# CE in Fort Worth

**CourseMaster**  
Sheila Morrison, OD, MS, FLS

**Program Location**  
Dallas Fort Worth Marriott Hotel & Golf Club  
3300 Championship Parkway, Fort Worth, TX 76177

## SATURDAY, OCTOBER 20th

<table>
<thead>
<tr>
<th>Time</th>
<th>Activity</th>
<th>Lecturer</th>
<th>COPE ID</th>
<th>CEE Available</th>
<th>Credits</th>
</tr>
</thead>
<tbody>
<tr>
<td>7:00 am - 8:00 am</td>
<td>Registration, Continental Breakfast, &amp; Visit Exhibits</td>
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<tr>
<td>8:00 am - 9:45 am</td>
<td>Lectures presented by Blair Lonsberry, MS, OD, Med, FAAO</td>
<td>Diagnosing and Managing Ocular Emergencies and Urgencies</td>
<td>COPE ID # 59063-AS</td>
<td>CEE Available</td>
<td>2 DT</td>
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<tr>
<td>9:45 am - 10:15 am</td>
<td>Break &amp; Visit Exhibits</td>
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<tr>
<td>10:15 am - 12:00 pm</td>
<td>Oral Pharmaceuticals in Anterior Segment Disease</td>
<td></td>
<td>COPE ID # 59064-AS</td>
<td>CEE Available</td>
<td>2 DT</td>
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<tr>
<td>12:00 pm - 1:00 pm</td>
<td>Lunch &amp; Visit Exhibits</td>
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<tr>
<td>1:00 pm - 2:45 pm</td>
<td>Lectures presented by Joe DeLoach, OD, FAAO</td>
<td>Glaucoma: Fact or Fiction</td>
<td>COPE ID # 59075-GL</td>
<td>CEE Available</td>
<td>2 DT</td>
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<tr>
<td>2:45 pm - 3:15 pm</td>
<td>Break &amp; Visit Exhibits</td>
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<tr>
<td>3:15 pm - 4:05 pm</td>
<td>Compliance: Can’t I Just Be A Doctor Any More?</td>
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<td>COPE ID # 58552-PM</td>
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<td>1 GEN</td>
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<tr>
<td>4:05 pm - 5:00 pm</td>
<td>2018 Professional Responsibility Course for Texas Optometrists</td>
<td></td>
<td>COPE ID # 56356-EJ</td>
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<td>1 PR/GEN</td>
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Eye Care and the Emergency Department

• Non-injury related ocular ER visits comprised 51% of ocular-related visits
• Only 3% of ocular-related ER visits required hospitalization
• 75% of the time, there was a clinically significant change in the diagnosis when care was first delivered at the ED or PCP and then followed up by a visit to an eye care specialist

What Classifies an Emergency?

• Any condition in which the patient has the potential for:
  – vision loss,
  – currently experiencing vision loss,
  – permanent structural damage,
  – pain or discomfort,
  – or is an “emergency” for the patient.
• It is important to be able to triage a walk-in patient and, more importantly, a call-in patient.
What questions to ask?

<table>
<thead>
<tr>
<th>Onset</th>
<th>suddenly noticed or sudden onset?</th>
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<tr>
<td>Visual Loss</td>
<td>any loss of vision?</td>
</tr>
<tr>
<td></td>
<td>loss vs. blurry vision</td>
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<tr>
<td></td>
<td>one eye or both</td>
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<tr>
<td></td>
<td>part of visual field or all</td>
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<td></td>
<td>transient vs. permanent</td>
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<tr>
<td>Pain</td>
<td>is there pain? constant? scale (1-10)</td>
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<tr>
<td>Redness</td>
<td>is there any redness? location?</td>
</tr>
<tr>
<td>Associated Factors</td>
<td>contact lens wear? trauma?</td>
</tr>
<tr>
<td></td>
<td>discharge?</td>
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<td>photophobia? medical history (eg. DM)</td>
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Visual Loss

- Visual loss varies greatly in meaning from patient to patient
  - ranging from blur to complete blindness and may affect one or both eyes
- Components include:
  - acuity,
  - visual field,
  - color and brightness may be affected jointly or separately
- Detailed history and extent of vision loss crucial

Profound Loss of Vision

- Referring to a complete or greatly diminished vision affecting the whole field
- Common causes of severe vision loss:

<table>
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<tr>
<th>Vascular</th>
<th>central retinal vein occlusion, central retinal artery occlusion, vitreous heme</th>
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<tbody>
<tr>
<td>Inflammatory</td>
<td>optic neuritis</td>
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<tr>
<td>Infiltrative</td>
<td>optic neuropathy</td>
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<tr>
<td>Mechanical</td>
<td>retinal detachment</td>
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</table>
Monocular vs. Binocular

- Ocular or optic nerve pathology causes monocular vision loss
- Lesion at or posterior to chiasm causes binocular vision loss
  - VF defects become more congruous the further back in the visual pathway
  - Homonymous VF defects noted posterior to chiasm
- Difference between mono vs. bino usually straightforward, keeping the following in mind:
  - Patients occasionally mistake homonymous hemianopsia (similar loss of visual field in both eyes) for a monocular loss

Visual Defects

Monocular

- Differentiate between eyes that have lost all useful vision and those that have blurred vision
- Blurring of vision is not localized and may be caused by pathology anywhere from cornea to optic nerve
- Need to get anatomical diagnosis first before considering the cause
General Appearance

- Level of consciousness
  - When introducing yourself be aware of the patient’s gross level of consciousness?
    - Is the patient awake, alert and responsive?
- Personal Hygiene and Dress
  - Is it appropriate for the environment, temperature, age and social status of the patient?
  - Is the patient malodorous or disheveled?

General Appearance

- Posture and Motor control
  - What posture does patient assume while sitting in the exam chair
  - Are there any signs of involuntary motor activity such as tremors
    - E.g. damage to the cerebellum may produce a tremor that usually worsens with movement of the affected limb

Neurological Screening: Cerebrum

- Frontal lobe
  - Emotions, drive, affect, self-awareness, and responses related to emotional states
  - Motor cortex associated with voluntary skeletal movement and speech formation (Broca)
Right vs Left Brain Injury

• So what happens if one side of the brain is injured?
  – **People who have an injury to the right side of the brain** "don't put things together" and fail to process important information.
  • As a result, they often develop a "denial syndrome" and say "there's nothing wrong with me."

• The left side of the brain deals more with language and helps to analyze information given to the brain.
  – If you injure the left side of the brain, you're aware that things aren't working (the right hemisphere is doing its job) but are unable to solve complex problems or do a complex activity.
  – People with left hemisphere injuries tend to be more depressed, have more organizational problems, and have problems using language.

Case History

• 38 black male, complaining that the vision in his right eye is blurry.
  – Got the current Rx 3 weeks previously, and started out good but in last couple of days OD vision has become blurry
• Medical Hx: no current health concerns and no medications
Entrance Skills

- Va’s: OD: 6/7.5 (20/25), OS: 6/6 (20/20)
- Pupils: PERRL
- CVF: full to finger count
- EOM’s: FROM
- Amsler: central metamorphopsia OD
- HVF: 10-2 (see VF)
Central Serous Retinopathy

• an exudative chorioretinopathy characterized by an exudative neurosensory retinal detachment with or without an associated detachment of the retinal pigment epithelium (RPE)
• Patients experience blurry vision, metamorphopsia and micropsia
• individuals between 20 and 50 years of age

CSR vs RD

http://www.octscans.com/retinal-detachment.html
Central Serous Retinopathy

- incidence in men vs women is approximately 6 to 1
- associated with stress and stress hormones (ie, corticosteroids and epinephrine);
- individuals with a "type A personality" who are under stress
- recurrence in the ipsilateral eye is approximately 30% and CSR in the fellow eye was 32%

Central Serous Retinopathy

- 80% to 90% of cases resolve spontaneously within 3 months
- Treatment options:
  - include laser photocoagulation,
  - "safety-enhanced" PDT,
  - Acetazolamide reduced the time for subjective and objective CSR resolution, but it had no effect on final VA or recurrence rate. Most patients in the experimental group in that study had side effects from the acetazolamide, including paresthesias, nervousness, and gastric upset

Central Serous Retinopathy

- Treatment options:
  - Topical NSAIDs:
    - Conflicting reports
    - Michael Singer, MD, from Medical Center Ophthalmology in San Antonio reported an increase in resolution time by 50%
    - PRADEEP VENKATESH, MD reports that NSAIDS treatment could possibly slow down or cause a rebound CSR
Latest Treatment Under Investigation

• Eplerenone is a mineralocorticoid antagonist receptor currently used in the treatment of hypertension and congestive heart failure.
• Literature has demonstrated improved resolution of CSR with no serious adverse effects.
• Several randomized clinical trials are currently underway.

Aussie Patient Story

• Male 59 Anglo Celtic heritage
• Asymptomatic, accidental detection by daughter following island holiday Bali and further sun exposure August 2016
• Hx: surfer and excessive sun exposure - coconut oils etc for first 2 decades of life.

Aussie Patient Story

• Initial dermatologist opinion – BCC (basal cell carcinoma)
  – BUT biopsy confirmed aggressive malignant melanoma, 2.2 mm thick, 5 mm cell growth rate
Aussie Patient Story

• Initial excision September 14 2016.
  – Found to have invaded sentinel axillary node –
• further surgery October 6 - complete axillary dissection right underarm - pathology clear.
• Final dx - stage 3 malignant melanoma.

Which of the following lid nevi have the greatest chance to convert to a malignant melanoma?

1 2 3 4

Lid Nevi

• Lid nevi:
  – congenital or acquired
  – occur in the anterior lamella of the eyelid and can be visualized at the eyelid margin.
• The congenital eyelid nevus is a special category with implications for malignant transformation.
• With time, slow increased pigmentation and slight enlargement can occur.
• An acquired nevus generally becomes apparent between the ages of 5 and 10 years as a small, flat, lightly pigmented lesion
Congenital Nevus

• The nevus is generally well circumscribed and not associated with ulceration.
• The congenital nevus of the eyelids may present as a “kissing nevus” in which the melanocytes are present symmetrically on the upper and lower eyelids.
  – Presumably this nevus was present prior to eyelid separation.

Congenital Nevus

• Most nevi of the skin are not considered to be at increased risk of malignancy.
  – However, the large congenital melanocytic nevus appears to have an increased risk of malignant transformation of 4.6% during a 30 year period.

Acquired Lid Nevi

• Acquired nevi are classified as:
  – junctional (involving the basal epidermis/dermis junction), typically flat in appearance
  – intradermal (involving only the dermis), tend to be dome shaped or pedunculated
  – compound (involving both dermis and epidermis) tend to be dome shaped.
CHRPE vs Nevus

Nevi Trivia

• 31% of choroidal nevi show slight enlargement over time without the transformation to a melanoma (Ophthalmology 2011)
• The prevalence of choroidal nevi in the white U.S. population ranges from 4.6% to 7.9%
  – If it is assumed that all choroidal melanomas arise from preexisting nevi, then the published data suggest a low rate (1/6845) of malignant transformation of a choroidal nevus in the U.S. white population. (Ophthalmology 2005)
• Choroidal melanoma risk for metastasis, ranging from 16% to 53% (at 5 years of follow-up) depending on the size of the tumor at the time of diagnosis. (Arch Ophthalmol 1992)

TFSOM—“To Find Small Ocular Melanoma”

Thickness: lesions >2mm
Fluid: any subretinal fluid (suggestive of serous retinal detachment)
Symptoms: photopsia, vision loss
Orange pigment overlying the lesion
Margin touching optic nerve head (<3mm)

• None of these factors = 3% risk of a nevus converting to melanoma in five years.
  One of these factors = 8% risk of conversion in five years.
  Two or more factors = 50% risk of conversion in five years.
For any changes noted during the course of follow-up, refer the patient to a retinal practice or an ocular oncology service.
TFSOM-UHHD:
“

To Find Small Ocular Melanoma Using Helpful Hints Daily”

- Thickness: lesions >2mm
- Fluid: subretinal fluid
- Symptoms: photopsia, vision loss
- Orange pigment overlying the lesion
- Margin touching optic nerve head (<3mm)
- Ultrasound Hollowness
- Halo absence
- Drusen absence

- Choroidal nevi showing no features should be initially monitored twice yearly and followed up annually
- 1 or 2 features should be monitored every 4 to 6 months.
- Nevi with 3 or more features should be evaluated at an experienced center for management alternatives and possible treatment owing to the high risk of ultimate growth

From: Enhanced Depth Imaging Optical Coherence Tomography of Small Choroidal Melanoma: Comparison With Choroidal Nevus
Case

- 65 yr old white male
  - Notices spot in vision in his left eye
  - Diabetes for 15 years
- Vision: 20/20 (6/6) and 20/40 (6/12)
- Dilated exam:
  - Large lesion noted in left eye (not noted in exam 6 months previously)
  - See photo and B-scan

Ocular Tumors

- Astrocytic Hamartoma
- Amelanotic Melanoma
- Retinoblastoma
- Metastatic Choroidal Tumor

Choroidal Melanoma Metastases

- 80 to 90% of metastases from uveal melanoma occurred in the liver, less common sites being the skin and lung.
Melanoma and Mortality

• Tumor Size:
  – 5-year mortality after enucleation:
    • 16% for small melanoma,
    • 32% for medium melanoma, and
    • 53% for large melanoma.
  – the prognostic importance of tumor size:
    • each 1-mm increase in melanoma thickness adds approximately
      5% increased risk for metastatic disease at 10 years

• Tumor genetics:
  – Chromosome monosomy 3 (approx 50% of patients)
    • 50% of them develop metastasis within 5 years of diagnosis
    • 70% mortality within 4 years of ocular treatment
    • one of the most important independent risk factors of poor
      survival

New Treatment for Choroidal Melanoma

• light-activated AU-011 agent represents the first potential new therapy for choroidal melanoma
• AU-011 is a viral nanoparticle conjugate delivered by intravitreal injection, which targets tumor cells in the choroid and then is activated by ophthalmic laser to disrupt the tumor cell membrane, leading to necrosis.
• Initial phase clinical trials showed promise

30 YR WM

• Patient calls from his PCP office asking if we can see him today because he has had red/painful eyes for over a week and has not resolved
• Medical history:
  – Past week has been experiencing painful urination and discharge
  – New sexual partner approx 10 days ago, who also had developed a red eye
  – Chlamydia and gonorrhea testing were negative
  – Has tested positive for HSV2 but no current flare up
30 YO WM

• Medications:
  – In the past week patient:
    • 2 courses of azithromycin (1 gram each)
    • Injection of rocephin
    • Injection of penicillin G
    • Currently taking doxycycline 100 mg bid
    • Valtrex 1 gram 3 times per day for 7 days (d/c 1 day ago)
    • Was on Vigamox qid for 7 days (d/c 1 day ago)
  • VA: 6/7.5 (20/25) OD, OS
  • Entrance skills unremarkable though some pain on eye movement

30 YO WM

• SLE:
  – 2+ injection conjunctival both eyes
  – 1-2+ lid edema
  – Mixed papillary and follicular response
  – 1-2+ diffuse SPK (no staining noted above infiltrates)
  – No cells or flare noted

30 YO WM

• AdenoPlus:
  – Performed on the right eye (patient felt that was the worst eye)
  – Negative
30 YO WM

- Started patient on the miracle drop
  - Tobradex 4 times per day and scheduled patient to come back the next day
- 1 day f/u
  - Patient was feeling better
  - Less redness and much reduced photophobia and discomfort
  - No improvement on painful urination or discharge and is now seeing blood in his urine
  - Continue tobradex 4 times per day and RTC in 4 days for f/u with dilation and told to contact PCP to update on the blood in the urine

30 YO WM

- 4 day f/u:
  - Patient says his eyes are doing great and that all of his urogenital problems abruptly stopped on Saturday
  - Discussion with PCP: Kidney stone
  - What was going on with the eye?
    - Viral conjunctivitis likely EKC

What did we learn from this?

