography scan (CT scan) in cases of ocular trauma
3. Optical coherence tomography (OCT) to detect macular folds and CME

II. List the differential diagnosis

- A. Exudative retinal detachment
- B. Choroidal tumor (mimic choroidal effusion)
- C. Papilledema
- D. Epiretinal membrane

- E. Pathologically thin central corneal thickness (measuring artifact)

III. Describe the patient management in terms of treatment and follow-up

A. Describe the medical therapy options

1. Topical corticosteroids
2. Topical cycloplegia
3. Discontinue anti-glaucoma medications

B. Describe the surgical therapy options

1. Ocular trauma
 - a. Surgical repair of injury
2. Leak from surgical wound after intraocular surgery
 - a. Revise wound for watertight closure
3. After glaucoma filtering procedure (trabeculectomy and tube shunt)
 - a. Consider referral to glaucoma specialist
4. Cyclodialysis cleft
 - a. If topical cycloplegia fails, consider argon laser to cleft or referral to specialist for surgical repair
5. Flat chamber
 - a. Consider reforming the anterior chamber with OVD
 - b. If not able to fill with OVD, consider referral to specialist
6. Drain choroidals
 - a. Consider referral to a retina specialist
7. Persistent macular folds after hypotony
 - a. Consider referral to retina specialist

IV. Describe the complications of treatment

- A. Infection
- B. Uncontrolled IOP
- C. Glaucoma progression

V. Describe the disease-related complications of hypotony

- A. Hypotony maculopathy
- B. Blurred vision
- C. Pain, dellen formation from large bleb

VI. Describe appropriate patient instructions and management

- A. No heavy lifting
- B. No bending over
- C. No straining or Valsalva maneuver
- D. Ocular protection (protective eye wear and shield at bed time)
- E. Warn of signs of choroidal hemorrhage and infections

F. Discuss possibility for referral to specialist for management and surgical intervention

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 10: Glaucoma, 2015-2016.
2. Fineman MS, Katz LJ, Wilson RP. Topical dorzolamide-induced hypotony and ciliochoroidal detachment in patients with previous filtration surgery. *Arch Ophthalmol* 1996;114(8):1031-2.
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19. Bardak Y, Cuypers MH, Tilanus MA, Eggink CA. Ocular hypotony after laser suture lysis following trabeculectomy with mitomycin C. *Int Ophthalmol* 1997;21(6):325-30.
20. Wilson RP, Moster MR. The chamber-retaining suture revisited. *Ophthalmic Surg* 1990;21(9):625-7.
21. Garcia-Feijoo J, Cuina-Sardina R, Mendez-Fernandez C, et al. Peritubular filtration as cause of severe hypotony after Ahmed valve implantation for glaucoma. *Am J Ophthalmol* 2001;132(4):571-2.
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Essential anisocoria

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Benign condition characterized by difference in pupil size as normal variant in population (20 %)

B. List the pertinent elements of the history

1. Noted by patient, friend, family member, health care provider
2. Painless
3. No associated neurologic or ophthalmic symptoms
4. Size of pupil may vary

C. Describe pertinent clinical features

1. Relative difference of anisocoria remains constant in all levels of illumination and both pupils react well to light, dilate in the dark and react well to near
2. Absence of other neurologic/eye signs or symptoms
 - a. Normal eyelid examination
 - b. Normal motility examination

D. Describe appropriate laboratory testing for establishing the diagnosis

1. No laboratory testing is indicated

II. List the differential diagnosis

- A. Adie pupil
- B. Horner pupil
- C. Cranial nerve (CN) III palsy
- D. Pharmacologic mydriasis/miosis
- E. Benign episodic pupillary mydriasis (often associated with a history of migraine headache)

III. Describe patient management in terms of medical treatment and follow-up

- A. Complete eye examination with measurement of pupillary reactions
- B. Review of old photographs may be helpful

IV. Describe appropriate patient instructions

- A. Reassurance
- B. Report any new symptoms - diplopia, ptosis

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 5: Neuro-Ophthalmology, 2015-2016.
2. Slamovits TL, Glaser JS, Mbekeani JN. The Pupils and Accommodation. In: Tasman W, Jaeger EA, eds. *Duane's Clinical Ophthalmology*. Philadelphia, PA: Lippincott Williams & Wilkins. Revised edition 2005. Vol 2, Chapter 15: 11-12.

Traumatic mydriasis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Blunt trauma to globe with rupture of pupil sphincter muscle or damage to long ciliary nerves
2. Surgical trauma to iris

B. List the pertinent elements of the history

1. History of blunt or surgical trauma

C. Describe pertinent clinical features which may be apparent

1. Diminished pupillary reaction
2. Irregular pupil
3. Dilated fixed pupil
4. Sphincter rupture, multiple small tears, laceration
5. Iris tear
6. Iridodialysis

II. Define the risk factors

A. Trauma

B. Surgery

III. List the differential diagnosis

A. Cranial Nerve (CN) III palsy

B. Physiologic anisocoria

C. Adie pupil

D. Pharmacologic mydriasis

E. Ectropion uvea

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Prosthetic contact lens
2. Trial of pilocarpine or brimonidine
3. Observe

B. Describe surgical therapy options

1. Pupilloplasty (suture reduction of pupil diameter)
2. Surgical iris implants (not FDA-approved)

V. List the complications of treatment, their prevention and management

A. Contact lens associated problems

- B. Pupilloplasty - includes all the complications associated with intraocular surgery, including infection and bleeding**

VI. Describe disease-related complications

- A. Persistent glare, photophobia**
- B. Impaired cosmesis**
- C. Side effects of pilocarpine or brimonidine**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 8: External Disease and Cornea, 2015-2016.

Adie pupil

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Parasympathetic dysfunction causing diminished pupillary reaction

B. List the pertinent elements of the history

1. Anisocoria with unilateral larger pupil with patient occasionally complaining of light sensitivity
2. Loss of accommodation with decreased near vision
3. No associated diplopia or ptosis

C. Define the relevant elements of the epidemiology of this disease

1. Almost all cases are idiopathic

D. Describe pertinent clinical features

1. Large pupil
2. Anisocoria is greatest in light
3. Light-near dissociation
4. On refixation from near to distance, pupil redilation is slow (tonic)
5. Sectoral palsy of the pupillary sphincter
 - a. Vermiform movements at slit lamp
 - b. Atrophy of pupillary ruff with time
6. In Adie syndrome deep tendon reflexes are decreased
7. Decreased near vision due to impaired accommodation
8. Over time, mydriasis decreases but reactivity does not recover

E. Describe appropriate laboratory testing for establishing the diagnosis

1. Dilute pilocarpine testing
 - a. Denervation supersensitivity -- dilute pilocarpine constricts pupil
 - b. Test will be negative before supersensitivity develops

II. Define the risk factors

- A. Recent history of orbit trauma/surgery
- B. Panretinal photocoagulation
- C. Female predominance

III. List the differential diagnosis

- A. Essential anisocoria
- B. Horner syndrome (contralateral eye)
- C. Pharmacological mydriasis/miosis
- D. Diabetes
 1. Autonomic pupillary dysfunction
 2. Previous panretinal photocoagulation

- E. Cranial nerve (CN) III palsy
- F. Previous trauma

IV. Describe patient management

- A. Describe medical therapy options
 - 1. Pilocarpine for miosis if light sensitive
 - 2. Spectacle correction for reading add for near vision

V. Describe appropriate patient instructions

- A. Education and reassurance
- B. Report any double vision or ptosis
- C. Tonic pupil may become smaller over months to years
- D. Second eye may develop findings

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 5: Neuro-Ophthalmology, 2015-2016.

Functional visual loss

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Visual complaints that have no physiologic or organic basis are due to:
 - a. Malingering, or willful exaggeration of symptoms, sometimes when litigation involving monetary compensation or disability are involved or other personal gain is sought
 - b. Hysteria, or a subconscious expression of nonorganic signs or symptoms (conversion reaction)
 - i. True Hysteria/conversion disorder is extremely rare

B. List the pertinent elements of the history

1. Monocular diplopia
2. Unilateral or bilateral decreased vision
3. Unilateral or bilateral visual field loss
4. Onset in relationship to antecedent trauma
5. Pertinent medical history
 - a. Psychosocial stress
 - i. Family/peer relationships
 - ii. Academic or social pressures
 - b. Secondary gain from litigation
 - i. Car accident
 - ii. Slip and fall
 - iii. Work-related disability
 - iv. Other personal injury

C. Describe pertinent clinical features

1. Monocular diplopia (may be bilateral)
2. Unilateral or bilateral decreased vision
3. Nonphysiologic patterns of visual field loss
4. Pupil findings
 - a. Normal reactivity in the absence of associated ocular trauma with traumatic mydriasis or other iris pathology
 - b. Absence of an afferent pupillary defect (APD) in spite of gross asymmetry of visual fields
5. May have component of functional loss superimposed upon organic visual loss related to trauma

D. Describe appropriate testing and evaluation for establishing the diagnosis

1. Complete ophthalmologic examination including visual fields
2. Appropriate concern when clinical findings do not match degree of vision complaints
3. Evaluation of monocular diplopia
 - a. Sensorimotor exam to confirm normal ocular alignment in all fields of gaze and verify monocular nature of diplopia
 - b. Rule out contributory corneal, refractive, lenticular, and retinal pathology
4. Evaluation of monocular decreased visual acuity
5. Evaluation of non-physiologic visual field loss

- a. Observe patient performing tasks outside of stated visual field (e.g. ambulate throughout office without difficulty, shake hand beyond stated visual field, locate objects outside of stated visual field such as trashcan to throw away tissues)
- b. Have patient look at your fingers in eccentric quadrants of field when "testing motility" after they've previously denied ability to count fingers in this region on confrontation field
- c. Absence of an APD in face of grossly asymmetric visual fields
- d. Normal appearing optic nerve in setting of longstanding monocular visual loss

II. Define the risk factors

- A. Personal injury/worker's compensation litigation
- B. Impending disability determination
- C. Psychosocial stress
- D. Dissatisfaction with recent medical care

III. List the most common or critical entities in the differential diagnosis

- A. Organic visual loss or superimposed organic component with non-physiologic embellishment
- B. Cancer-associated retinopathy or optic neuropathy
- C. Leber hereditary optic neuropathy

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 5: Neuro-Ophthalmology, 2015-2016.

