SOCCEP Q/A
Monday, March 30

Time	Session Details	Question	Answer
10:00 – 11:00 am	*Marisa Ciamacca, OD, MS, FAAO* *When and How to Perform Corneal Culturing*	Are these videos recorded? can we watch these videos later?	Yes! Following the conclusion of the entire program, faculty members will have access to the content. You will need to request the recordings from your faculty member.
		Why do we need to culture if there is several infiltrates? Thanks!!	Several infiltrates can represent the ability of a microbe to "spread" or invade multiple locations of the cornea and so accuracy in treating these microbes is key – it can also raise your suspicion for fungal keratitis (i.e. satellite lesions). If a patient has several central or mid-peripheral infiltrates, the decision to culture becomes easier from the location of the ulcer alone. However, if they have several peripheral infiltrates that you feel you can explain (pt slept in CL's last night and woke up with painful red eye and you are suspicious of CL associated infiltrates, or pt has significant blepharitis with infiltrates where the lid margin meets the cornea) consider treating first and then culturing if no response or if the patient worsens. If you cannot explain why the patient has several peripheral infiltrates, consider culturing right away as herpes and MRSA can create this appearance. I hope this helps!
		Will you re-explain why mucus is not helpful in a culture?	Mucous is essentially necrotic tissue made as a by-product from the bacteria, fungus, etc. It contains dead epithelial cells, a lot of WBCs and sometimes RBCs. Unfortunately, the actual microbe is rarely found in the mucous itself (they hang out inside the infiltrate) so culturing the mucous doesn't provide any benefit from identifying the source of the

			infection.
		do you have patient's waking up at night to put fortified drops in q1h when they start?	Yes – we try to hit it hard! If the patient does not wake up at night, they can go 6-8 hours without any medication. There is no rule for how long they need to wake up at night, but even just doing this for the first 48-72 hours can be a huge help.
		On case 1 for day 6, did you still continue cyclopentolate in addition to pred?	This patient was done using her cyclopentolate by Day 6 because her hypopyon and AC reaction had resolved (she also did not feel that her comfort was improved very much with the cyclo). But, she was still taking Pred (with Gentamicin) to help reduce long term scarring.
		When you specify using the fortified antibiotics every 1 hour or every 30 minutes, is the patient using both drops at those times, 5 minutes apart? Or alternating? (I've seen some fortified antibiotics that are combos and some in individual bottles so just wanted to clarify your plan! Thanks!)	The compounding pharmacies in this area fill the medications in separate bottles, so we have the patient use them 5 minutes apart!
		Are you continuously culturing even after your dx? TIA!	We aren't – unless the patient is not responding to their current therapy or starts to develop atypical clinical features. Here are a few scenarios below that I hope help: Initial Culture does not need repeated when: Positive OR Negative culture returns and the patient is responding well to current therapy. Initial Culture does need repeated when: Positive OR Negative culture result returns, but the patient is NOT responding to current therapy.
		is there a reason you would wait a few days before starting on doxy and	You can start these medications ASAP. Sometimes the patient (and,

		Vit C?	admittedly, the clinician) can become overwhelmed with the number and frequency of medications needed at the initial visit(s) to treat their corneal ulcer which causes them to hold off at first. But, you can absolutely add these medications on Day 1 (one of our OMDs always starts Doxy/Vit C right away).
		Is there a reason why you started on TWO topical anti-fungals? Is that because they're weaker in effectivity to natamycin?	Topical Voriconazole is effective against filamentary types of fungal keratitis (such as Fusarium, which are also most common), but will not treat non-filamentary keratitis (i.e. candida/yeast, more common in immuno-comprimised patients). Amphotericin B is non-selective and treats both fungal types. By adding Amph. B, we gain coverage for non-filamentary keratitis, but we also add a new mechanism of action for how we "attack" filamentary keratitis as it works differently than Voriconazole. There are a few case reports showing patients who were proven by culture to have Candida albicans keratitis (non-filamentary) that were treated successfully with Voriconazole alone, and oral Voriconazole is used in systemic Candida cases, so this medication may have broader spectrum coverage than initially speculated. You do not have to start with both, but the added coverage and double MOA is why both were used here!
		Easy being a Monday morning quaterback and we are generally reluctant to use antifungals without some evidence of mycotic infection…..but with an endothelial plaque why not think fungal (co-infection) from the onset in Case 2?	Endothelial plaque-ing is ultimately a non-specific WBC response that points to deep stromal involvement. Unfortunately, it is not pathoneumonic for one type of keratitis over the other. However, both fungal keratitis and pseudomonas are two of the most likely culprits to create a significant AC reaction (including endothelial

			plaque) and hypopyon overall. I think because pseudomonas made the most sense for this patient (given his history of CL wear and that he was topping off his solution – both which are major risk factors for pseudomonas) we suspected that the AC reaction and subsequent hypopyon were from bacteria. Admittedly, I think we could have considered fungal involvement sooner for this patient – a classic case of maybe relying on the initial culture result to much! You would not be wrong to add fungal coverage right away on this patient – the only thing to consider is the cost of extra medication to the patient and maybe not knowing which medication is "working" to help dictate how/when you taper.
		Do you do a wash out period before reculturing?	Typically we do not – if the patient is not responding well to their current medication than it is unlikely that medication will significantly alter the culture results (with the exception of AK unfortunately). Additionally, almost all patients with an ulcer and large epithelial defect require antibiotic coverage even just as prophylaxis and it could be risky to stop this prior to reculturing.
		Can you email the links for the studies that you referenced?	SCUT: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3830549/ SCUT 12 month: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3946996/ MUTT: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3769211/ MUTT1: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6044431/ AK Review Study: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4330640/

		Why two different biocides in your AK case? Usually Brolene + one biocie (either PHMB or chlor. .02%)	AK is a tough one to beat and unfortunately we do not fully understand the nature of this amoeba just yet. In the U.S. the most accepted, current therapy is treatment with both Chlor. and PHMB – the main reason for this is that, while both medications act on the trophozoite and cystic form, their individual efficacy depends on a large number of things (the stage of the disease being treated, strain of amoeba the patient has contracted, ability of the medication to adhere to that patient's cell molecules, etc). So, both are typically used to increase the chance that the patient will respond. Additionally, because it is difficult to eradicate AK cysts, combination therapy is thought to be beneficial not only medically, but also from a toxicity standpoint to break down the cystic walls. Researchers actually feel BAK could help treat AK in the future because of how toxic it is (but this of course will affect healthy host tissue as well). Brolene can certainly be used (typically with Chlor. as some studies suggest this medication may be slightly superior to PHMB in most cases), but it is not currently available in the U.S. Patients can order the medication online I believe (without a prescription!) through an Australian or New-Zealand website where the medication is approved. We used this protocol on a patient in our office once, and he resolved without recurrence. I wish I had a better answer for why we only use one biocide with Brolene – I cannot find any reasoning behind this in the literature. If you start with Brolene and one biocide, and the patient is not responding well, maybe consider switching to or adding the

			second biocide given the information above!
11:00 – 12:00 pm	*Joseph Kane, OD, FAAO* *Take A Second Look: Subtle (And Not So Subtle) Eye Disease*	due to the asymmetry of the glaucoma in patient #1, are you ordering a head scan or carotid ultrasound?	Hello, and thank you for your question. We did not order ancillary systemic testing/work-up for this patient. Her cupping was the most significant optic disc abnormality, which is most likely related to glaucoma. If there was evidence of optic disc edema or pallor, I think that further investigation into underlying systemic etiologies would be reasonable.
		would you say the nerves are tilted or malinserted? and how does what complicate assessing the vessels at the nerve?	Hello, and thank you for your question. Yes, I agree that her optic nerves do have a myopic and tilted/malinserted appearance. This does complicate assessment of her cupping/optic nerve integrity. It's one of the reasons I shared this case! I think even still, you can appreciate that the neuro-retinal rim is quite thin inferiorly, as evidenced by the appearance of the vasculature at the disc in this area. Fortunately, we don't have to rely on only one test and can see if there is agreement across multiple tests/exams: DFE, OCT, HVF, photos, etc.
		Without an IOP>21mmHg, What are some tell-tale signs to prompt you to start testing (OCT&fields) for early NTG besides drance heme, systemic conditions, and family Hx?	Hello, and thank you for your question. I think any investigation into glaucoma testing (OCT, HVF, etc) has to be initiated based on the appearance of the optic nerves. The other things you mention (family history, systemic disease, etc) are risk factors that might heighten suspicion for glaucoma, but the decision to pursue testing typically is based on suspicious features of the optic nerve itself (in this case, inferior cupping/notching in the left eye).

		Would it be worth it to order a CRP and ESR on this patient too?	Hello, and thank you for your question. We did not order ancillary systemic testing/work-up for this patient. Her cupping was the most significant optic disc abnormality, which is most likely related to glaucoma. If there was evidence of optic disc edema or pallor, I think that further investigation into underlying etiologies would be reasonable.
		For case 1, how long before the VF was retested to confirm the previous results? Did you have a conversation about SLT/drops at the initial exam or did you wait for the follow-up field? Being that it was a relatively young new patient presenting with noticable thinning on DFE and confirmed via same day OCT/VF I would be tempted to initiate treatment sooner rather than later. Would it be too soon to initiate a drop day 1 if she would have been ok with drops or better to wait and confirm VF? I also understand not wanting to overwhelm her day one since she needed to be referred to retina due to macular issues.	Hello, and thank you for your questions. Good questions! I believe we asked her to return in about 1 month to repeat the visual field, obtain an additional IOP reading, and re-visit the topic of how/when to treat. As you mentioned, the more "urgent" matter to address was the concern for sub-retinal fluid, implying a conversion from dry to wet AMD. So the first thing we wanted to do was connect with ophtho-retina. Since her IOP was not severely elevated, I think we have some time to repeat testing and obtain a few IOP measurements before deciding on treatment. I probably wouldn't start drops/treatment based on one visit alone unless there was an emergent concern, but these decisions have to be individualized based on the case as a whole.
		Would it also be appropriate to address the fact that the PCP/Neurologist prescribed sumatriptan to a 79 year old? Could it be a factor in contributing to her vascular eye problems?	Hello, and thank you for your question. It's an interesting thought. My understanding was that this patient was on this medication for many years (was not recently prescribed). We felt that her sub-optimally controlled blood pressure was more likely to be the underlying etiology for BRVO. But it's smart to look at if the medications may be playing a role.
		How urgent was the referral to	Hello, and thank you for your

		ophtho for the BRVO-induced ME? What about referrals for CRAO/BRAO/BRVO?	questions. She was asymptomatic, so the timing/onset was uncertain. I typically try to connect patients like this within 1-2 weeks, but this depends on a great many factors (access to specialty care, transportation issues, etc). I ended up calling the specialist in this case, and she ended up being seen same day. But I think within 1-2 weeks would have been reasonable. The other etiologies you mention each require individualized management decisions based on various factors. There are great preferred practice patterns available online that I would encourage you to reference (these are updated periodically): https://www.aao.org/preferred-practice-pattern/retinal-vein-occlusions-ppp https://www.aao.org/preferred-practice-pattern/retinal-ophthalmic-artery-occlusions-ppp
		If we saw an asymptomatic plaque in a private practice setting , how urgent is referral for cardiac work up and do we send to PCP or ER?	Hello, and thank you for your question. It's a good question. If the patient is acutely symptomatic with an ophthalmic artery occlusion (OAO), central retinal artery occlusion (CRAO), or branch retinal artery occlusion (BRAO) – these patients should be immediately referred to a stroke center for acute work-up. If the patient has an asymptomatic, non-occluding retinal embolus, these patients should still be investigated but the guidance is less clear (non-emergent typically). Of course, these discussions have to be individualized to the specific case. Typically, I communicate with the patient's primary care provider via phone call or letter when I see a retinal embolus on exam.

