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Beyond IOP: Integrating Ocular Surface Resilience Into Glaucoma Management

**Chapter 1: Introduction and Opening Remarks**

**Dr. Whitley:**
Well, good evening. Good evening, everybody.

Tonight, we're going to talk about glaucoma, and we're going to talk about dry eye. And it's all beyond IOP, integrating ocular surface resilience into glaucoma management.

My name is Walt Whitley. I am the Director of Optometric Services at Virginia Eye Consultants in Norfolk, Virginia. Our practice is a tertiary referral care practice, all the various subspecialties. And my role within the practice is anything we do within ophthalmology is to make sure optometry is well represented. I'm here with my good friends, Jessica Steen, as well as Nate Lighthizer. You want to introduce yourself?

**Dr. Steen:**
I know some of you in the room. Thank you for coming out as a fairly local-ish Floridian. I'm in Fort Lauderdale, Florida, at Nova Southeastern University. I serve as Director of the Glaucoma Service, which means that from an optometric perspective, these are entry patients into a specialty secondary service where we work closely with our community doctors as far as advancing treatments and ensuring optimal interventional care, even in an academic environment.

**Dr. Lighthizer:**
Nate Lighthizer. I am a faculty member and serve as Dean at the College of Optometry. I've done a variety of roles over the last16 or 17 years in laser clinic, OD surgery clinic, dry eye clinic, and glaucoma clinic. And we all see patients that have ocular surface disease, and we all see patients that have glaucoma, and often they mix, so.

**Dr. Whitley:**
And that’s what we're going to talk about today.

So our objectives for this evening is to identify glaucoma patients with ocular surface disease and ocular surface inflammation—and Jessica is going to talk a little bit about that—, we're going to discuss novel mechanisms for increasing natural tear production in the context of a glaucoma patient, design management strategies for individualized ocular surface rehabilitation while maintaining optimal IOP control, and then match appropriate patients to ocular surface-sparing first-line glaucoma intervention, including SLT and DSLT—and Nate will take us through that.

To get us going, it's all you, Jessica.

**Chapter 2: Seeing the Unseen: Ocular Surface Disease and Inflammation in Glaucoma**

**Dr. Steen:**
Thank you, Walt. So when we think about this connection—seeing the unseen, or appreciating the underappreciated, or diagnosing the underdiagnosed—it's ocular surface disease and ocular surface inflammation in the context of our patients who have a glaucoma diagnosis and are currently being treated, I'll say, for ocular hypertension or glaucoma.

So what's our goal in management of the disease process that is open-angle glaucoma? It's to slow. We can't ultimately stop disease progression at this point. When we watch a patient for a long enough period of time, there will be evidence of progression. But the goal is to slow down the disease process, to alter—meaningfully alter—visual field progression and optic nerve and retinal nerve fiber layer damage, with the goal of preserving functional vision and functional vision-related quality of life.

And I always like to think about this—at what cost? That cost needs to be sustainable, meaning that, yes, we think about, of course, underdiagnosis in glaucoma, but I do think it's relevant to appreciate sometimes the overdiagnosis that can happen which really relates to the fact that glaucoma is a challenging condition to ultimately diagnose.

The Thessaloniki Eye Study understood or described that at least 50% of patients who had a glaucoma diagnosis with further evaluation were determined to not have disease.

Our goal really is, unfortunately, to prevent this type of progression. We're looking at progression analysis through an additional 5 years of where this particular patient had been, and you know he goes down to 0% of the visual field index in the right eye and is on this very steep downward trajectory in his only remaining eye. This is true functional visual function loss and associated quality-of-life loss, unfortunately associated with disease progression despite our best efforts and our surgical colleagues' best efforts. This is what we try to avoid, and yet it still happens in 2026, unfortunately.

When we think about how frequently we encounter dry eye disease in patients who are being treated for glaucoma, you know depending on the estimate, generally around 50%. I think about this in my practice, and is it higher? Is it lower? No. Overall, I would say it's probably higher than 50%.

And we think about this as ultimately not only medications that are tipping patients over the edge in being symptomatic of ocular surface disease, but also the fact that many of the features or risk factors for development of glaucoma are also the same risk factors for development of dry eye disease. So what's the biggest one? Age. We think about our female patients. We think about patients who have underlying existing systemic autoimmune dysfunction or those with inflammatory active disease, whether that is systemic disease that has an inflammatory or potential inflammatory component, and like type 2 diabetes mellitus. So we think about these overlapping features.

Topical ophthalmic medications, I do not believe induce dry eye disease or cause dry eye disease but they often unmask the symptoms related to it. And we look at this individual. She's had a trabeculectomy in each eye. She has lupus. She is on two immunomodulators, and she is only on topical ophthalmic intraocular pressure-lowering medications in one eye. Which eye is it? The eye with much more advanced surface staining on the right side of the image. It's her left eye. But the image on the left side of the screen—that right eye—she has significant active inflammation, superficial punctate keratopathy. There's a reduced tear meniscus height, and yet this is an eye that is not being treated with a topical ophthalmic medication.

Thinking about benzalkonium chloride, it is difficult to avoid in topical ophthalmic medications in the United States. This is different in other countries around the world. And we try to understand at the level of the ocular surface, what is the impact of benzalkonium chloride. And this particular study evaluated the number of BAK-preserved drops on a number of markers. And overall, you know Ocular Surface Disease Index—the OSDI index—increased with an increasing number of BAK-preserved agents.

But I think what I take away from this is that there is significant variability—Schirmer's test, lissamine green staining—important features. Do either of you guys perform Schirmer's testing at all in clinical practice any longer?

**Dr. Lighthizer:**
We do not and haven't for quite some time.

**Dr. Whitley:**
We didn't. And I did a lecture the other day, and I had a couple doctors say they did. And the reason why they said, like, 'is because I can show patients,' and for education purposes, I mean, I see that. But I kind of joke, I don't do it anymore because the drug companies do it for us in their clinical studies. They already proved it for us.

**Dr. Lighthizer:**
Yeah.

**Dr. Steen:**
In an academic institution, yes, we have Schirmer strips that exist. They are not something that I utilize. I tend to look more at tear meniscus height, either just behind the slit lamp in a qualitative way. I've also started on a number of patients to evaluate tear meniscus height using OCT, so as another additional potential feature.