Viral Conjunctivitis

- Most common infectious keratitis presenting on emergent basis
- 62% caused by adenovirus
- Two major types:
  - Pharyngoconjunctival fever (PCF)
  - Epidemic keratoconjunctivitis (EKC)
Viral Conjunctivitis

- **PCF:** history of recent/current upper respiratory infection
  - classic triad of fever, pharyngitis, and acute follicular conjunctivitis.
  - occurs more commonly in children, is caused by serotypes 3 and 7, and is spread by respiratory secretions.
  - tearing and foreign body sensation that is initially unilateral.

Viral Conjunctivitis

- **PCF:**
  - corneal involvement is not a key feature, there is occasionally a punctate keratitis;
  - SEIs are rare.
  - self-limiting condition that varies in severity and may last from 4 days to 2 weeks
  - Treatment if symptomatic though topical steroids are rarely needed.

Viral Conjunctivitis

- **EKC:** highly contagious with a history of coming in contact with someone having a red eye.
  - Adenovirus 8 common variant leading to “rule of 8’s”
    - First 8 days red eye with fine SPK
    - Next 8 days deeper focal epithelial lesions
    - Following 8 potential development of infiltrates
    - Resolution
  - AdenoPlus available to use for adenoviral confirmation
Viral Conjunctivitis: Signs and Symptoms

• Gritty sensation
• Watery discharge
• Sticky in mornings
• Follicular response
• Chemosis
• Injection
• SPK
• Infiltrates possible
• Positive lymph nodes

Management

• Consider the use of anti-inflammatory treatment to relieve patient symptoms and improve comfort
  – Alrex QID OU
  – Lotemax QID OU
    • New: Lotemax gel (indicated for post-op cataract but has longer contact time than standard lotemax)
• EKC patients are typically very uncomfortable and would benefit from anti-inflammatory treatment
  – especially if infiltrates or pseudomembrane present

Management

• Betadine (Melton-Thomas Protocol):
  – Proparacaine
  – 4-5 drops of Betadine 5%
    • Get patient to close eye and gently roll them around
    – After one minute, lavage the eye
    – Lotemax 4 times a day for 4 days
• Alternative: Betadine swabsticks.
  – 5% Betadine solution only comes in 30 ml bottles cost $14.00.
  – Case of 200 Betadine swabsticks apprx. 45 dollars.
Available in Canada!

Management

• Antivirals used in HSV keratitis are ineffective in treatment of viral conjunctivitis
  – Update: in conversation with several colleagues, Zirgan 4-5 times/day has shown significant improvement in patients over a 7-10 time period
• Hypochlorous acid???
• Important to stress limited contact with others, frequent hand washing, not sharing of towels, etc.

Efficacy of Hospital Germicides against Adenovirus 8, a Common Cause of Epidemic Keratoconjunctivitis in Health Care Facilities. ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, Apr. 2006, p. 1419–1424

An important finding from our study was that of the four disinfectants recommended by the CDC and Association for Professionals in Infection Control and Epidemiology for elimination of adenovirus type 8 from ophthalmic instruments, two (70% isopropyl alcohol and 3% hydrogen peroxide) were found to be ineffective. Based on these data, 3% hydrogen peroxide and 70% isopropyl alcohol are not effective against adenovirus that is capable of causing epidemic keratoconjunctivitis and similar viruses and should no longer be used for disinfecting applanation tonometers.
EKC Disinfection

- Commercial grade disinfectants that include compounds such as:
  - peracetic acid,
  - aldehydes [glutaraldehyde and ortho-phthalaldehyde],
  - chlorine-based products [1,900 to 6,000 ppm available free chlorine],
  - ethanol mixed with quaternary ammonium compounds
- E.g. Cidex, DisCide

Pre-Malignant Eyelid Lesions: Actinic Keratosis

- Also known as solar or senile keratosis
- Most common premalignant skin lesion
- Develops on sun-exposed areas and commonly affect the face, hands and scalp (less commonly the eyelids)
  - Predominately white males

Pre-Malignant Eyelid Lesions: Actinic Keratosis

- Appear as multiple, flat-topped papules with an adherent white scale.
- Development of SCC in untreated lesions as high as 20%
- Management is surgical excision or cryotherapy (following biopsy)
Malignant Eyelid Lesions: Basal Cell Carcinoma (BCC)

- Most common malignant lesion of the lids (85-90% of all malignant eyelid tumors)
- 50-60% of BCC affect the lower lid followed by medial canthus 25-30% and upper lid 15%

Malignant Eyelid Lesions: Basal Cell Carcinoma (BCC)

- Etiology is linked to excessive UV exposure in fair-skinned, ionizing radiation, arsenic exposure and scars
- Metastases is rare but local invasion is common and can be very destructive

Malignant Eyelid Lesions: Squamous Cell Carcinoma (SCC)

- Much less common than BCC on the eyelid but has much higher potential for metastatic spread
- Typically affects elderly, fair-skinned and usually found on the lower lid
Malignant Eyelid Lesions: Squamous Cell Carcinoma (SCC)

- Environmental and intrinsic factors initiate cell growth
  - Many SCC arise from actinic lesions

Malignant Eyelid Lesions: Squamous Cell Carcinoma (SCC)

- Presents as a erythematous, indurated, hyperkeratotic plaque or nodule with irregular margins
- Lesions have a high tendency towards ulceration and tend to affect lid margin and medial canthus

Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

- Highly malignant neoplasm that arises from the meibomian glands, Zeis and the sebaceous glands of the caruncle and eyebrow
- Aggressive tumor with a high recurrence rate, significant metastatic potential and notable mortality rate
  - Rates of misdiagnosis have been reported as high as 50%
Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

• Relatively rare, 1/3 most common eyelid malignancy
• Uncommon in the Caucasian population and represents only 3% of eyelid malignancies,
  – most common eyelid malignancy in Asian Indian population, where it represents approximately 40% or more of eyelid malignancies

Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

• Upper lid origin in about 2/3 of all cases
• Typically affects older individuals, women more so than men
• has also been reported in younger individuals who are immunosuppressed or who have received radiation treatment.

Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

• Presents as a firm, yellow nodule that resembles a chalazion.
• May mimic:
  – chronic blepharoconjunctivitis,
  – meibomianitis or
  – chalazion that does not respond to standard therapies
Malignant Eyelid Lesions: Sebaceous Gland Carcinoma

- Diagnosis is by biopsy
- Treatment is surgical excision with microscopic monitoring of the margins

Malignant Eyelid Lesions: Malignant Melanoma

- MM of the eyelid accounts for about 1% of all eyelid malignancies
- Incidence been increasing and it causes about 2/3 of all tumor related deaths from cutaneous cancers
- Incidence increases with age

Malignant Eyelid Lesions: Malignant Melanoma

- Risk factors include congenital and dysplastic nevi, changing cutaneous moles, excessive sun exposure and sun sensitivity, family history, age greater than 20 and white.
- History of severe sunburns rather than cumulative actinic exposure thought to be a major risk factor
Malignant Eyelid Lesions: Malignant Melanoma

- Flat lesion with irregular borders and variable pigmentation typically occurring in sun-exposed areas
- Confirmed diagnosis by biopsy

Malignant Eyelid Lesions: Malignant Melanoma

- Prognosis and metastatic potential are linked to the depth of invasion and thickness of the tumor
- Treatment is wide surgical excision confirmed with histological monitoring
Case

- 65 year old Caucasian patient presents with sudden onset loss/blurring of vision in the right eye
- PMHx: HTN for 15 years, takes “water pill”
- VA’s: 20/60 OD, 20/25 OS
- Pupils: PERRL –APD
- CVF: Inferior defect right eye, no defects noted in the left eye

Vision Loss Without Pain: Diabetes/Diabetic Retinopathy

- Microvascular complications resulting in capillary closure & abnormal permeability
- S&S include:
  - blurring of vision (maculopathy and refractive error shifts),
  - sudden drop in vision (vitreous heme),
  - dot and blot hemes,
  - exude,
  - cotton wool spots,
  - neovascularization (iris, retina and disc)

Diabetic Retinopathy
Vision Loss Without Pain: Vein Occlusion

• Associated with:
  - hypertension,
  - coronary artery disease,
  - DM and
  - peripheral vascular disease.
• Usually seen in elderly patients (60-70), slight male and hyperopic predilection.
• Second most common vascular disease after diabetic retinopathy.

Branch Retinal Vein Occlusion: Signs/Symptoms

• BRVO: sudden, painless, visual field defect.
  • patients may have normal vision.
  • quadrantic VF defect,
  • dilated tortuous retinal veins with superficial hemes and CWS
  • typically occurs at A/V crossing (sup/temp)
BRVO

- BRVO more common than CRVO and has more favorable prognosis
  - Overall 50-60% of BRVO patients will maintain VA of 20/40 or better
- Visual loss results from:
  - Macular edema
  - Foveal hemorrhage
  - Vitreous heme
  - Epiretinal membrane
  - RD
  - Macular ischemia
  - Neovascularization complications

http://www.healio.com/ophthalmology/journals/osli/

Study Design (n=397) BRVO

- BRAnch retinal Vein Occlusion study safety/efficacy
- Macular Edema Secondary to BRVO
- 1:1:1 Randomization
- Sham (n=132)
  - Ranibizumab 0.3 mg (n=132)
  - Rescue Laser (if eligible beginning at Month 9)
- Ranibizumab 0.3 mg (n=134)
  - Monthly Injections (last at 5M)
  - Rescue Laser (if eligible beginning at Month 3)
- Ranibizumab 0.5 mg (n=131)
  - Month 6
- Primary Endpoint
- Secondary to BRVO
- Ranibizumab 0.3 mg
  - Ranibizumab 0.3 mg
  - Ranibizumab 0.5 mg
  - 12M
Central Retinal Vein Occlusion: Signs/Symptoms

- CRVO: thrombus occurring at lamina is classical theory but new evidence indicates that the occlusion is typically in the optic nerve posterior to the lamina cribrosa
  - decreased VA ranging from near normal to hand motion with majority 20/200 range
  - dilated tortuous vessels, with numerous retinal hemes and CWS

Central Retinal Vein Occlusion

- Visual morbidity and blindness are primarily from:
  - persistent macular edema,
  - macular ischemia and
  - neovascular glaucoma
- CRVO’s can be ischemic or non.
  - Classical definition of ischemic is 10-disc area of non-perfusion found on angiography
  - RAPD and ERG maybe better predictor
  - VA’s typically worse in ischemic
  - Increased number of cotton wool spots with decreased VA maybe predictive
Central Retinal Vein Occlusion

- Ischemic CRVO may lead to iris neovascularization and neovascular glaucoma
  - Estimated approx 20% of CRVO’s are ischemic with 45% of those developing neo
- Regular examinations (1-2 wks) to monitor for ischemia or neo development
  - should include gonio as angle neo can precede iris rubeosis

Study Design CRUISE (n=392)
- CRVO: Central Retinal vein occlusion Study: Efficacy & safety
- Macular Edema Secondary to CRVO
- 1:1:1 Randomization
- Sham (n=130) Ranibizumab 0.3 mg (n=132) Ranibizumab 0.5 mg (n=130)
- Monthly injections (last at 5M) 6M tx period
- PRN Lucentis available for all patients 6M tx period
- 0.5 mg Ranibizumab 0.3 mg Ranibizumab 0.5 mg

Mean Change from Baseline BCVA
- CRVO
- Mean Change from Baseline BCVA (ETDRS Letters)
- Pts with >/= 3 line improvement was noted in 48% of 5 AVT, 26 of 3 AVT & 17% of sham
Vision Loss Without Pain: Artery Occlusion

- Primarily embolic in nature from cholesterol, calcifications, plaques.
- Usually occurs in elderly associated with:
  - hypertension (67%),
  - carotid occlusive disease (25%),
  - DM (33%) and
  - cardiac valvular disease.
- Sudden loss of unilateral, painless vision
  - defect dependent upon location of occlusion

Vision Loss Without Pain: Artery Occlusion

- BRAO typically located in temporal retinal bifurcations.

CRAO

- CRAO has profound vision loss with history of amaurosis fugax.
  - Vision is usually CF (count fingers) to LP (light perception) with positive APD.
  - Diffuse retinal whitening with arteriole constriction, cherry red macula.
Ophthalmic Emergency

• Treatment is controversial due to poor prognosis and questionable benefit.
• Treat immediately before workup, if patient presents within 24 hours of visual loss:
  ◦ Digital ocular massage,
  ◦ systemic acetazolamide (500 mg IV or po),
  ◦ topical ocular hypertensive drops (Iopidine, B-blocker),
  ◦ anterior chamber paracentesis,
  ◦ consider admission to hospital for carbogen Tx (high carbon dioxide)

Flashes and Floaters

• Patients often present complaining of “spots” or “cobwebs” in front of their eyes
• Causes of floaters include: posterior vitreous detachment (PVD), retinal tear, vitreous heme, uveitis.
• Since PVD and retinal tears present the same way, a RT has to be eliminated
• Ask the patient whether spots move with eye and continue to move after the eye has stopped
• Large spots could be blood clots
Flashes and Floaters

- Sudden onset typically means a PVD, retinal tear or heme
- If the spots appear after flashing light, then retinal tear must be eliminated
- Myopes tend to have floaters and will notice them for a long time
- Key is to rule out potentially sight threatening condition for the floaters, i.e retinal tear.
- Patients with retinal condition such as lattice degeneration and myopes need to be educated about 5-8% of RD (flashes and floaters)
  - 8-11% population has lattice
  - Risk of RD with lattice is <1%
  - 30-50% of patients with a RD have lattice
Flashes and Floaters: Management

• A patient who presents with a sudden onset PVD without retinal breaks or hemorrhage requires repeat peripheral examination in six weeks, as the risk of retinal complications is highest within the six weeks following vitreous detachment.

• If no retinal breaks are seen at that point, routine yearly examination is all that is needed.

Macular hole

• Unilateral, decreased vision
  – Often in 60-80 year old women
  – Anyone w/ a history of trauma

• Symptoms:
  – Decreased vision, metamorphopsia
    • 20/200 for full thickness holes

• Signs:
  – Red hole in the macula
  – (+) Watzke-Allen sign

• Stages
  – Stage 1a -> impending hole. Normal foveal depression with yellow spot/dot in fovea.
  – Stage 1b -> Abnormal foveal depression with yellow ring.
Macular hole

- Stages
  - Stage 2: Small full-thickness hole. 20/80 - 20/400.
  - Stage 3: Full-thickness hole w/ cuff of SRF. No PVD
  - Stage 4: Full-thickness hole with cuff of SRF, with complete PVD.

Stage 2 macular hole

Stage 3 macular hole

Stage 4 macular hole

New Macular Hole Staging
**New Macular Hole Staging**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
<th>International Macular Hole Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Impending new hole</td>
<td>VH</td>
</tr>
<tr>
<td>2</td>
<td>New hole</td>
<td>VH</td>
</tr>
<tr>
<td>3</td>
<td>Large hole</td>
<td>Small or total FTMH w/o VMT</td>
</tr>
<tr>
<td>4</td>
<td>Traction</td>
<td>Medium or large FTMH w/o VMT</td>
</tr>
<tr>
<td>5</td>
<td>Perforation</td>
<td>Large or total FTMH</td>
</tr>
</tbody>
</table>

- **Small FTMH w/o traction**
  - 154 microns

- **Medium FTMH w/o traction**
  - 250-400 microns

- **Large FTMH**
  - 400+ microns
New Macular Hole Staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>Definition</th>
<th>International Vitreomacular Traction Study Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Large FTMH with traction (&gt; 400 microns)</td>
<td>Large or medium FTMH with VMT</td>
</tr>
<tr>
<td>2</td>
<td>Small FTMH with VMT</td>
<td>Medium or large FTMH with VMT</td>
</tr>
<tr>
<td>3</td>
<td>Large or medium FTMH without VMT</td>
<td>Small, medium, or large FTMH without VMT</td>
</tr>
</tbody>
</table>

> 400 microns

13 YR Female

CC: noticed that her left eye became blurry and objects were “wavy” a couple of days ago. Sudden onset and she had experienced a headache over the left eye just prior to the vision going blurry.