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			There are great preferred practice patterns available online that I would encourage you to reference (these are updated periodically): https://www.aao.org/preferred-practice-pattern/retinal-vein-occlusions-ppp https://www.aao.org/preferred-practice-pattern/retinal-ophthalmic-artery-occlusions-ppp
12:00 – 1:00 pm	*Andrew Mick, OD, FAAO* *Bilateral Sequential Non-Arteritic Anterior Ischemic Optic Neuropathy*	Is it possible to re-watch this lecture, and if so where?	
		Hi Andrew…..sleep apnea??	Good question. There several systemic diseases and systemic states (sleep apnea, renal dialysis, cardiac surgery, orthopedic surgery, hematologic malignancy, hyper coagulable states, etc), along with ocular states (disc drusen, peri-ocular surgery, etc) are linked to NAION. You are correct that sleep apnea has been shown in several studies to be linked to NAION, and the level of evidence is probably getting close to the threshold to be included in the "traditionally accepted risk factors" along with diabetes, hypertension, hyperlipidemia, and smoking. I just didn't know how many of these less established risk factors I should include in the talk. Thanks for listening and asking the question.
2:00 – 3:00 pm	*Alice Grasso McCaslin, OD, MS, FAAO** *Bilateral Optic Nerve Damage Mimicking Glaucoma*	Was any MRI/imaging done?	No imaging was done on this patient since the RNFL loss was so stable and the visual field did not appear to have any neurologic appearing defects. See below.

		Was the potential of an infectious disease considered as a differential, such as tuberculosis, syphillis, or other inflamatory conditions?	We had looked through the patient's other health records (both recent and historic) within the hospital system and there was no evidence of any infectious disease or other inflammatory condition systemically. Because of that, and the stability of the RNFL thinning, we did not consider any infectious or inflammatory causes. Had we seen active optic neuritis, we may have looked further at infectious/inflammatory causes.
		why not scan to see if there's any evidence of demyelinatiopn from the past?? or the rare chance he has a pituitary involvment? Also red cell folate, methymelanic acid should be included in bloodwork	While imaging (MRI) would be the best way to determine if there was any tissue damage, tumors, etc. posteriorly from optic nerve to chiasm to brain, since this patient's RNFL thinning was stable and VF defects did not appear neurologic (plus normal VA, color vision, pupils) an MRI was not considered necessary at that time. The patient was also having regular CBCs done, which were all normal. Since his systemic health records did not show any malnutrition or alcoholism, and CBC was normal, we did not feel we needed to address B12 or folate levels in bloodwork at that time – but those would be worth investigating!
		How long do you want to keep the pt on glaucoma monitoring	We plan to monitor him regularly for the rest of his life. With that said, if the RNFL thinning and VF defect stay stable over the course of years, it would be worth reevaluating the return interval. That is, switching him from every 6 months to every year if these defects are not changing.
3:00 – 4:00 pm	*Sowmya Srinvas, OD, MS, FAAO*	Do you perform B-scan over closed or open eyelid?	Closed eyelids

	Choroidal Melanoma	when do you use HVF 120? whats the difference between HVF 120 v. 10-2 v. 20-2 v. 30-2?	120 point screen was to get an overview of the VF loss versus narrowing it down with 10-2/30-2/24-2
		Are you looking for anything specific on the liver panel for possible evidence of metastasis?	Liver Function Tests (alkaline phosphatase, AST, ALT, or bilirubin) are done and elevated findings (1.5 to 2 times upper limit of normal) on LFT prompted a diagnostic or imaging test to confirm or rule out metastasis
		How emergent is referring the patient according to the size?	Size is only one component however it is important to follow the TFSOMUHHD mneumonic to refer patients.
		would it be pertinent to do an OCT over the melanoma?	Yes because it can help identify subretinal fluid which is a risk factor for conversion to melanoma
		Hi Dr. Srinivas, thank you for the lecture. So I just saw a melanoma this past fall and was wondering how common is it to see rapid growth from nevus to melanoma?	You're welcome. Here is an article from JAMA that answers your question. Shields CL, Furuta M, Berman EL, et al. Choroidal Nevus Transformation Into Melanoma: Analysis of 2514 Consecutive Cases. *Arch Ophthalmol.* 2009;127(8):981–987. doi:10.1001/archophthalmol.2009.151
		I was just wondering how common it is to see a rapid progression of a nevus into a melanoma?	Here is an article from JAMA that answers your question. Shields CL, Furuta M, Berman EL, et al. Choroidal Nevus
4:00 – 5:00 pm	*Denise A. Valenti, OD, FAAO* *Cannabinoids: Retina Model of Cholinergic Function*	Is this only assessing smoking marijuana and not the other forms that are available?	This research was only for smoked marijuana and it was to have been either simple vape or plant. But as noted we did have one person who used a dab product. Ingested, edibles have a time delay that can

			be very unpredictable. Future work will include edibles and dab products.
5:00 – 6:00 pm	*Tamara Petrosyan, OD** *Dark Without Pressure*	Can dwp cause vf defects?	From what I have read and seen, no it does not because it is so small an area and the photoreceptors are not necessarily dead, just damaged.
		First, thank you. I first ran across dark without pressure a few months ago. I am still trying to figure out how it can change shape or disappear. As in the PIL moves?	No the PIL stays damaged, the theory is that blood flow re-appears in the area and so the appearance changes - but again, there is really not a lot of research or published data on this.
		How would you differentiate dark Without Pressure from prominent choroid?	DwP is usually a smaller, well demarcated area and if you are able to get an OCT through it you will find a missing PIL
6:00 – 7:00 pm	*Jack Phu, OD, PhD, FAAO* *Glaucoma or Not Glaucoma: A Rapid Fire Review*	Can you please go over what you are looking for with retinal nerve fiber layer refelectivity?	In general (with reference to glaucoma): • A “wedge” shape – wide far away from the disc, tapering towards the disc • Contiguity to the disc: loss should extend in towards the disc • With reference to the comment below, a good idea may be to use a red free (green) filter in some cases • Fundus photograph helps, but be careful not to bleach the photo • Concordance with the NFL heat map on OCT or similar imaging devices • The notion of “feathering”: this is when the nerve fibre layer reflectivity becomes naturally sparse or less reflective far from the disc. Its key features are that there are little slits between the

			fibres, but the "defects" do not extend to the disc and thus are not suggestive of glaucoma Distinguishing between glaucoma and non-glaucoma: • If the NFL defect is very deep BUT the disc defect is not, then you may be more suspicious for a non-glaucomatous aetiology • If there is an accompanying RETINAL explanation, then you are thinking less of glaucoma… e.g. a cotton wool spot that may precede nerve fibre drop out, or vascular attenuation (early in the disease course… this may occur in late glaucoma, but if the disc looks fine, then it is not suggestive of glaucoma) Overall, glaucomatous RNFL loss should be accompanied by visible changes at the optic nerve head that are colocalised (e.g. cupping, notching, thinning etc).
		Any tips on identifying RNFL dropout when examining the fundus on DFE	• Red-free (green) filter as noted above have been suggested by some people • My preference is to start at the disc, have the beam height at least three times the height of the disc so you can see the adjacent areas (you are dilating the pupil so light should be no problem), beam width should be at least twice the width of the disc. Then, look at the disc: is there any focal loss suggestive of glaucoma? Then given the focal loss, follow into the retina, checking especially between the blood vessels for loss of reflectivity. In a dilated pupil the reflections should be minimal, which in an undilated pupil, is the key source of difficulty identifying defects

			Apply the same principle when examining a fundus photograph (with respect to glaucoma examination): start at the disc and go into the retina… If there is an obvious retinal lesion of interest, start there instead and examine for a "non-glaucomatous" optic neuropathy
		How do you know that this patient has glaucoma vs old BRAO?	The easiest way is to check for disease progression after removing treatment. However, the other clues why this is not a classically glaucomatous presentation: • Visual field defect very deep – not as common in glaucoma • Ganglion cell analysis map showed a very deep and focal loss not in an arcuate pattern typical of glaucoma, i.e. that the locus of change must be a focal inner retinal change (related to the inner retinal vasculature), rather than a progressive loss of structure connected to the optic nerve head • Attenuation of the retinal artery in a focal region only – not as common in non-endstage glaucoma (this can happen as per Paul Mitchell's work in the BMES and the Singapore group, but not to the extent visible here and on OCT-A) • Very unilateral and asymmetric (didn't show the other eye, but it was grossly asymmetric…. Note that around 10% of glaucoma cases can be unilateral or very grossly asymmetric, and hence those cases would be diagnoses of exclusion) As noted above, glaucoma is a diagnosis of exclusion – the ideal situation would have been to exclude a vasculopathy prior to a

			diagnosis of glaucoma
		I may have missed it, but what does drinking water has to do with IOP measures?	• Water drinking test is thought to measure trabecular outflow facility. As you know, aqueous outflow facility is the primary driver of intraocular pressure in the eye, and its impairment is a risk factor for pressure elevation and therefore glaucoma • Water drinking test is a measure of osmotic stress… similar to the glucose tolerance test performed in people suspected of having diabetes. In patients with suspected trabecular outflow facility impairment (i.e. glaucoma), it is an index that raises suspicion. See further details below
		What was the diagnosis for the last patient? I may have missed it.	In this case, we had a tentative/presumed diagnosis of non-glaucomatous ischaemic optic neuropathy
		Can you go into more detail about the water drinking test? I am not familiar.	I'll go through the process of water drinking test here. Unfortunately it's not a fully standardised test yet, but we follow some of the more recent literature in terms of how to do it: • Patient to liquid fast (no fluids) for 2+ hours prior to clinic visit • Baseline pressures (fasted) taken • Patient given 10 ml/kg body weight to drink in the space of 5 minutes • Pressures taken every 15 minutes following water drinking • Spikes in pressures signify suspected trabecular outflow impairment Uses of the water drinking test: • Diagnosis: if suspicious for glaucoma but want to identify

			another risk factor suggestive of glaucoma • Treatment: identifying best initial treatment – does the pressure peak high and suitable for SLT? • Post-treatment: identifying whether fluctuations are still occurring… is the treatment working Surrogate measure of pressure peak compared to home intraocular pressure phasing, which around 20% of people cannot perform on their own
		If there is GCA thinning but RNFL is normal, beginning of VF loss, pressure wnl, do you consider it as serious as RNFL thinning but GCA normal with a beginning of VF loss? Thx !!	Essentially this question is asking which we'd to be more serious: RNFL or GCA loss (presumably in the context of glaucoma?). This can be answered in a number of ways. Firstly, there is little conclusive evidence to say one might be more affected compared to the other in early glaucoma. Depending on the anatomy of the eye, some individuals may have true RNFL loss first prior to having true GCA loss… e.g. in the case of a high RNFL projection or trajectory. The opposite can also occur. I stress here that the neural loss needs to be "true"… you can have mimickers in the form of, say, a tilted disc syndrome or high myopia, which would not be concerning because they are non-progressive and non-glaucomatous. Secondly, we can consider what effect either an RNFL or a GCA loss may have on the visual field. In GCA loss, one would presumably have central visual field loss, versus more peripheral loss occurring in the context of the RNFL projections. To answer this question, we would

			need to look at the visual field loss in question: many visual field grading schemes and national guidelines refer to the presence of significant central visual field loss to be “advanced” glaucoma and hence would be more concerning. Therefore, yes, in the context of GCA losses with concordant central visual field losses, the glaucoma may be graded as more severe. However, you would also need to consider the question at the patient level: does the patient even use their central vision? My most commonly used example is a person who is illiterate but works on a farm. They require excellent peripheral vision, but do not spend any time reading. In their case, they may value their peripheral vision more than their central vision. Understanding this is critical. Finally, I would also be cautious about the relative dynamic ranges of the RNFL and GCA measurements. Statistically significant GCA losses may trigger more caution simply because the measurement variability is smaller, hence statistical reduction may be more likely to occur. In contrast, RNFL measurements around the optic nerve head can be more variable, affected by the peripapillary structures such as PPA and the retinal vasculature.
		For the first case, did we refer out for the retinal detachment OS since it was mac-on? Also, did we give him a new spectacle Rx for his myopic RE or do we bring him back after the RD repair to issue a new spectacle Rx?	The retinal detachment was part of their ocular history and before they presented to our clinic. It had been treated using retinopexy. We don’t refract patients routinely in our clinic (we are a referral service for ocular diseases, and so this remains the responsibility of the

			referring ECP). Good question about the spectacle rx though. The prediction would be that retinopexy that far off in the periphery should not really affect the spectacle rx, but a scleral buckle no doubt would. So yes: refract after RD repair
		Can you reiterate your 3 take home tips?	Each message was "built" on the previous: 1) In the glaucoma examination, examine not just the optic nerve head but the surrounding retina and macula as well (there may be another source of inner retinal atrophy) 2) Utilise imaging modalities to look for patterns of glaucomatous loss. If the pattern on imaging is NOT typical of glaucoma (but more like retinal/vascular loss), then consider an alternative, non-glaucoma diagnosis. Use other clinical tests to assess the risk factors for glaucoma (effectively a Bayesian approach where you weigh up the evidence)… this includes intraocular pressure profiling, OCT-A and longitudinal monitoring.