Alright, so when I think about a patient who has open-angle glaucoma who's on topical ophthalmic intraocular pressure-lowering medications, adherence is core to successful treatment through that patient's life. And this is challenging. Of course, we appreciate the gaps, the disruptions in treatment, and really we think about how we can do it better.

So what's our biggest concern for patients who are utilizing topical IOP-lowering medicines? Well, hyperemia has been described as the most common adverse effect of prostaglandin analogs, our first-line medical therapy. Overall adherence has been described to be about 57% through just under 2 years of therapy. So is 2 years short term in glaucoma, or is that long term? That is short term. If we're diagnosing a new patient at the age of, let's say, 55, I think that I've got 45 more years to manage that patient.

And what will treatment through that patient's lifetime look like? We have to understand and be realistic that there will be gaps, restarts, discontinuations altogether of medicine, and that's where careful follow-up—but honestly careful assessment and careful discussion with your patient—to find those potential challenges. If we know about them, we can address them.

So how do we clinically assess patients? It starts with a full medical history. I truly do want to understand, with accuracy, the medications that the patient is taking and the medical conditions that that individual has, not only for consideration of topical medication and trying to avoid a contraindication, but also to appreciate potential systemic risk factors for glaucoma and for ocular surface disease.

And the way that I ask patients about how they use their medications is exactly that. It's not, how do you use the branded whichever medication they're on? Did you use it last night before bed? It's really that open-ended—tell me how you use your medicine, sir. Now, what about the big bottle with the dark blue cap? What about that little bottle with the green cap? How do you normally use that medicine? Is that one eye or both eyes? How many times a day do you use it? And when did you last use it? That's where you will find gaps or unfortunately even errors in medication use.

Just last week, a very well-experienced glaucoma patient had been using her prostaglandin analog for the last about 3 months twice a day. She's been on a PGA for about 5 years, and 3 months ago she said, 'You know what, I just started—I thought it would be better if I used it two times a day.'

So finding and asking even our experienced, well-understanding patients how they use their medicines at every visit, you find things.

And how do we assess for symptoms? I do use a SPEED questionnaire. This is something that any downtime in the exam, either as the patient is dilating or as I'm evaluating the data after the student or resident has evaluated the patient, we're talking about the case together, or in the waiting area. Any downtime in the examination, that's where I keep the patient busy with a SPEED questionnaire, and I come back into the room walking in with the results that I can quickly add up to have an additional symptomatic assessment. It's not so much of a yes-or-no question, but truly I want to know more about their burning, redness, tearing, or other symptoms, fluctuating vision that they may have.

So what we understand is that even when patients are provided medication at no cost and know that their physician is watching and evaluating their medication use, that there are real gaps and challenges in adherence to medical therapy.

On the left side of the screen—I think we're all going to pull something away from this particular study—on the left side of the screen, what do we see? Adherence increase immediately before a visit, and then take a fairly steep drop off in that lower tertile group immediately after the visit. On the right-hand side, this is evaluating the physician estimate of how adherent that patient is to therapy versus what actually happened. And we know that we're not very good. We're actually quite inefficient in evaluating how well our patients are adhering to topical therapy.

So what do we do clinically? Sodium fluorescein staining, assessment of tear meniscus height, tear osmolarity, and MMP-9 testing. This is where, as we're thinking about inflammation, these markers come into play. I think about this basic, from a basic perspective, in a glaucoma clinic. Sodium fluorescein, tear meniscus height are absolute requirements here. I do also look at tear breakup, not just time, but also the pattern of tear breakup. On occasion, I'll incorporate lissamine green, not on every visit, but to evaluate conjunctival staining and lid margin or lid wiper epitheliopathy, and then carefully looking at the eyelids. Look, lift, push, pull, and look down. This is a low-hanging fruit for treatment and to think about symptoms, even maybe before that person is started on any therapy or treatment for open-angle glaucoma or ocular hypertension.

And finally, you know this was a group that came together from around the world, but did not include Canadian or American practitioners, who had developed a recommendation pattern for the assessment of ocular surface disease or inflammation in glaucoma, thinking about symptoms and clinical signs. And what's the difference between ocular surface disease and ocular surface inflammation? That's where the incorporation of tear osmolarity and matrix metalloproteinase testing comes into play to differentiate the true inflammatory component that's there.

**Dr. Whitley:**
Jessica, that was a great presentation there. And you know when she's differentiated the ocular surface inflammation and ocular surface disease, a couple slides that stuck out was the look down, that gross one. You all saw those collarettes on those, and they got mites. And actually, studies have shown about 65% of patients with glaucoma also have Demodex.

And then the other picture that you had, one that was on drops on the picture on the right, and the other one, the inferior staining there, when we're looking at ocular surface disease, that's not really dry eye. We have to look at an incomplete lid seal, doing that Korb-Blackie light test to see is there a complete closure, or is there exposure due to poor anatomy? If it's an anatomy issue, then we're going to have to treat that accordingly, and that's what I'm going to talk about here.

Question for you, though, is, we have dry eye, we have glaucoma, you got to check the pressure, what is your protocol? And Nate, if you want to chime in too, after Jess.

**Dr. Steen:**
So sodium fluorescein without proparacaine, so sodium fluorescein strip first, quick assessment, ideally waiting about 60 seconds after instilling sodium fluorescein before assessing for the corneal staining pattern, then instilling proparacaine, checking the pressures, undilated 90. It's actually a fairly smooth workflow pattern. It doesn't slow the examination down, but just taking that extra step, again assessing the lids, again very quick, it doesn't disrupt your glaucoma examination, it just adds to the clinical profile.

**Dr. Whitley:**
So Nate, do you have to tell your staff not to touch, or do they check pressures anyway?

**Dr. Lighthizer:**
We don't have staff, we have students.

**Dr. Whitley:**
Oh, okay.

**Dr. Lighthizer:**
So our students, they know what to do there. We tell them not to touch right away, look at the surface, just like she described, and then check your pressures. We do it with Goldmann. That remains the standard in our glaucoma clinic. Yours, same thing?

**Dr. Steen:**

Yes.