Culdar Hx: she currently wear glasses for distance

Medical Hx: she is currently not diagnosed with any health problems and is not taking any medications

Entrance Skills

- VA with current Rx: 20/30 OD and 20/30 OS
- Entrance skills unremarkable
- Amsler: metamorphopsia OS
- BCVA: 20/20 OD with increased minus, no improvement possible in the left eye
- IOP’s: 13 mm Hg OD and OS
Retina Consult

- Referred patient to retina and they confirmed the diagnosis of VKH.
- She was begun on oral prednisone 60 mg per day and she was re-evaluated in 1 week.
- At the follow up, there was reduction in her serous retinopathy and vision was improved.
From the Experts

- Vogt-Koyanagi-Harada (VKH) disease is a multisystemic disorder characterized by granulomatous panuveitis with exudative retinal detachments that is often associated with neurologic and cutaneous manifestations.
- VKH disease occurs more commonly in patients with a genetic predisposition to the disease, including those from Asian, Middle Eastern, Hispanic, and Native American populations.

From the Experts

- VKH:
  - Patients have no prior history of ocular trauma or surgery
  - Patients have no evidence of another ocular disease based on clinical or laboratory evidence
  - Patients have bilateral ocular involvement.

From the Experts

- VKH:
  - The neurologic and auditory signs include the following:
    - Malaise, fever, headache, nausea, abdominal pain, stiffness of the neck and back, or a combination of these factors; headache alone is not sufficient to meet the definition of meningitis
    - Tinnitus
    - Cerebrospinal fluid pleocytosis
  - Integumentary signs include the following:
    - Alopecia: loss of body hair
    - Poliosis: loss of pigment in hair
    - Vitiligo: loss of skin pigmentation in blotchy pattern
VKH Treatment

• For most patients with bilateral serous detachments and severe visual loss, begin therapy with systemic prednisone (1-2 mg/kg/day).
• The length of treatment and subsequent taper must be individualized for each patient.
  – Most patients require therapy for 6 months and occasionally up to 1 year before successful tapering of systemic corticosteroids.
  – Systemic therapy should not be discontinued during the 3 months following the onset of the disease because of the risk for recurrence.
Oral Pharmaceuticals in Anterior Segment Disease

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Case

• 20 year old male presents with a red painful eye
  – Started that morning when he woke up
  – reports a watery discharge, no itching, and is not a contact lens wearer
• SLE:
  – See attached image with NaFl stain

Herpes Simplex

• Most common virus found in humans
  – 60-99% are infected by 20 years old
• Double stranded DNA virus
  – HSV type 1 (HSV-1)
  – HSV type 2 (HSV-2)

• Primary infection
  – Occurs in childhood via droplet exposure
  – Subclinical infection in most
• Secondary infection (recurrence)
Herpes Simplex

• Recurrent infection:
  – After primary infection the virus is carried to the trigeminal ganglion where a latent infection is established.
  – Latent virus is incorporated in host DNA
  – Stress (trauma, UV light, fever, hormonal changes, finals week, etc) causes reactivation of the virus

Herpes Simplex Keratitis

• Epithelial Keratitis:
  – Symptoms:
    • Ocular irritation, redness, photophobia, watering, blurred vision
  – Signs:
    • Swollen opaque epithelial cells arranged in a course punctate or stellate pattern
    • Central desquamation results in a dendrite***
      1. Central ulceration
      2. Terminal end bulbs
    • ***Corneal sensation is reduced***

Dendritic Ulcers
Pediatric HSV Keratitis

- pediatric herpes simplex keratitis has an 80% risk of recurrence, a 75% risk of stromal disease, and a 30% rate of misdiagnosis
- 80% of children with herpes simplex keratitis develop scarring, mostly in the central cornea
  - results in the development of astigmatism
  - 25% of children have more than 2 D of astigmatism, most of which is irregular
- consider pediatric HSV when a patient has unilateral recurrent disease in the anterior segment

Herpes Simplex Keratitis Management

- Topical:
  - Viroptic (trifluridine) q 2h until epi healed then taper down for 10-14 days.
  - Viroptic is toxic to the cornea.
  - Zirgan (ganciclovir) available, use 5 times a day until epi healed then 3 times for a week (US only)
Anti-Viral Medication

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Bioavailability</th>
<th>Dosing</th>
<th>Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acyclovir</td>
<td>Acyclovir interferes with DNA synthesis</td>
<td>10-30% gets</td>
<td>Simplex: 400 mg</td>
<td>Overall very safe</td>
</tr>
<tr>
<td></td>
<td>inhibiting viral replication</td>
<td>absorbed Short</td>
<td>5x/day</td>
<td>Nausea, vomiting, headaches, dizziness, confusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>¾ life *Metabolized in kidneys</td>
<td>Zoster: 800 mg</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5x/day</td>
<td></td>
</tr>
<tr>
<td>Valacyclovir</td>
<td>Valacyclovir pro-drug equivalent to acyclovir but better for pain management</td>
<td>95% converted to acyclovir * Better bioavailability and longer 1/2 life.</td>
<td>Simplex: 500 mg tid Zoster: 1 g tid</td>
<td>Same as acyclovir</td>
</tr>
<tr>
<td>Famciclovir</td>
<td>Famciclovir Inhibits DNA chain elongation</td>
<td>Superior to acyclovir*</td>
<td>Simplex: 250 mg TID Zoster: 500 mg TID</td>
<td>Same as acyclovir</td>
</tr>
<tr>
<td>(Famvir)</td>
<td>It is metabolized to penciclovir where it is active 10-20x as long as acyclovir</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

HSV Stromal Disease

- HSV Stromal disease is an immune-mediated disease
- Increased risk of scarring and high risk of poor visual prognosis
- Requires corticosteroids (HEDS: corticosteroid reduced risk of progression by 68%)
  - Without epithelial defect: corticosteroids and prophylactic anti-viral dosage
  - With epithelial defect: active infection anti-viral dosage with judicious corticosteroids

How much to dose steroid?

- HEDS used QID of prednisolone phosphate
- Current Recommendations:
  - Mod – severe (especially with neo): 1%
    - Prednisolone or Lotemax QID to 6x/day
  - Want the lowest dose needed to control the inflammation
  - AAO EBM Treatment Guideline 2014
    - Topical steroid for 10 weeks (this is based on HEDS results) with oral antiviral
Herpes Simplex Epithelial Keratitis

- **Treatment Regimen:**
  - Zirgan 5x/day until the ulcer heals, then 3x/day for one week
  - Oral Valtrex 500 mg 3x/day for 7-10 days
  - Artificial tears
  - L-Lysine 2 grams daily?
    - Proven to “slow down” and retard the growth of the herpes virus and inhibit viral replication
  - Debride the ulcer?
    - Prior to topical antiviral therapy debridement was treatment of choice
    - Generally try to avoid use of sharp instruments and use of cotton swab and anesthetic

- **RTC 1 day, 4 days, 7 days**

---

Herpes Simplex Keratitis

- **Prophylactic Treatment:**
  - Reduces the rate of recurrence of epithelial and stromal keratitis by ≈ 50%
    - Acyclovir 400 mg BID
    - Valtrex 500 mg QD
    - Famvir 250 mg QD
  - L-Lysine 1 gram/day:
    - Proven to “slow down” and retard the growth of the herpes virus and inhibit viral replication
  - Frequent debilitating recurrences, bilateral involvement, or HSV infection in a monocular patient

---

**Prophylaxis??**

- **Pitfalls to Prophylaxis:**
  - Reduction of recurrence does not persist once drug stopped
  - Resistance?????
    - van Velzen, et. al., (2013) demonstrated that long-term ACV prophylaxis predisposes to ACV-refractory disease due to the emergence of corneal ACVR HSV-1.
Pain Management: Oral Analgesics

• Conditions potentially requiring use of oral analgesics:
  – Corneal ulcers
  – Herpes simplex/zoster
  – Post-surgical
  – Trauma

Acetaminophen

• Mechanism of Action is not well understood.
  – Possibly some CNS component
  – Very weak inhibitor of prostaglandin synthesis

• One of the most commonly used analgesics for mild to moderate pain.
  – Equal analgesic properties to ASA unless associated with inflammation, where it is less effective.

  Take home: Good for pain; Good for fever; No effect on inflammation

Dangers of Acetaminophen

• Acetaminophen overdose is the leading cause of liver failure in the U.S.
  – It sends 56,000 people to the emergency room annually and causes approximately 400 deaths yearly.

• Acetaminophen is used in so many products, people are often unaware that they are taking it, leading to more overdoses.
  – Combined with agents to get wide range of symptom coverage.
    • Antihistamines such as diphenhydramine – Tylenol PM
    • Diuretics such as Pyrilamine maleate – Midol Complete
    • Cough Suppressants such as Dextromethorphan - Nyquil
Combining Meds for More Severe Pain Relief

- Acetaminophen and Aspirin are often combined with each other and various agents to increase their analgesic effect.
  - Frequently seen in combination with narcotic analgesics.
  - Caffeine is also commonly used, especially in the treatment of migraines.
    - Excedrin Migraine
      - Acetaminophen 250 mg
      - Aspirin 250 mg
      - Caffeine 65 mg

Consider Combining APAP with NSAID’s for Mild to Moderate Pain Relief

1:00 pm: Two 325mg Tylenol
3:00 pm: Two 200mg Ibuprofen
5:00 pm: Two 325mg Tylenol
7:00 pm: Two 200mg Ibuprofen
Alternated every 2 hours while awake
  - Each medication is q 4 hours.

Ibuprofen

- Adult analgesic dose: 200-400mg q4hours
  - Maximum Dosage: 2400 mg/day for pain (approved for 3200 mg/day in arthritis treatment)
- OTC: 200 mg tabs
- Rx: 300, 400, 600, 800mg tabs
- Peak levels 1-2 hours
- Most renal toxic of all the NSAID’s
- Brand Names: Motrin, Advil, and Nuprin
Indoleacetic Acids: Indomethacin

- Adult Dosage: 25-50 mg TID
- Rx Only: 10mg - 75mg capsules
- Mainly used as a short term anti-inflammatory especially for conditions that do not respond to less toxic NSAIDs.
  - Indomethacin has a very high level of intolerance compared to other NSAID's.
- Oral NSAID most widely used in Tx of ocular inflammation.

Cox-2 Inhibitors

- Selective agents for only COX-2 designed to protect the GI system from the side effects seen with NSAID's.
- Major agent available on the market is Celecoxib (Celebrex).
  - Other agents Valdecoxib (Bextra) and Rofecoxib (Vioxx) were removed from the market due to increased risk of heart attacks and strokes.
  - It is approved for the treatment of osteoarthritis and rheumatoid arthritis.
- Dosage: 100 mg BID or 200 mg daily

Oral Analgesics: Guidelines

- Never exceed maximum recommended dosages:
  - ASA: 8 grams/day
  - Acetaminophen: 4 grams/day
  - Ibuprofen: 1200 mg/day OTC and 2400 mg/day prescription
  - Codeine: 360 mg/day
Oral Analgesics: Guidelines

- Make the proper diagnosis first (ie. Don’t prescribe without knowing what you are prescribing for!)
- Treat the underlying cause for the pain
- Treat the pain at presentation..don’t wait!
- Treat pain continuously over a 24 hour schedule
- Non-prescription drugs should be first choice and tend to be low cost
- Treat patients with the simplest and safest means to alleviate pain

Opioids Information

- Drug of first choice for the treatment of severe acute pain.
  - Block the body’s natural protective mechanism for protecting areas in pain – thus never prescribe unless you know the direct cause of the pain.
- Often administered in combination with acetaminophen or aspirin to enhance the analgesic effect.
  - FDA recommended in 2011 that all prescription narcotics containing acetaminophen standardize and limit the dosage to 325 mg.
    - This is to be slowly phased in over three years (just required in January 2014).

Opioids Side Effects

- Side Effects are very hard to predict because opioids can cause CNS depression or stimulation.
- CNS Side Effects
  - Dizziness, lightheadedness, sedation, and drowsiness are the most common.
  - Mood elevation (euphoria) and disorientation can occur in some patients.
  - Exacerbated if used in combination with alcohol, depression medications such as tricyclic antidepressants, anticholinergics, antihistamines, anti-seizure medications, or muscle relaxants, etc.
  - Visual symptoms such as blurry vision, miosis, and diplopia can occur.
Opioid Side Effects

• GI Side Effects:
  – Nausea and Vomiting (more common in ambulatory pts.)
  – Constipation
    • Opioids inhibit intestinal trace motility.
    • Very commonly found side effect.
      – Can be relieved by OTC docusate sodium (Colace).

Respiratory Side Effects:

• Respiratory Depression
  – Most serious side effect of the opioids
  – Opioids suppress the brainstem respiratory centers
  – Alter tidal volume, respiratory rate, rhythmicity, and responsiveness to CO2
  – Does not commonly occur at therapeutic doses in healthy patients, but must use caution in patients with pulmonary disease.

• Cardiovascular Side Effects:
  – Peripheral vasodilation can result in orthostatic hypotension, decreased BP, and changes in pulse rate.

• Others Include: Urinary retention, cough suppression, headaches, rashes, itching.

Tolerance to Opioids

• Patients experience shorter durations of analgesia from similar dosages, followed by increased levels of pain. Requires dosages to continually be adjusted to provide desired effects.

• Withdrawal can occur if long term use is discontinued abruptly resulting in increased heart rates and blood pressure, nausea, vomiting, dilated pupils, photophobia, shivering, etc. These symptoms peak approximately 2 to 3 days after the last dose and will subside over weeks.
Patient Education

- Avoid all depressants – especially using along with alcohol.
- Must educate all patients of risks of these symptoms and caution them for driving or operating dangerous machines.
- Stomach upset can be helped by consuming the medication with food.
- Watch for signs of breathing difficulty or changes in blood pressure.

Opioids Contraindications

- Avoid in patients with history of hypersensitivity to narcotics.
  - True allergic reactions are rare and often involve skin rashes or contact dermatitis.
- Avoid in patients with acute bronchial asthma or COPD.
- Avoid in patients with history of depression or suicidal tendencies.
- Avoid in patients with history of addiction.
- Avoid in pregnancy (Most opioids are pregnancy category C).
  - Drug Effects seem to be insignificant in nursing infants, but should recommend waiting at least 4 – 6 hours to nurse.
- Use caution in kidney or liver dysfunction due to increased accumulation of the medication.
- Must be very cautious of drug interactions and always review medications with your patient prior to prescribing.

Scheduled Medications – Most Opioids

<table>
<thead>
<tr>
<th>Schedule</th>
<th>Description</th>
<th>Opioid Medications</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Not commercially available, no approved indication</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>Very addictive medications that are accepted for medicinal use</td>
<td>Oxycodone = OxyContin, Oxyfast, Oxycontin + APAP = Percocet or Tylenol, Oxycodone + ASA = Percodan, Oxycodone + NSAID = Combunox, Hydromorphone (Dilaudid), Codeine Sulfate = Codeine Generic, Meperidine (Demerol)</td>
</tr>
<tr>
<td>III</td>
<td>Significant abuse risk, but less potent than I or II. May still contain narcotics.</td>
<td>Codeine + APAP = Tylenol 3 and Tylenol 4</td>
</tr>
<tr>
<td>IV</td>
<td>Relatively low abuse potential and limited risk</td>
<td>Propoxyphene (Darvon), Propoxyphene with APAP = Darvocet (Removed from Market in November 2010), Percocet (APAP with oxycodone) (Takiron), Tramadol</td>
</tr>
<tr>
<td>V</td>
<td>Very limited abuse potential. May be OTC in some states</td>
<td>Acetaminophen</td>
</tr>
</tbody>
</table>
Schedule III Opioids: Codeine

- Prodrug that relies on the cytochrome P-450 system to be metabolized to active drug morphine.
  - Schedule II medication if prescribed alone (Codeine Sulfate 15, 30, 60 mg generic.)
- Analgesic effect occurs within 20 minutes of ingestion and reaches a maximum at 1 – 2 hours.
  - Ceiling effect occurs.

Schedule III Opioids: Codeine

- Usually administered in combination with .
  - Tylenol 3 = Codeine 30 mg and Acetaminophin 300 mg
    - Dosage: 1-2 tablets every 4 hours.
  - Tylenol 4 = Codeine 60 mg and Acetaminophin 300 mg
    - Dosage: 1 tablet every 4 – 6 hours
  - Also available as generic with 15, 30, or 60 mg of Codeine with 300 mg of Acet. or elixer of 12 mg codeine + 120 mg Acet. per 5 mL.
    - Elixer can be used in children for pain management if >3 years.

