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Scottsdale, AZ

CHALLENGING CASES: FRONT TO BACK

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Disclosure

- Presenter is on speakers panel of Alcon, Allergan, JJ, Bausch + Lomb, TearLab, OcuSoft, Kala, Bruder, Reichert, Eyevance, Novartis, Sun Pharma
- OCCRS-Past President
- Presenter has NO financial interest in any products mentioned

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MY LIFE!

- 67 year old male
- -2.50 +1.75 X 065 20/20 OS
  - Previous history of cataract surgery
  - Left with undesirable astigmatism
- Consider PRK
  - Pre-op evaluation

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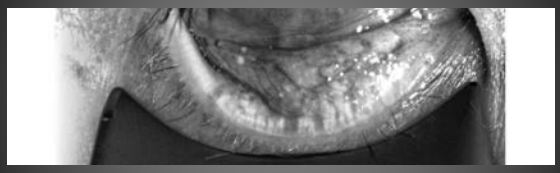
Lid Structure-Lipiview



OS

A Lipiflow treatment was scheduled and patient treated prior to PRK

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More Surprises!

- 1-day post-op
  - VA 20/80 with BCL
  - Treatment
    - BCL
    - Pred Forte qid/Besivance tid/Prolensa qd/Restasis bid/Tears..

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Painful and Red So…

- 1 week post-op
  - Patient complains that vision is worse
  - Tearful and red
  - Continues to use drops…
- What I expect:

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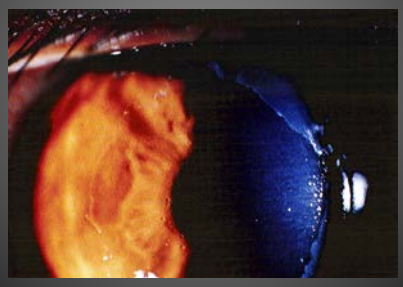
Less than 24 hours post operative PRK

~15% healed



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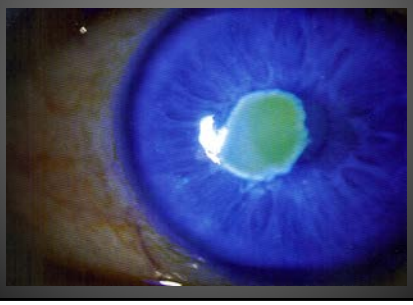
72 hours post operative PRK

~70-80% healed



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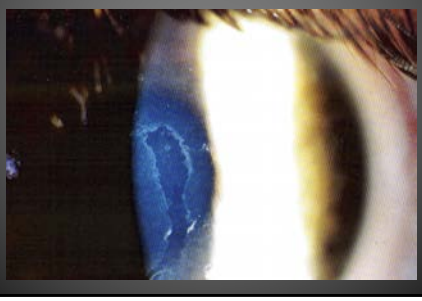
4 days post operative PRK –

90% healed



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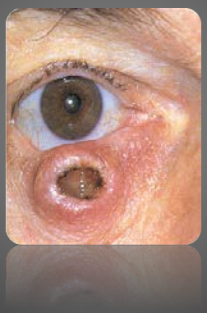
Not This…

10

Never gonna give you up..



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Eyelid Neoplasms

- May arise from epidermis, dermis or eyelid adnexal structures
  - Keratinizing epidermis
  - Prominence of sebaceous glands and blood vessels
- Epidermal origin most common
- Main goal: identify and diagnose malignancy

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Benign or Malignant?

- Most periocular epithelial lesions non-malignant
- Clinical judgment < 100% accurate
- Whenever in doubt -> BIOPSY:
  - absolutely necessary for the definitive Dx

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Clinical Evaluation: History

- Hx prior cancer
- Sun exposure
- Past radiation
- Smoking
- Skin type

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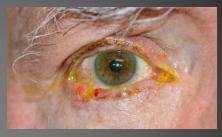
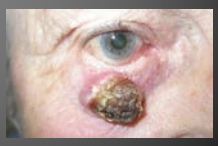
Clinical Signs

- Slow painless growth
- Ulceration, bleeding, crusting
- Irregular pigmentary changes
- Destruction of normal architecture
  - Lash loss, meibomian orifices
- Pearly edge, central ulceration
- Telangectasia
- Loss of cutaneous wrinkles





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Other Clinical Signs

- Palpable induration: infiltration into dermis, subcutaneous tissue
- Lesions near punctum: possible lacrimal invasion
- Fixation to deeper tissues/bone
- Lymph nodes
- Restricted EOM, proptosis: orbital invasion

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Final & Definitive Diagnosis

BIOPSY

- Incisional – when we suspect a malignant lesion
  - Shave Bx
  - Punch Bx

- Excisional - ideal when we suspect a benign lesion
  - Margins are not checked

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Keratoacanthoma

- Self-healing carcinoma
  - Pseudo carcinoma
  - Rapid enlargement
    - Different than SCC
- Sun damage may lead to this
  - Predominantly in elderly patients >45 y.o.
- Starts as pimple or boil
- M>F
- Involutes spontaneously
  - Excision often performed

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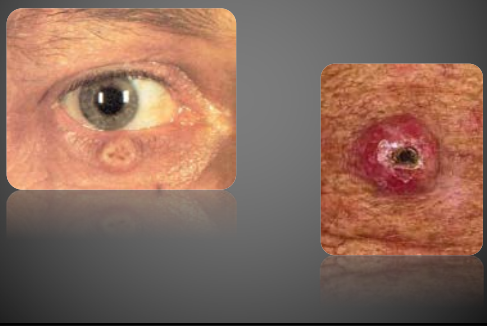
Keratoacanthoma



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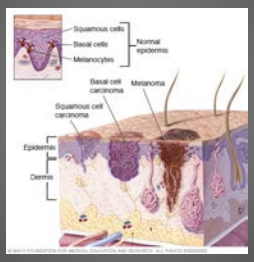
Basal Cell Carcinoma (BCC)



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Basal Cell Carcinoma (BCC

- 90-95% of eyelid malignancies
- Most common malignant tumor of the eyes
- Arise from hair-bearing skin
- Cystic type resemble a benign inclusion cyst
  - Fibrosing difficult to Dx
    - Lie beneath and lose lashes
    - Entropian/ectropian
    - Lid notch/retraction/chalazia
    - Chronic blepharitis

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Basal Cell Carcinoma (BCC)

- Location
  - LL: 50-60%
  - MC: 25-30%
  - UL: 15%
  - LC: 5%
- Hx: fair skin, sun exposure, smoking, prior BCC
- Forms: nodular, morpheaform
- Rarely metastasize
  - Recurrent or neglected may invade orbit and need exenteration

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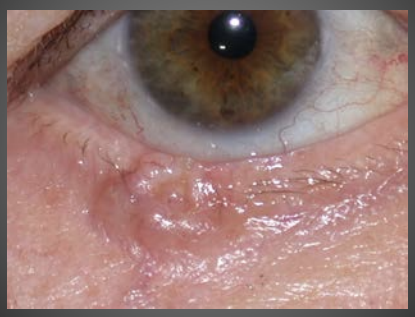
Nodular BCC



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Infiltrative BCC

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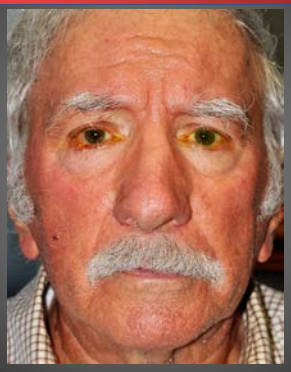
Clinical Case



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Ulcerated lid margin lesion

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After Excision

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2 days PO

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2 mo PO

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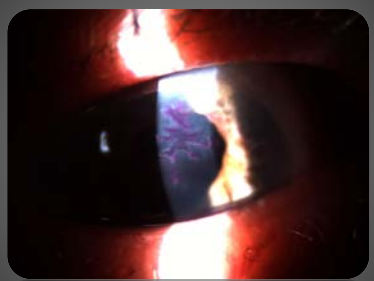
This ain't no disco..