**Dr. Lighthizer:**
So yep.

**Dr. Whitley:**
Yeah, for us, I have to tell my staff don't touch it, do a questionnaire, but you do the osmolarity, but don't touch anything else, because if they do, that's going to disrupt that tear film. And then we're putting that fluorescein, looking at that stain, it's starting to disrupt that tear film breakup time as well. We know with the non-invasive tear film breakup time, the normal number should be about 10 seconds. If we're just doing a fluorescein strip, should be about 5 seconds is what normal ison that. That's what was just in that TFOS DEWS III.

**Chapter 3: Individualized Management Strategies for Glaucoma with OSD/OSI**

**Dr. Whitley:**
But we're going to talk about individualized management strategies for glaucoma with ocular surface disease and ocular surface inflammation. And so I always say, if you have a glaucoma practice, you have a dry eye practice. I used to think it was 100%, and I was wrong, because it's not 100%, so it's 50%. We know with dry eye, well, when it comes to the glaucoma, the studies show it's up to 79% of patients with glaucoma have dry eye as well. But the main issue is make sure we're looking.

And so this math may not add up right, but all I'm going with here is if you have drops—the middle is the more mature patient, as Jessica just mentioned, age—we have to consider ocular surface, whether it's with inflammation or without. That patient in the middle, so talking about comorbidities, chronic medication, this patient had SLT in 2016, he had cataract surgery with a MIGS procedure in 2020, he had a trab a couple years later, still uncontrolled. So he's been on prostaglandins, alpha agonists, he's been on several medications, but cicatricial ectropion is what he developed over time due to the chronic medication. And so we're going to have to change our glaucoma treatments, but this is going to be a surgical evaluation treatment to address this patient's condition.

So if we look at the TFOS DEWS III, it's got broken down to several different algorithms. The one on the top left is tear film deficiencies, and that's looking at the various layers, whether it's the lipid layer, whether it's going to be the mucin layer. If we get that early staining with the conjunctival or with the lissamine green, we know that's early signs of patient with dry eye. And then there's evidence-based interventions for that.

On the right, the eyelid abnormalities, if it was Demodex, if a patient had a hordeolum, if they had something else going on with their glands, meibomian gland dysfunction, interventions for that.

But then on bottom, the ocular surface abnormalities, such as that ectropion patient I showed, that would be a surgical intervention.

And so, yes, they have glaucoma, yes, they have surface issues, but we need to break it down and identify which is going to be the best treatment option for our patients.

So triggers and treatments, we've all seen this vicious cycle, and it starts with the lids, and whether it's going to be with the bacteria that secretes lipases that increases the melting temperature of the meibomian glands, that leads to the blockage and dropout, to the internal circle of the dry eye disease, where we get tear film instability, hyper-evaporation, hyperosmolarity, inflammation, and ocular surface damage.

And you can see the various treatments, whether it's pharmaceuticals, whether it's going to be over-the-counter treatments, or if it's going to be MGD-type procedures that many of you are doing within your practice.

And so for any of those that are available, we need to look at what is the type of dry eye. We know 86% of all dry eye has an evaporative component. We know that almost 50% of patients have an aqueous deficiency when it comes to dry eye. Sambursky did a study, up to about 65% of patients with dry eye have inflammation, ocular surface inflammation.

And so when we're choosing our treatment options, what is going to be the best treatment for them? But how can we work upstream for these patients? Well, we've heard a dysregulation of a lacrimal functional unit. Many of you have heard of neuromodulation, whether it

was the device that you put in your nose, or this device you put outside your nose, the pharmaceuticals you spray inside your nose, or now we have a topical drop that can stimulate the TRPM8 receptors on the corneal surface. And so various ways that we can use our current therapeutics, but also the most recent treatment options.

When we're looking at dry eye, we know if there's inflammation, we have great treatments, our immunomodulators that have been around for over 20 years. We want to address quickly, we have our steroids. How often are y'all using steroids with your glaucoma patients on dry eye?

**Dr. Steen:**
When I really, really need to.

**Dr. Lighthizer:**
Yeah, and choose them wisely as well if you're going to go to them.

**Dr. Whitley:**
And what you'll hear from all of us, you prescribe a steroid, check the pressure, check the nerve, because if anyone's going to be a responder, it's going to be a glaucoma patient.

Anti-evaporatives. Actually, where does that fit in with, let's say, I'm on a PGA and you were going to address the anti-evaporative? there are several studies, 96% of patients that are on a prostaglandin have MGD. So how do you position an anti-evaporative for those patients?

**Dr. Lighthizer:**
I think it's got to have to be in there, involved. I think evaporation is such a key part of dry eye, and when we have those drops that cause so much MGD, you know you're going to have meibum issues. We know the job of meibum is anti-evaporative, so if you've got meibum issues, you're going to need an anti-evaporative on board.

**Dr. Whitley:**
Okay. And then neural stimulation. I'm going to get into this, but this is something that, if we can address the lacrimal functional unit, we can increase a basal tear. And so with the basal tear, the lipid layer, the aqueous layer, the mucin layer, there's over 2,000 components within the tears, and we know the purpose of the tear is for clear, comfortable vision. It's going to help protect that corneal surface and maintain homeostasis. And so whenever we're addressing a dry eye patient, we want a basal tear, not that reflex tear where it's due to a chemical or whether it's due to an eyelash in the eye. We want to help our patients produce better tears. Artificial tears aren't going to work. All it does, it washes out the 2,000 components of the antimicrobial proteins, the growth factors, the electrolytes.

But looking at this lacrimal functional unit, you're all familiar with the various components of it. And so essentially stimulating the nerves gives information to the trigeminal nerve that's going to send information to the meibomian glands, the goblet cells, as well as the lacrimal gland, to produce more of their tear.

We're hearing more about corneal neurons and tear production. We have the cold thermal receptors that can be stimulated, and those are the TRPM8 receptors, whether it's going to be on the cornea, or on the lids is typically, we're going to find the TRPM8.

The other receptors, I briefly mentioned, the mechano nociceptors and the polymodal, and that's due to the chemical or mechanical irritants that can cause the tearing.

How do we activate the trigeminal nerve? How do we activate the lacrimal functional unit? Whether we're targeting the ethmoid nerve within the nasal cavity, whether we're targeting it externally with some type of mechanical device, or we can do it on the ocular surface as well. And so what this does, it once again gets that lacrimal functional unit to produce a basal tear.