Schedule II Opioids: Hydrocodone

- Approximately 6X more potent than codeine.
- Milder Side Effects than Codeine: Less constipation and sedation.
- Clinically believed to cause more euphoria than codeine, but this is not backed by clinical studies.
### Schedule II Opioids: Hydrocodone

- Used in combination with APAP and ibuprofen.
  - Lortab: Hydrocodone 5, 7.5, and 10 mg with APAP 325 mg
    - Dosage: 1-2 tablet every 4-6 hours
  - Lortab Elixir: Hydrocodone 10 mg with APAP 300 / 15 mL
    - Dosage: 3 tsp every 4-6 hours
  - Vicodin: Hydrocodone 5 mg with Acetaminophen 300 mg
  - Vicodin HP: Hydrocodone 10 mg with Acetaminophen 300 mg
    - Dosage: 1 tablet every 4-6 hours
  - Vicodin ES: Hydrocodone 7.5 mg with Acetaminophen 300 mg
    - Dosage: 1 tablet every 4-6 hours
  - Norco: Hydrocodone 5, 7.5, and 10 with 325 mg APAP

### Schedule II Opioids: Oxycodone

- Approximately 10-12X more potent than codeine
  - As potent as parenteral morphine when given orally.
- Lower level of side effects in comparison to morphine, but high level of euphoria produced, thus higher level of abuse risk.

### Schedule II Opioids: Oxycodone

- Available in combination with APAP, ASA, or ibuprofen.
  - Percocet Tablets
    - 2.5, 5, 7.5 or 10 mg Oxycodone with 325 mg Acetaminophen
    - Dosage: 1 tablet every 6 hours
  - Tylox Capsules
    - 5 mg Oxycodone with 300 mg Acetaminophen
    - Dosage: 1 tablet every 6 hours
  - Percodan Tablets
    - 4.5 mg Oxycodone HCl
    - 0.38 mg Oxycodone terephthalate
    - 325 mg Aspirin
    - Dosage: 1 tablet every 6 hours
  - Combunox
    - 5 mg Oxycodone with 400 mg Ibuprofen
    - Dosage: 1 tablet daily to QID
**Newly Schedule IV: Tramadol (Ultram)**

- Central acting narcotic
  - Synthetic analogue of codeine.
  - Binds to mu receptors and inhibits norepinephrine and serotonin reuptake.
  - Potential for abuse is very low, but has occurred.
- Available as 50 mg tablets.
- **Dosage:** 50 – 100 mg q4 – 6 hours.
  - Analgesia occurs after 1 hour.
  - Maximum dose: 400 mg/day

---

**First Step in Managing Acute Pain: Acetaminophen**
- If inadequate analgesia with 1000 mg TID

- Consider NSAID:
  - Ibuprofen 400 mg every 4-6 hours

- If NSAID inadequate or contraindicated:
  - Hydrocodone + APAP

- If inadequate consider in combination with Ibuprofen or may need to consider stronger options/comanagement.

---

**Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)**

- Abnormal basement membrane production
- Not all patients are symptomatic (range 10-69%)
- Most common symptom is mild FB sensation which is worse in dry weather, wind and air conditioning
- Blurred vision from irregular astigmatism or rapid TBUT
- Pain is usually secondary to a RCE (recurrent corneal erosion) in apprx 10%
Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

• Easy to overlook:
  – typically bilateral though often asymmetric,
  – females>males,
  – often first diagnosed b/w ages of 40-70

Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

• Most common findings are:
  – chalky patches,
  – intraepithelial microcysts, and
  – fine lines (or any combination) in the central 2/3rd of the cornea

Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD)

• Often referred to as:
  – maps,
  – dots or
  – fingerprints
Epithelial (Anterior) Basement Membrane Dystrophy (EBMD or ABMD): Treatment

- Typically directed towards preventing RCE
- If RCE’s develop:
  - awake with painful eye that improves as day wears on
  - chalky patches/dots in lower 2/3rd of cornea

RCE: Treatment

- Initial treatment includes:
  - use of hyperosmotic ointment at bedtime,
  - bandage contact lens and
  - lubrication.

Recurrent Corneal Erosion: Treatment

- If severe enough to cause vision loss or repeated episodes:
  - oral doxycycline with/without topical corticosteroid
    - Doxy 50 mg bid and FML tid for 4-8 weeks
    - both meds inhibit key metalloproteinases important in disease pathogenesis
    - Azasite (topical azithromycin)
  - debridement,
  - stromal puncture, or
  - PTK
  - Latest development: amniotic membrane transplant e.g. Prokera
CORNEAL DEBRIDEMENT

- Soften epithelium
- 1-2 gtt topical anesthetic
- q 15-30 seconds for 2-3 minutes
- Use cotton swab, spatula, spud
- or jewelers forceps
- Remove flaps by pulling edges toward center
- Don’t pull directly up or out
- Remove flaps down to tight, firm edges.
- Tx abrasion (>50-100%)
  - Recurrence Rate 18%

Diamond Burr Polishing

- Removes abnormal basement membrane
- May also promote scarring

Amniotic Membrane Transplant

- Amniotic membrane is a biologic tissue with:
  - antiangiogenic,
  - antiscarring,
  - antimicrobial, and
  - anti-inflammatory properties that promotes healing of the ocular surface
- Amniotic membrane grafts have been used for a variety of ocular conditions including:
  - Corneal burns
  - Neurotrophic ulcers
  - Stem cell damage
  - Persistent epithelial defects
**Tetracyclines**

- This group includes:
  - Tetracycline (250mg - 500 mg cap BID-QID) needs to be taken 1 hour before or 2 hours after a meal.
  - Minocycline (100 mg cap BID)
  - Doxycycline (20mg - 100 mg cap or tab BID)
    - In Canada: Apprilon (30 mg doxy + 10 mg slow release doxy)

- Rules of Thumb with Doxy:
  - Do not take before lying down (>2 hours before)
  - Do not take with calcium and avoid antacids
  - Do not take with dairy
  - Do take with food
  - Do educate on sun protection

**Side Effects of Tetracyclines**

- Side effects include gastric discomfort, phototoxicity, effects on calcified tissues, vestibular problems, pseudotumor.
- Pregnancy Category D.
  - Tetracyclines are attracted to embryonic and growing bone tissue.
    - Depress growth of long bones in pregnant women/children.
    - Cause changes in both deciduous and permanent teeth during the time of tooth development (includes discolored and increased cavities)

- Contraindicated in:
  - Women in the last half of pregnancy
  - Lactating women
  - Children under 8 years of age

**Meibomian Gland Dysfunction**

- Meibomian gland dysfunction:
  - Also referred to as meibomitis and patients experience dry eye problems secondary to increased evaporation of the tears.
  - Signs include noticeable capping of the glands and frothing of tear film.
- Standard treatment includes:
  - Good lid hygiene with warm compresses and lid scrubs in conjunction with
  - Doxycycline 50 mg po BID for 2-3 months
    - Erythromycin ung (Ilotycin) can also be used externally.
Acne Rosacea

- Acne rosacea:
  - affects females > males after 30 with peak incidence 4th-7th decade of Celtic/Northern European descent. Males more disfigured.
- 4 subtypes with classic signs of flushing, papules or pustules usually in crops, telangiectasia.
  - secondary ocular complications (85% of patients) and often precede other skin manifestations include erythema, itching and burning.
  - Lipases secreted by bacteria on the skin metabolize sebum and produce metabolites that result in inflammation of the skin.

Acne Rosacea and Demodex

- Demodex is a natural part of human microbiome
- *Demodex folliculorum* live in hair follicles, primarily on the face, as well as in the meibomian glands of the eyelids;
- *Demodex brevis* live in the sebaceous glands of the skin.

Acne Rosacea and Demodex

- *Demodex folliculorum* frequently occur in greater numbers in those with rosacea and this overabundance is thought to trigger an immune response or possibly certain bacteria associated with the Demodex.
Acne Rosacea

- Mainstay oral Tx is **Oracea (40 mg in morning)** or
  - doxycycline 50 mg po or minocycline 100 mg po for 4-12 wks.
- **NOTE:** Oracea is subantimicrobial therapy.
- May want to consider Tea Tree oil wipes/foam for the face and lids to try and reduce the role Demodex plays.

LipiView

Hordeola

- Acute purulent inflammation
  - Internal occurs due to obstruction of MG
  - External (stye) from infection of the follicle of a cilium and the adjacent glands of Zeiss or Moll
- Painful edema and erythema,
Hordeola

- Typically caused by Staph and often associated with blepharitis
- Treatment includes:
  - hot compresses (e.g. Bruder)
  - topical antibiotics (?)
  - possibly systemic antibiotics
    - Augmentin 500 mg bid-tid
    - Doxycycline 100 mg bid
  - Treat concurrent blepharitis

Preseptal Cellulitis

- Infection and inflammation located anterior to the orbital septum and limited to the superficial periorbital tissues and eyelids.
- Usually follows sinus infection or internal hordeolum (possibly trauma)
- Eyelid swelling, redness, ptosis, pain and low grade fever.

Preseptal Cellulitis

- Tx:
  - Augmentin 500 mg TID or 875 mg BID for 5-7 days
  - Keflex 500 mg QID 5-7 days
  - or if moderate to severe IV Fortaz (ceftazidime) 1-2 g q8h.
  - If MRSA possible, consider Bactrim/Septtra
ARMOR

- Antibiotic Resistance Monitoring in Ocular Microorganisms (ARMOR)
- Approximately 42% of isolates were determined to be MRSA
- Newer fluoroquinolones have better activity than earlier generations
- Besivance has the lowest MIC values of all the fluoroquinolones
- Vancomycin is drug of choice if MRSA present
- Azithromycin had very poor activity against Staph

Penicillins: Augmentin

- Augmentin is amoxicillin with potassium clavulanate (clavulanic acid 125 mg).
  - Clavulanate is a B-Lactamase inhibitor which reduces a bacteria’s ability to negate the effect of the amoxicillin by inactivating penicillinase (enzyme that inactivates the antibiotic affect).
  - Dicloxacillin can also be used in infections due to penicillinase-producing staph.

Penicillins: Augmentin

- Augmentin is very effective for skin and skin structure infections such as:
  - dacrocystitis,
  - internal hordeola,
  - pre-septal cellulitis.
  - Treatment of:
    - otitis media,
    - sinusitis,
    - lower respiratory and urinary infections.
    - Given prophylactically to dental surgery patients.
Penicillins: Augmentin

- It has low:
  - GI upset,
  - allergic reaction and anaphylaxis.
- Serious complications include:
  - anemia,
  - pseudomembranous colitis and
  - Stevens-Johnson syndrome.

Penicillins: Augmentin.

Adults:
- 250-500 mg tab q 8hr (tid)
  (also available in chewable tablets and suspension)
- or 875 mg q 12hr (bid)
- 1000 mg XR q 12 hr and not for use in children <16
Peds: <3 mos 30mg/kg/day divided q12hrs using suspension
  • >3 mos 45-90mg/kg/day divided q12hrs (otitis media 90mg for 10 days)

Cephalosporins

- Closely related structurally and functionally to the penicillins,
  - have the same mode of action,
  - affected by the same resistance mechanisms.
  - tend to be more resistant to β-lactamases.
- classified as 1st, 2nd, 3rd, 4th and now 5th generation based largely on their bacterial susceptibility patterns and resistance to β-lactamases.
- Typically administered IV or IM, poor oral absorption.
Side Effects and Contraindications

- Hypersensitivity Reactions are common.
  - Risk of cross sensitivity with PCN's is higher for 1st generation, but often overestimated for later medications.
  - Used to state the cross sensitivity was ~10%, but now believed to be closer to 3%.

Cephalosporins

- 1st generation: cefadroxil (Duricef), cefazolin (Ancef), cepalexin (Keflex), and cephalothin
- 2nd generations: cefaclor (Ceclor), cefprozil, cefuroxime (Zinacef), cefotetan, cefoxitin
- 3rd generation: cefdinir (Omnicef), ceftriaxone, ceftaxime (Claforan), ceftriaxone (Fortaz), cefuroxime, ceftriaxone, ceftriaxone (Rocephin IM/IV)
- 4th generation: cefepime
  - Omnicef, Keflex, Ceclor (all orally administered) are effective against most gram positive pathogens and especially good for skin and soft tissue infections.

Cephalosporins

- Keflex (cephalexin):
  - treatment of respiratory, GI, skin and skin structure, and bone infections as well as otitis media
  - Adults: 250-1000 mg every 6 hours
    - typical dosing 500 every 6 hours
  - Children: 25-100 mg/kg/day divided 6-8 hours
Co-Trimoxazole (Bactrim/Septra)

- Combination of trimethoprim and sulfamethoxazole
  - shows greater antimicrobial activity than equivalent quantities of either drug alone.
- Has broader spectrum of action than the sulfa’s and is effective in treating:
  - UTIs and respiratory tract infections
  - often considered for treatment of MRSA skin infections

Co-Trimoxazole (Bactrim/Septra)

- Resistance is more difficult because has to develop resistance to both drugs.
- Adverse effects include:
  - severe potential for dermatologic reactions,
  - GI upset,
  - blood disorders, and
  - drug potentiation.

Co-Trimoxazole (Bactrim/Septra)

- Available:
  - Bactrim/Septra tablets:
    - contains 80 mg trimethoprim and 400 mg sulfamethoxazole
    - dosing 2 tablets every 12 hours
  - Bactrim DS/Septra DS (Double Strength):
    - contains 160 mg trimethoprim and 800 mg sulfamethoxazole
    - Dosing 1 tablet every 12 hours
Herpes Zoster

1. Primary infection – Chicken pox (Varicella)
   • Usually in children
   • Highly contagious***
   • Very itchy maculopapular rash with vesicles that crust over after ≈ 5 days
   • 96% of people develop by 20 years of age
   • Vaccine now available

Herpes Zoster

2. Reactivation – Shingles (Herpes Zoster)
   • More often in the elderly and immunosuppressed (AIDS)
     – Systemic work up if Zoster in someone < 40
   • Can get shingles anywhere on the body
   • Herpes Zoster Ophthalmicus (HZO)
     – Shingles involving the dermatome supplied by the ophthalmic division of the CNV (trigeminal)
       » 15% of zoster cases

Herpes Zoster

• Symptoms:
  – Generalized malaise, tiredness, fever
  – Headache, tenderness, paresthesias (tingling), and pain on one side of the scalp
    • Will often precede rash
  – Rash on one side of the forehead
  – Red eye
  – Eye pain & light sensitivity
Herpes Zoster

• Other Eye Complications (Acute):
  – Anterior uveitis (most common ocular manifestation)
  – Acute epithelial keratitis (pseudodendrites)
  – Conjunctivitis
  – Stromal (interstitial) interstitial keratitis
  – Endotheliitis (disciform keratitis)
  – Neurotrophic keratitis

Herpes Zoster

• Associated factors include increasing age, immune deficiency and stress.
• Only people who had natural infection with wild-type VZV or had varicella vaccination can develop herpes zoster.
• Children who get the varicella vaccine appear to have a lower risk of herpes zoster compared with people who were infected with wild-type VZV.

Herpes Zoster

• A person’s risk for herpes zoster increases sharply after 50 years of age.
• Almost 1 out of 3 people in the United States will develop herpes zoster during their lifetime.
• A person’s risk of developing post-herpetic neuralgia also increases sharply with age.
Herpes Zoster

- Management includes:
  - oral antivirals:
    - 800mg acyclovir 5x/day
    - valacyclovir (Valtrex) 1g TID
    - famciclovir (Famvir) 500 mg TID
  - effectiveness of therapy is best started within 72 hours
  - oral steroids (clinical trials show variable results but often prescribed with antiviral to reduce pain)
  - management of pain (capsaicin, tricyclic antidepressants, gabapentin).
  - If ocular complications, consider topical steroids (Pred Forte QID).