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10:00 – 11:00 am	*Kelsey Moody Mileski, OD, FAAO** *Double Vision Rapid Fire*	Can you redefine reverse deviation?	In regards to a right cranial nerve 3 palsy, they have a left hypertropia (right eye can't go all the way up) in up-gaze and a right hypertropia (right eye can't go all the way down)
		Student here - if I'm correct, a 3rd nerve palsy is usually either microvascular or from an ICA/PCoA aneurysm. Are there other conditions that might affect only the superior or inferior divisions in isolation?	Yes! Those are the two most common causes however other etiologies do exist, like a compressive mass or inflammation/infection. CN III splits into the superior and inferior division as it enters the superior orbital fissure. The superior division could be affected if a mass was in the superior orbit or vice versa. A compressive etiology would likely be the most common culprit.
		How can we differentiate between a CN4 palsy and a IO overraction?	It can be challenging. A true IO overraction is actually controversial. If it does occur it is typically bilateral. Most IO overraction is secondary to SO palsy. Here is a great article reviewing this: https://www.hindawi.com/journals/joph/2019/9713189/
		Can you explain Cn 3 pattern in more detail? Also what is PSP again?	Pattern of a CN3 palsy. Let's say we have a **right** cranial nerve 3 palsy. There may be a mild or significant deficit in elevation, infraduction and adduction. Therefore, on up-gaze, you will have a left hypertropia (right eye can't go all the way up). On down-gaze, you will have a right hypertropia (right eye can't go all the way down). On adduction (looking away from vertically limited eye AKA looking to the left) exotropia will increase (compared to primary gaze). Also likely diplopic at both distance and near. PSP is progressive supranuclear palsy. It is considered a parkinsonism syndrome. It is more severe than Parkinson's and most patients die within 5 years of diagnosis due to respiratory distress. They will have restricted vertical gaze on ductions which improve with Doll's head. Due to this, they can have a hard time

			with ADLs, even seeing their food. Yoked prism can sometimes help but the amount that would need to be moved upwards is typically too significant. Elevating things typically is the most helpful. Similar to Parkinson's, they often have convergence insufficiency as well.
		Would you consider a HVF as a part of your neurological workup?	Yes! Absolutely. We perform HVF on all new patients with diplopia. Also optic nerve OCT. You want to rule out any potential orbital process or evidence that something may be at the cavernous sinus.
		Can we get a copy of the diplopia chart or the citation to look it up? Thanks!	Dinkin M. Diagnostic Approach to Diplopia. *Continuum (Minneap Minn)* 2014;20(4):942-965 Great review article too!
		Do ODs order their own MRIs? If so, does a radiologist interpret them? OR do we need to refer to a neurologist to get the MRI instead? Private VT practice here.	It depends on your comfort level with them. I do, as do some of my other colleagues. Some states do have regulations on this, most do not. A prescription for the exact test needs to be written (for example, MRI of the brain and orbits W/W/O contrast). If your patient is over 50 or has DM/HTN, they may need kidney function testing to get contrast. It then needs to be precertified by their insurance. A neuro-radiologist typically reads the film and then sends you the report (include your contact info on the Rx). However, I always review it myself as well. I ask the patient for a copy of the CD if it is not available on my system.
		In case one- was the eso measurement a phoria or tropia? Would you only refer for surgery if it's a tropia or also in a phoria? Thanks!	In primary gaze at distance it was a tropia and phoria at near. When I am measuring in multiple positions of gaze, I am only doing an alternating test, so I am only measuring a 'phoria' however it is more so to record the pattern. For surgical referral, I typically only refer tropia's (although they can be intermittent), however, if they have binocular vision, they would need to be able to fuse with prism. Sometimes, an intermittent tropia has diplopia when

			prism is given which would indicate that surgery may now make them diplopic where they previous were not. If a patient is monocular, and are interested in a cosmetic result, then testing for fusion is not indicated. In general, most surgeons will operate on a horizontal deviation (eso or exo) of 10 PD or more and a vertical deviaton of ~6PD or more. However, this is surgeon dependent. If less than this, there is a higher chance of over-correction.
11:00 – 12:00 pm	*Alexandra K. McArthur, OD, FAAO** *Conjunctival Crisis: Uncovering Diagnosis in a Case of Chronic Follicular Conjunctivitis*	Do we get these lectures send to us in the emails. Every lecture I have attended and ones I haven't been able to attended. Are sooo helpful to study from for boards part 2.	Please reach out to a professor or faculty member. They are using the recordings for instruction during this period and have access to the video links.
		Good morning, where would we find the recordings for these lectures? Thanks!	
		Why do you think the patient's symptoms were worse in the morning?	This follows the pattern of any other condition that would cause mucoid discharge; worsening of irritation and crusting in the morning has also been reported in both allergic and bacterial conjunctivitis as well. This is likely due to a build-up that occurs overnight when our blink reflex is not working to clear away mucus and debris.
		If we believe the lesion is a pyogenic granuloma, and we begin treating with a topical steroid, would any differential potentially respond?	I think this question is referencing the slide where I compare the "salmon patch" appearance in a pyogenic granuloma, benign reactive lymphoid hyperplasia, and MALT lymphoma. There is literature to suggest that a benign reactive lymphoid hyperplasia would also repond to steroid treatment. I would not expect a response from a true conjunctival lymphoma.
		Great case. Can you please share studies referenced?	Thank you! The studies that were referenced specifically during the presentation are below: ■ Ferreri AJM, Cecchetti C, Kiesewetter B, et al. Clarithromycin as a

			"repurposing drug" against MALT lymphoma. *Br J Haematol.* 2018;182(6):913-915. ■ Kiesewetter B, Raderer M. Antibiotic therapy in nongastrointestinal MALT lymphoma: a review of the literature. *Blood.* 2013;122(8):1350–1357. ■ Lagler H, Kiesewetter B, Dolak W, et al. Treatment of mucosa associated lymphoid tissue lymphoma with a long-term once-weekly regimen of oral azithromycin: Results from the phase II MALT-A trial. *Hematol Oncol.* 2019;37(1):22–26. ■ Tanimoto K, Kaneko A, Suzuki S, et al. Long-term follow-up results of no initial therapy for ocular adnexal MALT lymphoma. *Ann Oncol.* 2006;17(1):135-140. ■ Shields CA, Alset AE, Boal NS, et al. Conjunctival Tumors in 5002 Cases. Comparative Analysis of Benign Versus Malignant Counterparts.*Am J Ophthalmol.* 2017;173:106-133. Shields CA, Shields JA, Carvalho C, et al. Conjunctival lymphoid tumors: Clinical analysis of 117 cases and relationship to systemic lymphoma. *Ophthalmology*.2001;108(5):979-84.
		the patient in this case never had the typical salmon patch on bulbar conjuctiva ?	That is absolutely true and was the reason that MALT lymphoma was not our top differential prior to biopsy. If you recall, our top differential was follicular lymphoma based only on the appearance of the conjunctiva. This demonstrates even further the need for biopsy in atypical conjunctival lesions and that while clinical observations can guide your thought process, this diagnosis cannot be made on appearance alone.
		If you were in private practice not near a Wills eye institute, who would be ideal for you refer to for biopsy? - PcP? -dermatology	To clarify, the biopsy was performed in our office by our corneal and anterior segment specialist. The sample was mailed from our location to the Wills Eye Hospital pathology lab and the results

		- oculoplastics?	were mailed back to us. There are a handful of ocular pathology labs which exist around the country and accept samples through the mail. (For an example, here is the link describing how to submit a specimen to Wills Eye Hospital:https://www.willseye.org/medical-services/subspecialty-services/ocular-pathology/). In terms of obtaining the sample, I would recommend either an oculoplastics or anterior segment specialist to perform the biopsy procedure. If you were in a very rural or remote area, it is possible that a general ophthalmologist who is comfortable with performing conjunctival biopsy could do this as well.
12:00 – 1:00 pm	*Stephanie Pisano, OD, FAAO* *The use of Scleral contact lenses for Corneal Scars*	Can you go over why acanthamoeba was suspected? I may have missed it! I guess I'm thinking we would see pain way out of context with the signs.	The confocal microscopy images performed in the ophthalmology clinic showed signs of cysts within the lesion that are usually seen with Acanthamoeba. Pain is usually a predominant symptom with Acanthamoeba keratitis as well, which the patient did note during his visits.
		In case 1, why do they not use an amniotic membrane?	An amniotic membrane was considered, but may need to be repeated to achieve full defect closure.
		Would an amniotic membrane function similarly? Why is a conjunctival flap used instead?	An amniotic membrane would be a reasonable alternative treatment plan. The corneal surgeon chose cryotherapy plus a conjunctival flap instead of amniotic membrane treatment as this has been a successful treatment modality for AK patients in our clinic in the past. Another consideration is insurance coverage, amniotic membranes are a fairly new procedure and may not always be covered by insurance. We usually perform a pre-determination with various treatment codes. The patient and surgeon usually discuss which option would be best based on their ocular condition, but the patient may be influenced by their out of pocket costs.
		Is using a conj graft similar to	See above.