So the TRPM8, this is a receptor that's always surveilling the ocular surface. In between blinks, that interblink interval, anytime it decreases anywhere between 0.5 to 2 degrees Celsius, that amount of cooling stimulates the nerves to stimulate a blink. And so, you know if we can stimulate this with a pharmaceutical, it's one of the latest innovative ways for us to help increase a tear and a full basal tear.

And so we have the various pharmaceutical treatment options, but treatment options aren't alone when it comes to dry eye. We also have to look at the interventional glaucoma, and whether we're talking about SLT—which Nate's going to talk about—whether we're talking about minimally invasive glaucoma procedures, we need to help take control of the disease state. We're trying to prevent vision loss for our patient. We're trying to get better control.

You saw Jessica had slides on adherence and compliance. I'm going to have a couple after this. Nate's going to have the exact same ones, because that is a key issue when it comes to glaucoma, is the adherence. If it's not feeling good, patients also forget, they're not doing their drops. That's why you know we're seeing patients progress, is because of this adherence and compliance. And so is there a way we can intervene, just like we do with dry eye, interventional dry eye, doing IPL, doing thermal pulsation, or LLLT.

So a proactive approach to managing glaucoma that focus on early intervention, minimizing the impact of both the disease and its treatment. It involves proactive diagnostics, active disease monitoring, and using procedures early in the treatment to help preserve vision.

And so the same study that Jessica just showed, and if you all can, does anyone remember the travoprost dosing monitor? So these patients were paid to be in a study. My partner was the lead author on that. And what they found is 44% of patients use their drops less than 75% of the time. They were paid to be in a study, and all they had to do was do their drops, and they still didn't do it all the time.

**Dr. Lighthizer:**
And you know they're more compliant in studies than they are in real life, especially when they're being paid. So like, those numbers are inflated even in the favor of compliance, and it still was bad.

**Dr. Whitley:**
Yeah, and so, you know, even with once-a-day dosing, we're only getting 79% compliance with our patient. Four times a day, it's roughly flip a coin, it's about 51% compliance.

And so what does interventional glaucoma mean in the practice? Being proactive. Don't wait for progression. Do something to get control of the pressure. Do something to help prevent vision loss. And so traditionally, drops, more drops. For a moderate glaucoma patient, typically they're putting in three to four drops a day, and they all have BAK in there, and so they're all beating up the surface, and they're growing up, so the age is causing it to be more dry. And then as it progresses, that's where they need the various procedures.

But what's the standard? And Nate's going to talk about SLT and DSLT, drug delivery system, doing intracameral injections, the MIGS procedures. First-line therapy we're familiar, SLT which is becoming first line, and it’s within the various guidelines from the Academy, as well as AOA.

But we're all familiar with the drops, but drops can cause issues, and that's why we're having this discussion. So we have to think about what are some of the second-line issues, or what can we do next?

Cost is always going to come into account, because more drops cost money, procedures cost money. Are we at the target? Are they doing their drops? And the side effects, once again, because if it doesn't feel good, patients aren't going to do it.

Some of you may have used this. This is the Nanodropper. Essentially what it does, it takes a normal size drop, which is about 45 to 50 microliters, and with that top, it decreases it down to about 10 to 15 microliters. And so there's a study looking at regular timolol versus timolol with this little device, and what they found is that the majority of the time points, that it was non-inferior, so it worked just as well.

The graph on the right, that's looking at all of our different dry eye medications, we know that the eye can only hold 10 microliters of a drop, so with this dosing device, you can get enough drug to the target to render its effect and lower the pressure.

But some of our secondary options when patients have issues is going preservative free, and we have various treatments that are available. We can go orally. We can do this intracameral injection of bimatoprost if you work in Oklahoma, like Nate, or some of the other states, where essentially you’re going to put a pledget of bimatoprost into the anterior chamber that's slowly going to dissolve over

time.

And so Nate, in your experience, you put these in, we know in the clinical studies, it was approved, it was one pledget every 12 weeks. After one, in your experience, how long has that been lasting?

**Dr. Lighthizer:**
So this is within the scope of practice of optometrists in four states. Obviously, ophthalmology does this wherever they would like. When I put these in, I'm typically finding somewhere between 6 and 12 months is kind of the middle of the bell curve. Sometimes it's a little bit longer, sometimes it's shorter. That medication eludes out for 4 months at a time, but we're finding about a 6-to-12-month effect. So if patients need a drop holiday, you know if they're struggling with drops, their ocular surface is beat up, it's in rough shape, let's put the medicine inside the eye instead of having them take the drops for a certain period of time.

**Dr. Whitley:**
Yeah, and so just trying to find other ways to drop holiday, I mean that's something that many of our patients—the good news, well, the only good news with glaucoma, it's a slow, progressive condition, so we can take that holiday. There are studies showing one intracameral injection can last a year. Some say it lasts even longer, up to 2 years.

We could also do minimally invasive glaucoma procedures, and I used to always say, you have glaucoma and cataract, always think MIGS. But you have glaucoma and dry eye, we really need to consider the MIGS procedures. And so normally the stents are done with cataract surgery, but there's various other ways, whether it's goniotomy or canaloplasty.

This is my glaucoma partner, Connie Okeke here, and this is the Streamline procedure, where essentially she's going into the trabecular meshwork into Schlemm's canal, and what she's doing is injecting some viscoelastic at several different clock hours, 3 or 4 clock hours of treatments, a clock hour or two apart from each other, to help address and open up the trabecular meshwork. But with the viscoelastic, it's to help open up the drain to lower the pressure. We can get about 20-25%lowering with these procedures but getting patients off the various medications. And the more severe conditions, then that's where looking at our tubes and more advanced procedures.

But when it comes to glaucoma and dry eye management, we know that these coexist, and so we do have to treat them accordingly. Be proactive with the glaucoma treatment, interventional glaucoma, aggressive dry eye treatment. The earlier we treat a dry eye patient, whether it's with whatever treatment you want, then that prevents that chronic inflammatory condition that can be very difficult to treat for some patients.

Promote adherence, education, why the patient's here, you know what their condition is, what we're going to do about it, and why they need to own their condition. And that's what adherence is, right? Patients have to be self-motivated to do their own treatments, but then also consider ocular-sparing treatments, preservative-free, SLT.

And I'm going to let you go into SLT.