NEW!! Shingrix HZ Vaccine

- Approved in US/Canada as of October 2017
- non-live antigen, to trigger a targeted immune response, with a specifically designed adjuvant to enhance this response and help address the natural age-related decline of the immune system
- Shingrix is 97% effective against shingles for people between the ages of 50 and 69 and 91% effective for people 70 or older.
- It is 91% effective against postherpetic neuralgia for people 50 and older.
- These rates are based on evidence presented to the committee from clinical trials with over 38,000 total participants.

NEW!! Shingrix HZ Vaccine

- recommended for healthy adults aged 50 years and older to prevent shingles and related complications
- recommended for adults who previously received the current shingles vaccine (Zostavax®) to prevent shingles and related complications
- the preferred vaccine for preventing shingles and related complications
Case

• 30 BF presents with eye pain in both eyes for the past several days
  – Severe pain (8/10)
  – Never had eye exam before
• PMHx:
  – Has chronic bronchitis
  – Rash on legs
  – Has recently lost weight and has a fever
  – Taking aspirin for pain

Ocular Health Assessment

• VA: 6/9 (20/30) OD, OS
• PERRL
• FTFC
• EOM*: FROM with eye pain in all quadrants
• SLE:
  – 3+ injection,
  – 3+ cells and trace flare,
  – deposits on endo (see photo)
• IOP: 18, 18 mmHg
• DFE:
  – see attached fundus image and fluorescein angiography.

Classification of Uveitis

• 4 main questions we need answered
  – Where is the inflammation located?
  – Is disease acute or chronic?
  – Granulomatous or non-granulomatous?
  – Unilateral or bilateral?
  – Granulomatous uveitis: typical work up includes ruling out sarcoid, tuberculosis and syphilis
Classification of Uveitis

- Secondary Questions:
  - Demographics of the patient
  - Has this happened before? If so did it respond to treatment?
- Systemic questions:
  - Lung/breathing problems?
  - Rashes/skin problems?
  - Joint problems or low back pain?
  - Urination issues?
  - Digestive problems – diarrhea? Bloody stools? Cramps?
  - Have you been out of the country recently?
  - Have you been in a wooded area? Ticks?
  - Any other systemic/autoimmune diseases?

Classification

- Classification is the key to the proper diagnosis and management of the uveitic patient
- Most common classifications
  - Anterior vs. Intermediate vs. Posterior vs. Panuveitis
  - Acute vs. Chronic/Recurrent
  - Granulomatous vs. Non-granulomatous
  - Infectious vs. Autoimmune

Anterior Uveitis Classification

- Acute, unilateral (or bilateral), non-granulomatous anterior uveitis
  - Idiopathic, HLA-B27, Herpetic, Behcet’s
- Chronic, bilateral (or unilateral), non-granulomatous anterior uveitis
  - JIA, Fuch’s Heterochromic, Idiopathic, Herpetic
- Chronic, bilateral (or unilateral), granulomatous anterior uveitis
  - TB, Sarcoid, Syphilis, VKH
Helpful Mnemonic

- Mnemonic for acute forms of non-granulomatous uveitis: **BLAIR G**
  - B: Behcet’s disease
  - L: Lyme disease
  - A: Ankylosing spondilitis
  - I: Inflammatory bowel disease (Crohn’s/ulcerative colitis)
  - R: Reactive arthritis
  - G: Glaucomatocyclitic crisis

Uveitis

- Clinical findings of:
  - circumlimbal hyperemia,
  - cells and flare in the aqueous and anterior vitreous, and
  - keratic and trabecular precipitates

Uveitis: Treatment

- “Classical treatment”:
  - Pred forte: every 1-2 hours, ensure taper
    - Pred forte: prednisolone acetate formulation which allows penetration through cornea to anterior chamber
  - Newer treatment option:
    - Durezol
Treatment Options

• Durezol:
  – Difluprednate
    • only difluorinated steroid
  – Steroid emulsion
  – BAK free
  – Increased “potency” so dosing needs to be less than “classical treatment” with Pred Forte
    • rough recommendation is 1/2 dosing of Pred Forte

Cycloplegics

• Cycloplegia:
  – used for reduction of pain,
  – break/prevent the formation of posterior synechiae
  – also functions in the reduction of inflammation

Treatment

• Topical administration is most common though periocular injections and systemic meds are useful for posterior uveitis and difficult cases
• Dosing is dependent upon severity of the inflammation
  – typically you want to hit the uveitis hard and fast!
    • E.g 1 gtt q 2hrs until the inflammation is gone!
    • If you have a minimal anterior chamber reaction then steroid may not be necessary at all
Treatment

• NOTE: it is crucial to taper your steroid treatment!
  – You will have a rebound inflammation if you simply remove your patient from their steroids...
  – The taper will be dependent upon how long you have had them on the steroid to get rid of the inflammation!
  – Typically, a slow taper is better in order to prevent rebound inflammation
  – If the patient has been on the steroid for less than a week a faster taper can be considered.

Follow-up

• Every 1-7 days in acute phase depending upon severity and every 1-6 months when stable.
• On each f/u visit the AC reaction and IOP should be evaluated
  – DFE should be performed for flare-ups, when VA affected, or every 3-6 months.

Follow Up

• If AC reaction improving, then steroid drops can be slowly tapered.
  – cycloplegia can also be tapered as the AC reaction improves.
  – slow taper recommended for chronic granulomatous uveitis.
Systemic Corticosteroids

• Prednisone
  – Available as Oral: 1, 2.5, 5, 10, 20, 50 mg tablets and 1 and 5 mg/mL solution and syrup

• Ocular Treatment Guidelines:
  – Mild to Moderate: Initial dose of 20-40 mg
  – Moderate to Severe: 40 – 60 mg
  – Severe: Begin with 60 mg and increase if necessary

Specific Conditions: Giant Cell Arteritis
  • 80-100 mg Prednisone
  • Consider IV Methylprednisolone 250 mg IV q6hours for 12 doses

Case: Gonzalez

• 33 HF presents with a painful, red right eye
  – Started a couple of days ago, deep boring pain
  – Has tried Visine but hasn’t helped the redness

• PMHx: patient reports she has been diagnosed with rheumatoid arthritis 3 years ago
  – Takes Celebrex for the joint pain
  – Patient reports she occasionally gets a skin rash when she is outdoors in the sun

• POHx: unremarkable
• PMHx: mother has rheumatoid arthritis
Case: Gonzalez

- VA: 20/30 OD, 20/20 OS
- Pupils: PERRL – APD
- VF: FTFC OH
- EOM’s: FROM OU
- BP: 130/85 mm Hg RAS
- SLE: see picture
  - 2+ cells, mild flare
- IOP’s: 16, 16 mm HG
- DFE: see fundus photo

Scleritis

- chronic, painful, and potentially blinding inflammatory disease that is characterized by edema and cellular infiltration of the scleral and episcleral tissues
- Symptoms of scleritis can include pain, tearing or photophobia, tenderness, and decreased visual acuity. The primary sign is redness.

Ocular Manifestations-Scleritis

- classified into anterior and posterior.
- Anterior:
  - Diffuse and nodular forms
  - Necrotizing (with/without inflammation) less frequent
    - Have the most serious systemic implications
  - Scleromalacia perforans
- Posterior:
  - characterized by flattening of the posterior aspect of the globe, thickening of the posterior coats of the eye (choroid and sclera), and retrobulbar edema.
Treatment and Management: Scleritis

- Scleritis treatment depends on both the type and severity.
- Aggressive treatment is necessary in order to prevent structural damage.
- Topical steroids (e.g. Pred Forte) have ease of use and relatively minimal side effect profile when compared to systemic therapy however, scleritis does not usually respond to topical corticosteroids alone.

Treatment and Management: Scleritis

- Subconjunctival/subtenon’s triamcinolone:
  - A multicenter retrospective case series of 68 eyes with either diffuse or nodular scleritis showed that 89.7% of eyes had complete resolution after a single injection.
  - There were no reported cases of perforation or scleral melt and the most common attributed side-effect of the triamcinolone was elevated intraocular pressure in 20% of eyes.
  - Only indicated in non-necrotizing forms.

Treatment and Management: Scleritis

- Oral NSAIDs:
  - considered first-line therapy for scleritis for their ease of use, cost, and relatively mild side effect profile for both anterior and posterior scleritis.
  - E.g. Ibuprofen 400-600 mg QID, Naproxen 250-500 mg BID, or Indomethacin 25-50 mg TID.
  - short term use of an NSAID is often well tolerated, NSAIDs can cause adverse effects which include peptic ulcer disease, hypertension, increased heart disease, bleeding, fluid retention, renal disease, and mood change.
Revised Recommendations on Screening for Retinopathy

- 2002 recommendations for screening were published by Ophthalmology
- Revised recommendations on screening published in Ophthalmology 2011; 118:415-42
  - Significant changes in light of new data on the prevalence of retinal toxicity and sensitivity of new diagnostic techniques
  - Risk of toxicity after years of use is higher than previously believed
    - Risk of toxicity approaches 1% for patients who exceed 5 years of exposure

“New” New Recommendations

- Recommendations on Screening for Chloroquine and Hydroxychloroquine Retinopathy – Ophthalmology 2016; 123:1386-1394
  - Released March 2016 from American Academy of Ophthalmology
  - Revised in light of new information about the prevalence of toxicity, risk factors, fundus distribution, and effectiveness of screening tools.

2016 Recommendations

- Maximum daily HCQ use of 5.0 mg/kg real weight, which correlates better with risk than ideal weight.
- Risk of toxicity is dependent on daily dose and duration of use.
  - At recommended doses:
    - Risk of toxicity up to 5 years is under 1%
    - Up to 10 years is under 2%
    - Rises to almost 20% after 20 years. However, even after 20 years, a patient without toxicity has only a 4% risk of converting in the subsequent year.
2016 Recommendations
• High dose and long duration of use are the most significant risks.
  – Other major factors are concomitant renal disease, or use of tamoxifen
• A baseline fundus examination should be performed to rule out preexisting maculopathy.
• Begin annual screening after 5 years for patients on acceptable doses and without major risk factors.

2016 Recommendations
• primary screening tests are automated visual fields plus spectral-domain optical coherence tomography (SD OCT)
• most patients of Asian descent will show initial damage in a more peripheral extramacular distribution near the arcades (require a 24-2 as opposed to 10-2 and OCT scans need to be analyzed further out)
Revised Recommendations on Screening for Retinopathy

- Parafoveal loss of visual sensitivity may appear before changes are seen on fundus evaluation
  - Many instances where retinopathy was unrecognized for years as field changes were dismissed as "non-specific" until the damage was severe
  - 10-2 VF should always be repeated promptly when central or parafoveal changes are observed to determine if they are repeatable
  - Advanced toxicity shows well-developed paracentral scotoma

Paracentral Scotomas

Normal Retina: VF/OCT/ERG

Copyright restrictions may apply.
Mild Maculopathy

![Diagram of Mild Maculopathy](image)


Bull’s Eye Maculopathy

![Diagram of Bull’s Eye Maculopathy](image)


Major Risk Factors

Table 1. Major Risk Factors for Toxic Retinopathy

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily dosage</td>
<td>High daily dosages of HCQ and CQ.</td>
</tr>
<tr>
<td>HCQ</td>
<td>&gt;5.0 mg/kg real weight</td>
</tr>
<tr>
<td>CQ</td>
<td>&gt;2.3 mg/kg real weight</td>
</tr>
<tr>
<td>Duration of use</td>
<td>&gt;5 yrs, assuming no other risk factors</td>
</tr>
<tr>
<td>Renal disease</td>
<td>Reduced glomerular filtration rate</td>
</tr>
<tr>
<td>Concomitant drugs</td>
<td>Tamoxifen use</td>
</tr>
<tr>
<td>Macular disease</td>
<td>Mir or affects screening and susceptibility to HCQ/CQ</td>
</tr>
</tbody>
</table>

CQ = chloroquine; HCQ = hydroxychloroquine.
Screening Recommendations

Table 2. Screening Frequency

Baseline Screening
- Fundus examination within first year of use
- Add visual fields and SD OCT if maculopathy is present

Annual Screening
- Begin after 5 yrs of use
- Sooner in the presence of major risk factors

SD OCT = spectral-domain optical coherence tomography.
Corneal Ulcers

- Infective bacterial and fungal corneal lesions cause severe pain and loss of vision
- Signs and Symptoms:
  - Pain, photophobia, tearing
  - Mucopurulent discharge with generalized conjunctival injection
  - Decreased VA (esp if on visual axis)
  - Possible AC reaction and hypopyon
  - Dense infiltrate
  - Satellite lesions around main lesion may indicate fungal infection

Associated Factors

- Contact lens wear, especially soft and extended wear lens
- Recent history of corneal trauma
- Topical steroid use
- History of exposure to vegetative matter (fungal etiology)

When to culture?

- 1,2,3 Rule:
  - 1 mm from visual axis
  - 2 infiltrates (or more)
  - 3mm or greater in size
  - Nosocomial infections
  - Immuno-compromised patient
  - Post-surgical
## Sterile vs Infectious Infiltrates

<table>
<thead>
<tr>
<th>Sterile Infiltrates vs. Infectious Infiltrates</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Sterile Infiltrates</td>
<td>Infectious Infiltrates (IMI)</td>
</tr>
<tr>
<td>Smaller lesion (&lt; 1 mm)</td>
<td>Larger lesion (&gt; 1 mm)</td>
</tr>
<tr>
<td>More peripheral</td>
<td>More central</td>
</tr>
<tr>
<td>Minimal epithelial damage</td>
<td>Significant epithelial defect</td>
</tr>
<tr>
<td>(Defect size compared to underlying infiltrate)</td>
<td>(Size of staining defect closely resembles size of underlying stromal lesion)</td>
</tr>
<tr>
<td>No mucous discharge</td>
<td>Mucopurulent discharge</td>
</tr>
<tr>
<td>Less pain and photophobia</td>
<td>Pain and photophobia</td>
</tr>
<tr>
<td>Little to no anterior chamber reaction</td>
<td>Anterior chamber reaction</td>
</tr>
<tr>
<td>No lid involvement</td>
<td>Lid edema</td>
</tr>
</tbody>
</table>


## Peripheral (Sterile) Corneal Ulcer

![Peripheral (Sterile) Corneal Ulcer](image)

## Infectious Corneal Ulcer

![Infectious Corneal Ulcer](image)
Corneal Ulcers

- The Steroids for Corneal Ulcers Trial (SCUT)
- Conclusions:
  - no overall difference in 3-month BSCVA and no safety concerns with adjunctive corticosteroid therapy for bacterial corneal ulcers
  - researchers did find significant vision improvement for one specific subgroup of the study by using steroid therapy on patients with severe ulcers
- Application to Clinical Practice:
  - Adjunctive topical corticosteroid use does not improve 3-month vision in patients with bacterial corneal ulcers unless in the severe category

ARMOR

- Antibiotic Resistance Monitoring in Ocular Microorganisms (ARMOR)
- Approximately 42% of isolates were determined to be MRSA
- Newer fluoroquinolones have better activity than earlier generations
- Besivance has the lowest MIC values of all the fluoroquinolones
- Vancomycin is drug of choice if MRSA present
- Azithromycin had very poor activity against Staph

Management

- Infective ulcers need to be cultured!
- If contact lens wearer, consider culture of contact lens
- Intensive topical antibiotic regimen, consider fortified preparations, subconjunctival injections.
  - loading dose of Vigamox/Moxeza/Zymaxid/Besivance 2gtt q 15 min x 1 hr,
  - 1gt q 30 min x 6 hours,
  - 1 gt q 1 hr until f/u in 24 hours.
Pseudomonas case report

“Doxycycline as an adjunctive therapy…may help to stabilize corneal breakdown and prevent subsequent perforation.”