		implementing a prokera/other amniotic membrane? Is one preferred in certain situations to the other? TIA	
		how do you calculate how to change the toricity to achieve a more centered fit?	This depends on the lens manufacturer. Some scleral lens designs work in micron changes, other work in diopter step changes. Taking pictures or OCT images and sending them to the lab consultant can help them assess how much toricity to add if you are unsure. With more scleral assessments, it becomes more obvious how much toricity to change. Another way to assess the amount of toricity that is needed is to look at the amount of decentration in the form of how large the disparity is between the vault superiorly versus the vault inferiorly when the lens is on the eye.
		How do you determine whether you need to increase back surface toricity vs steepening the edge(s) for cases like this?	In this lens design, adding back surface toricity is achieved by steepening the superior and inferior quadrants. In other lens designs, the consultants may recommend to only steepen the inferior haptic to achieve improved centration. I find the scleral lens consultants extremely helpful, always discuss what you are finding with them and take pictures if you can.
		Do we need an axis when ordering the 1.5D of back surface toricity or how does that work? Thank you!	If the lens is decentering inferior, the lab will usually add the back surface toricity to the vertical meridian, as that is where the lens is too flat. For this lens design you do not need an exact axis. Other lens designs can require you to measure lens rotation, ex: quadrant specific fitting sets. Each fitting set/design will require different parameters when ordering. Fitting guides are very important in learning what each company would like you to assess prior to calling consultation.
		In these situations, if the patients can tolerate corneal GP and there's proper fit, would it theoretically provide the same visual benefits?	Yes, if the patient is able to wear the lens, the fit is appropriate, vision is improved to the level needed by the patient, and the ocular health stays stable it is a good option. If you have a great fitting lens and improved acuity, but the patient will not wear the lens due

			to comfort, an alternative design should be considered.
		Why do you typically try a GP lens before jumping straight to a scleral? It seems (at least in your cases today) that most people didn't have success when trying the GP first.	In my experience, GP diagnostic lenses can give a very quick assessment of vision potential. Also, remember corneal GP lenses are usually easier on the patient from an application/removal standpoint and provide much better oxygen to the corneal surface vs. a scleral lens. Corneal GP lenses may not be a great option if the patient has extensive elevation changes on the cornea, from a stability standpoint. If the corneal GP lens decenters significantly, the vision will not improve as much as it would with a scleral lens.
		Is there any benefit to trying GP lenses first? Is it an insurance requirement?	As above, usually application and removal is easier with corneal GP, better tear exchange, and they are more cost effective compared to scleral lenses. In the case of determining medical necessity, where an insurance company is considering paying for the lens, I do apply a GP lens first and document in the chart if it was not adequate (patient comfort, vision, stability, etc). Insurances usually dot not specifically require it. I also like to trial it for the referring provider as well, so they are aware why I ended up fitting the patient in a scleral lens specifically.
1:00 – 2:00 pm	*Pat Segu, OD, FAAO** *CMV Retinitis*	Do you do anything special for patients with HIV?	You should follow Universal precautions when examining patients. Some patients may not know they have HIV or Hepatitis B. Have to use personal protective equipment when required such as gloves, masks etc. Tears are considered a non-infectious body fluid unless there is blood in the tears than it is consider infectious and universal precautions will apply. The CDC has guidelines for disinfection of the tonometer tip.
		Does sexual history have any relevance in the optometric setting. For clarificaiton, we see CMV retinitits in the patient: is it our job to council this	At times, sexual history does have relevance in the optometric setting for example in cases of Chlamydia and in cases when you suspect HIV in a patient

		patient on sexual practices or do we reserve that conversation for the ID specialist/PCP. When does the OD get involved with probing questions on sexual history/behavior, if at all?	that has a unremarkable medical history. Our job is to determine the risk factors associated with the ocular condition and providing guidance as well as coordinating care with the specialist.
		The questions came pretty fast and I didn't get to answer them all. Is that going to effect my participation in getting my certificate?	You will still get credit for attending the course.
		how can kaposi sarcoma in the eye be differntiated from subconjunctival hemorrhage?	A subconjunctival hemorrhage will resolve within 1-2 weeks and a kapsoi sarcoma lesion will not resolve and continue to grow.
		Are the yellow regions within the RPE mottlling after CMVR resolution considered areas of exposed sclera due to atrophied Retina/RPE tissue? Thank you!	The slide I showed was almost resolved but not completely. The exudative areas were still in the absorption process but majority of the retina had healed. You can get chorioretinal atrophy and in this case you would be looking at the sclera.
2:00 – 3:00 pm	*Susan Cotter, OD, MS, FAAO* *Accommodative Esotropia: The Ins & Outs*	How did she diagnose the first patient with accommodative ET if she wasn't able to uncover the ET during the exam with CT and Bruckner?	Mother quite confidently described seeing the esotropia and associated behavior when it happened (cried, clumsy, ect). So this gal had an intermittent ET that just did not occur when she was in the office,
		When you cycloplege these patients, do you expect their deviation to get better or worse? Does it depend on the underlying etiology (i.e. accommodative type or high AC/A type)?	Not sure what you mean – with the drops in the eyes while still cyclopleged? If that, then it can go either way. Sometimes eyes look straighter because accommodation is paralyzed. Sometimes worse – kid has constant larger-angle ET after drops put in. (You also see this sometimes with hyperopic kids without accommodative ET). This would be because the kid is blurry because cannot accommodate and the visual system is trying to clear it up by trying to accommodate. Cannot accommodate but because of the effort of trying to accommodate the innervation is going through the AC/A and the eye turns MORE than did without drops. (or the eye turns now when it was never seen before). Sometimes IXT's look better when cyclopleged for the same reason.

		For diagnosis of AET, do you use a +3.00 or +2.50 lens to assess alignment?	Not sure I understand question. To diagnose refractive accommod ET, need to assess eye alignment with refractive correction on when not cyclod. That could be the full cyclo or less than the full-cyclo. For high AC/A, would put on trial frame with distance SRx on and then determine least plus to eliminate the ET at near. That could be any plus but most likely in the +1.50 to +3.00D range. If this is not what you are asking or if still does not make sense contact me at scotter@ketchum.edu
		What was the AC/A equation again for calculation?	AC/A = IPD(cm) + Fix Dist(m) (<Dn − <Df) Signs: Eso/BO = plus; Exo/BI = minus <D is based on the Prism & Alternate Cover test measure
		Do these ETs resolve as the patient reaches presbyopia	Not necessarily because even if you cannot accommodate well, if your vision is blurred without your hyperopic correction, the visual system can try to make it clear. In doing so (trying to accommodate) you can stimulate accommodative-convergence through the AC/A – innervation still stimulated. Similar to what can happen when you cycloplege a kid. That said, you do not get a lot of presbyopes complaining about accommod ET. Because they are presbyopic , they wear their hyperopic glasses all of the time (to be able to see clearly) and when you examine them in the office you do all of your testing through their glasses. So, even if they had an accommodative ET, since they always have their CLs or specs on, it is a non-issue.
		Can you give lens and do vision therapy as well?	You can do VT to increase fusional divergence ranges. If successful, then you could prescribe less plus. But highly unlikely that you would eliminate the need for optical correction of accommodative ET entirely.
		Do you ever put patients into multi focal contact lenses for the add power?	I have not done much personally, but there are reports of this, but mostly in

			teens and adults with accomm ET. Because the optics are not as good and also less precise in multifocal CLs, don't usually do in young kids. Initially, you want to give it your best shot and that would be a flat-top bifocal for young kids. But if you have a kid who is doing well with bifocal specs and the ET is well controlled then could consider trying to switch over to multifocal CLs. Would also be good if had a kid who refused to wear Specs for some reason (maybe autistic and does not like things touching him) or had some anatomical reason could not wear specs. Then multifocal CLs would be good option rather than bifocal specs. So the answer is yes you can, but not something typically done as "first-line" treatment for young children.
		Is cyclotherapy usually a one-time instillation, or do you issue an Rx?	More often a 1 -time thingy, particularly if you use 1% atropine.
		Can you go into detail with cyclo therapy.how to use it	If child has hard time accepting the hyperopic Rx (blurred at distance) because she cannot relax accommodation fully, THEN…… Bring child in and instill cyclo drops (can be cyclopentolate or 1% atropine). The idea is that as accommodation gradually recovers while the drops gradually wear off…..this helps the child to keep his/her accommodation relaxed because it is a gradual recovery. When we dispense a +4 or +5.00 Srx and the child puts glasses on, they have to relax all of that plus right then and there (immediately) to have clear vision. My experience is that young kids often do this, but when you have a problem or suspect there will be a problem with this happening, you can do cylcotherapy. I have done this for particular cases, but not with any regularity – only every once in awhile. Maybe 1 of every 100. But I rarely prescribe full plus for school-age kids, so that is also why I don't have to do something like this very often.
		How often do you recommend Contacts for these kids given their age?	If purely refractive accommodative ET (i.e., don't need add at near) kids can wear SV contacts at any age, provided

			child wants to wear CL's and parent are also okay with it. If very young, parents have to be able to do insertion and removal for the child. Usually want kids to be fairly self-sufficient with personal hygiene, so can care for CLs on their own. For combined accomm ET, can wear SV CL's and then throw on Specs that are Plano with the add, when needed.
3:00 – 4:00 pm	*Sowmya Srinivas, OD, MS, FAAO* *OCT Retina Interpretation*	Is neurosensory retina everything above the RPE?	Yes, and it includes 9 layers. Here is a detailed description from AOA Neurosensory Retina
		Hi Dr. Srinivas, I had a question on your choroidal melanoma case from yesterday. After your lecture it'd be great if I could ask you then. Thanks!	Hi there, I am happy to answer your question if you email it to the organizers. They can send it to me. Thanks for listening.
		Looking at PIL, can you estimate a numeric VA range versus just knowing if the VA will be good or bad?	You can estimate a VA with experience from reviewing OCT scans
		About 3-5% of CSCR pts. get PEDs…do they portend SRNV??	Yes, here is one interesting observational study in JAMA Bonini Filho MA, de Carlo TE, Ferrara D, et al. Association of Choroidal Neovascularization and Central Serous Chorioretinopathy With Optical Coherence Tomography Angiography. *JAMA Ophthalmol*. 2015;133(8):899–906. doi:10.1001/jamaophthalmol.2015.1320
		would the PED resolve with the CSCR? and what is the general management/ referral plan with PEDs along with and when they present on their own.	RPE atrophy and mottling are seen in chronic CSCR. The management of PED is observation without referral for treatment.
		Have you managed patients on spironolactone as a treatment for chronic CSR? And have you seen success with this treatment option.	I have not utilized this as a treatment but studies have shown benefits with this therapy. Meta-Analysis of Randomized Controlled Trials below: Mineralocorticoid Receptor Antagonists in Central Serous Chorioretinopathy Wang, Sean K. et al. Ophthalmology Retina , Volume 3, Issue 2, 154 - 160

SOCCEP Q/A
Tuesday, March 31

		What about Spironolactone and eplerenone? I've been told that is commonly first line for chronic CSCR, is that accurate?	I have not utilized this as a treatment but studies have shown benefits with this therapy. Meta-Analysis of Randomized Controlled Trials below: Mineralocorticoid Receptor Antagonists in Central Serous Chorioretinopathy Wang, Sean K. et al. Ophthalmology Retina , Volume 3, Issue 2, 154 - 160
4:00 – 5:00 pm	*Richard Shuldiner, OD, FAAO** *Low Vision and COVID-19*	No questions	
5:00 – 6:00 pm	*Jennifer Gould, OD, MS, FAAO**	Can you comment on the timing of using anti-veg-F injections with tractional detachment when surgery is indicated?	Treatment is going to be surgeon dependent. Some surgeons will perform anti-VEGF prior to surgical repair, and some will repair the TRD first and then perform AntiVEGF. It is important to remember that AntiVEGF can cause contraction of the fibrovascular tissue. From an OD perspective, we should educate our patients that both surgery and injection are likely but not specify the order unless you know the preference of the surgeon you are working with.
		Using the colormaps on OCT, do the cooler colors show more edema?	The following answer applies to thickness analysis on Zeiss OCT: The coloration of the maps depends on which map you are looking at: The ETDRS analysis, cooler colors (pink, pale yellow) correspond to thickening, where warmer colors (Red, yellow) correspond to retinal thinning. On the thickness analysis, the warmer colors (red/yellow) correspond to thicker areas of the retina.
		Using the colormaps on OCT, do the cooler colors show more edema?	See Above