Cutaneous pigmented lesions

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Nevi are benign, melanocytic lesions of the skin, commonly occurring on the eyelids
2. Malignant melanomas are neoplastic transformation of normal skin melanocytes into a malignant cell line

B. Define the relevant aspects of epidemiology of this disease

1. Nevi
 - a. May be congenital or acquired
2. Malignant melanomas
 - a. Decreased skin pigmentation is a risk factor
 - b. Living in areas with high ultraviolet (UV) exposure

C. List the pertinent elements of the history

1. Pigmented lesion present since birth or acquired
 - a. Nevi may be present since birth or acquired
 - b. Melanomas may occur de novo or at the site of a pre-existing lentigo maligna or nevus
 - c. Change in the appearance of the lesion
 - d. Pigment density, distribution or color
 - e. Increase in size
 - f. New nodules in a previously flat lesion
 - g. Surface bleeding
2. Unprotected extensive sun exposure
3. Family history of melanoma

D. Describe pertinent clinical features

1. Congenital nevus
 - a. Usually larger than acquired nevi and may be of substantial size
 - b. Usually deeply pigmented
 - c. Border often irregular
 - d. Frequently covered with hair
 - e. A chance of malignant degeneration exists proportional to the size of the lesion
2. Acquired nevi vary in size and degree of pigmentation
3. Malignant melanoma
 - a. Variation in pigmentation density and/or color
 - b. Irregular, vague border with pigmentary infiltration into "normal" skin
 - c. Elevated pigmented nodule in a flat pigmented area

E. Describe appropriate laboratory testing for establishing the diagnosis

1. Usually diagnosed based on clinical appearance
2. Biopsy is performed for any suspicious lesion to confirm diagnosis and rule out malignancy

F. Preoperative metastatic work-up for melanomas greater than 1.5 mm thick

1. Systemic evaluation for liver, lung and brain metastatic disease
2. Increased incidence of metastatic disease with increased melanoma thickness or deeper cutaneous invasion

II. Define the risk factors

- A. Sun exposure in early childhood
- B. Decreased skin pigmentation
- C. Genetic predisposition
- D. Environmental mutagens

III. List the differential diagnosis

- A. Nevus
- B. Malignant melanoma
- C. Freckle
- D. Seborrheic keratosis
- E. Squamous papilloma
- F. Lentigo maligna
- G. Lentigo senilis (multiple small 3-5 mm uniformly pigmented lesions)
- H. Pigmented basal cell carcinoma

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Nevi
 - a. Observation
 - b. Recommend sunscreen use for prevention
 - c. Congenital nevi require close monitoring
 - d. Photography to document lesion appearance
2. Malignant melanoma
 - a. Immunotherapy

B. Describe surgical therapy options

1. Biopsy any suspicious lesion (those with irregular border, growth, bleeding, ulceration, variable pigmentation)
2. Surgical excision may also be performed for cosmesis or mechanical irritation
3. Wide surgical removal with clear margins in the case of malignant melanoma

V. List the complications of treatment, their prevention and management

- A. Scarring with eyelid and/or facial dysfunction
- B. Recurrence, local or metastatic

VI. Describe disease-related complications

A. Nevi

1. Development of malignant melanoma
 - a. More common in congenital nevi
 - b. Risk is directly related to size of congenital nevi
 - c. Close serial follow-up or excision warranted for congenital nevi

B. Malignant melanomas

1. Direct tumor extension into orbit with functional compromise (vision, diplopia, pain)
2. Direct tumor extension in adjacent facial tissues (lacrimal drainage, perineural)
3. Regional or distant metastasis

VII. Describe appropriate patient instructions

- A. Prompt evaluation of any lesion that has a change in character including growth, bleeding, ulceration, uneven pigmentation or irregular borders**
- B. Avoid unprotected sun exposure (hat, topical sun block)**
- C. Periodic examination of all sun exposed skin areas**
- D. For patients with malignant melanoma, continued systemic evaluation with interdisciplinary team**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 7: Orbit, Eyelids and Lacrimal System; Section 4: Ophthalmic Pathology and Intraocular Tumors; Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. AAO, Surgery of the Eyelid, Orbit & Lacrimal system, Vol. 2, 2011.
3. Boulos PR, Rubin PA. Cutaneous melanomas of the eyelid. *Semin Ophthalmol* 2006;21:195-206.

Eyelid trauma

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Traumatic injury to eyelids, midface, orbit, or eye
2. Source of injury may be any event that results in the transfer of kinetic or thermal energy into soft tissue or bone

B. List the pertinent elements of the history

1. Usually associated with blunt trauma to the face
2. Neurologic status
 - a. Inquire regarding associated loss of consciousness, diplopia or other neurologic complaints
3. Age of patient
 - a. Witnesses should be questioned in pediatric cases
4. Visual loss
5. Contact lens wear
6. Foreign bodies
7. Bites
8. Thermal or chemical burns
9. Tetanus immunization status

C. Describe pertinent clinical features

1. Treatment of concurrent, life threatening injuries is given priority
2. Hemorrhagic eyelid edema
3. Possible occult penetrating eyelid, lacrimal, ocular, or orbital injury
 - a. Note if there is any laceration that extends through the orbital septum
4. Possible ruptured globe or other intraocular injury - hyphema, subluxed lens, cataract, retinal detachment, commotio retinae
5. Possible facial, nasal or orbital fractures
6. Possible traumatic optic neuropathy with loss of vision
 - a. Note if there is a relative afferent pupillary defect
7. Possible retained orbital foreign bodies
8. Possible orbital emphysema

D. Describe appropriate testing for establishing the diagnosis

1. Pupillary examination
2. Axial and coronal computed tomography scan to evaluate possible facial and orbital fractures or hemorrhage, head or spinal injury, if clinically indicated
3. Magnetic resonance imaging is generally not needed. It may be helpful in unusual cases, such as suspected non-metallic foreign body

II. Define the risk factors

A. Participation in high risk activities

B. Can occur at anytime, anywhere

III. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Observation only
2. Medical management of ocular injuries
3. Possible high dose corticosteroid treatment of optic neuropathy
4. Ice packs, head of bed elevation
5. Possible oral antibiotics and avoidance of nose blowing if fractures are present
6. Avoid aspirin intake in the event surgery is needed

B. Consider referral for surgical therapy

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 7: Orbit, Eyelids, and Lacrimal System, 2015-2016.
2. AAO, Surgery of the Eyelid, Orbit & Lacrimal system, Vol. 2, 2011.

Dermatochalasis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Laxity (chalasis) of the skin of the eyelids
2. Most commonly involutional

B. Define the relevant aspects of epidemiology of the disease

1. Usually age-related
2. Stretching can be exacerbated by conditions causing swelling and fluid retention

C. List the pertinent elements of the history

1. The patient may complain of a full appearance of the eyelids
2. Forehead straining may be caused by lifting of the excess skin by active frontalis muscle flexion to counteract ptosis or pseudoptosis
3. Reduced visual functioning and constricted visual fields may be present
4. History of eyelid swelling and lid retraction should alert the surgeon to the possibility of thyroid eye disease (Graves disease)
5. Irritation may be present at skin folds

D. Describe the pertinent clinical features

1. Fullness in the upper or lower eyelids
2. Draping of the upper eyelid skin down to or over the eyelashes
3. Associated forehead/eyebrow ptosis
4. Contraction of the frontalis muscle to raise the eyebrows and redundant eyelid skin
5. Herniation of the eyelid fat may be associated
6. Ptosis of the upper eyelid often is associated
7. Horizontal laxity of the lower eyelids may also be present

E. Describe appropriate laboratory testing for establishing the diagnosis

1. Visual fields are performed to measure superior field loss
2. Ptosis evaluation if appropriate

II. Define the risk factors

- A. Age
- B. Allergy
- C. Fluid retention

III. List the differential diagnosis

- A. Acute, reversible edema
- B. Chronic swelling associated with systemic disease (e.g., thyroid, renal failure, allergy)
- C. Blepharochalasis syndrome

IV. Describe patient management in terms of treatment and follow-up

- A. Treat the reversible causes of edema
- B. Surgical reduction of the excess eyelid skin (blepharoplasty)
- C. Forehead/eyebrow lifting procedures

V. List the complications of treatment, their prevention and management

- A. Inadequate or excess skin/fat removal

VI. Describe appropriate patient instructions

- A. Do not rub the eyelids, as this will inflame and further stretch the skin
- B. Surgery is indicated if symptomatic

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 7: Orbit, Eyelids, and Lacrimal System, 2015-2016.
2. AAO, Surgical Anatomy of the Ocular Adnexa, Vol 2. 2012.

Upper eyelid blepharoplasty

I. List the indications/contraindications

A. Indications

1. Excess skin in the upper eyelids that causes an anatomical, cosmetic or visual problem for the patient.
 - a. Vision including superior and lateral visual fields may be compromised
 - b. Skin resting on the eyelashes may annoy the patient and push the eyelashes downward
 - c. Chronic blepharitis may be present in the area beneath the skin fold
 - d. Chronic frontal headache may result from constant elevation of the eyebrows in an effort to pull the redundant skin fold away from the eyelashes
 - e. Often associated with concomitant brow ptosis
2. Cosmetic concerns

B. Contraindications

1. Normal skin volume present, with the apparent excess caused by forehead ptosis
2. Severe dry eye conditions
3. When the skin will be needed for a grafting source in the future, as in a patient with recurrent periocular neoplasm

II. Describe the pre-procedure evaluation

A. Patient history

1. Effect on driving, reading and other close activities
2. Frontal headache
3. Dry eye problems
4. Ocular irritation
5. Aesthetic concerns

B. Clinical examination

1. Identify the upper lid skin fold inferior extension with the forehead in habitual and relaxed positions
 - a. Skin resting on the lashes or below indicates a functional indication for the procedure
2. Measure
 - a. Upper eyelid position and levator function
 - i. Rule out or treat coexisting blepharoptosis
 - b. Tear meniscus
 - c. Lid closure/blink status (lagophthalmos)
 - d. Measure tear production

C. Preoperative assessment

1. Documentation with photographs, visual fields with and without tape elevating the redundant skin away from the lash margin
2. Evaluation of lacrimal function
3. Prior lid surgeries

III. List the alternatives to the procedure

- A. Forehead lifting, brow plasty**

IV. Describe the technique

- A. Mark the upper eyelid crease**
- B. Determine the amount of upper eyelid skin to be removed**
- C. Excise skin with or without the underlying orbicularis muscle**
- D. Open or tighten the orbicularis septum to address prolapsing pre-aponeurotic fat pads**
 - 1. Address lacrimal gland prolapse with fixation to orbital rim periosteum

V. List the complications of the procedure, their prevention, and management-

- A. Inadequate skin removal**
 - 1. May require more surgery
- B. Excess skin removal preventing closure of the eyes**
 - 1. Careful measurement with the brow in neutral position prevents this
 - 2. Lubrication of the eyes and watchful following of the patient will support the patient while orbicularis muscle function returns
 - 3. In extreme cases, skin grafting may be required
- C. Wound separation may occur in the first week postoperatively**
 - 1. Prevention involves proper choice and placement of sutures and avoidance of wound tension
- D. Asymmetric eyelid creases**
 - 1. Meticulous marking is critical to avoid this complication
 - 2. Crease reformation sutures may be useful in creating symmetric creases
- E. Dry eye symptoms**
 - 1. Lubrication
 - 2. Bandage contact lenses
 - 3. Severe dry eye patients should undergo judicious, if any, skin removal
- F. Allergy to the antibiotic ointment**
 - 1. Redness, swelling and itching are present
 - 2. Differentiation from infective cellulitis and wound separation is essential
 - 3. Cessation results in improvement within a few days. May respond more quickly with topical and or oral steroids
- G. Infection**
 - 1. Rare, as the blood supply to the eyelid is excellent
 - 2. Sterile technique and treatment with antibiotic ointment are standard protocol
 - 3. Broad spectrum oral antibiotics if cellulitis is present
- H. Hematoma**
 - 1. Rare, and can be vision threatening
 - 2. Patient noting a sudden increase in pain should inform surgeon immediately
 - 3. Prevention is by careful intraoperative hemostasis
 - 4. If possible, prescription and over-the-counter medications which prolong bleeding time should be stopped

preoperatively for appropriate amount of time depending on medication

VI. Describe follow-up care

- A. Follow up is usually done at 1-2 weeks postoperatively
- B. Follow up is complete when the patient is satisfied and when the lid and cornea are stable

VII. Describe appropriate patient instructions

- A. Cold compresses are used for one or two days postoperatively
- B. Follow-up immediately for sudden pain, swelling and decrease in vision
- C. Antibiotic ointment is used on the incision until it epithelializes or until absorbable sutures are no longer present
- D. Frequent and adequate lubrication of the eyes is stressed in postoperative care

Additional Resources

- 1. AAO, Basic and Clinical Sciences Course. Section 7: Orbit, Eyelids, and Lacrimal System, 2015-2016.
- 2. AAO, Surgery of the Eyelid, Orbit & Lacrimal system, Vol. 2, 2011.
- 3. AAO, Ophthalmic Technology Assessment: Functional Indications for Upper and Lower Eyelid Blepharoplasty. Ophthalmology 2011.
- 4. Bosniak S. Reconstructive upper lid blepharoplasty. Ophthalmol Clin North Am 2005;18:279-89,vi.
- 5. Purewal BK, Bosniak S. Theories of upper eyelid blepharoplasty. Ophthalmol Clin North Am 2005;18:271-8,vi.