AM. McElvanney

Cephalosporins: Hyperacute Conjunctivitis

- Hyperacute conjunctivitis:
  - usually secondary to gonorrhea or chlamydia.
  - profuse purulent discharge,
  - pain,
  - redness,
  - chemosis,
  - papillae,
  - positive nodes

Chlamydia: Treatment

- Recommended Treatment Regimens:
  - Azithromycin 1 g orally in a single dose
  - OR
  - Doxycycline 100 mg orally twice a day for 7 days
- Alternative Treatment Regimens:
  - Erythromycin base 500 mg orally four times a day for 7 days
  - OR
  - Erythromycin ethylsuccinate 800 mg orally four times a day for 7 days
  - OR
  - Levofloxacin 500 mg orally once daily for 7 days
  - OR
  - Ofloxacin 300 mg orally twice a day for 7 days
Gonorrhea Conjunctivitis Treatment

- For patients with uncomplicated gonorrhea conjunctivitis:
  - Single 250 mg IM injection
    Ceftriaxone (Rocephin) for the treatment of the gonococcal infection
  PLUS
  - Azithromycin (1g) for possible additional activity against gonorrhea plus possible chlamydia coinfection

Sinusitis Red Eye

- Sinus infections (rhinosinusitis), are an inflammation of the nasal and sinus passages that can cause uncomfortable pressure on either side of the nose and last for weeks
- The increase in mucus creates pressure in the sinuses that leads to pain.
- The sinuses surround the ocular region
  - Pressure from sinuses may feel like eye pressure.
  - Swollen sinuses and nasal membranes can push against ocular nerves resulting in pain.
- Most develop during or after a cold or other upper respiratory infection, but allergens and environmental irritants may also trigger them

Sinusitis Treatment

- The infection is likely bacterial and should be treated with antibiotics if:
  - Symptoms last for 10 days without improvement, or
  - Include fever of 102 degrees or higher,
  - Nasal discharge and facial pain lasting three to four days
- Because of increasing resistance to the antibiotic amoxicillin — the current standard of care — the ISDA recommends Augmentin
- Augmentin 250/500 TID for 5-7 days for adults, 10-14 days for children
Glaucoma
Fact or Fiction?

Joe DeLoach, OD, FAAO
Clinical Professor, UHCO
CEO, Practice Compliance Solutions

Financial Disclosures  Joe DeLoach, OD, FAAO
I Have Received Honoraria From or Served as a Consultant for:  (Partial Listing)

Vision Source
Alcon
Laboratories
Carl Zeiss
Meditec
Optos
Diopsys
Kowa
Optovue
AllDocs
OfficeMate
Marco
TSO
NVision
Cleinman
Partners
Vision Trends
Konan

Essilor of America
Pearle Vision / SNAPP
Vision West
UHCO, NOVA, RSO,
UAB, Berkley, and
other optometry
schools

Over half the state optometric associations in the United States

If you were here two years ago, you saw this quote - it is still true, maybe more true

“There is very little truth in our knowledge of glaucoma, or medicine in general for that matter. Even the accepted evidence of fact is highly suspect.”

Harold Quigley, MD
One of the Yodas of Glaucoma
Here’s another memorable quote from a very smart doctor to add to demonstrate our core knowledge of glaucoma

“To manage glaucoma you are being asked by yourself and your patients to be a modern day soothsayer. And your crystal ball is very foggy.”

George Spaeth, MD

So...WHERE’S THE BEEF?

This presentation is based on:

- Evidence based medicine (NEI studies)
- Consensus statements from the World Glaucoma Association (heavy emphasis)
- My opinion - based on 37 years of practice including treatment of countless suspects and confirmed cases
- Peer reviewed literature

The Glaucoma Studies

- Ocular Hypertension Treatment Study (OHTS), Early Manifest Glaucoma Trial (EMGT), Advanced Glaucoma Intervention Study (AgIS), Collaborative Initial Glaucoma Treatment Study (CIGTS), Rotterdam Study (RS), Diagnostic Innovations in Glaucoma Study (DIGS), Baltimore Eye Study (BES), Los Angeles Latino Eye Study (LALES), European Glaucoma Prevention Study (EGPS), Collaborative Normal Tension Glaucoma Study (CNTGS)
- The “youngest” of these studies is over twenty years old - 20 YEARS OLD
- Sorry...most of them just weren’t great studies

Singh et al. The Randomized Clinical Trial: Beware of Limitations. J Glaucoma. 12(2); 87-95. April 2004
So who can you trust?

Kuldev Singh MD – Director Glaucoma Services Stanford Medical School

“Evidence is either not available or incomplete to support much of what we do in the clinical management of glaucoma. Even the highest quality evidence may be limited by the artificial nature of the trials. There is clearly a place for clinical judgment and opinion to supplement the available evidence.”

Hence, my opinion, and yours matter!

Let’s Look At It This Way

- Who really deserves our attention
- What testing helps us tell them apart
- What really constitutes proper management of glaucoma
- Where is diagnosis and management headed
Why Should We Discuss Suspects

- Over-testing, especially in glaucoma, is a significant audit issue for optometry.
- Over-testing creates a negative billing profile (see Optometric Management February issue).
- PCS audit analysis showed 36% of all diagnoses of “glaucoma suspect” had no validity based on medical record documentation.
  Biggest issues:
  - C/D or IOP asymmetry...that wasn’t
  - “Cupping”...so what...
  - History...that wasn’t and doesn’t even count in Medicare
  - OHT...that wasn’t
  - No identifiable evidence at all...oh my

Just a little coding advice...

- “Cupping” does not mean glaucoma
- C/Ds are relatively meaningless (more later)
- Dad’s uncle had glaucoma...so what
- If you’re going to claim asymmetry (C/D or IOP), know what clinically significant means
- A “spot” on your FDT screening VF does not make for glaucoma suspect

Suspects – Too Obvious To Talk About

- Age
- Ocular hypertension
- Hyperopes
- Narrow angles
- Family history (but...)
- Race - Black, Hispanic, ASIAN (maybe we should talk)
Risk Factor: Family History

- Confirmed as risk fact only in the Rotterdam and Baltimore Eye studies
- Risk limited to first line blood relatives
- Highest risk – sibling (10-12X greater risk)
- Current KNOWN genetic factors account for only 5% of glaucoma
- Run a bunch of test because patient’s mother’s aunt had glaucoma…pay the money back

Risk Factor: Asian Ethnicity

Subtitle: We May Be Looking At The Wrong Suspects

- **Quigley 2010: 50% of glaucoma blindness in the world worldwide from Chinese populations**
- Eastern Asians (China, Japan, etc) have known anatomical risk for chronic narrow angle glaucoma due to flatter corneas and shallow / abnormal anterior chambers - MAYBE WE SHOULD PAY ATTENTION TO THAT!
- I contend our newest SECOND most significant glaucoma risk factor – the first comes later

Suspects - Need To Be More Obvious

Optic Nerve Anomalies

- The poles are still the target area - but why and what did Quigley say?
- ISNT isn't always right anymore. *(Pradhám etal ] Glaucoma Jan 2016 - ONLY 42% AT RNFL LAYER! “makes use of this parameter unlikely to be useful clinically”)*
- Most importantly, despite what VSP thinks (and too many doctors) C/D ratios without VDD are meaningless recordings
Remember, everyone pretty much has +/- the same amount of rim tissue (these are to scale!)

Suspects - Need To Be More Obvious
Vasculopathic Individuals
- In glaucoma - high is good...kinda, low is bad
- ANY vascular compromise can be an issue - diabetes, BP issues, migraines, Raynauds, etc
- But the biggie may be obesity

REALLY?

Let's run the statistics
- 36% of the population is considered obese (160 million)
- 80% of obese individuals have sleep breathing disorder (SBD) (128 million)
- 30-40% of SBD will develop glaucoma (51 million - at 8% risk factor there are only 15 million AA + Latino suspects)
- CONCLUSION: Obesity is likely the most significant risk factor for glaucoma

QUESTION: Should optometrists be counseling their glaucoma patients about losing weight?
**Obesity, sleep apnea and smoking are a deadly, blinding trio.**

**So let’s not ignore the facts**
1. Obesity is a significant vascular risk
2. Obesity - 10-14% higher in AA/ Latino
3. No correlation of obesity with income
4. BUT...72% of smokers are low income

**And remember what we’re dealing with...**

All vascular related glaucomas typically result in low/normal tension glaucoma...

**The hardest glaucoma to detect, confirm and treat**

**QUESTION**
For obese patients, or all low tension glaucoma patients for that matter, is glaucoma their most significant health concern?

**Misc. Risk Factors**
- Pigment Dispersion Syndrome - 30-50%
- Pseudoexfoliation Syndrome - 10-30%
- Chronic uveitis - 20%
- Iridocorneal Endothelial Syndromes - 50%
- Anatomical angle anomalies (narrow, plateau)
- Angle recession? Yes but - recession <180 degrees only 5% develop glaucoma - risk overall poor correlation with severity
- Phacomorphic/ Spherophakia (common - glaucoma is inevitable!)
The most common and risky anatomic risk factor

Spherophakic or phacomorphic lenses

This is what prompted Dr. Richard Lindstrom to make the statement “cataract extraction is the best glaucoma surgery we have available”

Risk Factor: Medications

- Steroids yes...95% in POAG BUT average increase only 4-5mmHg
- Incidence of true steroid response is generally unknown. Estimates as high as 5% of general population reported but true incidence probably MUCH lower
- Systemic beta blockers (surprised? LOW BP!!)
  - HERE’S THE REAL CONCERN: Any muscarinic or mydriatic agent in a narrow angle situation (antihistamines, antidepressants, H-2 blockers, incontinence drugs, vasoconstrictors)

CCT Risk Factor? Not so fast...

Central Corneal Thickness

1. Is an independent risk factor for glaucoma
   NO
2. Contaminates your IOP reading?
   Depends...and likely minimal concern
Is Corneal Thickness an Independent Risk Factor for Glaucoma?
Felipe A. Medeiros, M.D., Ph.D.

In conclusion, the results of Brandt et al suggest that the use of CCT correction formulas for GAT measurements is probably of little value in clinical practice. Instead of attempting to use these formulas, clinicians are probably better off incorporating risk information as provided by validated predictive models for glaucoma development. However, the conclusion that CCT is a true independent risk factor for glaucoma is not validated at this time and requires further investigations.

More???

Ophthalmology 2012 - Weinreb MD

"The results of this report (OHTS) have been mistakenly interpreted by some as demonstrating that CCT is an independent risk factor for the development of glaucoma."

Also conclusion of the Early Glaucoma Manifest Trial (EMGT), Diagnostic Innovations in Glaucoma Study (DIGS) and Collaborative Normal Tension Glaucoma Study (CNTGS)
So – what about CCT **NOW**

- Hommer et al, Wang et al, Asoaka et al, Mitz et al (all reporting as far back as ARVO 2008) – CCT **NOT** related to risk for glaucoma. Ocular rigidity (hysteresis) is which is not correlated to CCT
- Mee et al – No correlation between CCT and VF loss or progression of VF loss
- Medeiros 2011 – CCT had no correlation with deforming effects at level of optic nerve

*WE COULD GO ON AND ON AND ON*

And that messing up your IOP reading thing

The issues:

- Only applanation tonometers *(more later)*
- With the true accuracy of tonometers *(more later)*, does 2-4mmHg really matter?
- The conversion formulas do not work *(because Goldmann cheated)*

*LET’S LOOK AT THAT CONCEPT…*

CCT Correction Doesn’t Make That Much Difference and It Doesn’t Work

**ADJUST OF 12mmHg IOP (per Eilers formula)**

**MAX 4mmHg adjustment – at the extremely thin CCT**

**AND WHAT ABOUT THIS MESS?**
CCT Correction Doesn’t Make That Much Difference and It Doesn’t Work

ADJUST OF 20 mmHg IOP (per Ehlers formula)

MAX 5 mmHg adjustment – at the most extremely thin CCT

AND WHAT ABOUT THIS MESS?

CCT Correction Doesn’t Make That Much Difference and It Doesn’t Work

ADJUST OF 35 mmHg IOP (per Ehlers formula)

MAX 6 mmHg adjustment – same story at a CCT of 460!!!

AND WHAT ABOUT THIS MESS?

Not just my opinion...

NIHMSID - Brandt et al

“The calculation of individual risk for developing POAG in ocular hypertensive individuals is simpler and equally accurate using IOP as measured rather than applying an adjustment formula to correct IOP for CCT”

National Institute of Health….just sayin
In the past six months I read FOUR optometry articles stating pachymetry is a “standard of care” in glaucoma management

**REALLY?** “Trying to be more precise than this (standard applanation without adjustment or consideration of CCT as a risk) is not supported by the data and may be harmful to patient care”

Jamie Brandt MD - Director Glaucoma Services UCD and original OHTS investigator

Which Brings Us To Hysteresis!

- **CONFIRMED** risk factor for developing glaucoma
  - Wells et al. (IOVS, Aug 2008, 49(8))
- **CONFIRMED** risk factor for progression
  - Medeiros et al. (Optimal 2013, 20(8))
- **CONFIRMED** ORA as accurate as Goldmann but technician driven – technically more accurate as not influenced by biomechanical properties of cornea
- No disposables, no anesthetic
- Corneal hysteresis is billable (92145)

What Tests Make the Diagnosis?

How Reliable Are Those Tests?
IOP

- ACCURATE? In human hands, Goldmann accurate to +/- 2mmHg AT BEST
- Others are as essentially as accurate – ORA, ICare, Tonopen (severe calibration issue!) – most all except standard NCT
- Goldmann NOT the most accurate – Pascal (DCC) is
- Target IOP?
- Remember – the goal is to prevent vision loss, NOT ACHIEVE AN ARBITRARY GOAL IOP

IOP – Current Truths

Lui 2012
- IOP highest at night? Not really...actually only 60%
- Daytime IOP highest in AM? Not really...actually only 55%
- Regulation of IOP may be more of a factor of cerebrospinal fluid than ocular dynamics – translaminar CSF proposed to be auto-regulatory component of IOP
- Over 70% of Blacks and 90% of Whites presenting with IOP >25mmHg will NOT have glaucoma, at least on initial presentation
- Long term variability, circadian variation not established as risk.

Main diagnostic factor associated with MEAN IOP

Gonioscopy

- Purpose is to determine the facility of outflow
  WE NEED TO REDEFINE THIS – WHETHER THE TRABECULUM IS OPEN IS MORE IMPORTANT THAN WHETHER THE ANGLE IS OPEN
- Anterior segment OCT analysis almost NEVER a substitute for Gonioscopy – can augment (remember the Asians?)
- Accuracy and repeatability dependent mainly clinician competency but patient cooperation and corneal clarity can be issues
- THIS IS A STANDARD OF CARE ISSUE IN GLAUCOMA!
RNFL Analysis

- Direct observation (fundus cameras with red free are great at this! AND your BIO!)
- Scanning laser (peripapillary & papillomacular)
- Repeatability well documented
- Significant “data base” issues (red and green disease)
- What about when what YOU see and COMPUTER sees do not agree???

MORE ON THIS LATER!

And Blood Flow?

WGA says will be the most important concept of glaucoma diagnosis and management
Once we are able to clinically measure it

SO CAN WE?

We Do Have Our First Clinical Tool

OCTA – OPTIC NERVE BLOOD FLOW (pretty impressive)
Side by Side

COMING SOON! Overview Report
Provides Disc Health at a Glance

One scan generates report showing:
- OCT Intensity
- RPC
- RPC Density
- RNFL
- Cup/Disc

COMING SOON: AngioDisc Trend Analysis
Comments / Opinions on the Concept of Pre-Perimetric Glaucoma

- Do structure and function really coincide and we just are not smart enough to figure out how to show that? **Answer:** Probably yes! BUT?
- That 40% of NFL loss before first visual field defect idea? **No longer valid** - says who?
- Is there any scientific evidence to support that treatment of “pre-perimetric” disease prevents vision loss?

Visual Fields - 2018

FINALLY - technology has now made acquisition of reliable, repeatable visual fields a piece of cake

Visual Fields - 2018 Truths

- World Glaucoma Association consensus statement - white on white strategy still the standard
- Rest based on testing an isolated group of RGCs. **Truth Update** - glaucoma does not effect isolated groups of RGCs but that strategy sometimes can increase sensitivity due to isolating a smaller set of test cells
- Historic consensus - HFV is the Gold Standard. **Truth Update** - not so fast!
Visual Fields – 2018 Truths

- What is a defect – common truth is any contiguous, non-edge points depressed by at least 6dB (repeated 3X?)
- What is a change in the defect – defect must decrease by an additional 3dB (repeated 3X?)
- Historic consensus (?) – diagnosis by indicies is a valid concept. **Truth Update** – VF data is already screwed up. The more you massage it, the more screwed it likely gets.