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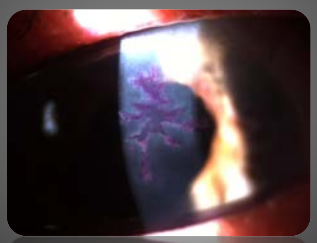
This ain't no fooling around…



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More Surprises

- Vasc 20/100; ph NI
- Started Zirgan q5x's day
  - Considered an amniotic membrane
  - Remarkable healing
- D/C Steroids
- Continue NSAID
- Artificial tears

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Other Options

- Amniotic Membrane
  - Dry
  - Cryopreserved
- Neurotrophic?
  - Oxervate

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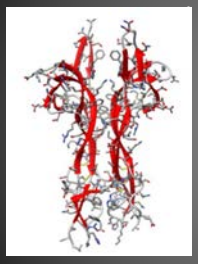
Active ingredient structurally identical to human nerve growth factor produced in ocular tissues



Naturally occurring neurotrophin is responsible for differentiation, growth, and maintenance of neurons[1]

The regenerative potential of nerve growth factor (NGF) was discovered by Nobel-prize winning scientists in the early 1950s[1]

Cenegermin-bkbj, a novel recombinant human nerve growth factor (rhNGF), is **STRUCTURALLY IDENTICAL** to the NGF protein[2]

34 Bonini A, Rama P, Bonini S, Caprioglio G, Aloe L. Topical treatment with nerve growth factor for corneal neurotrophic ulcers. Arch Ital Biol 1998;136:157-63. 2. Voelker R. New Drug Treats Rare, Debilitating Neurotrophic Keratitis. JAMA. 2018;320(16):1656.

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(cenegermin-bkbj) ophthalmic solution 0.002% Weekly Device Kit

- OXERVATE™ is supplied in a weekly carton containing 7 multiple-dose vials*
- A separate weekly Delivery System Kit contains the supplies needed to administer treatment

The Delivery System Kit Contains:

- 7 vial adapters
- 42 pipettes
- 42 sterile disinfectant wipes
- 1 dose recording card
- 1 extra adapter, 3 extra pipettes, 3 extra wipes are included as spares

**Extra drug is available in each vial to take into consideration for loss or spillage during treatment administration*

35 OXERVATE™ (cenegermin-bkbj) ophthalmic solution 0.002% (20 mcg/mL) [US package insert]. Boston, MA: Dompe U.S. Inc.; 2018.

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More Surprises

- After 3 weeks the cornea healed
  - No scarring
  - No haze
  - Just 20/100 Vasc!
    - Now what!??

36

Rule out Abnormal

- POST PRK
  - Check topography
- POST Cataract
  - 5 C's!
    - Correction
    - Corneal surface
    - Capsule
    - Centration
    - CME

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So what now?

- Correction
  - -0.25 +0.50 X090 20/100
- Corneal Surface
  - Mild staining, tear miniscus low, Osmolarity: 290
- Capsule
  - Open
- Centration
  - Centered

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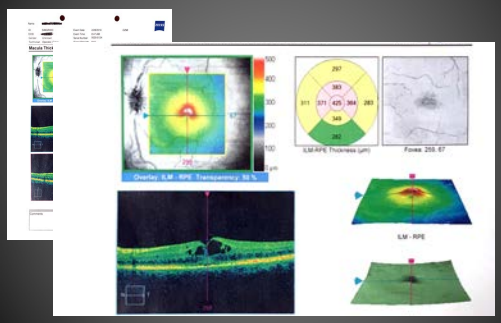
CME



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Don't be surprised!

- Occam's Razor
- Möbius strip
- Manage the disease at hand

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SF CASE

- 68 year old male
- Presents with c/o flashes floaters OD x 2 days
  - No pain
  - No change in acuity
- Med hx: Type 2 DM x 2 years, well controlled; HTN; ED
- Meds: Metformin, HCTZ, Lipitor, Viagra
- Oc Hx: Unremarkable

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SF CASE

- Entering VA: 20/25 OU
- SLE: WNL
- IOP 14 mm OU
- DFE:

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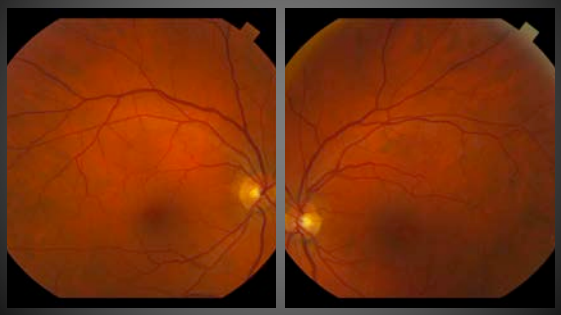
SF CASE



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SF CASE

- Assessment:
  - Acute PVD OD
- Plan:
  - Pt education
    - Signs/symptoms of RD
  - **RTC when?**

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SF CASE

- Really no consensus
- Symptomatic PVD without retinal break
  - AOA:1-2 weeks
  - **AAO: depending on symptoms, risk factors and clinical finings:**
    - **1-6 weeks**
    - **Then 6 mos to 1 year**
  - Cleveland Clinic: 4-6 Weeks
  - Others: if no heme or other issues, very low risk so no need to see to back

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PVD

- Floaters are typically most common symptom
  - Cobwebs
  - Files
  - Hairs
- Flashes
  - Indicative of traction on retina, but not necessarily a tear or break

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Hello….Mr. Wiess

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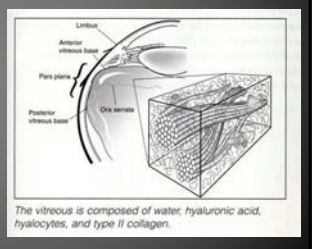
The Vitreous Humor

- Vitreous attached most firmly at
  - Macula
    - VMT
  - Vitreous base
  - Around optic nerve head
    - Weiss' Ring
  - Also, some traction on blood vessels
    - Vit heme



The vitreous is composed of water, hyaluronic acid, hyalocytes, and type II collagen.

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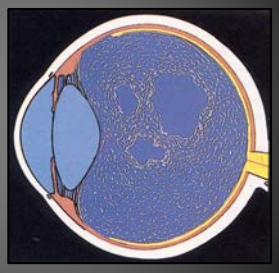
Physiologic Changes

- With age, liquifaction due to reduction in hyaluronic acid causes loss of support.
- This process is referred to as synchesis.



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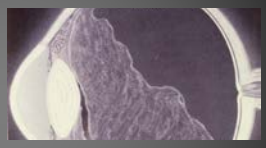
Physiologic Changes

- Vitreous shrinkage, contraction and collapse can cause traction.
- This process is referred to as syneresis.