Medical management of acute dacryocystitis

I. List the indications for medical management

A. Acute dacryocystitis

1. Symptoms: medial canthal pain, tearing, discharge
2. Signs: medial canthal erythema, edema, lacrimal sac distension +/- reflux, conjunctivitis, elevated tear meniscus, preseptal/orbital cellulitis

II. Describe the diagnostic evaluation

- A. External exam - lacrimal sac mucocoele, discharge with pressure on sac, punctal eversion, erythema
- B. Slit-lamp biomicroscope exam - enlarged tear lake, debris in tear film
- C. Avoid canalicular probing/irrigation in setting of acute infection; avoid nasolacrimal duct probing in adult patients
- D. Consider culture to guide antibiotic therapy
- E. Computed tomography if associated with orbital cellulitis/abscess, evidence of globe displacement, or associated with ocular motility impairment

III. Describe patient management in terms of treatment and follow-up

- A. Warm compresses
- B. Topical antibiotics
- C. Systemic antibiotics
 1. Oral antibiotics
 2. Parenteral antibiotics in patients with associated preseptal/orbital cellulitis or with infection unresponsive to oral antibiotics, particular concern in pediatric patients
- D. Possible surgical referral for dacryocystorhinostomy (DCR) in adult patients

IV. List the complications of the procedure/therapy, their prevention and management

A. Complications

1. Recurrent/persistent dacryocystitis
 - a. Consider methicillin resistant *Staphylococcus aureus* (MRSA) and atypical mycobacteria
2. Progression to orbital cellulitis/abscess
3. Cutaneous fistula formation

B. Management of complications

1. Alternate choice of antibiotic treatment
2. Alternate route of antibiotic administration
3. Surgical management

V. Describe appropriate patient instructions

- A. Warm compresses**
- B. Antibiotics if indicated**

Additional Resources

1. AAO, Basic and Clinical Sciences Course. Section 7: Orbit, Eyelids, and Lacrimal System, 2015-2016.

Medical treatment of amblyopia: refractive correction

I. List the indications/contraindications

A. Indications

1. Presence of poor vision or amblyogenic refractive error
2. Poor fixation

B. Relative contraindications

1. Anatomical abnormality that precludes improvement in vision

II. Describe the pre-therapy evaluation

A. Full exam including

1. Age-appropriate visual acuity, each eye
2. Strabismus evaluation
3. Evaluation for structural abnormalities of the eye or visual pathways
4. Determination of cycloplegic refractive error

III. Describe the instrumentation and technique

A. Dispense the appropriate prescription for eyeglasses with polycarbonate lenses

IV. List the complications of this therapy, their prevention and management

A. Poor compliance

1. Counsel families on the importance of treatment
2. Allow adequate time for education to allow family engagement and treatment success

B. Unresponsiveness

1. Recheck refraction
2. Look for abnormalities of the macula or optic nerve
3. Institute additional amblyopia therapy such as occlusion or atropine to the sound eye
4. Can terminate primary therapy after an appropriate length of time if there is no response in a compliant patient

C. Recurrence

1. Reinitiate treatment or continue maintenance regimen

D. Development of amblyopia in the originally better eye

1. Recheck refraction
2. Initiate appropriate amblyopia treatment

V. Describe the follow-up care

A. Appropriate follow-up care based on age and severity of amblyopia

VI. Describe appropriate patient instructions

- A. Encourage compliance with eyeglasses and explain the importance of polycarbonate lenses**
- B. Stress importance of follow-up as instructed**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. Clarke MP, Wright CM, Hrisos S, et al. Randomised controlled trial of treatment of unilateral visual impairment detected at preschool vision screening, BMJ, 2003, 327: 1251.
3. Atkinson J, Braddick O, Robier B, et al. Two infant vision screening programmes: prediction and prevention of strabismus and amblyopia from photo and videorefractive screening. Eye, 1996, 10: 188-89.
4. AAO, Preferred Practice Pattern Committee, Pediatric Ophthalmology Panel. Amblyopia Preferred Practice Pattern, 2012.

Medical treatment of amblyopia: occlusion and optical degradation

I. List the indications

- A. Indication: reduced visual acuity secondary to amblyopia, in appropriate age range

II. Describe the pre-therapy evaluation

- A. Evaluation of visual acuity
- B. Evaluation for strabismus
- C. Complete eye exam to rule out structural causes of visual loss
- D. Determination of cycloplegic refractive error

III. Describe the instrumentation and technique

- A. Dispense the appropriate prescription for eyeglasses
- B. Patching
 - 1. Place adhesive patch on skin or occlusion of glasses over non-amblyopic eye for prescribed time
 - 2. Titrate patching schedule based on patient response
- C. Optical penalization by atropine drops

IV. List the complications of therapy, their prevention and management

- A. Patching
 - 1. Occlusion may cause amblyopia in the occluded (better) eye
 - a. Monitor vision closely in the occluded eye
 - 2. Contact dermatitis
- B. Atropine
 - 1. Systemic absorption (fever, redness, hyperactivity)
 - 2. Photophobia
 - 3. Possible retinal/lens phototoxicity - use ultraviolet protection, hat, etc.
 - 4. Conjunctival reaction
 - 5. Iatrogenic amblyopia
- C. Treatment failures-generally due to non-compliance (see below) or late diagnosis
- D. Strabismus may either worsen or improve with therapy

V. Describe the follow-up care

- A. Follow-up periods to be determined by age of patient, treatment prescribed, severity of amblyopia, and response to treatment but generally require frequent visits
- B. When plateau reached, period of maintenance patching may be helpful to prevent recurrence
- C. Treatment generally terminated when no response in compliant patient after appropriate length of time

VI. Describe appropriate patient instructions

A. Patching

1. Encourage patching
2. Emollients for skin irritation

B. Atropine - compliance with drops. Watch for side effects

C. Wear prescribed spectacles

D. Non-compliance is a significant issue—parental education on importance of therapy, as well as suggestions for improving compliance (e.g., rewards, restraints, materials to cover patch, getting through the toughest first few weeks, etc.)

E. When patient has difficulty with spectacle use, care must be taken to re-evaluate refractive error to discover cause

F. Educate on possibility of recurrence and need for follow-up and possible reinstatement of treatment

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. Newsham D. A randomized controlled trial of written information: the effect of parental non-concordance with occlusion therapy. *Br J Ophthalmol.* 2002, 86: 787-91.
3. Leguire LE, Rogers GL, Walson PD, et al. Occlusion and levodopa-carbidopa treatment for childhood amblyopia. *J AAPOS.* 1998, 2: 257-64.
4. AAO, Preferred Practice Pattern Committee, Pediatric Ophthalmology Panel. Amblyopia Preferred Practice Pattern, 2012.

Infantile esotropia

I. Describe the approach to establishing the diagnosis

A. Define the relevant aspects of epidemiology of the disease

1. No gender predilection
2. Family history of strabismus and amblyopia

B. List the pertinent elements of the history

1. Present in first six months of life
2. Cross-fixation common
3. May demonstrate alternate fixation

C. Describe pertinent clinical features

1. Equal visual acuity common; amblyopia in a minority
2. Full abduction in each eye (may need doll's head maneuver)
3. Large angle of deviation (often greater than 30 prism diopters)
4. Absence of high hyperopia

II. Define the risk factors

- A. Family history of strabismus and decreased binocular function common**

III. List the most common or critical entities in the differential diagnosis

- A. Cranial Nerve (CN) VI palsy**
B. Pseudoesotropia
C. Accommodative esotropia

IV. Describe patient management in terms of treatment and follow-up

- A. Treatment of amblyopia if present**
B. Treatment with spectacles if significant hyperopia is present
C. Early surgical correction is almost always necessary

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. AAO, Preferred Practice Patterns Committee, Pediatric Ophthalmology Panel. Esotropia and Exotropia Preferred Practice Pattern, 2012.

Refractive accommodative esotropia

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Accommodation causes excess convergence
2. Driven by choice of clear vision over alignment
3. Esotropia leads to amblyopia via suppression of non-preferred eye

B. Define the relevant aspects of epidemiology of this disease

1. Usually occurs in early childhood
2. Positive family history

C. List the pertinent elements of the history

1. Orthophoric initially
2. Esotropia begins in early childhood
3. Adults may become symptomatic again near onset of presbyopia

D. Describe pertinent clinical features

1. Comitant esotropia
2. Two types: refractive and non-refractive
3. Refractive accommodative esotropia is caused by significant hyperopia with normal AC/A ratio; correction of hyperopia alone can resolve esotropia
4. Non-refractive accommodative esotropia caused by high AC/A ratio without significant hyperopia; bifocals are necessary for realignment

E. Describe appropriate laboratory/clinical testing for establishing the diagnosis

1. Must do cycloplegic refraction/retinoscopy
2. Motility exam

II. Define the risk factors

A. High hyperopia

B. Family history of strabismus

III. List the most common or critical entities in the differential diagnosis

A. Infantile esotropia

B. Cranial nerve (CN) VI palsy

C. Pseudoesotropia

IV. List treatment options

A. Full cycloplegic spectacle correction

B. Amblyopia treatment as indicated

C. Surgery not indicated unless significant esotropia present with full spectacle correction (infantile esotropia concurrent with accommodative esotropia)

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. Coats DK, Avilla CW, Paysse EA, et al. Early-onset refractive accommodative esotropia. J AAPOS. 1998. 2:275-8.
3. AAO, Preferred Practice Pattern Committee, Pediatric Ophthalmology Panel: Esotropia and Exotropia Preferred Practice Pattern, 2012.