SOCCEP Q/A
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6:00 – 7:00 pm	*Alex Hynes, OD, FAAO** *Optic Disc Pit Maculopathles*	Would having an optic disc pit make it difficult to diagnose/monitor for glaucoma?	Yes- first of all if we are speaking of a congenital ODP, then you may have a VF defect (ex. arcuate defect)caused just from the pit itself (therefore getting a baseline VF is critical); furthermore the serous fluid from the fit could potentially 'artificially' thicken your RNFL or GCC OCT scans , with potential future reduction in fluid then possibly being misinterpreted as a glaucomatous change when its really not (just like when vitreous traction is released from the RNFL and RNFL thickness decreases); because of this monitoring IOP as well as other glaucoma risk factors (pachs, fhx, gonio, NTG factors (apnea/BP) is important in addition to careful interpretation of the field/OCT → for an acquired optic disc pit- this is almost certainly associated with glaucoma (in the absence of pathological myopia) and therefore you need to monitor this area of the disc for progression very closely and treat accordingly
		When you analyzed the RNFL cross section- did you say the image helped you visualize the pit itself or the subarachnoid space filled with fluid?	It helped me visualize the continious tract of intraretinal fluid from the optic nerve head (anterior part of the pit) into the nasal macula ; note the image on my old Topcon SD OCT didn't have enough imaging depth to visualize the pit itself or the subarachnoid space which would be more posterior to the pit. One study showed that swept source OCT is able to image both the pits and subarchnoid space+fluid (ex. Fig 3 D –F). Ohno-Matsui, Kyoko, et al. "Evaluation of Congenital Optic Disc Pits and Optic Disc Colobomas by Swept-Source Optical Coherence Tomography." *Investigative Ophthalmology & Visual Science.*, vol. 54, no. 12, 2013, pp. 7769–7778., doi:10.1167/iovs.13-12901.
		do you use ONH RNFL in helping the	I don't think the RNFL thickness plot itself

		diagnosis of an occult ODP ?	could conclusively diagnose an occult ODP. however if you look at the individual B scans aka cross sections of the optic nerve head you would likely be able to see the pit if your OCT is able to image deep enough into the optic nerve (swept source better than SD OCT better than TD OCT)
		Good day! So… can we clearly see the disc pit with a disc OCT? I'm not sure since we mostly looked a fundus photo	It depends on the OCT you are using. My old Topcon SD-OCT wasn't able to get enough depth in cross sections (not the RNFL plot itself). However if you have a better OCT you will be able to. 1 study at the bottom does an excellent job with their new swept source OCT at imaging both the entirety of their disc pits including even the subarachnoid space in some figures ex. Fig 3. Ohno-Matsui, Kyoko, et al. "Evaluation of Congenital Optic Disc Pits and Optic Disc Colobomas by Swept-Source Optical Coherence Tomography." *Investigative Ophthalmology & Visual Science.*, vol. 54, no. 12, 2013, pp. 7769–7778., doi:10.1167/iovs.13-12901.
		Is 'acquired disc pit' the same or different from notching?	- optic nerve head notch is a localized/focal loss of neuro-retinal rim tissue - if it is a very 'deep' notch sometimes we call this an acquired optic disc pit, however in general I do think of a pit as not just a lack of neuroretinal rim tissue but an area of deep excavation into the space where the lamina cribrosa would be located ; furthermore you can't really have a notch centrally but you can have an acquired disc pit centrally - both notches and acquired pits are associated with high risk for glaucoma progression
		In the diagrams showcasing the OCTs of the retinoscheses, there was a designation of PMC. What does that	Pit-macula communication ; as in vitrectomy + gas tamponade can close the pit-macula comunication resulting in

		stand for?	reduced retinal thickness/fluid in the retina

SOCCEP Q/A
Wednesday, April 1 (Happy April Fool's Day!)

Time	Session Details	Question	Answer
10:00 – 11:00 am	Don W. Lyon, OD, MS, FAAO Are you sure you have double vision?	No questions.	
11:00 – 12:00 pm	Sarah MacIver, OD, FAAO Systemic considerations in normal tension glaucoma	Does diurnal range have to be taken on the same day to be considered useful?	Yes and no. There's not great evidence about the reliability of a diurnal/modified - diurnal. What a person's diurnal looks like one day is not what it will look like the next day. The main value we get in doing this all on the same day is an idea of the max and mins of an individuals IOP fluctuations.
		why are the disc hemes more common in NTG cases, if it is vascular- why is it localized to the nerve	This is not well understood – there is likely a vascular etiology behind this but not fully understood because glaucoma is not an ischemic diseae.
		are re-occurring drance hemes indicative of progression?	Yes, that's generally the thought process. If we see a heme consistently in the same location then the disease is likely not well controlled
		Was it a DBP of less than 56 is at risk or a OPP of less than 56?	OPP of less than 56
12:00 – 1:00 pm	Maria Walker, OD, MS, FAAO Scleral Lenses in Keratoconus	What is the etiology of Vogt's striae?	Occurs due to the mechanical stress of the ectasia. Remember that the striae occur in the posterior stroma, which is important to consider if you really want to understand the etiology. Basically this posterior tissue folds in on itself due to the stress of the "outward collapse" that occurs in ectasia. Think about something being stretched (like a balloon) but stretched more on the anterior surface than the posterior. That can help to understand why the posterior cornea folds in on itself. The reason why the striae are vertical is thought to be because the HVID is slightly wider than the vertical height of the cornea, which physically stretches it

			so that the stress lines are aligned vertically.
		How do you choose what power of lens to use due to the fact that the conrean has differning powers?	Power of lenses for Kc eyes should always be determined by using a diagnostic lens (either GP or SL). You can't empirically figure out the power for these individuals because they have so many powers in the area overlying the pupil. The diagnositic lenses (aka trial sets) should neutralize most of the higher order aberrations and allow for a spherical-cyl over-refraction.
		could i please get that video explaining the fit over the entire cornea	Yes, email me at mkwalker@central.uh.edu and I will send it
		also, would you use a back surface toric to reduce the clearance in the periphery areas to ensure not more than too much clearnace in areas other than the apical cone?	You could, but you wouldn't necessarily call it a back surface toric. What you'd probably do in the situation of reducing clearance in the periphery would be to change the curvature for the transition zone (aka the limbal zone).
		Is it possible to get a copy of the slides/videos discussed today? Thanks!	Email mkwalker@central.uh.edu
		Do we want the apical clearance to be that 100-150 microns after settling to be located at the thinnest area (i.e. on the cone)?	Yes, that would be acceptable. The range is about 100-300, but it depends on the patient response. Most of them don't do as well with more clearance because of discomfort, or debris accumulating in the fluid. But you don't really want less than 100, because then it can settle in to touch over time (or the patient cornea can progress outward and hit the lens). You always want to leave some "wiggle room". So 100 is minimum…but up to 300 will usually work for most people. Certainly, if one of the side effects of excess fluid reservoir depth is occurring (debris), you'd want to reduce that depth by as much as possible (to 100 if possible).
		Does the anterior stromal haze post CXL improve or resolve?	It usually resolves within a year, but I've seen it persist. It can be treated with steroids which often reduces it. Fortunately, it doesn't usually affect vision even when it is observable from our view. However, in a small percent of patients, it persists and patients are

			affected by it…so this is a risk factor when referring. That is why patients who are low risk for progression with clear corneas and 20/20 vision are not always referred for CXL.
		A follow up lecture on cross linking would be great	Good suggestion! I'll see if there is room for that.
		What specifically led you to not refer patient "George" for cross linking?	There are a couple reasons we haven't sent him yet: - Given his age, he is still relatively low risk to progress much further beyond where he is now. The amount that he has progressed is not much more than the standard deviations of the instrument (meaning changes like this could almost be considered the same). We need to see more consistent progression (which, if real, is not going so fast that we will miss the drastic changes). We still may refer him if we see changes in the future. - It is his less severe eye that we are watching, and often the less severe eye does not progress as aggressively. But note that I say "yet", because we are still watching him every 4-6 months to monitor for more changes in topography and the emergence of any Kc signs (this patient currently has a completely clear cornea with no striae, haze, or visible stromal thinning). I have forewarned the patient of this, and told him to be saving his money and preparing for a time when we say, okay, it's time to get it done.
		In what scenario would you fit with a GP first?	There are a few general situations when I go to a GP first: - If a patient is already established with GP lens wear - If they are a known failed SL candidate – important to ask - If they are very very apprehensive about putting a SL in their eye (and their eyes force shut and roll back when a lens comes at it)

			To be clear though, anytime a Kc patient comes in, GPs are one of my options. They are especially useful in mild Kc patients with difficulties with application. However, I have found that for most patients, they end up being more successful, for longer, with the SL. And given that it does not touch the cornea, it causes less mechanical disturbance of the cornea. There's a lot more to this, but that's the best answer I can write out ☺
		Can you please go over who you would refer for crosslinking?	So, most Kc patients (who are progressing) are going to be candidates, but it also depends on age, risk for more progression, and status of current cornea. Also, keep in mind that there are really two different types of candidates: urgent referral (should have CXL asap) and non-urgent referral (should have CXL within 2-3 years). Below is an example of an urgent referral and one that's non-urgent referral. Hopefully this helps: Great candidate: 18yo HM, brother with Kc, max K OD: 64D, max K OS: 51D. Min Pachs: 415 OD, 440 OD VA in specs: 20/40 OD, 20/25 OS. Progression in past 3 mo: 2D OD, 12um thinner pachs; 0.5D OS This patient is a pretty urgent referral, and should have CXL asap on his right eye. He has a recorded fast progression (2D in 3mo), young age, genetic component. And he is right about at 400 um so should be done as soon as possible before his cornea gets too thin. Left eye should be watched close and probably done after right eye. Poor candidate: 38yo BM, Dx with Kc age 19, max K OD: 64D, max K OS: 51D. Min Pachs: 415 OD, 440 OD VA in specs: 20/40 OD, 20/25 OS. Progression in past 1 year: 0D OD, 5um thicker pachs; 0.2D OS This patient has the same exact
		do you consider scleral lenses the	That's a good point, and it is certainly a

SOCCEP Q/A
Wednesday, April 1 (Happy April Fool's Day!)

		standard of care even for patients who may be financially struggling? what is your best plan for patients who may not be able to afford sclerals as a first line of care?	point of consideration….usually the fitting fee is the same for both lenses, but GP may cost $250 per pair wheras SL would cost $800 or higher. Insurance should cover both for Kc patients (If they have it). But yes, this is part of the consideration, and I always offer the pros and cons of both options, tell the patient what I'd recommend (but be sure they know cost differences), and they make the choice. I will say, even with the cost, most patients go for my recommendation.
		I would like to get a copy of ppt , If possible	Email mkwalker@central.uh.edu
		Hi, thank you for the presentation, you mentioned posting the powerpoint, how do we get that?	Email mkwalker@central.uh.edu
1:00 – 2:00 pm	Barbara Mihalik, OD, FAAO So you have a patient with an inherited retinal disease (IRD), now what?	What's the easiest way to look for vitreous cells?	Use retro-illumination. Just like you would use the slit lamp to look at a PSC cataract, but, push the beam of light past the lens into the vitreous. Then have the patient look and up, down, and then straight ahead. This will stir up the cells and you will see them swirl around. You can pick up even the most trace of cells this way.
2:00 – 3:00 pm	Anna Bedwell, OD, FAAO Paracentral Acute Middle Maculopathy	Have you seen more patients with pamm since this case?	Other than these two cases, I have mostly seen PAMM lesions with other retinal vascular disease specially in a few patients with diabetic retinopathy (severe and proliferative) as well as CRVO. In those other cases, the acuity wasn't affected (from PAMM at least) because the PAMM lesions were not near the fovea.
		Does vision remain relatively stable or can there be a further drop in acuity due to atrophy?	The vision generally will remain stable from onset of symptoms. The degree of visual acuity loss depends on how much the fovea is involved.
		Is it more common for PAMM to appear unilaterally?	Since there are no large case studies to go off, it is hard to say with absolute certainty. Based off reviewing other case reports in the literature, it seems