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Incidence of PVD

Age	Incidence
>30	RARE
30-59	10%
60-69	27%
>70	63%
>80	75%

- 65%>65 HAVE A PVD

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Incidence of PVD

- Incidence may be accelerated by
  - Myopia
  - Trauma
  - Prior vitreoretinal disease
  - Surgery
  - Inflammation
- Symmetrical 90% of the time
- Happens to second eye with 1-2 years

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PVDs

- Good News:
  - Retinal Tears/Breaks *Relatively* uncommon
    - One study: only 7-15% of symptomatic PVDs have a retinal break
  - 8-26% acute PVDs have an associated RB/RD at the time they present (Ophthalmology AAO 2014)
- Bad news:
  - 7-15% have a retinal break
  - The chances of RB there after is <2-5%

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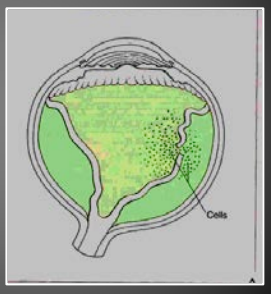
Risk Factors

- Pigment
  - Schaeffer's Sign
    - Indicates break is possible
- Hemorhage
  - 90% have break
- Inflammatory cells



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My recommendations

- DFE **WITH SCLERAL DEPRESSION**
- DISCUSS SIGNS/SYMPTOMS OF RD
- RTC 6 WEEKS
  - SEE UNTIL FLASHES SUBSIDE
  - Less than 5% risk with NO Flashes
- IF RISK FACTORS, CONSIDER REFERALL TO RETINA
  - Vitreous heme
  - Pt is Lawyer/father-in-law, etc
  - Just doesn't feel right

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HOLIDAY AT HOLIDAY INN

May I have some more please?

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Are you sure?

- 62 yo w/progressive decrease in DVA in OD
  - "My Dr. told me I have keratoconus starting in my 60's"
  - "I had a bad reaction one morning, painful red eye"
- Refered for CXL
- MedHx: Hpertension,Thyroid Dz, Arthritis
- OMHx: None

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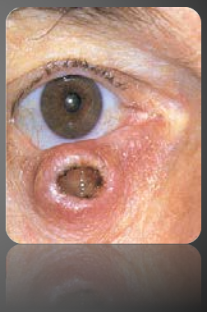
Are you sure?

- BCVA
  - OD: -3.00 +2.50 X 77 20/40
  - OS: -.25DS 20/20
- BAT
  - OD: 20/25/30
  - OS: 20/20/25
- Potential Acuity: 20/20 OD & OS
- SLE :
  - K: OD central opacities, OS clear
  - Lens: NSC-OU
  - Fundus:



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OptoVue Avanti

RULE OUT ABNORMAL

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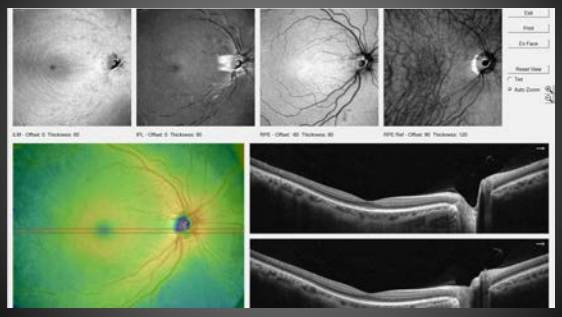
Optovue Avanti-OD



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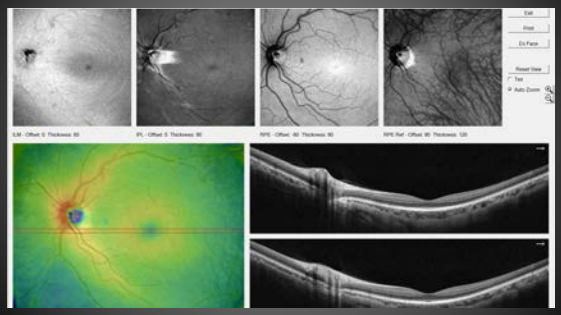
Optovue Avanti-OS



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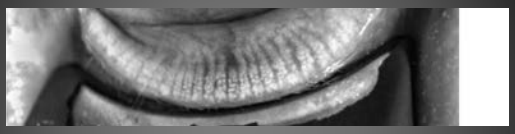
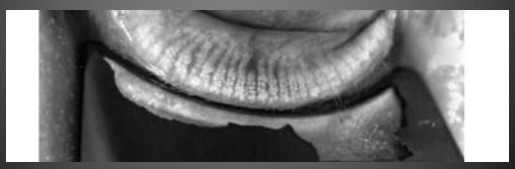
Lid Structure-Lipiview

OD



OS



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Orbscan

TOPOGRAPHY

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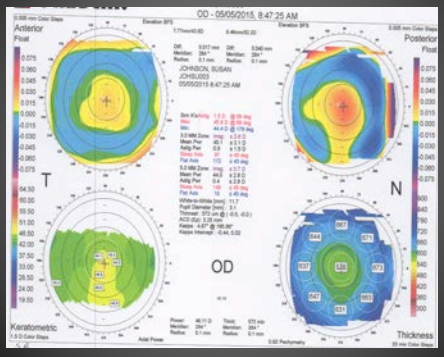
Orbscan OD



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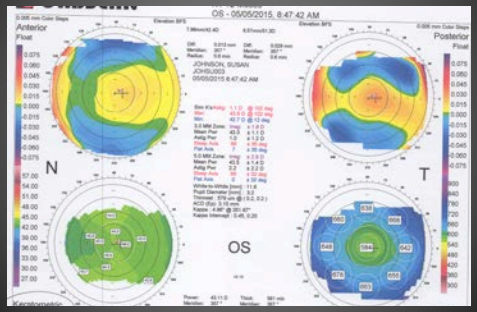
Orbscan OS



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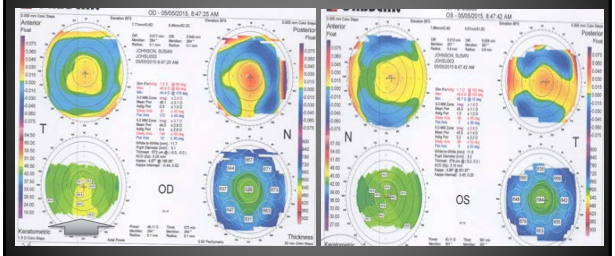
Asymmetry?