Exodeviations

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Unknown etiology for infantile exotropia, exophoria, and intermittent exotropia but can be associated with neurologic disease
2. Sensory exotropia is due to reduced visual acuity in one or both eyes due to anisometropia or injury/degeneration of ocular structures

B. Define the relative aspects of epidemiology of the disease

1. Infantile exotropia
 - a. Much less common than infantile esotropia
 - b. High association with neurological problems
 - c. May be associated with craniofacial syndromes
2. Intermittent exotropia
 - a. Onset in childhood, may decompensate with age
 - b. Most common type of exodeviation

C. List the pertinent elements of the history

1. Infantile exotropia
 - a. Large angle deviation present in first year of life
2. Exophoria and Intermittent exotropia
 - a. Worse with fatigue, illness, or injury
 - b. Asthenopia may be present, especially with reading
3. Sensory exotropia
 - a. Develops weeks to months after visual loss occurs
 - b. May cause diplopia

D. Describe pertinent clinical features

1. Infantile exotropia
 - a. Full versions, perhaps mild adduction deficit
 - b. Alternate fixation common
 - c. Normal refractive error
2. Exophoria
 - a. Usually controlled by fusion mechanisms under conditions of normal binocular vision
 - b. May be associated with myopia
3. Intermittent exotropia
 - a. Full ductions and versions
 - b. Stereopsis present during periods of orthotropia
 - c. Amblyopia and diplopia uncommon
4. Sensory exotropia
 - a. Large angle exotropia
 - b. Adduction deficits common (due to lateral rectus contraction)

II. List the differential diagnosis for exotropia

- A. Cranial nerve (CN) III palsy**
- B. Duane syndrome**
- C. Internuclear ophthalmoplegia**
- D. Infantile exotropia**
- E. Intermittent exotropia**
- F. Sensory exotropia**
- G. Consecutive exotropia**
- H. Ocular myasthenia gravis**

III. Describe patient management in terms of treatment and follow-up

- A. Infantile exotropia, exophoria, and intermittent exotropia**
 - 1. Treatment of refractive errors
 - 2. Treatment of amblyopia
 - 3. Prisms
 - 4. Orthoptic exercises for exophoria and intermittent exotropia
 - 5. Surgery for infantile exotropia and intermittent exotropia, especially if progressing toward constant exotropia
 - 6. Evaluation for neurologic disease in infantile exotropia
- B. Sensory exotropia**
 - 1. For diplopia can use occlusion, prisms, or fogging of non-preferred eye
 - 2. Treatment of accompanying amblyopia, if possible
 - 3. Surgical option of recess-resect procedure on non-preferred eye for cosmesis
 - 4. Sometimes resolves if binocularity restored (e.g. cataract surgery)

IV. Describe disease-related complications

- A. Infantile exotropia**
 - 1. Amblyopia
 - 2. Loss of stereopsis
- B. Exophoria**
 - 1. Progression to intermittent or constant exotropia
- C. Intermittent exotropia**
 - 1. Progression to constant exotropia

V. Describe patient appropriate patient instructions

- A. Sensory exotropia**
 - 1. Protection of sound eye in functionally monocular patient (polycarbonate lenses)
 - 2. Because fusion is compromised, exotropia may recur

1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.
2. AAO, Preferred Practice Patterns Committee, Pediatric Ophthalmology Panel. Esotropia and Exotropia Preferred Practice Pattern, 2012.

Strabismus following ocular surgery

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Binocular diplopia
 - a. Scarring of fascial tissues
 - b. Mechanical restriction due to implanted material (e.g., scleral buckle or glaucoma drainage device)
 - c. Direct injury or anesthetic toxicity to extraocular muscle(s) or nerves
 - d. Disruption of fusion due to dissimilar sensory input between the two eyes (e.g., monovision, aniseikonia)
 - e. Uncovering or worsening of pre-existing strabismus
 - f. Induced prismatic effect with eyeglass lenses for anisometropia/anisoastigmatism
 - g. Restored binocular vision after prolonged disruption of fusion
2. Monocular diplopia

B. List the pertinent elements of the history

1. Characteristics of double vision
 - a. Monocular vs. binocular
 - b. Vertical, horizontal, and/or torsional
 - c. Duration and rate of onset
 - d. Perceived stability
2. History of double vision, eye misalignment, high refractive error or amblyopia prior to surgery
3. Previous ocular surgeries, including use of extraocular implants and type of anesthesia (injection vs topical)

C. Describe pertinent clinical features

1. Vertical, horizontal, and/or torsional strabismus
2. Usually incommittant
3. Frequently limitation of ductions due to mechanical restriction
4. After retrobulbar or peribulbar injection, most common presentation is hypotropia of affected eye with restricted elevation
 - a. Affected muscle often initially paretic with hypertropia, later becomes fibrotic with hypotropia
 - b. History of reversing deviation common
5. If strabismus absent, anisometropia (e.g., after cataract or refractive surgery) or findings associated with monocular diplopia (e.g., after cataract surgery or retinal detachment repair)

II. Define the risk factors

- A. History of strabismus or amblyopia
- B. History of surgery involving placement of extraocular implant
- C. Retrobulbar or peribulbar injection
- D. Prolonged disruption of fusion
- E. Multiple surgeries

III. List the differential diagnosis

- A. Pre-existing strabismus
- B. Thyroid eye disease
- C. Myasthenia gravis
- D. Acquired cranial nerve palsy

IV. Describe patient management in terms of treatment and follow-up

A. Define medical therapy options

- 1. Prisms sometimes useful for small deviations or as temporizing measure while waiting for stable alignment
- 2. Monocular occlusion
- 3. Observation alone if no diplopia in common positions of gaze

B. Define surgical therapy options

- 1. Stability of alignment for several months before surgery is desirable

V. List the complications of treatment, their prevention and management

- A. Overcorrection / undercorrection after strabismus surgery/persistent diplopia
- B. Scarring of conjunctiva due to multiple surgeries
- C. Inadequate control of glaucoma if drainage device removed

VI. Describe appropriate patient instructions

- A. Goal of treatment is to restore single vision in primary and /or downgaze, but double vision in other positions of gaze often persists
- B. More than one strabismus surgery may be required

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 6: Pediatric Ophthalmology and Strabismus, 2015-2016.

- F. Dry eye syndrome**
- G. Flap-related complications following LASIK**
- H. Difficulty accurately assessing intraocular pressure**
- I. Inaccuracy of intraocular lens power calculations among patients eventually requiring cataract surgery**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 14: Refractive Surgery, 2015-2016.
2. AAO, Preferred Practice Pattern Refractive Errors and Refractive Surgery. 2013.
3. AAO, Ophthalmic Technology Assessment: Laser in-situ keratomileusis for Myopia and Astigmatism: Safety and Efficacy, Ophthalmology 2002; 109: 175-87.
4. AAO, Ophthalmic Technology Assessment: Laser in-situ keratomileusis for Hyperopia, Hyperopic Astigmatism and Mixed Astigmatism. Ophthalmology 2004; 111:1604-17.

Keratorefractive surgery

I. List the indications/contraindications

A. Indications

1. Refractive error treatable by the excimer laser system
2. Absence of ocular disease
3. Adequate corneal thickness
4. Good general health

B. Contraindications

1. Unstable refraction
2. Insufficient corneal thickness (laser in situ keratomileusis (LASIK))
3. Corneal abnormalities
4. Visually significant cataract
5. Uncontrolled glaucoma
6. Abnormal medical examination
 - a. Uncontrolled diabetes mellitus
 - b. Pregnancy
 - c. Uncontrolled connective tissue disease
 - d. Severe dry eye syndrome
7. Unrealistic expectations

II. Methods of refractive management

A. Nonsurgical

1. Eyeglasses
2. Contact lenses - including soft, toric or gas-permeable types

B. Surgical (cornea based)

1. Laser vision correction
 - a. Photorefractive keratectomy (PRK)
 - b. LASIK
2. Incisional surgery
 - a. Astigmatic keratotomy (AK)
 - i. Flattens a corneal meridian with arcuate or transverse incisions

III. Complications and sequelae of the procedures

- A. Incomplete refractive correction
- B. Optical side effects (glare, halos, starburst, monocular diplopia)
- C. Poor quality ablation
- D. Microbial keratitis
- E. Postsurgical scarring or steeping

Issues of intraocular pressure measurements after laser in situ keratomileusis and laser surface ablation

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the situation

1. Laser eye surgery removes corneal tissue, resulting in a thinner cornea
2. Devices used to measure eye pressure (Goldmann tonometer/Tono-Pen, etc) measure eye pressure with the assumption of a normal corneal thickness
3. Corneas that have become thinner following laser eye surgery will seem to have a lower measured pressure, although their actual intraocular pressure (IOP) should be unchanged following surgery
 - a. Most studies suggest approximately 15-20 microns of thinning reduces measured IOP by one millimeter of mercury (mmHg)

B. Describe pertinent clinical features

1. IOP measures lower than prior to surgery
2. IOP measurement may be in the low or normal range when it is actually elevated
3. Eyes suspicious for glaucoma based on optic disc cupping or visual field loss should be evaluated despite low IOP measurements

C. Describe appropriate testing for establishing the diagnosis

1. Pachymetry: to measure the corneal thickness

II. Define the risk factors

A. Patients with risk factors for glaucoma (family history, optic disc cupping, optic disc asymmetry, visual field loss) despite low or normal IOP

B. Frequent use of topical corticosteroids

III. List the differential diagnosis

A. Unrelated IOP reduction

B. Postoperative optic neuropathy

IV. Describe appropriate patient instructions

A. Alert all doctors who measure eye pressure of previous laser eye surgery

B. Educate patient on effects of laser vision correction on IOP measurement

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 14: Refractive Surgery, 2015-2016.

Optical coherence tomography

I. Describe the underlying physical principles

- A. Non-contact technique (different from ultrasonography)
- B. Requires optically clear media (different from ultrasonography)
- C. Requires cooperative patient (fixation)

II. List the indications/contraindications

A. Indications

1. Macular diseases -OCT may be of value in the evaluation of
 - a. Vitreomacular interface disorders
 - i. Posterior vitreous detachment
 - ii. Vitreomacular traction
 - iii. Epiretinal membrane
 - iv. Macular hole
 - b. Macular diseases
 - i. Cystoid macular edema
 - ii. Diabetic retinopathy and macular edema
 - iii. Retinal vascular disease and macular edema
 - iv. Subretinal fluid
 - v. Central serous chorioretinopathy
 - vi. Age-related macular degeneration, nonexudative and exudative forms
 - vii. Other causes of choroidal neovascular membrane and SRF including
 - i) Myopic degeneration
 - ii) Presumed ocular histoplasmosis
 - iii) Choroidal rupture
 - viii. Macular toxicity disorders
 - i) Hydroxychloroquine
 - ix. Retinal and macular dystrophies
 - x. Chorioretinal inflammatory diseases
 - xi. Retinal detachments, tractional and rhegmatogenous
 - xii. Trauma
 - xiii. Other
2. Optic nerve diseases - OCT may be of value in
 - a. Vitreopapillary traction
 - b. Glaucomatous optic neuropathy
 - c. Papillitis
 - d. Ischemic optic neuropathy
 - e. Optic disc edema

3. Anterior segment evaluation - OCT may be of value in evaluation of
 - a. Anterior chamber depth
 - b. Angle structure/angle pathology
 - c. IOL positioning

B. Contraindications: none

III. Describe the pre-procedure evaluation

- A. Explain procedure**
- B. Pupillary dilation provides optimal posterior segment imaging**
- C. Media clarity required for optimal imaging**
- D. Adequate patient fixation is required**

IV. List the alternatives to this procedure

- A. Macular contact lens examination at the slit-lamp biomicroscope**
- B. Fluorescein angiography**

V. Considerations in interpretation of procedure

- A. Acquisition of a good quality OCT scan**
 1. Adequate pupillary dilation
 2. Clear ocular media
 3. Steady patient fixation
- B. Accurate OCT scan interpretation - quantitative information**
 1. Retinal thickness/volume measurement/map
 2. Retinal and optic disc nerve fiber layer thickness/volume measurement/map
 3. Serial scan analysis
 4. Identification of morphological changes in tissue layers - atrophy, thickening, distortion

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. Carl Zeiss Ophthalmic Systems, Inc. Optical Coherence Tomographer - Model 3000, User manual. Germany, 2002.
3. Joel S. Schuman, Carmen A. Puliafito, James G. Fujimoto. Optical Coherence Tomography of Ocular Disease, Second Edition. Slack Incorporated, 2004.