			like it is pretty equal between unilateral and bilateral presentations. It would mostly depend on the underlying etiology.
		Any idea what was found on vavle…vegetative matter? Hard to imagine not from IV drug use.	Our records were limited as we were going off the information from the stroke rehab hospital which was not the same facility for his treatment. The cultures were positive for MSSA. Bacterial endocarditis in a relatively young patient would be most suspicious for IV drug use, but that was denied.
		Could we please get a copy of the lecture ppt?	
3:00 – 4:00 pm	Sowmya Srinivas, OD, MS, FAAO Ocular Manifestations of Medications	Hi. The audio is going in and out for me. Will there be access later?	Hi there. Yes, I believe the lectures are recorded and the organizers can provide the link to the recording.
4:00 – 5:00 pm	Richard J. Madonna, MA, OD, FAAO Mild-to-Moderate POAG - Or is it?	Speaking of "polling", I do not see where or how to access that feature. This has been the case for the whole time of my participation. I'm guessing I'm simply missing a tab or a button or something	
		Did he say for the average person without glaucoma the VFI would change 1-10% per decade?	No. I said that on average, for a person without glaucoma, the average reduction in average RNFL thickness is 1-5 microns/decade, about 10x faster than an "average" glaucoma patient. The loss of mean deviation for normals is about 1 db per decade. I suspect that VFI would change similarly.
		Does the ERM throw that off? the progression analysis	Epiretinal membranes often affect the measurement of the inner retina. Of course, that will affect our measurements of any component of ganglion cells. When looking at progression analysis, if any of the measurements in the progression are affected, then the progression analysis will be affected.
		Considering the ERM in OS, how reliable	The VF pattern deviation may be

		is the pattern deviation?	effected.
		Hi, do you use Tmax or the last 3 IOP readings when determining IOP targets?	Tmax is important as it's the IOP that is most likely to cause damage. That said, I will always try to base my decision-making on at least IOPs.
		Is there an event/trend analysis available for 24-2Cs?	Interesting question. There is not for the 10 new points. However, if you run the normal GPA analysis you will get the analysis for the 56 points on the regular 24-2.
		What is your opinion on using ERG and/or OCT-A in glaucoma management?	I think electrodiagnostic testing has great potential in identifying patients with pre-OCT or VF damage i.e. identify patients with GC injury prior to GC death. The variability in results are still an issue, at least for some of us. OCTA also has potential, and there is a great deal of research being done indicating that there are differences between normals and patients. The question is whether OCTA abnormalities are causative or in response to glaucoma damage.
		If the GCA is thinner but RNFL normal, would you manage the px the same way if RNFL thinner and GCA normal?	That's a major question right now. I don't believe there is any consensus. I am a firm believer in the importance of measuring the macula, yet I don't think I can give you a definitive answer. Like all glaucoma patients, the big question is what is their anticipated lifespan, how severe is the damage you detect, and what is the rate of progression. Great question!
		Are most alternating between 24-2 and 10-2 the next year?	As above, I don't think there is a consensus. I am trying to do more central tests, be there 10-2 or a 24 degree field with increased central sampling. Additionally, it depends on the patient and their level of disease.
		What are your thoughts on usage of the octopus VF as opposed to the standard humphrey?	They are both great. Depends on which is best for your practice. With the addition of 24-2C on the Humphrey, you now have a test on that machine that samples the central field that is different but determines something similar to the Gtop or Mtop on the Octopus.

SOCCEP Q/A
Wednesday, April 1 (Happy April Fool's Day!)

5:00 – 6:00 pm	Tamara Petrosyan, OD Dark Without Pressure	She had outlined what the fundus, vision, visual field and OCT would show for mild, moderate and severe plaquenil toxicity and I thought this was really helpful but was not able to jot down what she was saying before she switched slides. This kind of staging is really helpful when classifying disease clinically so I was wondering if she could provide that information to me or refer me to the articles that she gathered that information from?	All of the information is in the American Academy of Ophthalmology Statement paper 'Recommendations on Screening for Cloroquie and Hydroxychloroquine Retinopathy" by Michael Marmor et al
6:00 – 7:00 pm	Dionne Moore, OD, FAAO BRAO	How did you rule out it was not a calcium plaque from the heart since it was so close to the ONH?	Good question. HHP are easy to spot because they are shiny. Sometimes the "refractile" nature is lost in the photo. However, if you see a shiny particle, in the branches or at the disc, it is most likely a HHP. Calcium plaques tend to be more white in nature. For reference, here is an article to review: Diagnosis and Management of Carotid Artery Disease
		is it common to have neo with BRAO?	One of the reasons I presented this case is because of the NVD, which is rare in BRAO. For reference, here is an article to review. Ocular neovascularization in eyes with a central retinal artery occlusion or a branch retinal artery occlusion

SOCCEP Q/A
Thursday, April 2

Time	Session Details	Question	Answer
10:00 – 11:00 am	Sondra Black, OD, FAAO Role of the OD in Refractive Cataract Surgery 2	What does the patient see when you use the laser to break the lens and while they wait for their extraction?	From personal experience I can tell you just a very psychedelic visual phenomenon. Doesn't go black, no pain or even discomfort
		Is there a way to see if a patient will get CME after surgery?	No way to predict
		I thought yag were done at 6 months for pco?	These days the majority of surgeons are totally comfortable by 3 months. There are some that feel comfortable as early as 2 months postop if the patient is otherwise happy with outcome and exchange not on horizon
		Do you have a script that you tell almost every patient about to get cataract surgery?	I use my eye model to show them where the lens is as they all think it is a corneal growth. I tell them that the lens has gotten cloudy. Then I tell them the lens is like an M&M, we are going to leave the candy coating and swap out the chocolate. Quick and easy explanation that they can relate to.
		We were taught to not dialte patients with ACIOL, what do you recommend if we need to see the posterior segment or if we need to dilate?	With all BUT an iris sutured IOL, dilation is totally fine with AC IOL's. They are sitting in the angle and not affected by dilation at all.
11:00 – 12:00 pm	David Lampariello, OD, FAAO Ocular Biometry and IOL Power Calculation	can you go over how much of a role the tear film play in iol calculation	The tear film, if not normal can negatively impact the pre-operative readings taken by the biometers and topographers. With an unhealthy ocular surface, the manifest refraction can also be incorrect. Studies also show that patients with tear film problems present as having either significantly or more or significantly less astigmatism that then resolves after the appropriate treatment.
		What are tension rings used for?	Capsular tension rings are used to stabilize the capsular bag of the crystalline lens during cataract surgery. Particularly for patients with

			zonulopathies.
		Is Dr. Lampariello willing to share these slides?	Thank you for your request for the materials. Due to the proprietary nature of the content, Johnson & Johnson Vision is unable to release the PowerPoint presentations. Additionally, these sessions were recorded, and you may request access through your faculty members.
		I would also really like a copy of the PowerPoint if possible!	Thank you for your request for the materials. Due to the proprietary nature of the content, Johnson & Johnson Vision is unable to release the PowerPoint presentations. Additionally, these sessions were recorded, and you may request access through your faculty members.
12:00 – 1:00 pm	Caroline Blackie, OD, PhD, FAAO Meibography Review	Did the Dr. say that the top or bottom lid overlaps the other during a blink?	The top lid slightly overlaps the bottom lid during blinking.
		When you talk about the numerical number of “glands loss”, are you including those that are truncated as “lost”?	‘Gland loss’ refers to the absence of visible gland structure which includes truncation as well.
		Okay, I have a technical question too—each time I watch and try to take the attendance survey at the end, it tells me I “already filled out the survey”. Is there someone I can message about this?	This is a question for the AAO. I will forward your request on to them
		Can you talk about pathophysiology of a concretion and its management?	The pathophysiology of eyelid concretions is varied. One of the possibly causes is chronic MGD. Aside from addressing any coexisting OSD, treatment for the concretion itself depends on whether or not, it is causing any problems. They are typically not removed if the patient is asymptotic.
		Can you elaborate on what, if any chnages, are reversible.	In terms of gland structure, what we learn from animal models is that provided the atrophy is not too advanced, there is a possibility of gland regrowth secondary to treatment. This is actively being explored in the scientific community.
		If there is significant loss of gland lets say	The patient treatment will depend on

		75 % loss , what would you start the patient on ?	what the problem is. In an advanced case on MGD, it is likely that both obstruction and inflammation require treatment. There are other comorbidities to consider, such as causes of blepharitis, ocular allergy, etc. It all depends on the patient presentation and the specific pathologies.
		1)Should we say that CL would increase the integrity of MG? 2) In px with severe gland dropout, would you still recommand Lipiflow to increase the expression of lipid? If not, what would be your recommandation?	1. The data in the literature indicate that CL may have a negative and measurable impact on the meibomian gland structure and function. LipiFlow is indicated for the treatment of MGD. 2. The medical literature shows that in patients with greater than 67% gland loss, symptomatic relief after MGD treatment may be reduced. This result is obvious. However, the patient would still need treatment to improve any remaining gland function. The decision to select a particular treatment device would depend on the complete patient presentation.
		what is the go to treatment for gland drop out? especially for more severe	The MGD workshop recommendations are to always treatment gland obstruction and to incorporate adjunctive treatments as needed. With increasing severity, a patient is likely to need a larger variety of adjunctive treatments.
1:00 – 2:00 pm	Kurt Moody, OD, FAAO OSD in Clinical Practice: Case Reviews	Why wouldn't lissamine staining be shown later in the disease process compared to NaFL, since lissamine picks up the cells that are dead and devitalized?	According to the medical literature, lissamine green staining of the conjunctiva is seen earlier in the disease process in dry eye disease and NaFl later. Thus, in the later stages you typically see both.
		Do we know if the MG evaluation was done on lower lids only or both upper and lower (to yield only 2-4 glands secreting in case 1)?	Lower lids only for the MGE
		How would you counsel the patient to manage their expectations of Lipiflow, how long it takes to have an effect, whether they need to continue drops	The medical literature shows that the vast majority of patients treated with LipiFlow experience improved gland function. It is also true that the majority

		during the first few weeks, what drops, etc? Thanks in advance!	also experience symptomatic relief. (Reference: Pang SP, Chen YT, Tam KW, Lin IC, Loh EW. Efficacy of Vectored Thermal Pulsation and Warm Compress Treatments in Meibomian Gland Dysfunction: A Meta-Analysis of Randomized Controlled Trials. Cornea. 2019;38(6):690–697. doi:10.1097/ICO.0000000000001907) However, per the MGD workshop there are a variety of adjunctive treatments that can be very important in MGD management. These adjunctive treatments include artificial tears, warm compresses, topical and systemic medications, and others. The decision regarding what to use and when depends on the particular case and what is being treated in addition to MGD. (Reference: Geerling G, Tauber J, Baudouin C, et al. The international workshop on meibomian gland dysfunction: report of the subcommittee on management and treatment of meibomian gland dysfunction. Invest Ophthalmol Vis Sci. 2011;52(4):2050–2064. Published 2011 Mar 30. doi:10.1167/iovs.10-6997g)
		Would you consider adding a soft steroid (FLM or Lotemax) in the presence of inflammatory factors in addition to starting Restasis/Xiidra?	Adjunctive treatments should be targeted to the conditions presented based on the labeling recommendations of those products. This is determined on a case by case basis.
		Hello, I missed the first half of the lecture. Is that a way to access this (and other) recording? Thanks.	The lectures are all recorded
		For patients who want to choose to continue wearing contact lenses, what options with respect to contact lens parameters (sclerals, material, modulus) will be more beneficial to recommend?	The selection of a contact lens, including material, will depend on the particular case. Remember that the mere presence of a contact lens will increase desiccating stress on the ocular surface. And some contact lenses are contraindicated for patients with a diagnosis of dry eye disease.