Are Visual Fields Still Valid in World of Glaucoma

- Pre-perimetric diagnosis – obviously not
- Post-perimetric diagnosis standpoint, still a preferred practice pattern if not standard of care issue
- From a monitoring for disease progression standpoint, still a flawed essential

**WGA Consensus** – SAP is still considered “Gold Standard” and trend progression analysis is essential

Do You Need to Spend $20K+?

**iPad Visual Field App**

*AMJ Ophthal Nov 2017*

“Almost as accurate as SAP, especially for moderate to advanced disease”

**FREE of course**
What Tests Make the Diagnosis

Psychophysical testing
- Genetic testing? WGA says no way - not yet
- Contrast sensitivity (not just your chart)
- Color vision (extended)
- ERG (mainly asymmetric amplitude)
- VEP (delayed latency)

A WORD ABOUT ERGs

Five Limitations of Psychophysical Testing
1. Most of data is limited - no large, controlled, INDEPENDENT studies (except ERG)
2. No valid normative data base for diagnosis
3. No valid normative data base for progression
4. Poor coefficients of repeatability (mainly VEP)
5. Payers consider not medically necessary

SO THAT MEANS DON'T DO THEM???

But Joe....
- You didn’t mention provocative glaucoma testing - we use 92140 a lot and sometimes get paid!
- And you didn’t mention serial tonometry - we use 92100 on all our glaucoma encounters and get paid every time!
What Tests Make The Diagnosis

Very Important

Any analysis like this is difficult, variable and always based on the assessment of risk factors and the given findings.

_Glaucoma diagnosis is NOT always easy or clear_

Identifying Patients with Swine Flu

**SYMPTOMS**

- Elevated temperature
- History of exposure
- Headache
- Runny nose
- Sore throat
- Diarrhea or vomiting

**SIGNS**

Identifying Patients with Glaucoma

**SYMPTOMS**

None!

**SIGNS**

- IOP
- CD
- RNFL
- VF
- ERG

65 y/o BM - IOP 20mmHg OU
So What Do I Do?
- Establish your own belief system: pre or post perimetric; structure + function requirements; what tests mean the most to you
- Never rely on any single piece of data to make a decision related to glaucoma
- NEVER move fast. With the exception of angle closure, no one loses vision from glaucoma in a month or two - or more - or never?

I like to think of it like making a soup

Your Last, and Possibly Biggest Hurdle in Glaucoma Management:
PROGRESSION
Weinreb 2004

With minimal intervention (decrease IOP) and often NO intervention - the majority of glaucoma patients do not show progression that would cause a functional deficit.

SO, HERE’S THE DEAL

With minimal intervention (decrease IOP) and often NO intervention - the majority of glaucoma patients do not show progression that would cause a functional deficit.

"Glaucoma is like life...we know we can’t win but maybe we can lose very slowly"

Stephen Drance, MD

You do not know who is going to be a “yellow liner”!
Per Tony Rinaldi...another guru

Three kinds of “glaucoma” patients:
- Those who are diagnosed with glaucoma but don’t really have it
- Those who have glaucoma but will not lose vision even with treatment
- Those who have glaucoma and will likely lose vision even with treatment

Can’t a guy/girl get a break here?

And the experts say...

Anders / Hejil 2012

“Disease progression rates in glaucoma vary widely among patients...and cannot be predicted taking any risk factors into account”

That Means What?

WE ARE SPENDING BILLIONS OF HEALTHCARE DOLLARS AND TONS OF OUR TIME AND OUR PATIENT’S TIME MONITORING A DISEASE THAT WON’T REALLY HURT A GOOD PORTION OF CONFIRMED GLAUCOMA PATIENTS

BUT DO WE HAVE A CHOICE?
So how can we try to measure progression

- Intraocular pressure *(not really)*
- Optic nerve appearance
- Visual Fields *(REALLY difficult)*
- RNFL *(difficult, limited to mild to moderate disease and DEFINITELY over analyzed)*
  - Conventional RNFL imaging
  - Ganglion cell analysis

---

Progression by Visual Fields

Viawntham BJ Ophthal 2003

Panel of “experts” (MD glaucoma specialists) asked to independently evaluate a set of visual fields with 100% PERFECT reliability for potential progression. Results:
- 33% said the fields showed progression
- 33% said the fields showed no progression
- 33% said the findings were inconclusive

**IMPRESSED? And on the 4th retest?**

---

EVALUATING VISUAL FIELDS FOR PROGRESSION

**CONCLUSION – WGA CONSENSUS**

There is no accurate, independent, unbiased analysis of visual field results, much less changes in them. Best is TREND progression analysis.

Trend Analysis most useful predictor – but remember is still based on a house built on sand!
“It should be obvious that the presence of visual field loss by itself would obviate the need for using any imaging instrument to diagnose or monitor glaucoma in clinical practice” Lisoba 2012

REALLY? Maybe - and WHY? And some payers are thinking that way!

Progression by OCT

What constitutes change in OCT measurements

- Thinning change greater than 4-6um - Roh 2013
- Variability of 2-10um - Mwanza 2010
- Longitudinal changes greater than 11.7um (really?) - Leung 2008

CONCLUSION: Be suspicious of changes exceeding 10um

Keeping All This In Mind - Let’s Answer This. Do I Really Need To Be Worried About Changing From One OCT to Another?

- Got it yet??? Most ALL the data is suspect at best
- We are WAY too wound up in DETAILS with glaucoma and not looking at overall patterns of change
- Personally, I wouldn’t worry about it so much (and I have lived through FOUR different OCTs)
What did the glaucoma gurus say was **THE most valuable information needed to detect glaucoma progression**

- IOP?
- Visual Fields? *(8% SENSITIVITY)*
- OCT? *(14% SENSITIVITY)*
- Ganglion cell analysis?
- VEP/ERG?

*Despite limitations, it was **SUBJECTIVE, serial optic nerve analysis***

---

**Treatment of Glaucoma**

*Most of this can be fast...*

---

**Think Treatment is Complicated?**

- **DIAGNOSIS** is complicated. Treatment is easy.
- Our only CURRENT conventional treatment is lowering IOP
- All glaucoma meds do that in one of the following ways:
  - **Inhibition of aqueous secretion**
  - **Enhancing trabecular outflow**
  - **Enhancing uveo-scleral outflow (DEBATED)**
  - **Combination of the above mechanisms**
Significant Point

Don’t try to memorize. Think about what medications do to physiology – that will tell you what the mechanisms do and what most of the side effects are.

How They Work – Good and Bad

**Beta Blockers (production)**
- Slow down BP, heart, lungs, peripheral blood flow - *can this possibly be good for a glaucoma patient?* Next...

**Carbonic Anhydrase Inhibitors (production)**
- Regulates cell membrane transmission - kinda important!
- Topicals have few side effects
- TID / BID? About the same
- Work really well – especially one of them!

How They Work – Good and Bad

**Alpha-2 Adrenergic Agonists (production and outflow?)**
- Beta blocker plus CNS depression - *can this possibly be good for a glaucoma patient?* Next...

**Pilocarpine (outflow?)**
- No idea how it works – lots of side effects, kinda
- Actually works...good “non-compliance patient” choice
How They Work – Good and Bad

PROSTAGLANDINS

- The newest miracle drug? Perspective - it is 2018!
- They work by creating inflammation - are you confused???
- They work GREAT - 30+% - but all the time?
- Major, legitimate, totally overlooked side effects - what did Quigley say?

Get real – which do you think your patient is most concerned about???

OR #1 #2

AND ONE PGA IS BETTER THAN THE OTHER - RIGHT???

ACTUALLY PROBABLY YES...BUT NOT BASED ON EFFICACY
Believe What You Will…But Science Supports BAK is BAD for you!!

Quartenary Ammoniums - Used for WHAT?

- Organic solvents (Jiffy Lube!)
- Pesticides
- Wash down genetic labs
- Spermicide
- The biggie… *used to treat ICK!*

**Combination Products**

- **Cosopt**
  Combination of dorzolamide hydrochloride 2% and timolol maleate 0.5%
- **Combigan**
  Combination of timolol maleate 0.5% and brimonidine 0.2%
- **Simbrinza**
  Combination of brinzolamide 1% and brimonidine 0.2%

*ALL HAVE BAK! (except new Cosopt PF)*
Additive Medications

• First, make sure you have a really good reason to prescribe additional medication.
• Is there an order of increasing additive effectivity? Probably not – but CAI may have a slight advantage.
• Beta blocker AM only – esp. LTG
• OPTOMISTIC Expectations:
  - second drug – 2-3mmHg
  - third drug – 1-2mmHg
  - fourth drug – forget it

What About Alternative Medicine? Simply My Opinion

NO DOUBT ABOUT IT!!

• Omega-3
• Ginkgo biloba
• Melatonin
• Mirtogenol

Alternatives Update

Omega-3 (2-3gm QD)
Effect appears to be due to significant anti-inflammatory and anti-oxidant properties

Ginkgo (40mg TID)
Improves blood flow, reduces oxidative stress, improves mitochondrial activity in RGCs

Melatonin (10mg HS)
Anti-oxidant properties, regulates sleep cycle, proposed neuroprotectant effect

Mirtogenol (80mg QAM)
Anti-oxidant properties, but shown to decrease IOP equivalent to latanoprost at 24 weeks!
Now – the Future(?) of Glaucoma Tx

First interesting concept – **CHRONOTHERAPY**

Customized timing of treatment based on patient's highest IOP reading as determined by continuous IOP monitoring device – Triggerfish, Sensimed, Bionode, Equinox

New Available Medication

*(not many pharmacies yet)*

**latanoprostene bunod (Vyzulta) – Valeant/ B&L**

- PGA benefits plus…
- Nipradilol – nitric oxide (NO) donating beta blocker:
  - Decreases aqueous production
  - Increases TM outflow
- Progression control that exceeds the additional IOL lowering effect *(PROPOSED neuroprotective effect – likely more vasorelaxing effect of NO)*
- **ISSUES?** PGA side effects (none from NO); $375

Very Soon Available Medication

**netarsudil (Rhopressa) - Aerie**

- Novel triple action medication
  - Inhibits rhokinase (ROCK) and norepinephrine transport (NET)
  - ROCK and NET decrease episcleral venous pressure 35%
  - ROCK increases TM outflow
  - NET decreases aqueous production
- **ISSUES?** QD dosing; Almost NO SEs; could be over $400
Right on the heels of Rhopressa – the game changer

**netarsudil + latanoprost (Roclatan) - Aerie**
- Novel FOUR action medication
  - ROCK and NET decrease episcleral venous pressure 35%
  - ROCK Increases TM outflow
  - NET decreases aqueous production
  - **PLUS** however PGA lowers IOP
- 25-30% more IOP reduction than PGA alone – QD dosing
- **ISSUES?** PGA SEs; no idea on cost

**ROCLATAN EXPECTED TO BE DRUG OF CHOICE BY 2023 - EVEN AT EXCESSIVE COST**

---

**BUT...drops on their way OUT!**
Replaced by sustained release, lasers and MIGS

SEVEN sustained release products on their way to market – most notable:
- Latanoprost - punctal plug delivery (lasts 1-6 months)
- Travoprost - punctal plug delivery (lasts 1-6 months)
- Bimatoprost - subconjunctival depot (lasts a year)

---

**Anything new in lasers? YOU BET!**

**Annual SLT**
- Kinda like getting your teeth cleaned every year - yearly repeat of SLT
- Treat entire 360 degrees at reduced power
- Several studies show continuous IOP control at 200% better than one time SLT
- No idea on how many years you can repeat
But the biggie is....

**MIGS**
(minimally invasive glaucoma surgery)

**Why?**
- They work - duh...
- They are “minimally invasive” - MINIMAL COMPLICATIONS
- They erase compliance issues
- They are cost effective
- They are performed predominantly by general surgeons during cataract surgery

---

**MIGS Examples**

- **Endocyclophotocoagulation**
  - Disables ciliary process aqueous production
  - 20-30% sustained IOP reduction
- **Trans-scleral cyclophotocoagulation**
  - Similar to endocyclophotocoagulation but can be performed ab externo
  - Can be performed with MPL
  - 20% sustained IOP reduction

---

**MIGS Examples**

- **Trabectome**
  - Electropulse system destroys and aspirates TM unfolding upper Schlemm's canal
  - 30-40% sustained IOP reduction
- **Schlemm's canal scaffold implant**
  - Flexible tube (noodle) inserted into Schlemm's canal - pressure on inner wall increases outflow
  - 20-30% sustained IOP reduction
The MIGS Game Changer
Microshunt (InnFocus)

- Microshunt creates direct communication between anterior chamber and the subconjunctival space
- This is a trabeculectomy with FAR LESS complications
- Sustained IOP reduction of **50%**

Conclusions

- **KEEP UP** - this stuff changes all the time
- Be slow to assume a diagnosis or progression - you rarely have to make quick decisions in most glaucoma
- Consider the **PATIENT** - inconvenience, cost and lifestyle implications
- Collect serial data...as much as you need but no LISTS and you must follow practice patterns
- **MOST OF ALL** - do NOT be the doctor who runs a bunch of tests then just refers the patient to an OMD

Last Comment

Despite yours or anyone's best efforts, people go blind from glaucoma. As an optometrist, you are better suited than any ophthalmologist to manage the visual disability and blindness from glaucoma. “**Don't be the last one to see an eye before it goes blind**” is an antiquated concept
GLAUCOMA SHOULD BE OPTOMETRY’S DISEASE!
jwdeloach@uh.edu
THANK YOU!
Compliance: Can’t I Just Be a Doctor Any More?

Joe DeLoach, OD, FAAO
CEO, Practice Compliance Solutions
Clinical Faculty, UHCO

Disclaimer

Joe is the non-equity CEO of Practice Compliance Solutions and Clinical Professor at the University of Houston College of Optometry. My opinions do not necessarily reflect the opinions of UHCO. This presentation does not promote any product or service including Practice Compliance Solutions. I am not an attorneys (fortunately) and do not provide legal counsel.

Here’s the deal...
You have two choices
1. Comply with the law
2. Gamble
You are over 10X more likely to have a compliance violation filed against you than a malpractice claim...and the average compliance fine or lawsuit dwarfs the average liability claim penalty.

**WHAT ARE WE MISSING HERE?**

<table>
<thead>
<tr>
<th>Scary Facts 2014-2016</th>
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<tbody>
<tr>
<td>HIPAA 154,400 complaints in 2015</td>
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<tr>
<td>➢ 69% result in fine (OCR data)</td>
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<tr>
<td>➢ Estimated 60% increase in 2016</td>
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<td>OSHA - 5,162 OSHA/CDC small business violations 2014 (OSHA data)</td>
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<tr>
<td>HR – Biggest and FASTEST CHANGING compliance area. Feds estimates 77% of all small businesses are NOT in compliance</td>
</tr>
<tr>
<td>Dramatic increase in medical fraud and abuse - more on that later!</td>
</tr>
</tbody>
</table>

**Why the increase in the rest?**

IT’S VERY SIMPLE!

$4 BILLION in cash cow

GOVERNMENT CASH COW
Compliance the Hard Way

“Experience is something you don’t get until just after you need it”
Steven Wright

NONE of the many ODs who get in trouble never thought they would get in trouble with the government

HIPAA

“One of the most worthless laws ever written”
Joe – CEO PCS

How Most Doctors See HIPAA

By national statistics (OCR & OIG), about 60% of you listening are NOT HIPAA compliant!
Right Now... $10,000 MINIMUM fine for ANY, even minor HIPAA violation

Moderate ($5,000 - $150,000 per violation per day)
Compliant but significant omission or breach

“Reckless indifference”
That can cost you $250,000.00

• Criminal Penalties
Up to $500,000 fine and felony jail time

And its not just hospitals and big clinics?

Why the increase in HIPAA?

Medscape 4/28/14
Stolen EHR Charts Sell for $50 Each on Black Market
Credit cards are worth about a buck!

NEWS FLASH!
HIPAA is about IDENTITY THEFT – not health care information
So...Are YOU HIPAA compliant?