Macular hole

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Localized perifoveal vitreous detachment with persistent foveal attachment resulting in traction on the fovea

B. Define the relevant aspects of epidemiology of the disease

1. Typically idiopathic, but can be associated with other ocular conditions including
 - a. Myopia
 - b. Direct trauma to globe
2. Idiopathic holes are most prevalent in patients 50-80 years old and are more common in women
3. A patient with an idiopathic macular hole has a slightly increased risk of a contralateral macular hole

C. List the pertinent elements of the history

1. Central visual impairment
2. Metamorphopsia
3. Blurring of vision
4. Central scotoma

D. Describe appropriate testing for establishing the diagnosis

1. Optical coherence tomography
2. Fluorescein angiogram

II. Define the risk factors

A. Idiopathic holes

1. Age
2. Female gender

B. Traumatic holes

1. Direct globe trauma

C. Myopic holes

1. High Myopia

III. List the differential diagnosis

A. Cystoid macular edema

B. Lamellar or "Pseudo" macular hole associated with epiretinal membrane

IV. Describe the patient management in terms of treatment and follow-up

A. For traumatic macular holes

1. Observation for a period of months even with full thickness hole as spontaneous closure can occur (unknown what percentage will spontaneously close)
2. Consider pars plana vitrectomy if hole remains

B. For idiopathic and myopic holes

1. Observation
2. Pars plana vitrectomy with gas tamponade; best results if hole is less than one year in duration, but can obtain good results in some patients with holes of up to 3 years duration

V. List the complications of treatment, their prevention and management

A. Vitrectomy

1. Cataract (typically nuclear sclerosis)
2. Visual field defect, retinal tear, retinal detachment, ocular hypertension, vitreous hemorrhage, cataract (PSC)
3. Macular damage, especially macular pigmentary disruption

B. General

1. Late hole re-opening (rare)

VI. Describe disease-related complications

A. Loss of central vision due to foveal atrophy

VII. Describe appropriate patient instructions

A. Amsler grid

B. Risk of fellow eye involvement

Additional Resources

1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. AAO, Focal Points: Evaluation and Management of Macular Holes Module #1, 2003.
3. AAO, Focal Points: Macular Surface Disorders, Module #4, 1996, p.1-5.
4. AAO, Preferred Practice Patterns. Idiopathic Macular Hole, 2003.
5. Ophthalmic Technology Assessment: Surgical Management of Macular Holes, Ophthalmic Technology Assessment Committee Retina Panel, American Academy of Ophthalmology, *Ophthalmology* 2001; 108:1328-1335.

Hypertensive retinopathy

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Acute and/or chronic elevation of systemic blood pressure (BP)

B. Define the relevant aspects of epidemiology of the disease

1. Defined at systolic BP ≥ 130 or diastolic BP ≥ 85 , or treatment with anti-hypertensive medication
2. Hypertension is a risk factor for cardiovascular disease

C. List the pertinent elements of the history

1. History of hypertension may or may not be known
2. Usually without ocular symptoms
3. May have visual loss as a result of secondary complications

D. Describe pertinent clinical features

1. Retinopathy may be graded using the Modified Scheie classification
 - a. Retinal vascular changes include arteriolar narrowing, arteriovenous (A/V) crossing changes, hemorrhages, exudates, cotton wool spots, and optic disc edema, and serous retinal detachment
2. Changes may also affect the choroid
 - a. Ischemia of the choroid may manifest with RPE hyperpigmentation
3. Acute malignant hypertensive retinopathy/choroidopathy
 - a. Associated with severe hypertension, usually of rapid onset, typically can be seen in renal failure and eclampsia
 - b. Clinical features include cotton wool spots, intraretinal hemorrhages, serous retinal detachment, capillary obliteration, and optic disc edema
 - c. Treatment is emergent with referral to PCP or emergency room for aggressive blood pressure management
4. Late findings may include
 - a. Retinal telangiectasis
 - b. Severe retinal vessel sclerotic changes
 - c. Optic atrophy

II. Define the risk factors

- A. African-Americans at greater risk
- B. Age
- C. Obesity
- D. Smoking
- E. Poorly controlled systemic hypertension

III. List the differential diagnosis

- A. Diabetic retinopathy
- B. Central retinal vein occlusion

- C. **Toxemia of pregnancy**
- D. **Other retinal vascular diseases**

IV. Describe patient management in terms of treatment and follow-up

- A. **Describe the natural history, outcome and prognosis**
 - 1. Predisposes to ocular vascular complications noted below
- B. **Describe medical therapy options**
 - 1. Refer to primary care provider to manage BP and search for causes of secondary hypertension

V. Describe disease-related complications

- A. **Associated with**
 - 1. Branch vein occlusion
 - 2. Central vein occlusion
 - 3. Arteriolar macroaneurysms
 - 4. Non-arteritic anterior ischemic optic neuropathy
- B. **May worsen course of diabetic retinopathy**
- C. **May develop retinal ischemia**
- D. **May develop late optic nerve atrophy**

VI. Describe appropriate patient instructions

- A. **Maintain good BP control**
- B. **Seek regular ophthalmic care**
- C. **Consider referral to retinal specialist for complications listed above, or in cases of malignant hypertensive retinopathy**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 12: Retina and Vitreous, 2015-2016.
- 2. Wong T, Mitchell P. The eye in hypertension. *Lancet* 2007;369:425-35.
- 3. Grosso A, Veglio F, Porta M, et al. Hypertensive retinopathy revisited: some answers, more questions. *Br J Ophthalmol* 2005;89:1646-54.
- 4. Wong TY, Mitchell P. Hypertensive retinopathy. *N Engl J Med* 2004;351:2310-7.
- 5. Schubert HD. Ocular manifestations of systemic hypertension. *Curr Opin Ophthalmol* 1998;9:69-72.

Diabetic retinopathy

I. Classification

- A. Nonproliferative
- B. Proliferative
- C. Diabetic macular edema

II. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Diabetes mellitus especially if poorly controlled with high blood glucose levels

B. Define the relevant aspects of epidemiology of the disease

1. Prevalence of diabetes mellitus and retinopathy
 - a. Type 1 diabetes (5-10% of patients with diabetes)
 - i. Rarely have retinopathy at diagnosis
 - ii. 90% develop retinopathy after 10-15 years
 - iii. 25% develop proliferative disease
 - b. Type 2 diabetes (90-95% of patients with diabetes)
 - i. 30% have retinopathy at diagnosis
 - ii. 50% of **non-insulin dependent patients** have retinopathy after 15 years
 - iii. 80% of **insulin dependent patients** have retinopathy after 15 years
 - iv. Accelerating incidence in the US

C. List the pertinent elements of the history

1. Duration and type of diabetes mellitus
2. Glycemic control (HgA1C)
3. Visual impairment
4. Presence of floaters
5. Systemic hypertension
6. Cardiovascular disease
7. Renal disease
8. Neuropathy
9. Lipid disorder
10. Pregnancy

D. Describe pertinent clinical features

1. Microaneurysms
2. Dot and blot hemorrhages
3. Cotton wool spots
4. Intraretinal lipid
5. Venous beading
6. Intraretinal microvascular abnormalities

7. Neovascularization
8. Macular edema
9. Capillary nonperfusion
10. Vitreous hemorrhage
11. Pre-retinal hemorrhage
12. Tractional retinal detachment

E. Describe appropriate testing and evaluation for establishing the diagnosis

1. Fundus photographs
2. Fluorescein angiography
3. Optical coherence tomography

III. Define the risk factors for developing disease progression

- A. Poor glycemic control
- B. Increased duration of diabetes mellitus
- C. Increased blood pressure
- D. Increased serum lipid levels
- E. Severe diabetic retinopathy in the other eye
- F. Pregnancy
- G. Rapid normalization of blood sugar in patient with history of poor control
- H. Smoking

IV. List the differential diagnosis for diabetic retinopathy

- A. Branch or central vein occlusion
- B. Radiation retinopathy
- C. Parafoveal telangiectasis
- D. Retinal macroaneurysm
- E. Hypertensive retinopathy
- F. Ocular ischemic syndrome

V. Describe patient management in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. Diabetic retinopathy usually progresses
2. Diabetic macular edema may occur at any stage of diabetic retinopathy
3. Visual impairment can result from diabetic macular edema, macular ischemia, or complications of proliferative diabetic retinopathy (PDR)

B. Describe medical therapy options

1. Control of hyperglycemia, hypertension, dyslipidemia, and renal disease

C. Describe surgical therapy options

1. Laser photocoagulation
 - a. For those with PDR, especially those at high-risk (as defined by DRS), prompt panretinal (scatter) photocoagulation should be given

- b. Consider panretinal photocoagulation for those with severe nonproliferative diabetic retinopathy (NPDR)
 - c. Focal macular photocoagulation is generally inferior to intravitreal injection
 - i. Laser combined with injections is no better than injection of anti-VEGF agents alone
- 2. Referral to retinal specialist for vitrectomy in patients with nonclearing vitreous hemorrhages, tractional macular detachments, and in cases of chronic diabetic macular edema in which vitreoretinal traction can be identified

D. Diabetic macular edema

- 1. Intravitreal injection of steroids and/or anti VEGF medications
- 2. Corticosteroid-releasing implant
- 3. Laser photocoagulation

VI. List the complications of treatment, their prevention and management

A. Laser photocoagulation

- 1. Inadvertent treatment to the fovea, rare
- 2. Subretinal choroidal neovascularization
- 3. Worsening of diabetic macular edema
- 4. Peripheral field loss
- 5. Choroidal effusions and angle closure

B. Vitrectomy

- 1. Phthisis
- 2. Endophthalmitis
- 3. Corneal epithelial problems
- 4. Rhegmatogenous retinal detachment
- 5. Cataract

C. Intravitreal Injection

- 1. Endophthalmitis
- 2. Retinal detachment
- 3. Vitreous hemorrhage

VII. Describe appropriate patient instructions

A. Need for periodic eye examinations

B. Maintain good glycemic control

C. Maintain good blood pressure control

D. Maintain good lipid control

E. Symptoms of macular edema and vitreous hemorrhage (blurred vision and floaters) should trigger prompt consultation with the ophthalmologist

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 12: Retina and Vitreous, 2015-2016.
- 2. AAO, Preferred Practice Pattern Committee, Retina Panel. Diabetic Retinopathy Preferred Practice Pattern, 2014.
- 3. AAO, Laser Photocoagulation of the Retina and Choroid, 1997, p.63-95.