**Chapter 4: Implementing Ocular-Surface-Sparing Approaches: Focus on SLT/DSLT**

**Dr. Lighthizer:**
Alright, I guess I'm the third and final one before the case. We're going to talk on SLT and DSLT. Show of hands, how many of you have heard of DSLT before? You guys heard of DSLT? Okay, many of you have not.

So direct SLT, how many states? And you know you guys, many of you probably heard me lecture on SLT. One of my favorite topics to lecture on is SLT. Very passionate. I think it is an absolute travesty that all of you cannot perform an SLT, that your law does not allow you guys to do that.

Quiz, how many current states allow optometrists to do laser procedures? What's the answer? 5? 14? That answer was correct yesterday. Kansas became state 15 this morning when the governor signed it into law. So we now have— yeah, round of applause for that on Kansas.

**Dr. Steen:**
Yeah.

**Dr. Lighthizer:**
State number 15 where optometrists can do laser procedures, YAG capsulotomies, SLT among that.

So, you know this is something that has been proven time and time again to be first-line therapy. Who treats glaucoma first line? You guys do. So shouldn't you guys be able to offer the two wonderful first-line options, whether it's SLT or whether it's drops? These are amazing providers here. Walt now can do it in Virginia. You should be able to do this?

**Dr. Steen:**
Soon.

**Dr. Lighthizer:**
Soon.

**Dr. Steen:**
Soon.

**Dr. Lighthizer:**
Soon. There you go.

So, you know I think all of you probably agree this has been our historic treatment paradigm. Would you guys agree with this? Over the years and over the decades it's been drops are first, you maximize the drops, then you considered ALT back in the day, and then you saved the more invasive surgery for the end. We would argue now that this is your new paradigm. We've got MIGS mixed in, so don't forget about MIGS, but don't let me fool you on this one/two, or two/one. You've got two wonderful first-line options, wonderful medication patients, and we have a wonderful, gentle laser called SLT that has been proven in the SLT-MED study, the LiGHT study, among others, that it is first-line therapy.

So, you know why SLT? Let's wake you up after dinner and show you kind of a fun video. Okay, I don't know if we have the audio here. Can we hit play on that?

**Female Speaker:**
My asthma, they said they'd fix it, but it didn't make any difference at all.

**Male Speaker:**
Well, sometimes doctors make mistakes, Anna, you need to try twice as hard to fix them. Are you using your inhaler?

**Female Speaker:**
All the time. Go through one a week.

**Male Speaker:**
You sure you're using it right?

**Female Speaker:**
Do I look like an idiot?

**Male Speaker:**
No. Why don't you show me how your inhaler works?

**Dr. Lighthizer:**
Do I look like an idiot? Of course, I know how my inhaler works, and I know that's *House*, it's not even our neck of the woods, it's a TV show. But they've looked at this in the literature and say, 'Mrs. Jones, Mr. Jones, you say you're taking your drops, why don't you show

me how you take your drops?' Watch this.

This one's hard to see because of the shadow, but she literally puts like half the bottle in her eye. And here's my favorite one coming up here, let's drag it through the eyelid and the eyelashes, currently testing corneal sensitivity with the bottle tip right there.

Even the best of intention patients may be struggling. I know all of your glaucoma patients are athletic, 35-year-olds, is that correct? No, and they struggle. I see this with my father-in-law. He's 73, and he worked outside for UPS in the winters of North Dakota and his body looks like it's 85, and his current cupping is 0.95. I've done multiple SLTs on him. Vance Thompson Vision said cataract surgery and MIGS. He hates taking drops, and he just shakes, and he can't handle it.

I mean, this is the third time you guys have seen this study. Do you battle compliance? And every one of you says what?

**Dr. Steen:**
Absolutely.

**Dr. Lighthizer:**
Yes, we battle compliance. So you guys should be able to offer a treatment that eases the burden on these patients, and SLT does this. And it's not new. It's been around for a long time. SLT works via inflammation. It's biologic, or it's inflammatory. And I don't have time to go into all the details of this, but basically what happens during this laser, it is so quick. Look at the pulse duration of SLT, 3 nanoseconds. It takes the pigment in your body, it's something called the thermal relaxation time of melanin, 1 microsecond. That's how long it takes for pigment melanin to absorb laser energy and cause a burn.

How many of you have seen laser burns on the retina? Every one of you has. Those are with green lasers, thermal lasers that burn, that kill. SLT doesn't. You want to know why it doesn't? That slide right there, 3 nanoseconds. The pulse duration is so quick that there is no thermal destruction, there is no damage or coagulation that happens. It just puts a little stress on the drain, and when your body is stressed, what fights that? Your immune system does. When your body is sick, your immune system fights that. SLT is inflammatory, biologic. That's how we think this works. It's not destructive, it's not coagulation, but it all comes down to the pulse duration.

So when should it be indicated? What does the literature say? Well, you know if this was your patient that comes into your practice next week and their pressure is in the mid 20s, and you see their nerve and their visual field, let's say, let's call this glaucoma, what would you choose first line? What does the literature say?

This was the first big study in the United States that directly compared your number one glaucoma treatment, which is what, prostaglandins historically, that's your number one drop, versus SLT. This is out of Wills Eye, and this was in 2012, hard to believe, almost 14 years ago, and it compared SLT 360 degrees to a prostaglandin analog, first-line therapy, treatment-naive patients. And it said, what does better? Is it IOP reduction? Which one is better? And the number of treatment steps.

Okay, so I'm going to break you guys up. Okay, you guys on this half of the room, you were just diagnosed with glaucoma, you're getting an SLT first line. You guys on that half of the room, you were just diagnosed with glaucoma, and you're getting a prostaglandin. We're going to set target pressures. Do you guys believe setting target pressures in glaucoma?

**Dr. Steen:**
Absolutely.

**Dr. Lighthizer:**
And we're going to see you every 2 months in this study. And if you guys don't meet target that got SLT, you're getting more SLT. And those over here that got a drop, if you're not meeting target, you're getting more drop. That was the number of treatment steps. So IOP reduction and the number of treatment steps. IOP reduction was 26% for the SLT, 28% for the prostaglandin. That did not reach statistical significance between the two. So the results were what? The same. They were equal. You guys over here, the SLT group, 11% of you needed more treatment during the study, meaning you weren't meeting target, you needed more treatment. Over here, 27% of you in the med group needed more treatment during the one year of study.