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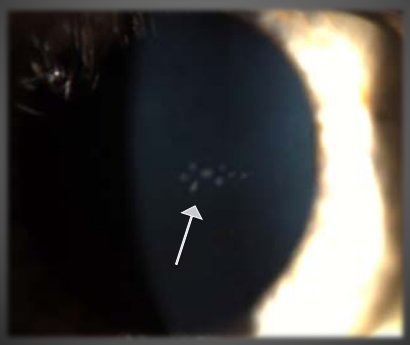
Right Corneal View



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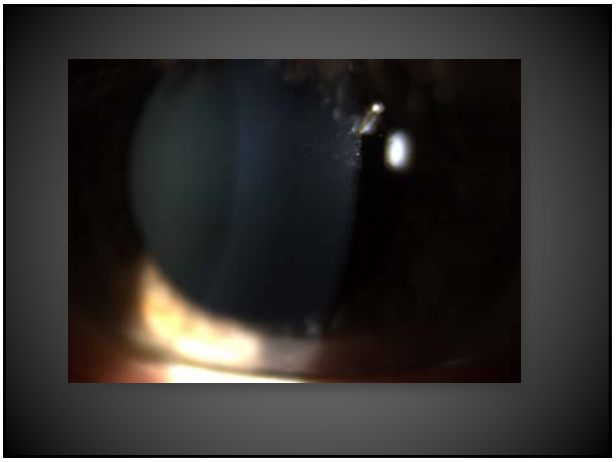

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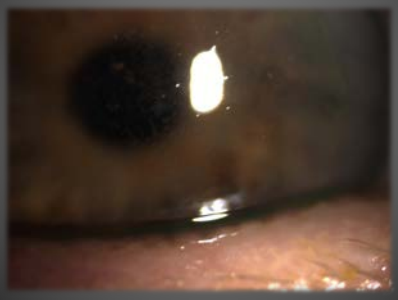
Epithelial Cells



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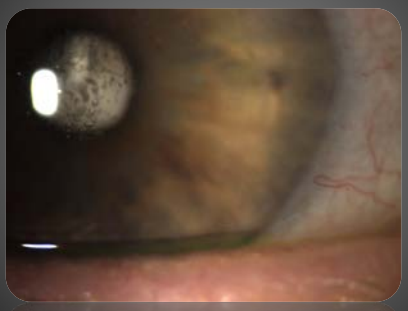
Epithelial Cells



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But why cells??

EBMD

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Hx of RCE

- Plan: RTC in 3 days/Debride Cornea/Insert Prokera Slim
- Plan:
  - Tape tarsoraphy
  - Zymaxid qid/Prolensa qd/Pred Forte qid
    - RTC:
      - 6 days

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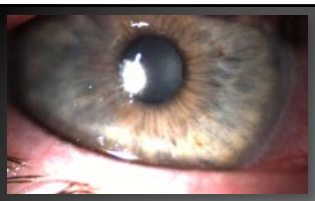

Prokera Removed

- VAcc 20/25
- “My eye feels great”
- Follow-up in 2 weeks
  - Restasis BID
  - Retaine MGD q1h

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CASE

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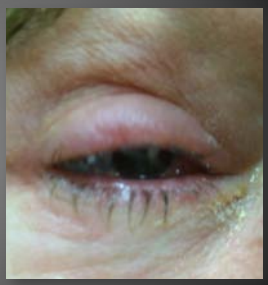
DIFFERENTIAL

- Allergic Conjunctivitis
- Bug Bite
- Bacterial Conjunctivitis
- Viral Conjunctivitis
- Preseptal Cellulitis
- Cellulitis
- Corneal Ulcer
- Foreign Body
- Hot tub
- Trauma



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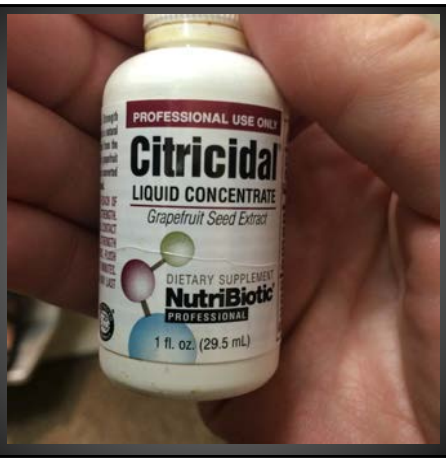

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3 Types of Eye Burns

- Alkali Burns: These burns involve high pH chemicals, and thus are the most dangerous. They are powerful enough to penetrate the eye, and cause damage to its vital inner components. In the worst cases, they can lead to conditions like cataracts and glaucoma and may cause vision loss or blindness.
- Acid Burns: Lower pH burns that are less serious than alkali burns, but still dangerous. These burns are unable to penetrate the eye, but still may cause significant damage to the cornea, with the potential to cause vision loss.
- Irritations: These burns are neutral in pH

79

Symptoms of Chemical Burns

- Eye redness
- Eye irritation
- Eye pain
- Swelling of the eye
- Blurred vision
- Inability to open the eye
- Feeling of foreign objects in the eye

80

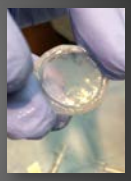
Telephone Triage Tips

- Irrigation process begins on site before the patient seeks care.
- Use shower or hose if outside work place
- Attempt to determine the type of chemical that entered the eye(s).
- Attempt to determine if the patient is wearing contact lenses. Irrigation should not stop in an effort to remove contact lenses.
- A minimum of 20 to 30 minutes before the patient is brought to the office.
- When the patient is ready to make the trip to the ER or office, remind them to bring the container that held the offending chemical. Important information may be obtained from the labeling.
- If the injury occurred in the workplace, ask the patient to bring the MSDS (material safety data sheet) if available.
- If the injury occurred where there is no or limited access to water for irrigation, refer them to the nearest emergency room or your office, whichever is closer.
- Assist with dispatching emergency services as needed.

81

Treatment

- Assess the cornea and conjunctiva
  - Cornea intact-mild SPK
    - Prophylactic Antibiotic
    - Topical Steroid (Lotemax Gel)
    - Preservative Free Tears
    - Cycloplege for Pain
  - Cornea haze/Necrotic
    - All the above
    - Consider debridment
    - Sodium ascorbate drops (10%) Q1H while awake
    - Vitamin C-1000mg/day
    - Prokera



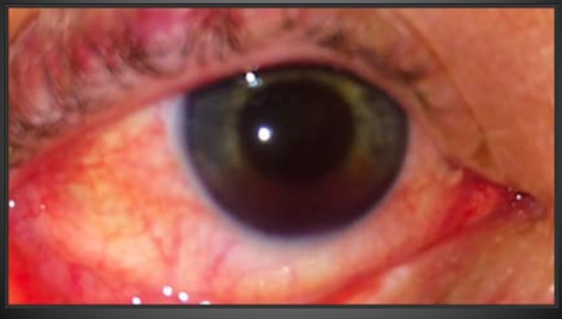
82

Batter Up!-Let's Go Dodgers (2022)

- 20-year-old collegiate baseball player was hit in the right eye when the ball was deflected off the bat.
- The athlete bled from the nose, and the right eye swelled shut from eyelid edema.
- Initial nasal hemorrhage was controlled

83

Patient Presents to you…



84

Hyphema

- *Microhyphema* is the term used when RBCs are in the anterior chamber but haven't settled inferiorly
- *Hyphema* is the name given once blood settles inferiorly in the anterior chamber
- Most commonly the result of blunt trauma to the globe
  - Force causes blood vessels of the iris or ciliary body to break

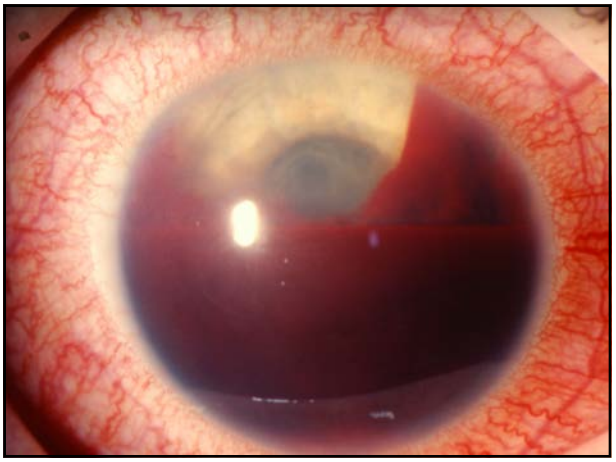
85

86

Traumatic Hyphema

- Check periorbital area and globe for injuries
  - Vision, anterior chamber depth, Seidel's sign, IOP, broken facial bones, EOMs, APD, lens, iris, retina, etc.
  - May need CT of orbit/face, B-scan, retinal consult
- Any sign of an open globe is an emergency requiring placement of shield and immediate referral