4. AAO, Diabetes and Ocular Disease: Past, Present and Future Therapies, 2000, p.4-5, 8, 13, 20-26, 36-40, 45-49, 69-202, 239-298.
5. Early Treatment Diabetic Retinopathy Study Research Group. Results from the Early Treatment Diabetic Retinopathy Study. *Ophthalmology*. 1991;98:739-840.
6. Wilkinson CP, Ferris FL 3rd, Klein RE et al. Proposed international clinical diabetic retinopathy and diabetic macular edema disease severity scales *Ophthalmology*. 2003; 110:1677-82.
7. Diabetic Retinopathy Clinical Research Network , Writing Committee, Elman MJ, Bressler NM, Qin H et al. Expanded 2-Year Follow-up of Ranibizumab Plus Prompt or Deferred Laser or Triamcinolone Plus Prompt Laser for Diabetic Macular Edema *Ophthalmology* April 2011;118;609-614.

Sickle cell retinopathy

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Sickle-cell hemoglobinopathy results from a genetic mutation in the hemoglobin A (HbA) protein that polymerizes in low oxygen environments leading to 'sickling' of erythrocytes and retinal vascular occlusion
2. Evolutionary pressure for this mutation stems from a protective effect in malarial infections

B. Define the relevant aspects of epidemiology of the disease

1. Sickle mutations are highest in African-American populations

C. List the pertinent elements of the history

1. Known hemoglobinopathy (see above)
 - a. Retinopathy more common in HbSC and HBS Thal variants than HbSS
2. African heritage
3. History of sickle crisis with severe pain of long bones, chest, abdomen, joints
 - a. Crisis more common in HbSS
4. Myocardial infarction and stroke
5. Symptoms
 - a. Blurry vision
 - b. New floaters
 - c. Photopsia
 - d. Scotoma (partial or complete)
 - e. Ocular pain

D. Describe pertinent clinical features

1. Signs
 - a. Non-proliferative
 - i. Conjunctival vasculature changes ('corkscrew or comma-shaped' vessels)
 - ii. Iris atrophy
 - iii. Fundus changes:
 - i) Salmon patch (retinal/pre-retinal hemorrhage)
 - ii) Black sunburst (RPE hyperplasia)
 - iii) Retinal vascular tortuosity
 - iv) Artery occlusion (branch or central)
 - v) Retinal vascular sclerosis (whitening)
 - iv. Choroidal vascular occlusions
 - v. Angioid streaks
 - b. Proliferative sickle retinopathy
 - i. Peripheral neovascularization "sea-fan"
 - ii. Vitreous hemorrhage
 - iii. Tractional retinal detachment (RD)

E. Describe appropriate testing and evaluation for establishing the diagnosis

1. Sickle prep (rapid test)
2. Hemoglobin electrophoresis (identifies hemoglobin chains)

II. Define the risk factors

- A. Family history
- B. African ancestry
- C. Mediterranean ancestry (less common)
- D. Hypoxia
- E. Dehydration
- F. Possibly smoking (increases risk of acute chest syndrome)

III. List the differential diagnosis

- A. Diabetic retinopathy and tractional retinal detachment
- B. Hypertensive retinopathy
- C. Central or branch retinal vein occlusion
- D. Radiation retinopathy
- E. Leukemic retinopathy

IV. Describe patient management in terms of treatment and follow-up

- A. Describe the natural history, outcome, and prognosis
 1. Many peripheral neovascular fronds regress
 2. Salmon patch resolves spontaneously
 - a. May leave hyperpigmented black sunburst
 - b. May create vitreoretinal traction or epiretinal membrane
- B. Describe systemic therapy options
 1. Maintain good oxygenation
 2. Maintain good hydration
- C. Describe therapy for elevated intraocular pressure
 1. Topical agents (multiple if necessary) to control IOP
 2. Avoid acetazolamide (Diamox®) which causes metabolic acidosis and increases sickling tendency
- D. Referral to retina specialist for treatment of neovascularization and retinal detachment repair

V. List the complications of treatment, their prevention and management

- A. Treat IOP elevation aggressively
- B. In patients with hyphema, consider AC washout to control IOP
- C. Recommend good hydration and oxygenation

VI. Describe disease-related complications

- A. Ocular

1. Vitreoretinal
 - a. Vitreous hemorrhage
 - b. Vitreoretinal traction
 - c. Epiretinal membrane
 - d. Retinoschisis
 - e. Retinal detachment
 - f. Retinal hemorrhage
 - g. Peripheral retinal vascular ischemia
 - h. Macular ischemia
 2. Choroidal
 - a. Choroidal infarction
 - b. Angioid streaks
 3. Optic nerve ischemia
 4. Cataract
 5. Iris atrophy
- B. Systemic**
1. Ischemia/infarction of multiple organ systems
 2. Medication toxicity (glaucoma medications, desferrioxamine)

VII. Describe appropriate patient instructions

- A. Recommend good hydration and oxygenation**
- B. Obtain dilated fundus examination annually and with onset of new visual symptoms**

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. Hemoglobinopathies in Retina ed. Ryan SJ, Section 5 Chapter 79: Emerson GG, Harlan JB Jr., Fekrat S, Luty GA, Goldberg MF. Mosby 2005.
3. Penman AD, Serjeant GR. Recent advances in the treatment of proliferative sickle cell retinopathy. *Curr Opin Ophthalmol* 1992;3:379-88.
4. Jacobson MS, Gagliano DA, Cohen SB, et al. A randomized clinical trial of feeder vessel photocoagulation of sickle cell retinopathy. A long-term follow-up. *Ophthalmology* 1991;98:581-5.
5. Farber MD, Jampol LM, Fox P, et al. A randomized clinical trial of scatter photocoagulation of proliferative sickle cell retinopathy. *Arch Ophthalmol* 1991;109:363-7.
6. Jampol LM, Farber M, Rabb MF, et al. An update on techniques of photocoagulation treatment of proliferative sickle cell retinopathy. *Eye* 1991;5 (pt 2):260-3.
7. AAO, Ophthalmology Monographs 11. Laser Photocoagulation of the Retina and Choroid, 1997.
8. AAO, Ophthalmology Monographs 2. Electrophysiologic Testing in Disorders of the Retina, Optic Nerve, and Visual Pathway, 2nd ed., 2001.

Retinitis pigmentosa

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Multiple different genetic defects which cause photoreceptor death

B. Define the relevant aspects of epidemiology of the disease

1. Most common hereditary chorioretinal dystrophy
2. Most common inheritance patterns are autosomal dominant, autosomal recessive, or x-linked recessive
 - a. Half have no family history

C. List the pertinent elements of the history

1. Nyctalopia
2. Constricted visual fields
3. Onset of symptoms commonly in first or second decade of life

D. Describe pertinent clinical features

1. Waxy disc pallor
2. Narrowed arterioles
3. Pigment within the retina, either as a generalized granularity, as discrete pigment clumps, or as deposits appearing as bone spicules
4. Fine pigmented cells in vitreous
5. May have cystoid macular edema (CME)
6. May have posterior subcapsular cataracts

E. Describe appropriate laboratory testing for establishing the diagnosis

1. Electroretinography
2. Dark adaptometry
3. Visual field testing
4. Fluorescein angiography
5. OCT if macular edema suspected
6. Genetic testing

II. Define the risk factors

A. Positive family history

III. List the differential diagnosis

- A. Infection (such as syphilis, congenital rubella)
- B. Inflammation (uveitis)
- C. Choroidal vascular occlusion
- D. Toxicity (chloroquine or thioridazine)

IV. Describe patient management in terms of treatment and follow-up

A. Describe natural history, outcomes, prognosis

1. Slowly progresses to visual loss over time

B. Describe therapy options

1. Vitamin A palmitate -- clinical benefit is questionable
2. Docosahexaenoic acid (DHA), an omega-3 fatty acid -- clinical benefit is questionable
3. Treatment of cystoid macular edema
4. Cataract surgery may be beneficial in some cases
5. Vision rehabilitation aids as needed (retina prosthesis)

V. Describe appropriate patient instructions

A. Genetic counseling

B. Assess driving abilities in relationship to visual acuity and field

Additional Resources

1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. AAO, Electrophysiologic Testing in Disorders of the Retina, Optic Nerve and Visual Pathway, 2nd ed., 2001, p.30-39,165,208-209.

Chloroquine and its derivatives (drug toxicity)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Chronically administered chloroquine phosphate (Aralen™) for malaria prophylaxis and hydroxychloroquine (Plaquenil™) generally for treatment of collagen-vascular related arthritis (e.g., rheumatoid arthritis, systemic lupus erythematosus)

B. Define the relevant epidemiology of this disease

1. Most cases in 20 to 50 year-olds and more common in females (likely because of distribution of diseases treated)
2. Cumulative continuous years of exposure increases risk, especially if longer than 10 years at standard dosing or higher daily doses
 - a. Cumulative risk at standard daily dose is less than 2% for up to 10 years; 20% after 20 years
3. Liver or renal failure increases risk because these drugs are cleared by these organ systems
4. No racial predilection

C. List the pertinent elements of the history

1. Treatment of collagen-vascular disease or malaria prophylaxis with a chloroquine derivative
2. Blurred vision, reading difficulties due to paracentral scotomas, central photopsia, photophobia; in more advanced cases, color vision disturbance
3. History of macular degeneration or retinal dystrophy
4. Weight and height
 - a. New information indicates that real body weight, rather than prior recommendation of ideal weight, should be used to calculate dosing
 - i. For hydroxychloroquine ≥ 5.0 mg/kg real weight is maximum recommended for typical patient
5. Liver failure

D. Describe pertinent clinical features

1. Macular pigment abnormality, most typically a bull's-eye of depigmentation or atrophy

E. Describe appropriate ancillary testing for establishing the diagnosis

1. Spectral domain OCT of the macula, evaluating continuity of the inner segment-outer segment junction
 - a. "Flying-saucer" sign (parafoveal thinning of photoreceptor layers and loss of the inner-/outer-segment line)
 - b. Time domain OCT is not useful for early detection
 - c. For Asian patients need to look outside the macular area as well
2. Fundus autofluorescence
3. Paracentral scotoma detected by Amsler Grid test or standardized visual field test (e.g., Humphrey 10-2)
4. Multifocal electroretinogram (mf ERG)
5. Color vision

II. Define the risk factors

A. Hydroxychloroquine has much less potential ocular toxicity than chloroquine

- B. **Duration: toxicity occurs after at least 6 months of treatment and risk increases with longer duration of drug use**
- C. **Dose: toxicity much more likely as dose increases**
- D. **Greatest risk is after 10 years in patients on standard dose**
- E. **Renal or liver insufficiency requires reduction in dose to prevent toxicity**

III. Describe patient management in terms of treatment and follow-up

- A. **Describe the natural history, outcome and prognosis**
 - 1. Progressive loss of visual acuity, visual field, and color vision if drug continued
- B. **Describe medical therapy options**
 - 1. Baseline examination within the first year of starting the drug to document any complicating ocular conditions, and to establish a record of the fundus appearance and functional status
 - 2. Follow-up examination and ancillary testing after first five years of drug use if no unusual risk factors is the current AAO-recommended interval for patient's age and annually thereafter
 - a. More frequent intervals for higher dose, hepatic/renal disease, tamoxifen use
 - 3. Discontinuation of the chloroquine derivative at the first sign of toxicity is recommended
 - 4. Toxic effects may progress even after drug is stopped

IV. Describe appropriate patient instructions

- A. **Description of potential ocular toxicity to all patients taking a chloroquine derivative**
- B. **Follow-up examinations annually if patients are at higher risk due to higher dose, a duration longer than 10 years, or have renal or liver insufficiency, tamoxifen use**
- C. **Ophthalmoscopic changes and measurable loss of visual function may precede visual symptoms**
- D. **Patients should understand that screening examinations help to identify toxicity earlier, but cannot prevent toxicity or guarantee that there will be no visual loss**

Additional Resources

- 1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
- 2. Marmor, MF, Kellner, U, Lai, TY et al. Revised Recommendations on Screening for Chloroquine and Hydroxychloroquine Retinopathy. *Ophthalmology* 2011; 118:415-22.
- 3. AAO, Preferred Practice Pattern Committee. Comprehensive Adult Medical Eye Evaluation Preferred Practice Pattern, 2015.