Why do you think more of them needed more treatment during the study? What's the answer? Compliance. They weren't taking their

drop. Their pressure went up. They didn't meet target, and in this study they had to put more treatment on them. So that was their ultimate conclusion. IOP reduction was similar.

Go down to the bottom. These results support the option of SLT as a safe and effective initial therapy in open-angle glaucoma and ocular hypertension.

Walt, this was in 2012, Jessica, and I thought, here it is, and SLT is going to be first line from here on out. But you know what? Change is hard, isn't it?

**Dr. Whitley:**
Yes, it is.

**Dr. Lighthizer:**
It is. So more studies had to come out. The American Academy of Ophthalmology preferred practice patterns in 2017 and 2022, laser trabeculoplasty can be considered, now it can be used as initial therapy or adjunctive therapy.

Here's the big one. I'm sure all of you have heard of the LiGHT trial, and this was released in 2019, and it really helped to answer some of the criticisms of the SLT-MED study, which was it's a 1-year study and it's a small n number. So let's do a big study, big study, longer term. I'm going to go right to the conclusion: SLT, not could be, should be offered as a first-line treatment for open-angle glaucoma or ocular hypertension—wait for this—supporting a change in clinical practice.

Jessica, change is easy, right, for all of us?

**Dr. Steen:**
One day at a time.

**Dr. Lighthizer:**
One day at a time. Change is hard for all of us, myself included. But gone are the days of drop one and then drop two and then drop three. How many of those drops is your patient taking probably? Not many. Okay. So supporting a change in clinical practice.

Some of the key data from the 3-year LiGHT trial. Again, 3-year study, multicenter, big n numbers, 325-ish patients went down the SLT arm and down the drop arm. Five times less adverse events, and again, the key bullet here is the fourth one from the left. That's what I told patients in 2019, 2020, 2021, and 2022. Mrs. Jones, if we decide SLT is best for you first line, there's a 75 to 80% chance we will keep you drop free for the next 3 years. This does not work in everybody. One of the biggest downsides, it's going to wear off at some point. But what's one of the biggest upsides of SLT? When it wears off, what can we do? We can do it again. Okay. So again, big data, should be offered as a first-line treatment.

There was two other things from this first release of the LiGHT trial. They made a comment that there's a 97% probability that SLT is more cost effective than drops, 97%, and there was no quality-of-life difference at 3 years between the drops. The longer it goes, though, it should favor what? SLT.

If I put Walt on a drop today and do an SLT on Jessica today, tomorrow their quality of life is essentially the same, but the longer it goes, drop, drop, drop, Walt, your quality of life may not be as good.

**Dr. Whitley:**
I already forgot, so.

**Dr. Lighthizer:**
There you go. So this trial just kept going. So at 6 years, in September of 2022, they released the 6-year data. Quality of life still close in this study, but now it's slightly tipping towards the SLT side with the glaucoma symptom scores.

Here's the number I tell patients now. Mrs. Jones, if we decide that SLT is best for you, there's a 70% chance that we can keep you drop free for the next 6 years—70% chance, that's a big deal—70% chance. Nothing's 100% in medicine. And yeah, I always tell docs when

we travel around the country in our laser and surgery course, you know what should be on every single consent form that you do for SLT, ineffectiveness of the procedure and the need for further treatment options, including further surgery. Just because we did an SLT today doesn't mean you won't need a tube or a trab at some point down the road, or a MIGS or whatever it is, or repeating SLT. 70% stayed drop free for 6 years. That's not the most impressive bullet here. The next one is.

What is the ultimate goal in glaucoma? To prevent what? To prevent progression. More eyes in the drop arm exhibited disease progression over 6 years in the drop arm versus the SLT arm. 27% in the drop arm versus 19.5% in the SLT arm. I always say we have two equal first-line options, drops and SLT, and I always kick it to our glaucoma guy and I say, do we? And he goes, 'Based on this data, we might not.' SLT may be slightly better. You're more likely to progress in the drop arm.

And why is that? What's the answer? Compliance. You got it.

By the way, you were more likely to have a trab in the drop arm versus the SLT arm. You were more likely to have cataract surgery in the drop arm versus the SLT arm. There was no serious laser-related adverse events, and that was their ultimate conclusion, better long-term disease control, reduced need for surgery. Powerful data, powerful, powerful data in the 6-year LiGHT trial.

Alright, let's go. Indications. Our five main types of glaucoma where laser trabeculoplasty is indicated. Notice what all of these have, open angles—open angles, POAG and ocular hypertension are your two strongest indications, but the others are indicated as well.

Contraindications, again, a whole slew of things where it's contraindicated for one reason or another. Let me go back here. There we go.

Narrow angle, yeah, if you can't see into the angle, you probably can't do an SLT in its traditional form. And then a variety of secondary glaucomas, like inflammatory, angle recession, neovascular, age, and prior laser trabeculoplasty that failed. You can cross those bullets off if you're taking notes, those are no longer contraindications for SLT.

Alright, let's watch some videos. We're going to go quickly through this. This is traditional SLT. This is a 4-minute video. I'm going to show about 30 seconds of this. SLT is all about what skill, folks? Gonioscopy. Are you comfortable with a gonio lens in your hand?

You guys know when you started training for laser procedures your first year of optometry school, you didn't know it, because I didn't know it either, but when you were slit lamp training and gonioscopy training, you were starting to train for SLT. You have most of the foundational skills already needed for laser trabeculoplasty. You put your eyes behind the oculars, your hand on a joystick. You do that every single day. Okay?

This is a very simple, again, I don't have time to go through all the ins and outs of SLT. You're seeing champagne bubbles during this. You see one great big beam, and Walt, Jessica, you don't see really any tissue destruction there, do you, at all? Very gentle, very straightforward there.

So this is another SLT here with the— there we go, I'm going to go back a slide. This is our new RAPID—I shouldn't say new, but the new RAPID lens. Can we hit play on this video here? It has four mirrors. This is great to have four mirrors, because now you only have to rotate the lens once versus the Latina lens, when you had to rotate that more often there.