You are legally obligated to do all this and have doctors AND STAFF that are prepared to answer patient’s questions about their rights and your policies

NOT UPDATED = NOT COMPLIANT

Laundry List of Other Privacy Issues

<table>
<thead>
<tr>
<th>Authorizations</th>
<th>Medical records review</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marketing</td>
<td>Medical records request</td>
</tr>
<tr>
<td>Minimum Necessary Rule</td>
<td>Request to change medical records</td>
</tr>
<tr>
<td>Incidental Disclosure Rule</td>
<td>Requests for disclosure documentation</td>
</tr>
<tr>
<td>Business Associates</td>
<td>Individual privacy accommodations requests</td>
</tr>
<tr>
<td>HIPAA Breach</td>
<td></td>
</tr>
</tbody>
</table>

You and your staff must understand all of these issues and they must be addressed in your Privacy Manual

HIPAA legislation totals 2712 pages of legal mumbo jumbo. You can try to do this yourself or work with a compliance company who has already done it!
Security Rules

Security Rule Requirements

• Appoint a Security Officer
• Conduct a risk analysis and risk management plan to determine threats or risks in your operational systems
• Complete the Organizational Requirements
• Documented policies and procedures for all applicable Security Standards – Security Manual

And that is how involved? Another 1,000 pages of legal mumbo jumbo

OCR Tenets of Security Risk Analysis and Management

• FLEXIBILITY - the Security Standards do not prescribe a specific policy, software and other course of action
• SCALABILITY – and do not hold large and small business to the same standard!
• UNIQUE – your risk analysis is unique to your business

Be wary of "experts" telling you that you MUST do certain things under the Security Rules

WHAT IS A SECURITY RISK ANALYSIS?

SECURITY IS MORE THAN COMPUTERS
SECURITY IS ABOUT YOUR OFFICE LAYOUT, LOCKS, ALARMS, FLOODS, HURRICANES, BACK UPS, LOCATION, PASSWORDS, PAPER RECORDS, EMPLOYEES, OFFICE POLICIES

COMPUTERS ARE JUST ONE COMPONENT OF THAT
YES IT IS COMPLICATED BUT IT CAN BE MADE EASY, UNDERSTANDABLE AND NOT A HUGE FINANCIAL BURDEN
So How Do I Get Caught? (true of all the compliance issues)

- Whistleblowers (90%)
  - 1st – unhappy patients
  - 2nd – unhappy current / former employees
  - Opportunists, trained by the government

- Actions of staff

- Random audits
  - Permanently funded in HIPAA
  - New to OSHA

The MAJOR HIPAA Issues That Result In Finding Yourself Meeting with the Feds – 2016 Data

1) No or incomplete compliance ("gambling doctors")
2) Lost laptops / back up tapes
3) Employee ignorance or stupidity
4) Stability (lack of) of your EMR / network
5) Inadequate / absent usernames & passwords
6) Hacks – main ones are identity theft and ransom

Summaries of New HIPAA Regs

- Breach responses – MUST have a plan
- Encryption in medical records
- Legal healthcare communications
- Medical records release – BIG UPDATE
- Patient non-discrimination
- Patient’s rights regarding YOUR use of THEIR insurance
- Marketing regulations

Don’t know ALL this? You’re still gambling!
MONDAY 2/6/17

$2.3 MILLION against a Dallas, Texas clinic for failure to encrypt (or equivalent) patient email communications. AND THERE WAS NO BREACH INVOLVED!

- Patient is informed method is not secure, AND;
- Patient is informed regarding consequences of non-secure transmission, AND;
- Patient provides authorization (written?)

Communication Issue #2
Communication to ANYONE else

MUST use:
- Secured practice/patient portal
- Text (not defined)
- Secured email (encrypted)
- FAX (allowed but not as secure)

Not so fast on that text thing...

JUST IN CMS 12/2017
Communication to other doctors ONLY by encrypted email, encrypted text service or FAX!
NO STANDARD TEXTING
Medical Records Update
HHS CFR 164.524 – Provider FAQ 11/16

• Is any authorization required from the patient to release medical records to the patient or to anyone else at the patient’s request? NO – actually discouraged – cited as a “barrier”. NOT the patient’s request, still a good idea. (Side note: What have they already consented to?)

• Can the patient demand the format of release? YES – paper or electronic no matter if YOU have paper or electronic

Medical Records Update
HHS CFR 164.524 – Provider FAQ 11/16

• Can I charge the patient for their records? Strongly discouraged – only actual copy cost; If you have EMR – NO charge or $6.25

• Can I charge someone else for a copy patient records? If requested by the patient or if involved in TPO – actual cost or if you have EMR NO charge or $6.25

A New 2016 HIPAA Issue

Section 1557 of the ACA

• Another jewel hidden inside a six year old law no legislator ever read

• Basically extends non-discrimination, like we already have with employees, to our patients
Requirements

Doctors should not discriminate against an individual for **ANY** reason – Section 1557 specifically makes it illegal to discriminate based on:

- Sex
- Age
- Race
- Color or national origin
- Disability status – the biggie!

The Big One...Disability

Can be physical or mental, yes...but this law more addresses a new concept called LEP

*Limited English Proficiency (LEP)*

LEP is classified as a disability, one you are now required by Federal law to accommodate. Unlimited civil penalties!

Think this is BS...one healthcare attorney firm cited this law as one of the greatest retirement programs in history

Human Resources

One of the most complicated, most dangerous and potentially most financially devastating compliance issues facing doctor employers
Let’s Start With What Has NOT Changed

How Most Doctors See HR

By EEOC (government) statistics, at least 77% of you listening are NOT HR compliant!

WE ARE HR COMPLIANT!

Federal and State agencies

• State Employment Commission
• State Commission on Human Rights
• State Communicable Disease Prevention and Control Act
• State Workers Compensation Act
• Equal Employment Opportunity Commission
• Americans with Disabilities Act
• Department of Labor
• Wage and Hour Act
• Family Medical Leave Act
• Fair Labor Standards Act
• Occupational Safety and Health Administration
• Center for Disease Control
• Federal Privacy Act
• Immigration Reform and Control Act

HR issues you MUST understand

• Necessity of employee manual
• Legal hiring / firing
• What you CANNOT ask in an interview
• Checking credit reports
• Criminal background checks
• New Hire reporting requirements
• Discrimination laws
• Smoking laws
• Guns on property laws
• Avoiding embezzlement
• Employee poster requirements

• FLSA—salary vs hourly
• Contract labor laws
• Breaks, meals, time off requirements
• Jury leave, military leave, voting leave
• Employee surveillance
• Disability requirements
• Family Medical Leave
• Harassment – Sexual and more
• Pay requirements
• Social media regulations
• COBRA
• Legal / proper terminations ... and many more...
Two most important HR issues

1. ALL these laws are written to “protect and serve” your employee – NOT YOU. We are in the age of “Minimal Employer Rights”
2. Your employees likely know more about HR laws than you do. And what they do not know attorneys are willing to teach them

Most Important Slide on HR

• You MUST have a complete, state law specific employee manual – and you have to keep up with the frequent changes (HR is fastest changing compliance area in small business law)
• Policies must be enforced fairly, consistently and without discrimination
• If you delegate HR responsibilities, that person MUST know what they are doing

JUST IN: January 18, 2018

EEOC issues directive to “go after” small healthcare businesses.

WHY?

QUOTE: The research indicates ignoring HR laws makes them a “big, fat, juicy target”, “low hanging fruit”, “like shooting fish in a barrel”
Specific to Wyoming law...

1. You hire a new employee Monday – you are required to report the new hire to the online state employee reporting agency?

2. Your employee works from 8:00am to 5:00pm – the voting polls are open from 7:00am to 7:00pm. You must adjust the employees schedule or pay them one hour of regular pay for time to vote?
   THAT IS TRUE!

3. Failure to display all the correct Federal and Wyoming employee posters can result in a fine of up to $7,500 per day?
   THAT IS TRUE! Get them FREE – https://www.dol.gov/elaws/posters.htm

4. You may restrict an employee from keeping a gun in their car in your parking lot?
   EVER PLAY SCRUPLES?

5. Wyoming is an “at will” state – you can hire and fire for any reason you want
   THAT IS ABSOLUTELY NOT TRUE

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Most Significant NEW HR Issues

- Lack of employee manual (policies)
- Discrimination
- Harassment – “me too”
- Improper employee classification
- Contract labor
- I‐9 regulations
- OTHER NEW STUFF!!!!

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Three fastest routes to Dante's Inferno

- Discrimination
- Harassment
- FLSA
Discrimination

So you all know you cannot discriminate on the basis of age, sex, religion, sexual preference, race, color, creed, national origin, disability status or history, military service status or history…on and on

What are the “new kids on the block”?

New Kids on the Block

• Appearance based on religious belief (national law)
• Pregnancy (national law)
• Obesity (national law…maybe for now)
• Prior criminal activity (state specific for now)
• Salary history – “equal pay” (state specific for now)

Male, Female, Transgender, Cisgender, Agender, Pangender, Bisexual, Gender Neutral, Gender Fluid – DISCRIMINATION ISSUE???

Is it ever! The questions have blown up and EEOC and OSHA have already answered – right down to restroom regulations.

In HR, an employee’s gender is what the employee believes it to be
A comment on “15 or more employees” and Discrimination

Sounds like a way out for some of you but DO NOT bank on it.
Discrimination is THE hot topic and courts are routinely upholding discrimination claims against employers with less than 15 employees. Be very careful with discrimination (DON'T DO IT!)

Harassment

Let’s not talk about this….let’s just so NO!

Fair Labor Standards Act

• Controls lots of things but one of main ones is classifying your employee as salaried or hourly
• The current system of classification is based on three tests – it is next to impossible to pass the three tests and classify any optometry employee as salaried!
• The first test, the SALARY TEST got a lot of airplay over the past two years but means NOTHING! It’s the DUTIES test that makes it next to impossible to pay on salary basis!
And the duties test is...

**Administrative Exemptions**

To qualify, all of the following tests must be met:

- The employee must be compensated on a **salary** basis (as defined in the regulations) at a rate not less than $455 per week.
- The employee’s primary duty must be the performance of office or non-manual work directly related to the management or general business operations of the employer or the employer’s customers; and
- The employee’s **primary duty** includes the exercise of discretion and independent judgment with respect to matters of significance. (Better read what this means – can they hire/fire; make large purchase decisions; sign your checks and control bank account; make operational changes... ALL without your approval!)

[https://www.dol.gov/whd/overtime/fs17a_overview.htm](https://www.dol.gov/whd/overtime/fs17a_overview.htm)

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**FLSA – A Federal Witch Hunt**

- **REALLY?** Yes really – messing with this cost an OD friend of ours $108,000 last year!
- Heard of “Bridges to Justice”?  
- FLSA does not apply to “licensed professionals” (they fall under a Professional Exemption)
- Employee works unapproved overtime – you can discipline them but you HAVE to pay them *(don’t believe the “experts” on optometry blogs)*

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**While we’re at it - other blog misinformation!**

- “ODs are exempt from OSHA – only MDs have to comply”
- “You can fire an employee for whatever reason you want – we’re ‘at will’”
- “You can bill a scanning laser and photo at the same time as long as you use different diagnoses”
- “When a patient returns for dilation only you can bill extended ophthalmoscopy and get paid”
- “It’s OK to bill fundus autofluorescence under the unlisted procedure code”
- “You can bill refractions only when the insurance will pay for it because it is a non-covered service”
I-9: 2017 Need to Know

- Form has changed – and an online version available
- MUST use new form for all new hires or re-hires after January 21, 2017
- Fines vary from $375 - $2,725 PER employee / PER DAY for failure to produce a valid I-9
- I-9 is simply identity confirmation – some states still require documentation of immigration status
- Must keep all old I-9 forms (3 yrs from date of hire or 1 yr from termination – whichever longer)


OH SNAP – and they changed it AGAIN effective 07/17/17!

Contract Labor

- Why? Clear as can be - avoid taxes and benefits. Nice idea, but...
- Government likes this idea? RIGHT!
- OD employers are exempt from this?
- The national trend is what?
- The “rules” aren’t clear?
- Who is at risk here?

Six Economic Realities Factors

1. Is the work an integral part of the employee’s business? If yes, not contract labor! [need to go any further?]
2. Can employee’s make management decisions that impact their profit or LOSS? If no, not contract labor!
3. Has the employee invested substantially (similar to employer) into the resources needed to complete the work? If no, not contract labor!
4. Are the particular employee’s special skills essential to the operation and management of the business? If yes, not contract labor!
5. Permanent or indefinite? If permanent (even by renewable contract), not contract labor!
6. Degree of employee control – hours, locations, type of work, fees, etc. If not total control, not contract labor!
MISC New Issues in the Cooker

• Contract labor, criminal history, salary history – all discussed

In the wings...

• Mandatory sexual harassment training for supervisors
• Mandatory Sick Pay laws

Having fun yet?

HAZARD (OSHA/CDC)
Let’s Start With What Has NOT Changed

By PCS survey, at least 90% of you listening are NOT OSHA/CDC compliant!

The Hazard Players

- Office of Health and Safety Administration (OSHA)
  ➢ maintaining a safe working environment for your employees and your patients
- Center for Disease Control (CDC)
  ➢ preventing spread of infection
- Clinical Laboratory Improvement Amendments
  ➢ ensuring quality of lab test results

Hazard Requirements

- Must have written policies that comply with OSHA safety and CDC Universal Precaution standards
- Must have documented evidence that your staff is trained on Federal and YOUR policies in these two areas (trained AND TESTED)
- Must now meet the extensive requirements of the Globally Harmonized System – the completely re-written OSHA (effective 6/1/2015 - oops)
A MAJOR focus of OSHA is dangerous chemicals in your office – like Windex

Optometrists have to comply with OSHA, CDC and CLIA?

- Yes, no matter how much you deny it
- Non-compliance penalties can include
  - fines in excess of $10,000 - $50,000 - $129,336 for “reckless indifference” or “repeat” violations (define “repeat” Joe)
  - civil law suites AND criminal charges
  - termination of your third party payor contracts including VSP and EyeMed (it’s in your provider contract!).

**BIGGEST ISSUE**: OSHA can padlock your door and not remove it until you can demonstrate complete compliance

How Is OSHA / CDC Gonna Git ya?

**Most Common Causes of Inspections**

- Employee compliant (34%)
- Targeted / random (22%)
- Referral from other Federal agency (18%) – WHAT???
2015 Daugherty Ruling

• *Paraphrase* – OSHA has given private health care providers a free ride too long – in 2016 we are going after them.
• They learned from HIPAA – ongoing random audit project
• And they have recruited the public to help!

OSHA in a nutshell...

One of the biggest PITAs you will encounter with a bite that is much worse than the roar

Don’t think they are serious? This is for YOUR employees and patients! You are willing to ignore this?

OSHA INSTRUCTION manual for the public! (including YOUR patients)

You would do this why?
Yes we know...
Much of this is really stupid
BUT IT IS
THE LAW!

Fraud and Abuse
The newest compliance pain – but not really

Fraud & Abuse
This is NOT Stupid
• There are five Federal fraud and abuse laws – can you name them?
• You swear, under conviction of perjury, you know them every time you file an insurance claim!
Ever read what you sign?

- In submitting this claim for payment from federal funds, I certify that 1) the information on this form is true, accurate and complete 2) I have familiarized myself with all laws, regulations and program instructions available from the Medicare contractor 3) I have provided or can provide sufficient information required to allow the government to make an informed eligibility and payment decision 4) this claim complies with all Medicare program instructions and...

lists all five Federal F/A laws you can’t name!

And...

“My signature is to certify that the foregoing information is true and accurate. I understand that any false claims or statements or concealment of a material fact may be prosecuted under applicable Federal and Stark laws.”

Good luck saying you did not know better

Five Main Fraud and Abuse Laws

1. False Claims Act
2. Anti-Kickback Statute
3. Provider Self-Referral Law
4. Exclusion Statute
5. Civil Monetary Penalties Law

If you don’t know what these laws say, you need to do some personal homework (or we can make it easy for you with the PCS Fraud and Abuse Manual)
Talk to someone, but get compliant!