Operculated retinal holes

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Dynamic vitreoretinal traction in the setting of a posterior vitreous detachment resulting in a full-thickness retinal defect

B. Define the relevant aspects of epidemiology of this disease

1. Most operculated retinal holes are asymptomatic retinal breaks and only rarely lead to retinal detachment
2. Retinal detachment, when it occurs, is due to residual vitreoretinal traction on or near the hole

C. Describe pertinent clinical features

1. Red blood cells or pigment granules on slit-lamp biomicroscopic examination of the anterior vitreous (tobacco dust)
2. Round or oval full-thickness retinal break on indirect ophthalmoscopy
3. Retinal operculum often visible floating in the vitreous cavity
4. Subretinal fluid may be present around the operculated hole
5. Pigmentary demarcation around operculated hole is evidence of chronicity
6. Retinal vessel bridging an operculated hole can be a source of vitreous hemorrhage

II. Define the risk factors

- A. Increasing age
- B. History of retinal tear or detachment in contralateral eye
- C. Family history of retinal tear or detachment
- D. Ocular trauma
- E. Axial myopia
- F. Aphakia, pseudophakia, status post laser posterior capsulotomy

III. Describe patient management in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. Operculated holes rarely progress to retinal detachment but the risk is greater if residual traction or surrounding subretinal fluid is present
2. Pigmentary demarcation around retinal tear is evidence of chronicity and, while the demarcation itself is not protective, its presence might reassure that the condition has not been progressive

B. Describe surgical therapy options

1. Symptomatic operculated holes do not necessarily require treatment unless there is evidence of persistent vitreous traction
 - a. If not treated close followup is required for several months
2. Asymptomatic operculated holes, especially if pigmentary demarcation is present, can be observed
3. History of prior retinal detachment in fellow eye, high myopia, aphakia, pseudophakia or upcoming cataract surgery, can bias toward treatment, especially in cases of symptomatic operculated holes
4. Retinopexy methods include cryopexy and laser photocoagulation

IV. Describe disease-related complications

- A. Progression to retinal detachment**
- B. Epiretinal membrane formation**
- C. Vitreous hemorrhage, possibly recurrent, from bridging retinal vessel**

V. Describe appropriate patient instructions

- A. Natural history description**
- B. Benefits, risks and alternatives of retinopexy**
- C. Importance of prompt consultation if patient develops sudden increase in floaters, photopsia or a shadow in the field of vision**

Additional Resources

1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. AAO, Laser Photocoagulation of the Retina and Choroid, 1997, p.212, 214.
3. AAO, Preferred Practice Pattern Committee. Posterior Vitreous Detachment, Retinal Breaks and Lattice Degeneration Preferred Practice Pattern, 2008.

Atrophic holes

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Progressive atrophy and thinning of the neurosensory retina resulting in a full-thickness retinal defect
2. Frequently seen in the thinned retina within areas of lattice degeneration
3. Vitreous traction is not a pathogenic mechanism

B. Define the relevant aspects of epidemiology of this disease

1. New holes may develop at a slow rate throughout life
2. About a third of patients with lattice degeneration have concomitant atrophic retinal holes
3. Patients with atrophic retinal holes are highly unlikely to later develop a clinical retinal detachment
4. Retinal detachments related to atrophic holes occur predominantly in young myopic patients with lattice lesions

C. List the pertinent elements of the history

1. Atrophic retinal holes usually are asymptomatic and found incidentally
2. Visual field defects, photopsias, and/or floaters should raise consideration of an associated retinal detachment

D. Describe pertinent clinical features

1. Round or oval full-thickness retinal break without an overlying operculum
2. Often found within a zone of lattice degeneration
3. Often surrounded by a cuff of subretinal fluid

II. Define the risk factors

- A. Increasing age
- B. Lattice degeneration
- C. Axial myopia
- D. Family history of atrophic holes or lattice degeneration

III. Describe patient management in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. With time, atrophic holes can enlarge and become more numerous
2. A small percentage can progressively accumulate subretinal fluid, leading to a subclinical retinal detachment
3. Subclinical retinal detachments usually do not progress or do so very slowly

B. Describe surgical therapy options

1. Treatment is rarely recommended
2. Retinopexy may be considered for atrophic holes with documented progression of subclinical detachment

IV. List the complications of treatment, their prevention and management

- A. Progression to retinal detachment

- B. Iatrogenic vitreous hemorrhage or choroidal neovascularization if laser photocoagulation performed**
- C. Inadvertent photocoagulation of the macula**

V. Describe appropriate patient instructions

- A. Natural history description**
- B. Importance of prompt consultation if patient develops sudden onset of floaters, photopsias, or a shadow in the field of vision**
- C. Return in 1-2 years for re-examination**
- D. Those associated with subclinical retinal detachments need closer follow up to monitor for potential progression**

Additional Resources

- 1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
- 2. AAO, Retinal Detachment: Principles and Practice, 2nd ed., 1995, p.14-15,18.
- 3. AAO, Focal Points: Retinal Breaks, Holes and Tears, Module #3, 1996, p.2, 7-8.
- 4. AAO, Preferred Practice Pattern Committee, Retinal Panel. Posterior Vitreous Detachment, Retinal Breaks and Lattice Detachment Preferred Practice Pattern, 2008.

Traumatic retinal breaks

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Transmission of blunt force to the vitreous may result in vitreoretinal traction that can cause a variety of retinal breaks including retinal dialysis, retinal flap tear, operculated retinal hole and macular hole
2. Open globe trauma can result in a tear of the retina either directly, or indirectly secondary to vitreoretinal traction or retinal incarceration in an ocular wound

B. List the pertinent elements of the history

1. History of trauma, usually recent, and whether one or both eyes involved
2. Floaters
3. Photopsias
4. Blurred vision
5. Loss of visual field or acuity
6. Mechanism of injury
 - a. Blunt
 - b. Penetrating
 - c. Projectile

C. Describe pertinent clinical features

1. If blunt mechanism of injury, retinal breaks are usually peripheral
 - a. Retinal dialysis most commonly inferotemporal or superonasal
2. Retinal break at the site of an entrance or exit scleral wound, or at the impact site of an intraocular foreign body
3. Increased chance of traumatic break in the presence of vitreous hemorrhage with possibility of hemorrhage obscuring break
4. Other signs of trauma may be present to any portion of the eye or periocular structures
5. Loss of vision due to blood in the visual axis, traumatic maculopathy, retinal detachment, or concurrent corneal, lens or optic nerve injury

D. Describe appropriate testing/evaluation to confirm the diagnosis

1. B-scan echography (if safe) to detect traumatic retinal break or detachment in the presence of vitreous hemorrhage or other media opacity secondary to anterior segment injury

II. Define the risk factors

A. Penetrating or severe blunt ocular trauma

B. Lack of appropriate eye protection

III. Describe patient management in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. Traumatic retinal breaks with vitreoretinal traction are usually treated
2. Penetrating trauma, giant retinal tears, vitreous hemorrhage, ocular inflammation and younger age increase risk for proliferative vitreoretinopathy and secondary complex retinal detachment
3. Retinal dialysis can slowly progress to symptomatic retinal detachment

4. Chronic asymptomatic traumatic breaks may occasionally remain stable without treatment, although this is the exception

B. Describe surgical therapy options

1. Laser retinopexy, or cryopexy if view obscured by opacity such as vitreous hemorrhage
2. Associated retinal detachment often requires scleral buckling surgery and/or vitrectomy

Additional Resources

1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. AAO, Retinal Detachment: Principles and Practice, 2nd ed., 1995, p.16-17.
3. AAO, Preferred Practice Pattern Committee. Posterior Vitreous Detachment, Retinal Breaks and Lattice Degeneration Preferred Practice Pattern, 2008.

Lattice degeneration

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Peripheral retinal condition characterized by
 - a. Local thinning of the inner retina
 - b. Strong vitreous adherence at its margins
 - c. Overlying vitreous liquefaction

B. Define the relevant aspects of epidemiology of the disease

1. More common in myopes
2. Familial in some reports
3. Can be associated with retinal detachments

C. List the pertinent elements of the history

1. Usually asymptomatic

D. Describe pertinent clinical features

1. Elliptically shaped, usually parallel to the ora, multiple, tendency to cluster in superior and inferior retina
 - a. Radial lattice
2. Usually located anterior to the equator
3. Named for the crisscrossing retinal vessels within the lattice patch
4. May have round atrophic holes within it

E. Describe appropriate ancillary testing for establishing the diagnosis

1. Scleral depressed exam to view retinal thinning

II. Define the risk factors

A. Positive family history

B. Myopia

III. Describe patient management in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. Lattice predisposes to retinal break formation
2. Small atrophic holes located within the lattice rarely lead to a retinal detachment
3. Lattice with horseshoe tears usually occurs in older patients, is associated with posterior vitreous detachment, and retinal detachment

B. Describe surgical therapy options

1. Treatment of lattice without a retinal break or with small atrophic holes without retinal detachment is usually not recommended
2. Associated retinal detachment should be treated appropriately

IV. Describe disease-related complications

- A. Retinal break with or without retinal detachment**

V. Describe appropriate patient instructions

- A. Warning signs of retinal detachment include photopsias, floaters, or a curtain or veil in the visual field**
- B. Discussion of natural history and outcomes**
- C. If no associated retinal breaks, follow-up yearly**

Additional Resources

1. AAO, Basic Clinical and Science Course. Section 12: Retina and Vitreous, 2015-2016.
2. AAO, Laser Photocoagulation of the Retina and Choroid, 1997, p.212-214.
3. AAO, Retinal Detachment: Principles and Practice, 2nd ed., 1995, p.9, 14, 23-26, 92.
4. AAO, Focal Points: Retinal Breaks, Holes and Tears, Module #3, 1996, p. 3-4.
5. AAO, Preferred Practice Pattern Committee. Posterior Vitreous Detachment, Retinal Breaks and Lattice Degeneration Preferred Practice Pattern, 2008.