87



88

Hyphema Management

- Watch daily until resolved/controlled
- Start steroid q2h to qid
- Homatropine/cyclogel bid to tid
- Control IOP if elevated to avoid corneal blood staining
  - Larger hyphema = larger risk of increased IOP
  - Use brimonidine, beta-blockers first-line
  - Avoid CAIs if risk/known sickle cell
  - Avoid prostaglandins when possible

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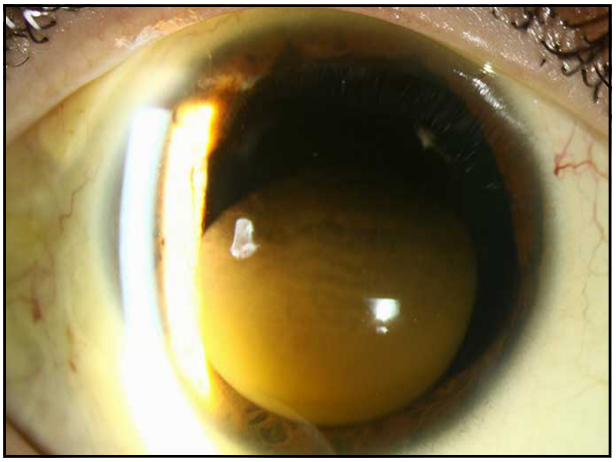

90

Hyphema Management

- Consider referral if:
  - Unable to control IOP
  - Corneal blood staining develops
  - Continued bleeding without clotting
  - Sickle cell patient
  - Spontaneous hyphema of unknown etiology
- Surgical treatment
  - Anterior chamber washout most common
  - Paracentesis for IOP control
  - Trabeculectomy with AC washout
  - Yag PI if pupillary block develops

91

Hyphema Counseling

- Counseling
  - Limit activities
  - Highest risk of rebleed in first 5 days
  - Keep head of bed elevated
  - No ASA/IB products/blood thinners if possible
  - Long-term glaucoma risk (75% will have angle recession)
    - Gonioscopy 3-6 week post-resolution
    - Baseline VF

92

Post-surgical Hyphema

- Post-peripheral iridotomy
- Following cataract surgery
  - MIGS
  - Fuchs heterochromic iridocyclitis (FHI)
- UGH syndrome
  - Uveitis-glaucoma-hyphema syndrome
  - History of ACIOL or poorly positioned PCIOL

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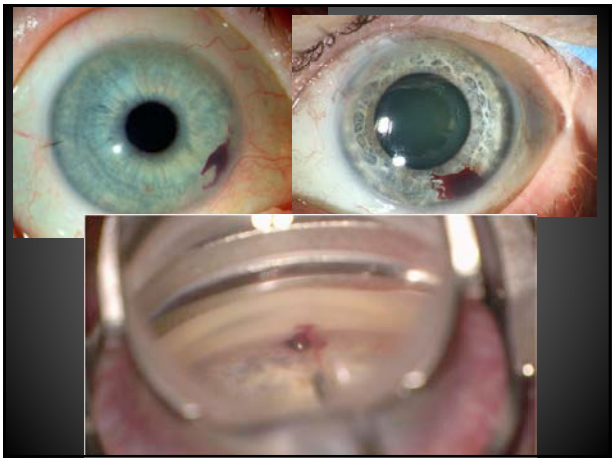


94

Spontaneous Hyphema

- Neovascularization of the Iris
  - PDR/Ocular Ischemic Syndrome (OIS)
- Sickle Cell disease and Sickle Cell Trait
- Ocular Melanoma/Retinoblastoma
- Herpetic uveitis/FHI
- Leukemia/hemophilia
- Anticoagulant use
- Others

95

Case

Glaucoma is my thing…

96

48 YO HF

- Diagnosed with POAG 1995
- Diagnosis made by ophthalmologist in Minnesota
- Relocated to Phoenix, I assume care
- Untreated peak IOP
  - OD=27mm Hg
  - OS=29mm Hg

97

48 YO HF
Treatment History

- Initial therapy Timolol 0.5%
  - Discontinued after 2 months
  - Side effects of bradycardia & fatigue
- Current Medical Regimen:
  - Xalatan ou qPm x 2 yrs

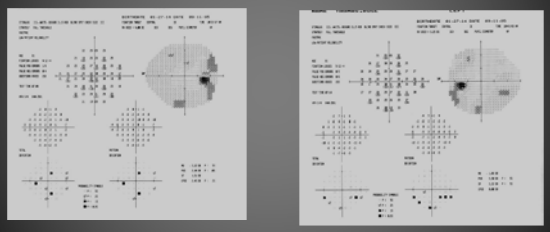
98

48 YO HF

- Since starting Xalatan IOP readings:
  - OD=17-19mm Hg
  - OS=18-20mm Hg
- Previous doctor felt that patient was being "safely" treated at this IOP level.

99

48 YO HF
VF 2 yrs ago



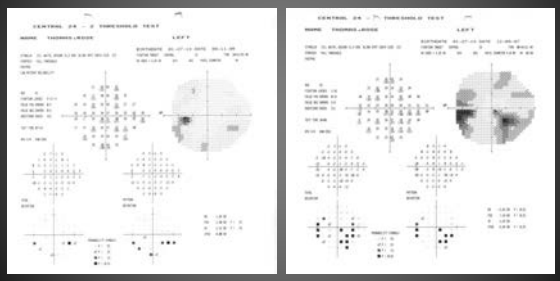
100

48 YO HF
Initial Exam in Phoenix

- BCVA 20/20 OU
- - RAPD
- IOP OD=17mm Hg OS=18mm Hg
- Subjectively, the patient
  - Reports excellent compliance
  - Denies any side-effects

101

Visual Field Progression
in upper teens



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48 YO HF
Left Eye: Phoenix ancillary tests

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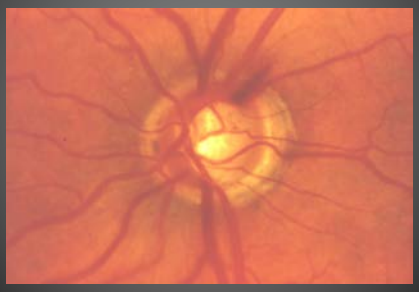
Disc hemorrhage suggests
Upper teens are NOT low enough



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WHAT WOULD YOU DO NEXT?