So that's the traditional form of SLT. This typically takes me somewhere between 2 and 4 minutes per eye to do an SLT. Typically takes our students somewhere between 4 and I won't even mention any higher amounts than that, but students take a little bit longer.

This is what I want you guys to remember, DSLT. This is exciting. It's intriguing. We're in our infancy stages. Again, how many of you have heard of DSLT? Show of hands. I want to see. Okay, so maybe 1/4 of the room. I was at SECO back in March of 2020, attending a subconference right before COVID ended the world, and there was a subconference called OIS, and this company up there came up that was called Belkin, and they showed an instrument that looks like this.

What does that look like in your office? An autorefractor. So this is automated direct transscleral SLT. What does that mean? Automated, you hit the button and it fires 120 shots for you automatically. It registers the limbus, and it's transscleral. There is no lens on the eye. It goes transscleral through the limbus. Automated direct transscleral SLT. So this has actually been studied, and this is actually FDA approved. It was FDA approved in December of 2023. Belkin sold this technology. It's now owned by Alcon, and it's known as the

Alcon Voyager.

Automated direct, it was studied in the GLAURIOUS trial. IOP reduction of 18 to 27%, fairly comparable to SLT.

Let's show you some videos. So again, there's the study there from the GLAURIOUS trial, why it was FDA approved, all of this and the good stuff.

Again, let's just show some videos. Procedure tips. You're going to have to put some proparacaine and tetracaine in, some artificial tears. You do need a lid speculum. You don't want them to blink during this.

By the way, guys, I forgot to mention, how long does this treatment take? Takes me 2 to 4 minutes to do an SLT per eye. This is 2.4 seconds, automated direct.

Okay, let's watch a couple videos. Alright, you want to hit play there. So chin in there, forehead up against the forehead rest, lid speculum so you don't blink, and there is the treatment session going right around there. Excellent. Look at that. SLT. Look how happy she looks. Doesn't she look so happy? Automated direct transscleral SLT, again, this is here, it's available. There's numerous practices around the country that have this.

It's interesting, early clinical experience. Does it induce possibly more inflammation? Is it slightly more painful? The answer is yes. They have to increase the energy a little bit in DSLT, because it's got to go transscleral through the sclera. The energy is about double regular SLT, so it's more painful. But are you willing to sit through a more painful 2.4 second procedure or a 2- to 4-minute procedure per eye? Again, that's kind of one of the trade-offs in that.

Darker pigmentation, may feel it more. Will not register the limbus with arcus and things like that. Similar IOP reduction to SLT, according to the studies, and we're still learning in that clinical experience. Okay.

Future directions. Again, guys, we're in our infancy of this. Anyway, there's the current instrument cost and kind of the treatment packs and things like that.

So Walt, back to you, buddy.

**Chapter 5: Interactive Case Dialogue: 72-Year-Old White Woman Presents for 6-Month POAG Follow-Up**

**Dr. Whitley:**
Yeah, that DSLT is really exciting. We can do SLT, as we have them at all of our clinics, but we're trying to get this in our main clinic just for those reasons. Efficiency, you can do it right there at that time in 2 seconds, and you saw those numbers or the efficacy in lowering that pressure.

And so my practice, we have about 34 providers, and sometimes you'll get a patient on your schedule that you may or may not have seen before. And so this was one of those cases. So here we have a 72-year-old white female who presents for 6-month open-angle glaucoma follow-up. And so here for her glaucoma and IOP check, daily grittiness, occasional blurry vision, has been using lifitegrast since the last visit, burns, uses timolol at night. You can see the ocular history, cataracts, dry eye. I saw her several months- a couple months ago, and she's had glaucoma since ‘97.Andyou can see her medical history, the medications they're on as well, and then the allergies that they have.

**Dr. Steen:**
Hold on, go back to that one moment. Alright, we got a couple of things that don't fit here. So how is she using her medicine? Timolol before bed. That's really the one time where you don't want to use timolol, right, related to ocular perfusion and salmeterol. I think we're missing something in her medical history. This is this gap. So does this patient have asthma that's not listed in her medical history? Yeah.

**Dr. Whitley:**
Yeah. And so, you know this case, this patient, looking back, she's been on that forever, and so you probably should have been

changing the timing of it, but we're going to address that either way here. So, that's a great point there.

Looking at the findings, vision's doing pretty good. Tear osmolarity, so I did no touch, of course, 315, so mild hyperosmolarity in the left eye. You can see the pachys, but also the staining that we're seeing here, you know 1+collarettes, 3+ SPK in the right inferiorly, dense in the left eye, has cataracts.

You can see the nerves as well and the staining. Thank you, Jessica, for the picture, since I didn't have one for this patient.

So other tests that we do, look at that lid seal. Anytime you have the inferior corneal staining, you know you want to make sure that they have a full closure when you put the transilluminator on their eye. Anytime you have 2+ staining or higher, that's when you also want to check, is it dry eye? They've already been on lifitegrast, and we know these dry eye medications work, and so we always have to think, consider NK, touching is it present, reduced, or absent? And it was reduced in the right, absent in the left. Then I did the pressures,14 and 13, and looked at the pressures there. OCT, you can see here, don't look at yellow, red, green disease, but trust me, there were issues here. The visual field, reliable fields, and then you can see the superior defects on both eyes. And she's already been on drops at night, but she's not supposed to be.

And so here's the assessment: dry eye, glaucoma, it's mild, and then cataracts as well.

And so the questions for you, how do you all approach that? Nate, I'll let you go first.

**Dr. Lighthizer:**
I'm looking at cataract surgery there. She's got some cataracts, and she's got some glaucoma, and let's take advantage while we're in there. Let's improve her vision, and you get that glaucoma surgery going as well. I think that's one of the places I would start.

**Dr. Whitley:**
What about if they're pseudophakic? We'll switch it.

**Dr. Steen:**
Pseudophakic. I mean, this is someone who should respond very well to SLT.

I'm absolutely stopping the timolol. This isa potential real risk for someone with asthma, never mind the time of day that she's taking it. Again, a prostaglandin analog, preservative-free prostaglandin analog, reasonable option for this woman too.