SOCCEP Q/A
Thursday, April 2

		What 'blinking exercises' are we recommending for patients, exactly? How many blinks, how hard, etc? Thanks in advance!	Blinking exercises have not been standardized in the medical literature. A commonly used recommendation is to use the 20-20-20 rule. Every 20 min take a 20 second break and blink 20 times.
		What accommodations would we have for management and patient expectations for a patient with comorbidities e.g. diabetes?	For patients at higher risk for MGD, the medical literature recommends that they be monitored for the condition on a more routine basis.
		What is the method you use to evaluate lagophthalmos?	Gently close their eyes and use your transilluminator on the superior lid to see if you notice a lid gap
		What is your opinion on IPL therapy for MGD? One of my externships did IPL sessions prior to Lipiflow for some MGD patients	We are limited in our ability to comment on other products. We can offer that when considering a treatment, it is helpful to know what is being treated. IPL is a vascular based treatment. LipiFlow, which is not an IPL therapy, is intended to treat MGD through the process of alleviating gland obstruction. Per the MGD workshop there may be multiple coexisting pathologies that require treatment, simultaneously. These should be addressed as needed based on the patient profile.
		How many sessions do patients usually require for moderate lipiflow treatment? Are patients usually charged per session or one set price for x amounts of sessions?	The medical literature shows in a randomized controlled trial that a single treatment can be effective at improving gland function for 12 months or longer in 86% of patients. (Reference: Blackie CA, Coleman CA, Holland EJ. The sustained effect (12 months) of a single-dose vectored thermal pulsation procedure for meibomian gland dysfunction and evaporative dry eye. Clin Ophthalmol. 2016;10:1385–1396. Published 2016 Jul 26. doi:10.2147/OPTH.S109663) However, the literature also shows that patients treated early in the disease process tend to experience improved outcomes – this is typical for progressive disease states in general. When it comes down to the individual patient, expectations depend on the complexity of the particular individual.

SOCCEP Q/A
Thursday, April 2

		Is there a way that I could access the other 2 cases?	We are looking into how to get them out
		Excellent lecturer. Would love to hear from him again on OSD. Thank you.	Thanks
		When he says 9 working MG, does that mean 9 working MG between the upper and lower lid or just the lower lid?	Evaluation is of the lower lid only so you need at least 9 glands yielding liquid secretion to the lower lid when using the MGE to be asymptomatic if wearing contact lenses and at least 6 is not a contact lens wearer.
2:00 – 4:00 pm	Chris Freeman, OD, FAAO & Brian Schwam, MD Refractive Surgery Post-operative Care and Complications Management - Part 1 & Part II	Was flap being displaced normal?	No, not at all normal. Typically, flap displacement is due to patient rubbing their eye in the first 24 hours after surgery. While rare, can also be due to trauma in the later post-operative period.
		Are the microstriae always horizontal?	Microstriae usually occur perpendicular to the flap hinge. However, they can be in any direction or orientation.
		Would ICL be an option for this patient as well?	We are unclear as to which case you are specifically referring. If you are referring to Case #1, the anterior chamber depth was too shallow for a posterior chamber phakic IOL according to the manufacturer's directions for use. If you are referring to Case # 2, posterior chamber phakic IOL was not an option since at that time no toric phakic IOL's were available- one manufacturer's toric posterior chamber phakic IOL wasO approved and available after Sept 2018 but only up to 4D of cylinder correction in the US. Therefore, the correct lens power needed wasn't available then and is not currently an option in the US.
		Is there a certain pupil size that you worry about the symptoms they may have especially at night due to the size of the LASIK flap?	Yes and no. Pupil size is only one of many factors considered for night vision complaints. Magnitude of correction is a significant factor due to change of curvature from untreated peripheral corneal tissue to flattened central corneal tissue (in myopic treatment). The larger the dioptric treatment amount, the smaller the diameter of the effective optical zone (the central

			"plano" area of treatment) allowing for increased spherical aberration outwardly toward the edge of the laser treatment zone. Some degree of halos/glare around lights at night is common for nearly all LASIK cases early postoperatively. However, improvement is commonly seen by one week post-operatively and continues over time. With modern LASIK surgery, many patients experience better overall night vision post-surgery (once fully healed) than they had with correction pre-operatively.
		when would you choose to perform PRK instead of LASIK?	The choice of when to do PRK vs LASIK could be a whole lecture unto itself. The answer of when to choose PRK instead of LASIK depends on surgeon preferences. Different surgeons have different preferences and feelings of what they are comfortable treating. There are varying levels of "suspicious" corneas based on certain parameters in the literature, including various software from corneal tomography companies that are available on their devices. Once you're in practice, we recommend searching out CE courses that go into detail on this topic and getting to know your referral surgeon's/surgeons'/center's preferences. Some surgeons believe that a patient with a suspicious cornea should not undergo any corneal refractive surgery, whether LASIK or PRK, but "suspicious" has different meanings and levels of severity to different surgeons and optometrists working in surgery centers. Common, general reasons for choosing PRK include such things as very thin corneas by pachymetry (varies by surgeon as to what is considered "very thin") and EBMD, where epithelium may be displaced or damaged during LASIK and PRK

			may have a therapeutic like-effect – similar to PTK. Also, corneal scars that may be too opaque for a femtosecond laser to penetrate and occupational/recreational risks, such as those that include high risk for ocular trauma, may contribute to a surgeon choosing to avoid flap-based surgery.
		how much cyl can custom wavefront LASIk treat?	This varies by laser manufacturer/platform. Our recommendation is, once in practice, to learn what your referral surgeon/center's platform treats. The amount of astigmatism measured by wavefront aberrometry may be slightly different from what is measured at the phoropter, and if measures more, it could be out of approved treatment range compared to the amount measured in a phoropter refraction. Also, FDA-approved dioptric amounts of astigmatism treatment vary by type of treatment as well, such as compound myopic astigmatism, mixed astigmatism, or hyperopic astigmatism.
		On day one post-op for LASIK, what is the worst allowable VA you would expect?	There really is no "worst allowable VA" for one day post-op. It's variable as it depends on many things. In our talk, on slide 27 we reference common findings of VA between 20/20 and 20/50, with a range of 20/15 to 20/100. It also depends on type of refractive error treated: as mentioned in the lecture, pre-op myopes will likely be temporarily slightly hyperopic early post-op, and pre-op hyperopes will likely be temporarily slightly myopic early post-op. And the degree of the temporary overcorrection is usually greater with a larger magnitude of pre-op refractive error. Varying degrees of flap edema and dryness may affect UCVA as well.

SOCCEP Q/A
Thursday, April 2

		How long should you expect to put the pt on Pred for Q30minutes before tapering?	The q30 min dosing is one sample regimen when prednisolone acetate is the steroid prescribed for DLK post-surgery. Routine medication regimens vary and may not utilize q30 min. dosing. The steroid dose depends on the course of the disease state and how significant inflammation is at the time of diagnosis. It's also dependent on the doctor's preferences for prescribing steroids, type of steroid the doctor is using (prednisolone acetate or another topical medication), whether or not the flap was lifted and interface rinsed, and if oral steroids are prescribed. In the sample treatment regimen referenced in the presentation for DLK, it's fair to say you'd keep them on that dosing frequency until you see the DLK improve. Always be sure to monitor IOP while the patient is on high-dose and long-term steroids and thus be aware of/watch for PISK (pressure-induced stromal keratitis, a.k.a. IFS (interface fluid syndrome)) that was discussed during the presentation. Be sure to consider measurement of IOP on the peripheral cornea using the tonopen vs. visual axis Goldmann tonometry if the DLK doesn't appear to be improving within 48 +/- hours. As a long-known, albeit rare, complication, there is a large body of literature covering various DLK treatment & management strategies and treatment regimens may vary greatly.
		Can we get this powerpoint it has useful information that we can review again when in clinic	
		Is it possible to get a copy of these notes?	

SOCCEP Q/A
Thursday, April 2

4:00 – 6:00 pm	Azaam Alli, MS & Leilani Sonoda, BS Contact Lens Materials Overview - Part I & Part II	No questions	

SOCCEP Q/A
Friday, April 3

Time	Session Details	Question	Answer
10:00 – 11:00 am	Kelsey Moody Mileski, OD, FAAO Bilateral Conjunctival Chemosis	Good morning, I've noticed the week 1 schedule is no longer listed on the website. Where can I get this information? Thanks!	Email SOCCEP.
		Does the European severity scale look at any exophthalmometry measurements above 25 (for the medium severity), regardless of ethnicity? Or does that number change based for dark-skinned patients?	
		How do you think through choosing a 24-2 instead of a 30-2? Thank you!	
11:00 – 12:00 pm	Jennifer Qayum, OD, FAAO Is This Papilledema?	In case 1, how would your plan have changed if this was a new patient and you did not have previous records of his nerves? Is there anything else that would make you more or less suspicious for edema?	If I did not have previous images of this patient's optic nerves, I would have had the patient return to clinic in 2-4 weeks to monitor nerve appearance with education for the patient to call the clinic with any new neurologic symptoms (nausea/vomiting, tingling in extremities, ringing in ears, etc.). Because he has very large optic nerve drusen and CNVM, we have a reason for his elevated nerves. Although there are areas of the nerve that are questionable for vessel obscuration, overall, the patient's symptoms are minimal (mild headaches), and his vision is 20/20, so I consider him low risk for true papilledema.
		I thought that was neo vs shunt vessels? Pics in case 2	Great question! As I review the images I shared in the presentation, I agree that there is neovascularization present as well. I do have the advantage of having seen the optic nerves "live" in stereo, which helps with distinguishing between neo and shunt vessels. To help distinguish between neo and shunt vessels, it is also helpful to have an FA, which we did not have on this patient. Here are two links with more

			information about neo vs shunt vessels: This article has great images of shunt vessels: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5762152/ This is a summary article of shunt vessel formation: https://www.reviewofoptometry.com/article/collateral-damage
		When looking at an ONH OCT, do you look for the "lazy V" sign to determine if appearance of the optic nerve is true papilledema or pseudopapilledema? What specifically are you looking at on an ONH OCT to differentiate between true papilledema and pseudopapilledema?	I am looking at many factors to help determine if the patient has true optic nerve edema: 1. RNFL thickness on optic nerve scan, more specifically the contour or pattern. The RNFL should maintain its normal contour if there is no edema present. 2. Subretinal hyporeflective space (this is the "lazy V" you are referring to). The subretinal hyporeflective space will be much thicker in patients with optic nerve edema. 3. "Lumpy bumpy" appearance is hallmark for optic disc drusen. This article has some great pictures with captions: https://jamanetwork.com/journals/jamaophthalmology/fullarticle/420977
		If they have optic nerve drusen and you want to follow-up to rule out papilledema, how long should you wait Until the follow-up?	I typically have the patient return to clinic in 2-4 weeks, depending on the patient's case. If the patient is very asymptomatic, 4 weeks, as I do not expect them to have true papilledema.
		Do you do VF too in your workup?	Because most of my patients are under the age of 10, visual field is not part of my typical work up as they are usually not reliable. However, for my patients who are teenagers or who seem capable of performing a visual field, I do typically order visual field testing. Diagnosing optic nerve edema takes many tools to diagnose and monitor – fundus exam, fundus photos, OCT, and VF.