105

ORA Reichert

MORE TESTING IS NEEDED

106

Understand the Cornea,
Understand the Pressure

Corneal Biomechanics and Accurate IOP in One Simple Instrument

107

Ocular Response Analyzer®

108

Key Features

- ORA is the only device in the world capable of measuring Corneal Hysteresis (CH), which is an indicator of bio-mechanical properties of the cornea (tissue properties)
  - Is it "strong" or "weak", "tough" or "soft"
- **CH is *independently* predictive of visual field progression, which helps clinicians make important treatment decisions earlier**
  - *CH is superior to Pachymetry (CCT) as a glaucoma risk assessment tool*
- **CH facilitates our "corneal compensated" IOP (IOPcc): an IOP measurement that is less influenced by corneal properties than other tonometers, including Goldmann.**
  - *This is superior to CCT-based IOP adjustment, which has been discredited*
- There are over 425 peer-reviewed publications about ORA in the literature
- ORA is fast, objective, non-contact, and operator friendly

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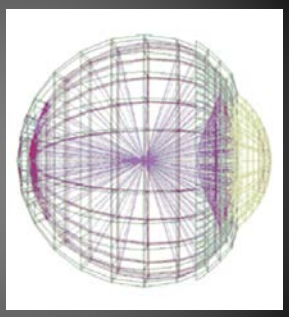
Bioengineering of the Eye

- Viscoelastic tissue with complex, interconnected microstructure[1]
- Geometrical attributes are not a surrogate for biomechanical properties[1]
  - eg: CCT does not describe viscoelasticity
- The eye appears to be a mechanical structural continuum[2]
  - Tissue properties may provide additional diagnostic information[3]



©2002 Park et al. *Invest Ophthalmol Vis Sci.*
1. Glass DH et al. *Invest Ophthalmol Vis Sci.* 2008;49:3919-3926.
2. Taylor DA et al. Corneal Biomechanics. In: Copeland RA Jr., Afshari NA, eds.: *Copeland and Afshari's Principles and Practice of Cornea. Two Volume Cornea Textbook.* Jaypee Brothers. 2012:148-157.

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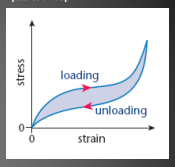
Hysteresis: Not a New Concept

Sir James Alfred Ewing
Identified the phenomenon of hysteresis and coined the term in 1890

Classic "Hysteresis Loop"



- A measurement that characterizes response to application and removal of force (load/unload)[1]
  - Found in materials or systems that do not instantly follow forces applied to them but react slowly, *or dissipate a portion of the applied energy* [1]
- More than 7500 papers published on hysteresis in a variety of medical fields[2]
  - Various tissues and structures (tendon, lung, arteries, etc)
  - The importance of Corneal visco-elasticity had been discussed and explored (*EX-VIVO*) prior to the ORA[3]

1. Vincent J. Basic elasticity and viscoelasticity. In: Vincent J, ed. *Structural Biomaterials.* 3rd ed. Princeton, NJ: Princeton University Press; 2012:1-28.
2. PubMed Search for "hysteresis" on October 3, 2014 returned 7696 results.
3. Hjortdal JO1. On the biomechanical properties of the cornea with particular reference to refractive surgery. Acta Ophthalmol Scand Suppl. 1998;(225):1-23.

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Basic Parameters

ORA
- **IOPG** - Goldmann Correlated IOP
- **IOPCC** - Corneal Compensated IOP
- **CH** - Corneal Hysteresis
- **CRF** - Corneal Resistance Factor

7CR
- **IOPG** - Goldmann Correlated IOP
- **IOPCC** - Corneal Compensated IOP

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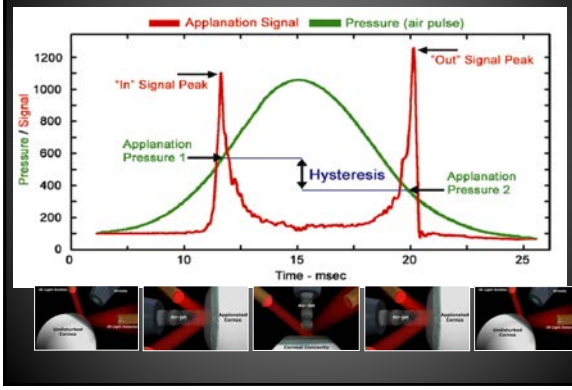
Signal Plot



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Normal CH values - Summary of published results

Authors	Ave CH
Kirwan, O' Keefe (Ireland)	10.8 ± 1.5
Shah et al. (UK)	10.7 ± 2.0
Ortiz (Spain)	10.8 ± 1.5
Hager et al. (Germany)	10.6 ± 2.3
Touboul et al (France)	10.26
Lam et al. (China)	10.9
Fontes, et al (Brazil)	10.17 ± 1.82
Ehongo (Belgium)	10.9 ± 1.3
Gonzalez-Meijome (Portugal)	11.4 ± 1.5
Kamiya (Japan)	10.2 ± 1.3
Carbonaro (UK)	10.24 ± 1.24
Montard (France)	10.25 ± 1.6
Kida et al (USA 50-80 adults)	10.4 ± 1.1
Kida et al (USA Young Adults 20-26)	11.8 ± 1.6
Song (Chinese Children)	10.7 ± 1.6
Lim et al (Singaporean Children)	11.8 ± 1.55
Kirwan, O' Keefe 2008 (Irish Children)	12.5 ± 1.35

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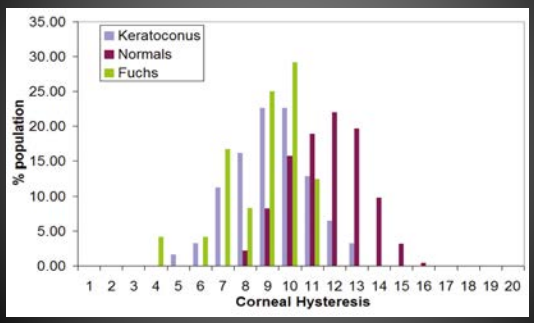
CH in Corneal Pathologies



Data courtesy Shah, Brandt, Pepose, Castellano

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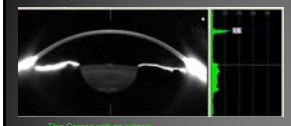
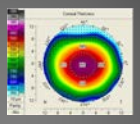
CH in Corneal Pathologies

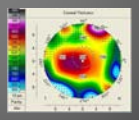


Thin Cornea with no ectasia



CCT 455
CH 11.2
CRF 10.8

Thin Cornea with Keratoconus



CCT 500
CH 8.1
CRF 7.9

Data courtesy Renato Ambrosio, MD - ASCRS 2006

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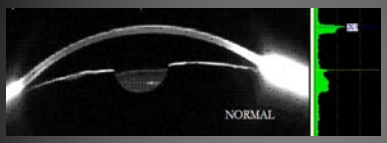
CH in Corneal Pathologies



CCT 605
CH 11.3
CRF 10.7

CCT 597
CH 8.4
CRF 7.2

Data courtesy Renato Ambrosio, MD - ASCRS 2006

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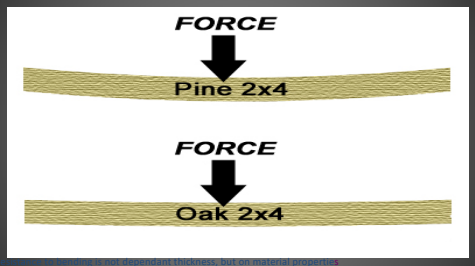
Why CCT-based IOP correction is flawed



Corneal thickness is not dependant thickness, but on material properties

Thick and thin is WRONG. Think weak and strong

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The Cornea and Glaucoma

Numerous studies, such as the Ocular Hypertension Treatment Study (OHTS) have found that corneal thickness is an independent indicator of glaucoma risk.