Nevus of the choroid

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. Congenital

B. List the pertinent elements of the history

1. Lesions are generally asymptomatic, become recognized on routine ophthalmologic examination
2. New visual symptoms suggest malignant transformation
3. Change in clinical appearance suggests malignant transformation

C. Describe the pertinent clinical features

1. Flat or minimally elevated choroidal lesion
2. Usually brown to grey in coloration, occasionally amelanotic
3. Overlying drusen or retinal pigment epithelium (RPE) changes suggest chronicity
4. Orange pigmentation, associated subretinal fluid, increased thickness, increased basal dimension, new subjective symptoms are atypical for nevus and suggest the diagnosis of choroidal melanoma
5. Peripapillary nevi are more likely to grow

D. Describe appropriate ancillary and laboratory testing for establishing the diagnosis

1. Fundus photography to document clinical appearance for subsequent serial comparison
2. A- and B-scan ultrasound to document basal dimensions, thickness, and internal sonographic features
3. Fluorescein angiography

II. List the differential diagnosis

A. Choroidal melanoma

B. Metastatic carcinoma

C. Atypical or eccentric disciform scar associated with age-related macular degeneration

D. Congenital hypertrophy of the RPE

E. Other tumors of the retina or RPE

III. Describe patient in terms of treatment and follow-up

A. Describe the natural history, outcome and prognosis

1. Choroidal nevi are distinguished from choroidal melanoma by relatively small size/thickness and by a natural history without documented change in clinical appearance
 - a. Nevi may grow very slowly over long periods of time and change slightly in surface pigmentation
2. Majority of nevi are stationary
3. Some choroidal nevi develop a choroidal neovascular membrane, drusen, or overlying RPE changes; these do not suggest malignant transformation
4. A small number of choroidal nevi undergo malignant transformation. Findings concerning for transformation include:
 - a. Increase in basal dimensions
 - b. Increase in thickness

- c. Development of associated subretinal fluid
 - d. Development of orange pigmentation
 - e. Development of visual symptoms or a decline in acuity
5. Patients who develop any of these features described above should be referred to a retinal specialist for consideration of treatment

B. Describe medical therapy options

- 1. Typical choroidal nevi should be drawn or photographed, with clinical estimates of basal dimension and height documented
- 2. Fundus photography is useful for serial comparison
- 3. Patients with atypical choroidal nevi should have A- and B-scan ultrasound to document basal dimensions and thickness and to exclude internal sonographic features which are more typical for choroidal melanoma
- 4. Atypical features mandate referral to a retina specialist

IV. Describe appropriate patient instructions

A. Patient should return periodically for assessment of nevus stability

Additional Resources

- 1. AAO, Basic Clinical and Science Course. Section 4: Ophthalmic Pathology and Intraocular Tumors, 2015-2016.
- 2. AAO, Laser Photocoagulation of the Retina and Choroid, 1997, p. 242-243.

Lens-associated uveitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Immune reaction to lens material
 - a. Phacoantigenic - rupture of the lens capsule
 - b. Phacolytic - leakage of lens protein through an intact capsule
 - i. Lens protein and swollen macrophages block the trabecular meshwork

B. Define the relevant aspects of epidemiology of this disease

1. Phacoantigenic
 - a. Rupture of the lens capsule following trauma or surgery
 - b. Retained lens fragments after cataract extraction
2. Phacolytic
 - a. Occurs in setting of mature or hypermature cataract

C. List the pertinent elements of the history

1. Phacoantigenic
 - a. History of trauma or intraocular surgery
 - b. Sudden or insidious onset of decreased vision, pain, redness, photophobia
2. Phacolytic
 - a. Progressive loss of vision due to gradually worsening cataract
 - b. Sudden or insidious onset of decreased vision, pain, redness, photophobia

D. Describe pertinent clinical features

1. Phacoantigenic
 - a. Anterior uveitis
 - i. Granulomatous or nongranulomatous
 - ii. Mild to severe
 - iii. Keratic precipitates; small, punctate, or mutton-fat
 - iv. Posterior synechiae
 - v. Hypopyon in some
 - b. Retained lens material in anterior chamber and angle possible
 - c. May have elevated intraocular pressure
 - d. Vitreous cells often present
2. Phacolytic
 - a. Large, refractile cells in the aqueous (lipid containing macrophages)
 - b. May have elevated intraocular pressure
 - c. Corneal edema possible
 - d. Lens capsule may appear wrinkled
 - e. Keratic precipitates rare
 - f. Posterior synechiae rare

E. Describe appropriate testing and evaluation for establishing the diagnosis

1. Phacoantigenic
 - a. History and clinical exam paramount
 - b. B-scan ultrasonography
 - i. Useful if lens material located in vitreous
 - c. Histopathology of lens material
 - i. Zonal granulomatous inflammation centered about the site of injury. Neutrophils with the lens material surrounded by lymphocytes, plasma cells, epithelioid cells, and macrophages
2. Phacolytic
 - a. Anterior chamber paracentesis may be considered
 - i. Aqueous cytology may reveal swollen macrophages

II. Define the risk factors

A. Phacoantigenic

1. Trauma
2. Cataract or glaucoma surgery

B. Phacolytic

1. Advancing age
2. Dense cataract

III. List the differential diagnosis

- A. Postoperative endophthalmitis (phacoantigenic)**
- B. Traumatic iritis (phacoantigenic)**
- C. Other anterior or intermediate uveitides**
- D. Posner-Schlossman syndrome (phacolytic)**
- E. Sympathetic ophthalmia**

IV. Describe patient management in terms of treatment and follow up

A. Describe medical therapy options

1. Phacoantigenic
 - a. Corticosteroids
 - i. Topical or regional. Severe cases may require systemic therapy
 - b. Cycloplegics
 - c. Aqueous suppressants to control intraocular pressure
2. Phacolytic
 - a. Aqueous suppressants to control intraocular pressure
 - i. Additional topical and/or systemic therapy may be required to lower the intraocular pressure
 - b. Topical corticosteroids

B. Describe surgical therapy options

1. Phacoantigenic

- a. Cataract extraction is typically curative and should be performed as soon as possible
- b. In cases with retained lens material
 - i. Removal of all residual lens material in most cases, consider vitrectomy for cases of lens material in posterior segment.
 - ii. Patients with very small amounts of lens material may improve with corticosteroid therapy alone
- 2. Phacolytic
 - a. Cataract extraction as soon as possible

V. Describe disease-related complications

- A. Pupillary membrane**
- B. Glaucoma**
- C. Corneal edema and decompensation**
- D. Cystoid macular edema**

VI. Describe appropriate patient instructions

- A. Use medications as prescribed**
- B. Adhere to postoperative instructions**
- C. Report changes in vision and/or pain immediately**

Additional Resources

- 1. AAO, Basic and Clinical Science Course. Section 9: Intraocular Inflammation and Uveitis, 2015-2016.
- 2. Michel SS, Foster CS. Lens-Induced Uveitis. In: Foster CS, Vitale AT, eds. Diagnosis and Treatment of Uveitis. Philadelphia: W.B. Saunders; 2002:817-21.

Chronic or delayed onset pseudophakic uveitis (P. acnes)

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of the disease

1. *Propionibacterium acnes* or other slow-growing organism

B. List the pertinent elements of the history

1. Late onset (months to years) after cataract surgery
2. Blurred vision
3. Recent laser capsulotomy

C. Describe pertinent clinical features

1. Anterior chamber flare or cells, may rarely result in hypopyon
2. Granulomatous or fine keratic precipitates
3. Capsular plaque
4. Inflammation becomes recalcitrant to increasing frequency of topical steroids

D. Describe appropriate diagnostic testing for establishing the diagnosis

1. Vitreous and anterior chamber "taps" (aspirations) for culture and cytology
2. Capsulectomy for culture

II. Define the risk factors

A. Posterior capsulotomy

III. List the differential diagnosis

A. Inflammation due to retained lens fragments

B. Other infectious or non-infectious causes of uveitis that may fit the particular patient's demographic and clinical features

IV. Describe patient management in terms of treatment and follow-up

A. Describe medical therapy options

1. Intraocular antibiotics
 - a. Culture dependent
 - b. May need combined therapy

B. Describe surgical therapy options

1. Pars plana vitrectomy
2. Partial or total capsulectomy
3. Removal of intraocular lens (IOL) may be needed in select cases

V. List the complications of treatment, their prevention and management

- A. Complications of intraocular antibiotics
- B. Complications of vitreous tap/vitreotomy surgery
- C. Complications of capsulectomy and/or IOL removal

VI. Describe disease-related complications

- A. Posterior synechiae and peripheral anterior synechiae
- B. Glaucoma
- C. Cystoid macular edema

VII. Describe appropriate patient instructions

- A. Medication instructions
- B. Follow-up instructions

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 9: Intraocular Inflammation and Uveitis, 2015-2016.

Syphilitic panuveitis

I. Describe the approach to establishing the diagnosis

A. Describe the etiology of this disease

1. Sexually transmitted multi-systemic disease caused by the spirochete *Treponema pallidum*
2. Acquired and congenital forms

B. Define the relevant aspects of epidemiology of the disease

1. Incidence
 - a. Increased risk among patients engaging in high risk sexual behavior and those with other STDs and acquired immune deficiency syndrome (AIDS)

C. Describe pertinent stages and features of systemic disease

1. Acquired disease
 - a. Uveitis is rare in secondary and tertiary syphilis
2. Ocular manifestations
 - a. Protean: may affect all ocular structures
 - b. Uveitis most common

D. List the pertinent elements of the ocular history

1. Onset weeks to years after primary systemic disease
2. Sudden or insidious
3. Variable severity
4. Variable pain, redness and photophobia
5. Blurred vision or floaters
6. May be bilateral

E. Describe pertinent ocular features

1. Interstitial keratitis
2. Nodular or diffuse anterior scleritis
3. Mild to severe iridocyclitis
4. Cystoid macular edema
5. Vitritis
6. Diffuse or localized choroiditis or chorioretinitis
7. Anterior or posterior uveitis
8. Mild to severe iridocyclitis
9. Lens dislocation

F. Describe appropriate testing and evaluation for establishing the diagnosis

1. Refer for appropriate serologic testing

II. List the differential diagnosis

A. Syphilis is the "great masquerader"

1. Retinitis pigmentosa
2. Necrotizing herpetic retinopathies (acute retinal necrosis, progressive outer retinal necrosis)

3. Cytomegalovirus retinitis
4. Tuberculosis
5. Lyme disease
6. Sarcoidosis
7. Toxoplasmosis (especially in AIDS patients)
8. Neuroretinitis

III. Describe patient management in terms of treatment and follow-up

A. Penicillin treatment by appropriate subspecialist

1. Referral to internist or an infectious disease specialist

IV. Describe appropriate patient instructions

A. Importance of long term serologic monitoring

B. Counseling for high risk sexual behavior

C. May be reportable disease to public health agencies

Additional Resources

1. AAO, Basic and Clinical Science Course. Section 9: Intraocular Inflammation and Uveitis, 2015-2016.
2. Aldave AJ, King JA, Cunningham ET, Jr. Ocular syphilis. *Curr Opin Ophthalmol* 2001;12:433-441.
3. Browning DJ. Posterior segment manifestations of active ocular syphilis, their response to a neurosyphilis regiment of penicillin therapy, and the influence of human immunodeficiency virus status on response. *Ophthalmology* 2000;107:2015-2023.
4. Centers for Disease Control and Prevention. Sexually transmitted diseases treatment guidelines 2002. *MMWR* 2002;51 (No. RR-6).
5. Gass JD, Braunstein RA, Chenoweth RG. Acute syphilitic posterior placoid chorioretinitis. *Ophthalmology* 1990;97:1288-97.
6. AAO, Focal Points: Infectious Posterior Uveitis, Module #3, 2005.

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