		Out of curiousity, what was the last case's final VA after treatment?	At her appointment in January 2020, her vision was 20/40 in each eye, with visual field defect superior nasal in the left eye and decreased color vision of the left eye.
		Good checklist for papilledema: 1. optic cup is V shaped, 2. tissue around disc is black (can be subtle with drusen however), 3. RPE is pushed back on OCT. Remedies in severe cases (pseudotumor): shunt (lumbo-peritoneal), gastric by-pass, and ON sheath fenestration	Yes, I agree!
		Would you start pt on diamox before or after imaging?	I start patients on Diamox after imaging, lumbar puncture, and any lab work.
12:00 – 1:00 pm	Shelby Leach, OD, FAAO Myopia Control: Back to the Basics & Beyond	How would your exam/thinking have changed if her development was slightly delayed?	In this case, my exam/thinking wouldn't have changed drastically since we weren't prescribing. In a Peds exam, I may consider the results of my testing or how I want to treat the patient to be slightly different (ex. Do they have a "20/20 brain"?)
		Will speak provide the reference to the study (outdoor time 40mins vs 80mins)?	The study is called "Time Outdoors as an Intervention for Myopia in Children" by Xu et al. It is unpublished. The URL is: https://clinicaltrials.gov/ct2/show/NCT02980445?cond=Myopia+outdoor+time&draw=2&rank=2
		for case 2 will you cut the astigmatism equally maintaining the aniso? will the reduced power solve his discomfort and unbalance improving his compliance in wearing glasses?	I would definitely cut the cyl while keeping the aniso. His symptoms are probably from repeatedly taking his glasses off. So I would enforce full-time wear with a slight back-off so that he can adapt fully.
		is there a minimum age you recommend for CL's, or do you recommend based on maturity?	Exactly, more on maturity! Also depends what you feel comfortable with. Most will do ortho-k starting around 5, and maybe slightly older for MFSCL since they need to know how to take them out themselves if something happens at school.

SOCCEP Q/A
Friday, April 3

		Would you send all high myopic kids for genetic counseling?	No, only if something in the case history or my exam findings would indicate it.
		What prescription would you have recommended for the -10.50 baby? Would you consider contacts if parents are competent and cooperative?	I would have prescribed either full or almost the full amount of myopia. She is probably mobile at 3, so we need to base it on her visual demands. Plus, if it is a progressive type, we don't want to underminus.
		what is your opinion on doing myopia control without measuring axial length, i am not sure of many ODs practicing with A scans, would appreciate your opinion on this matter, how do we differentiate between corneal curvature and axial length elongation without an A scan	While not ideal, it's doable! It will be the most difficult if you are doing ortho-k lenses, since you can't directly compare the change in Rx over time (only relatively compared to last visit). However, a topographer will be necessary with ortho-k because it tells you how the lens is fitting at night. You could potentially refer out for the A scan since it's only done once every 6-12 months.
		what is your opinion on bifocals for myopia control?	Bifocals are not as effective as the other 3 options. However, if the parents are against the main options, or the patient has an esophoria and we want to do a combo therapy, then I will prescribe.
		What add power would you give for the MF LENS	Most studies use either a +2 or +2.5D add. The theory is that the more peripheral myopic defocus, the better. However, if you listen to Dr. Kochik's myopia lecture from the first week, I believe she dives into that a little more. I tend to do as much plus as the patient can handle.
		If they haven't had genetic testing, would you start myopia control or wait for the testing to come back in order to determine if you think they will progress?	I wouldn't necessarily wait if I think that the myopia is progressing. While the genetic testing can give us answers, it won't answer whether the myopia is progressive or not. We still need more studies in this area.
1:00 – 2:00 pm	Pat Segu, OD, FAAO Ocular Ischemic Syndrome	Why does OIS cause a low grade anterior uveitis?	The uveitis is an inflammatory response to the ischemia. The patient may develop a low-grade iridocyclitis secondary to the lack of oxygen to the eye.

		What did you say was the differnce between to 50% blockage and 90% blockage of the CRA?	OIS develops when the internal carotid artery reaches 90% stenosis. When the patient develops OIS, then the blood flow to the CRA is reduced by 50%.
		Is the associated cataract in the affected eye typically of a certain type? NS vs. Cortical?	The eye with OIS will have more advance opacification of the lens. There is not s certain type of cataract associated with OIS. The lens becomes more opaque.
2:00 – 3:00 pm	Yin C. Tea, OD, FAAO Straighten Up! Abnormal Head Postures	Are we concerned about the behavior effects of using BU Yoked Prism?	Trial frame of Yoked Prism in office and allowing them to were it a bit, including walking around, will allow you to determine if the may have adaptation or mobility issues. My case was for prescribing yoked prism for optical treatment only, not behavioral.
		with vision therapy on the 15 year old, was vision therapy done with prisms or were they never used?	Vision therapy was initially performed with his glasses, so he had the yoked prism in them. Activities were increased in difficulty from primary gaze to upgaze OR with TF Rx without yoked prism. Also as he improved, activities were performed in upgaze (harder for him). For example vectos in free spaxe but placed in upgaze. OR Vectos placed in regular vecto holder, but TF Yoked BD prism so he would have to work fusion ranges in upgaze. We also did VT at end with training prism (so that would we prism opposite of what he normally likes, both yoked and/or horizontal training prism).
		Are we able to have access to these lecture slides afterwards for additional review?	Email directly for info.
		I am just watching (and very much enjoying) these lectures on my own time. It is not through or required by my university, so I don't have a faculty member to request it from	Please email SOCCEP
		The prism was placed on the right eye only ?	In the trial frame, yes 8 over paretic eye. In the final prescribed Rx, it was split. 6 over paretic eye, 2 over non-paretic eye.
		If you don't split the prism equally between the eyes like in this case, how	I only adjusted balance by a small amt to keep total prism low but get most

		do you determine what amount to put in the more paretic eye? Thanks!	therapeutic effect while maintaining good cosmetic balance to thickness of glasses.
		Could you please go over again why split the prism unequally?	It has to do with Primary vs. Secondary Deviations with paretic etiology. Think about what happens Prism Neutralization with ACT if the patient has a paretic muscle. If you place prism over the paretic eye, the prism moves the image for that eye and the paretic muscle can sit back and relax while the prism does the work (the prism amt that neutralizes the deviation is giving you the magnitude of the "primary deviation" when you do this). However, if you place prism over the non-paretic eye, you are moving the image for an eye that has no problem moving AND when it's the paretic eye's turn to fixate the straight ahead target, that poor eye has to work so hard to get that eye straight… the prism is over the other eye, the normal eye. So the brain needs to send extra signal to the paretic muscle just to get it to move and refixate the straight ahead target. When extra signal is needed for the paretic muscle to work, Herring's Law says excess innervation is sent to BOTH eyes, so the contralateral synergist in the non-paretic eye also gets the same excess innervation, pulling the normal eye behind the paddle, with the prism bar over it, in FURTHER, so you need more prism to neutralize. Your prism bar will read artificially high because it is measuring this excess innervation, aka secondary deviation. SO, long story short, in a paretic strabismus, you get more bang for your buck by placing prism over the paretic eye. I'm always looking to give the least amt of prism that gets the job done.
		Hi I was just wondering if these are being recorded and posted somewhere?	Yes. Your professors/faculty members are receiving the video links to use for instruction. Please reach out to them directly.
3:00 – 4:00 pm		Does rhopressa increase cornealscleral	Rhopressa works by enhancing

SOCCEP Q/A
Friday, April 3

	Sowmya Srinivas, OD, FAAO Glaucoma Suspect/Glaucoma Cases	outflow? I was under the impression that pilocarpine is the only drop that increases this route of outflow.	trabecular meshwork outflow
		How old was this patient?	Age is an important consideration when managing glaucoma.
		Shouldn't ganglions cells be considered as well?	Yes, indeed! Think of glaucoma as being a chronic disease in which all ganglion cells are insulted at about the same time with variable death rates for individual cell
		Is the age of the patient irrelevant?	Age is an important consideration when managing glaucoma
		The patient with drace heme, why was their so much hyperreflection of the ONH on the OCT?	Due to the peripapillary atrophy.
4:00 – 5:00 pm	Timothy E. Hug, OD, FAAO Peripheral Field Deficits Secondary to ROP tx	could a reverse telescope used like a bioptic be used for driving? to enlargen his field?	I think the minification from a reverse telescope would compromise his function…also I believe his functional field of vision would not improve much because of the retinal damage.
5:00 – 6:00 pm	Ava K. Bittner, OD, PhD, FAAO Sam I Am, I Do Like the OrCam!	Can low vision be clinically profitable-in private practice?	I have never been in private practice, but I reached out to my colleagues who are and do low vision. The answer is yes! (or else no one would do it, right?) It's more time consuming with difficult cases, so you'll need to schedule them at particular times with longer slots, but it's very rewarding. Patients self-pay for glasses and devices, so you'll need to be honest and clear about what they will do, as well as sign a form that indicates there are no refunds and their vision will still be reduced.
		What study was refered to, it started with Outcomes of the Veterans Affairs Low Vision ..... and it could not have time to read the rest. Thanks	Stelmack JA, Tang X, Wei Y et al. Outcomes of the veterans affairs low vision intervention trial II (LOVIT II): a randomized clinical trial. JAMA Ophthalmol 2017; 135: 96–104.

SOCCEP Q/A
Friday, April 3

6:00 – 7:00 pm	Torres Zulmaris, OD, FAAO Juvenile Glaucoma	1. What is the prognosis for juvenile glaucoma? Does a diagnosis at an earlier age influence the severity of the condition and visual outcomes? How can we counsel patients and parents what to expect? 2. What modifications would we need for dosing IOP-lowering medications to children with juvenile glaucoma? Where are the guidelines posted to follow?	1. Progression will depend on responses to treatment. Juvenile glaucoma by nature is more aggressive than adult glaucoma. Therefore, it is important to reinforce adherence to treatment to determine if it is slowing progression or if we need to refer the patient for a surgical option. The prognosis is definitely better if glaucoma is detected at an early stage. Regarding parents, I always present the case by talking about statistics and information based on the literature on the aggressiveness of glaucoma in the young population, and then I reinforce the importance of parental commitment and cooperation in terms of the use of drops and compliance with follow-up appointments. I set up an annual case progression discussion with parents where the patient will also participate to compare the findings of the previous year with the findings of the current year. Progression and prognosis is very individual. 2. I will give you a book reference. Pediatric Glaucoma Book. But in general, always use the lowest doses available, for

			example, Timolol comes in 2.5 mg / ml and in pediatric patients we choose 2.5 mg / ml. Weaver, D. T. (2012). Glaucoma. *Pediatric Clinical Ophthalmology: A Color Handbook*, *390*, 113–130.
		Why are you doing 30-2 instead of 24-2?	I usually use 24-2 in adults with glaucoma or suspects, but I like to use 30-2 in younger patients who can respond well to the fields because I like to r/o neurological involvement. It is important to me to r/o brain involvement in young patients with changes in the optic nerve. You can actually do either (30-2 or 24-2) on glaucoma patients, what is really important is to perform the same field parameters annually to analyze glaucoma progression.
		Where can we find some normative data for pediatric patients in regards to OCT findings: Av NFL, DA, RA, cup volume?	There are several studies on normative data on OCT in pediatric patients. I will provide you with some of them. Eslami (1).pdf Huynh (1).pdf Leung (1).pdf Salchow (3).pdf
		Would you treat an ocular hypertensive child? Or would you just monitor closely for any change?	It depends on many factors; this is a very individual decision of each case. First, to get a clear idea of how often I should

			monitor the patient, I make a list of the positive risk factors that the patient has. According to that I decide whether to f/up the patient for 3 months or every 6 months. At those visits I closely monitor the IOP to determine if there is an increasing trend. If the pressure has a positive tendency to increase or is above 30mmHG in combination with the functional and structural evaluation, I decide to start treatment. The patient in the case with Juvenile Ocular Hypertension has been on Latanoprost 0.005% HS for two years since pressures rose above 30mmHG , he is stable without damage to the nerve fiber or field, so he has not progressed to glaucoma and pressures have keep between 17mmHg and 19mmHg.