More recent research has indicated that the Corneal Hysteresis measurement appears to be be even more powerful in this regard.

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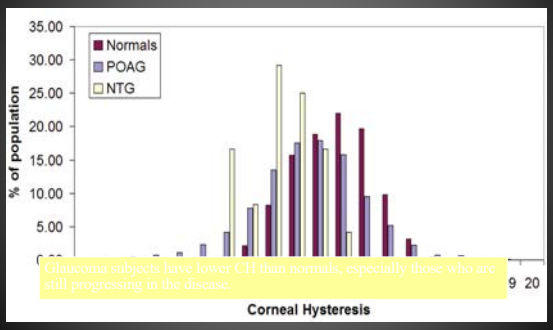
CH distribution - Normals & Glaucoma



Data courtesy New England College of Optometry and Mitsugu Shimmyo, MD

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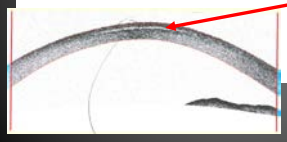
IOPcc Case Example

57 yo post LASIK female
- Complaining of blurry vision and pain in right eye
- GAT: 15 mmHg
- IOPcc: 46 mmHg!!
- OCT image showed fluid under the flap



Patient diagnosed with Angle Closure Glaucoma

Data Courtesy of William Wiley, MD
Cleveland Clinic

137

48 YO HF

- CH OD: 7.3 OS: 6.9
  - IOP g OD: 19 mm Hg IOPcc OD: 25 mm Hg
  - IOP g OS: 17 mm Hg IOPcc OS: 23 mm Hg
- Patient switched from Xal to Lumigan
- IOP at 2 wks after switch
  - IOPcc OD= 12mm Hg
  - IOPcc OS= 13mm Hg
- IOP 3 months after switch
  - IOPcc OD= 14mm Hg
  - IOPcc OS= 11mm Hg

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48 YO HF Summary

- AGIS 7 asserts that IOP reduction correlates with visual field preservation.
- Low teens preserve visual field better than upper teens
- Fewer medications improve compliance

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One more???

142

When it Rains

- 67 year old WM
- "My vision is not good…I have blurred vision. My eyes cry a lot too. They cry all the time."
- +NIDDM (diet controlled 15 years)
- NKMA
- History of skin lesion removed from cheek

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When it Rains

- VACC
  - 20/70 (PH-20/30) OD
  - 20/70 (PH-20/70) OS
- SLEX:
  - Lids: 1+ inspissated glands/turbid expression
    - OS-atrophy notied
  - 2+ NSC/Tr PSC-OD
  - 1 ACC/ 2+NSC/2+ PSC-OS

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My Patients Eye(s)

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LipiScan

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Pre-op

- Patient advised about Lids
  - "See it treat it!"
  - Lipiflow procedure performed
- Scheduled for cataract surgery
  - OD then OS
    - Uncomplicated OD Surgery
    - 1 week post-op 20/25

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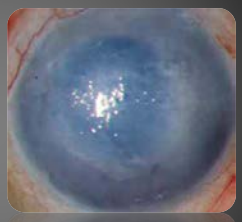
Cataract Surgery OS

- 1-day
  - Vasc: CF
  - 2-3+ Striae
  - 3+ POME
  - 1-2+ cells (tough view)
  - Lens centered
  - IOP-ORA
    - 34 mmHg IOPcc



- Diamox provided, Cosopt Bid, CPM
  - 2 day F/U: CF with 22 IOPcc

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5Day Postop

- Vasc
  - CF "Seems clearer"
- Slex:
  - Cornea: tr edema
  - A/C: 1+Cells with increased flare and deposits in the anterior chamber.
  - Iris –Round with vitreous prolapse
  - Lens: ?
  - 28 IOP cc

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Something is not right?

- CF vision
- Increased IOP
- Corneal Edema
- Complicated surgery with PSC adherence to capsule
  - Open capsule

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What to look for?

- In a retrospective study in AJO 2016:
  - Persistent iritis
  - Increased IOP
  - Corneal edema
    - Retained Lens material
  - Study concluded that delay in cataract diagnosis was the most common factor

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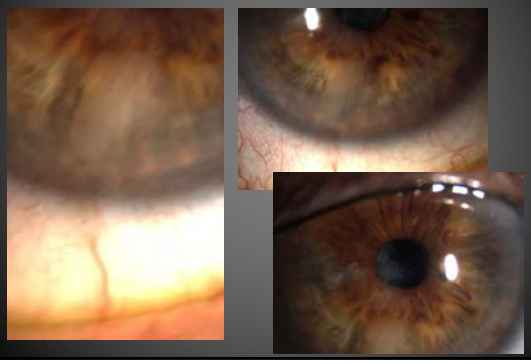
Retained Nucleus



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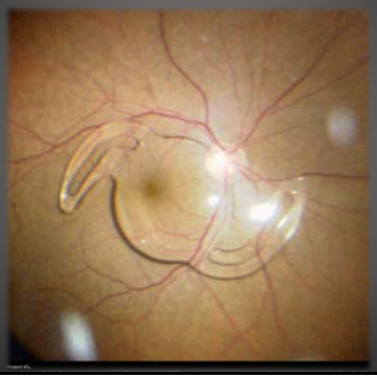
But why CF?



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Case SS

- 71 year old retired Military and secret service agent
- Hasn't had eyes checked in a few years
- VA 20/20 OU with low hyperopic/astigmatic RX
- SLE: mild bleph, trace NSC
- Posterior pole:

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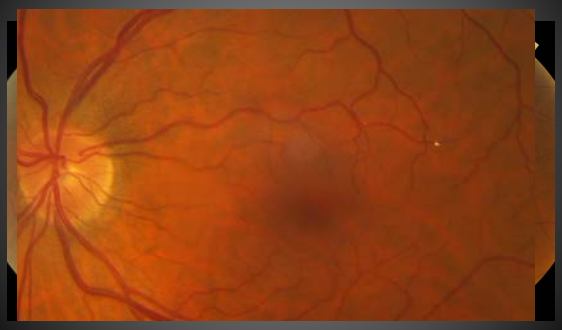
Case



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Case SS

- A: HH plaque OS
- P: refer for carotid doppler
  - Labs
  - refer to PCP for management of other risk factors
  - Vascular clinic dependent on carotid study

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Case SS: Labs

- Labs
  - BP: 134/88
  - Weight: 236
  - BMI: 38.2
  - A1c: 9.9 (H)
  - Triglycerides: 173 (H)
  - HDL: 31.2 (L)
- PCP: diet, education, start insulin

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Case SS

- Carotid:
  - Right: non hemodynamically significant soft calcific plaque at left carotid bifurcation
  - Left: 50-69% ICA Stenosis
- Vascular clinic:
  - Monitor left carotid q 6 mos as no symptoms in last year
  - Start ASA therapy

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Retinal Plaques

- Several different types of plaques can often be visualized in the retinal vasculature
- Pt is typically elderly, has HTN, CAD, hypercholesterolemia/hyperlipidemia, and/or atherosclerotic disease
- Often totally asymptomatic and found on routine exam

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RISK FACTORS

- Age
- HTN
- Vascular disease
- Past vascular surgery
- SMOKING
- High TOTAL cholesterol
- Men> women

185

Prevalence

- Beaver Dam Eye Study: 1.3%
  - smoking, HTN and DM
  - 9x more likely after age 75 vs. 43-54
    - after 75, 3.1% prevalence
    - Equates to 1.2 million people with emboli 43-86
      - » 450,000 are 75-86
    - Fatal stroke 3x as likely over 8 years in pts with emboli, adjusting for other factors
  - OD>OS
  - Bilateral very infrequently

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Prevalence

- Blue Mountain Eye Study1.4%
  - HTN, smoking, Vascular disease
- LA Latino Eye Study: 0.4%
  - Smoking, CAD, h/o MI, HTN
- Singapore Eye Study: 0.6%
  - Smoking, high cholesterol, h/o angina

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Retinal Plaques

- May present with amarosis fugax, transient episodes of monocular blindness
- Rarely, may report transient ischemic attack (TIA) , which is above with hemiparesis, parasthesia or aphasia

188

Retinal plaques

- Three different types of plaques, but all share strong association to significant cardiovascular disease
  - HH 80% > fibrino-platelet 14% > calcific 6%

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Retinal Plaques

- Cholesterol (Hollenhorst) plaque
  - Most common
  - shiny yellow-orange in appearance
  - from plaque in the ipsilateral carotid artery
  - Rarely causes occlusion, unless multiple
  - Typically occurs at bifurcations
  - Mobile in nature

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Cholesterol Plaques

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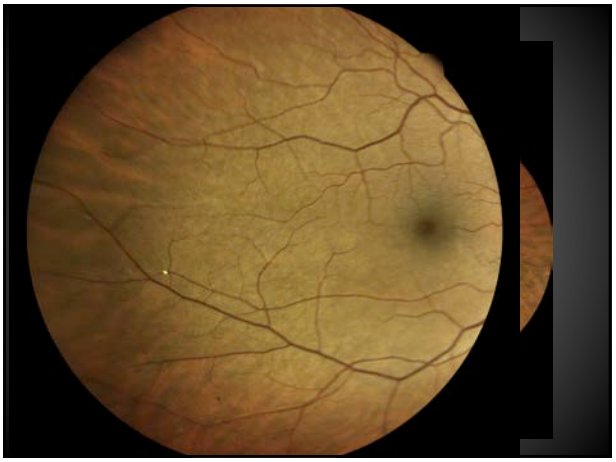

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Retinal Plaques

- Fibrino-platelet
  - Appear as dull white to gray, long plugs
  - Typically within arterioles, not at bifurcations
  - May break-up and dissolve with time
  - May lead to BRAO or CRAO
  - Often associated with carotid disease or mitral valve insufficiency

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Fibrino-platelet Plaques

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Retinal Plaques

- Calcific
  - Appears more whitish than HH
  - Dull, non-reflective, white
  - Classically within arteriole, not at bifurcation
  - Typically immobile
  - Most dangerous, as often cause BRAO
  - Often from cardiac arethromas of heart valves

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Calcific Plaques

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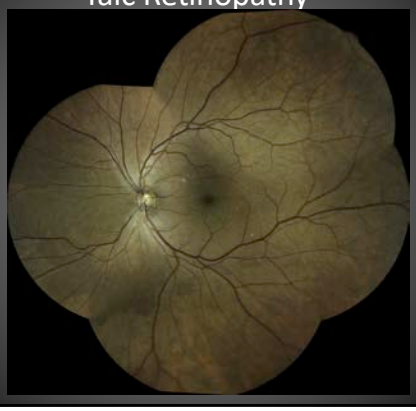
Retinal plaques

- Talc retinopathy
  - Represents an exogenous plaques as opposed to others
  - Appears typically as multiple shiny yellow plaques within capillaries in posterior pole
  - Typically smaller than other plaques
  - Typically seen in IV drug users
  - Rarely cause complications, but reported cases of associated NV and occlusions

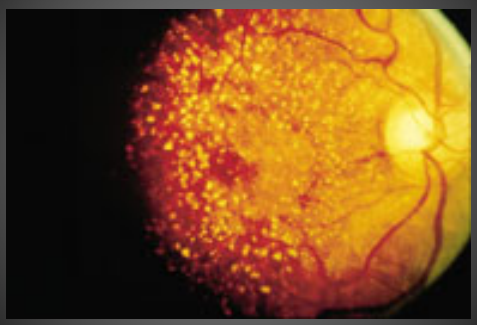
198

Talc Retinopathy



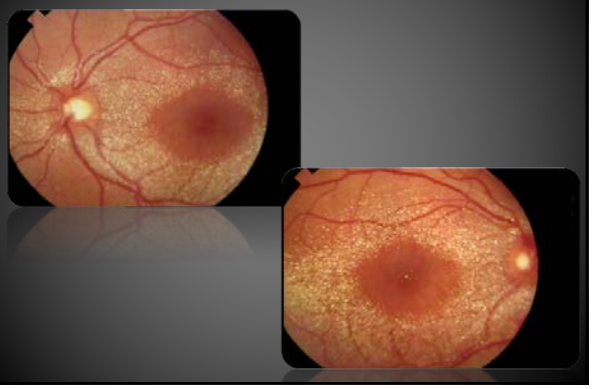
199

Tamoxifen Maculopathy (Nolvadex)



200

Canthanxine Maculopathy



201

Retinal plaques

- No direct management of plaques is needed
- Management is aimed at discovering source of embolus to decrease risk of other emboli, occlusion, or stroke
- Patients need referral to internist for complete physical

202

Retinal Plaques

- Assess risk factors with PCP
  - DN, HTN, lipid panels
- Carotid ultrasound
- MRA: non-invasive image with 2D/3D
- TEE: invasive, probe into esophagus to image heat valves
  - Helpful with calcific
- CTA: CT scan of arteries construct 3D images

203

Carotid Ultrasound

- First line screening test
- ORDER WITHIN TWO WEEKS!!
- Identifies flow rate and % stenosis
- Common, internal, and external
- Only ≈20% of asymptomatic emboli will have significant carotid stenosis

204

Retinal Plaques

<50-60% occlusion

- ORAL TREATMENT
  - Anti-Platelet
    - ASA
  - Anti-coagulation
    - Comadin, platelet
  - Cholesterol meds

>70-99%

- SURGICAL TREATMNET
  - Carotid edarterectomy
  - Angioplasty
  - Reduces risk of future stroke!

205

Retinal Plaques

- Assess risk factors with PCP
  - DN, HTN, lipid panels
- Carotid ultrasound
- MRA: non-invasive image with 2D/3D
- TEE: invasive, probe into esophagus to image heat valves
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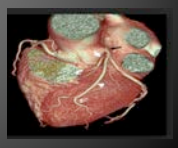

206

Carotid Ultrasound

- First line screening test
- ORDER WITHIN TWO WEEKS!!
- Identifies flow rate and % stenosis
- Common, internal, and external
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Retinal Plaques

- After ruling out underlying etiology, see patient regularly, q 6 -12 mos, to evaluate for additional plaques or other disease associated with vascular disease
  - BRVO/CRVO
  - BRAO/CRAO
  - NTG

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Is it worth working up these patients?

- 18% of pts with retinal emboli had internal or common carotid stenosis>75%
- Higher incidence of stroke
  - 8.5% with emboli vs 0.8% w/o per year
- Pts with cholesterol HH emboli have 15% mortality at 1 yr, 29% by year 3, and 54% by 7 years

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DONE?

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Thanks!

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