**Dr. Whitley:**
Would you do anything else for the dry eye or the surface?

**Dr. Lighthizer:**
Certainly, that ocular surface needs to be treated as well. I saw collarettes in there. I saw staining in there. I saw an elevated osmolarity. So while we cataracts and glaucoma, let's not forget the ocular surface as well, addressing it with a neuromodulator, an anti-inflammatory. When I see staining, I'm probably going to address that with an immunomodulator or steroid in the short term, but again, she's a glaucoma patient, so we may not, again, depending on how we want to choose that. But, you know let's get her ocular surface in a better state.

**Dr. Whitley:**
Yeah. And that's one of the issues we have, is that we can treat the Demodex, we can treat the dry eye. What are we going to do for the glaucoma? And so, if we do too much at once, then that can be an issue for our patients as well, because the more we give them, the less compliant they're going to be.

So stop the timolol, did the preservative-free latanoprost, and discuss the SLT, and that's what the patient did elect. For the dry eye, I went with the higher concentration of the cyclosporine with perfluorobutylpentane, which gets into the cornea, and that's been shown in various studies, but also helps with the symptoms.

But at the next visit, if they're still having those issues, if they have staining, we're doing some type of biologic, whether it's amniotic

membranes or SynergiMin. And then the cataract, discussed the cataract surgery as well, but she wasn't ready for it. She was still 20/20, so she wasn't as worried about that.

So after the SLTs, came back using the various drops, and you can see it was 13 and 14 before, and actually she got a better response, she's 10 and 11. Staining did improve in both eyes. Still had reduced sensitivity in both eyes. And so that's how she presented here for this evaluation.

So what are the next steps for this case? What would you do with the prostaglandin? Do you all stop it? Or do you keep them on it when you're scheduling SLT? Or do you just stop it? Or what do you do, Nate

**Dr. Lighthizer:**
So the answer that, like in many of glaucoma, is it depends. That's always the answer in glaucoma. But her glaucoma can withstand probably taking her off the drop, and I would take her off the drop when we started to do the SLT and just let the SLT kick in with the prostaglandin kicking out to equalize each other.

That's how I would do it. So, yeah.

**Dr. Steen:**
Ocular surface wise, looking back at the surface, I think that reassessment is really key, improvement, but maybe not to the level that we would expect.

**Dr. Whitley:**
Yep, and so that's just a case where you know there's several different variables there. Pick one, but the main thing is do something. And anything that you can do to preserve that surface, anything we do to help address the pressure is only going to benefit our patients.

We got a couple minutes left, so does anyone have any questions? If you do, please come up to the mic. If not, I love asking Jessica and Nate questions all the time, and so that's what I already prepared.

I started talking about the neuromodulation, and you know how's your experience been, whether it's going to be with varenicline or acoltremon and where does that fit into your dry eye armamentarium right now?

**Dr. Steen:**
No, I've had good success, really, with both medicines. And really the way that I think about it and describe it to patients is that this is increasing your natural tear production, so it's harnessing really what our body should be doing and just increasing that pathway.

So even especially for patients who were maybe even more cautious about any medical therapy, you know it's really giving more of the body its own natural tears.

So from a mechanism standpoint, patients are really on board even early on.

**Dr. Lighthizer:**
How many of you have used acoltremon at this point? It's been out for 6 months. How many of you used acoltremon, or TRYPTYR at this point? Show of hands. I think all of us would encourage you guys, you know the data is very overwhelmingly strong on this in terms of increased tear production on day 14, as early as day 1 and out to day 90, with symptom reduction or reduction in staining.

And I think to myself, when you're thinking dry eye, is there a patient where you wouldn't want to increase natural tear production? Your tears are so very important. Our tears are so very important, and we're going to treat those artificially a lot, or at least we have historically. If we can increase natural tear production, that's going to be a win for a patient. So I would encourage you guys to get some experience with that. They've been doing a really good job of getting it in the hands of our patients for the month, that first fill free.

**Dr. Whitley:**
Yeah, it's free. So Nate, it stings. What do you tell me about that?

**Dr. Lighthizer:**
Well, sure, get out in front of that with your patients. I always tell our patients, this tickles the nerve endings in your cornea. That's what I tell Mrs. Jones or Mr. Jones. This is a TRPM8 agonist—I don't go into that detail—but this is going to tickle the nerve endings. Well, if you touch or stimulate or tickle your nerve endings, you might sense that. You might feel that. So there's going to be some sensation, instillation site burning, stinging, or pain, 50% according to the studies. Most of it was mild, 98% was mild, and 86% lasted a minute or less.

Now, I've used it on my eyes, because I think we are better doctors when we experience the drops ourselves. And I will tell you, when I put it in my eyes, I went, whoo, I must be a wuss, because it was not mild for me. It was moderate to severe for me, but it was for about 30 seconds to a minute, and then my eyes had a cooling sensation for about a minute or 2, and then they felt better than they had in a long time.

So I'd encourage you guys to get some experience with our newer drops, whatever they are.

**Chapter 6: Closing Remark and Audience Q&A Session**

**Dr. Lighthizer:**
A question?

**Audience Member:**
Yeah, thanks. I have some questions. One, there was a study, I don't remember which one it was, that you quoted that your treatment pro- protocol resulted in a, it was like there were three different things that it resulted in. One of them was a lower incidence of cataract?

**Dr. Steen:**
SLT.

**Dr. Lighthizer:**
Oh, we're talking the LiGHT trial.

**Audience Member:**
Yeah. So could you elaborate on that cataract part? I didn't quite make that connection.

**Dr. Lighthizer:**
Yep, yeah. They had a lower risk of progression, like we saw in SLT, less trabs, which makes sense, and less rates of cataract surgery. Why do you think that is? Is cataract surgery, in a way, a glaucoma surgery? What's the answer?

**Audience Member:**
Absolutely.

**Dr. Lighthizer:**
Absolutely. So again, you're looking at those patients. Are they progressing? Well, we might as well take the cataract out, and maybe we get a couple of points lower on the pressure. So that's the explanation for why there was likely a lower rate, or there was a lower rate of cataract surgery.

**Audience Member:**
Okay, that makes sense. Thanks. And then the other question was I believe it was the DSLT that you said that melanin resulted, those patients had higher pain. So is that just the darker iris or the whole- well you know, the ciliary body, all that, or just your patients with darker skin, or it's kind of both?

**Dr. Lighthizer:**
SLT, remember, is selective for pigment. It's selective for melanin. So the more pigment, the more melanin you have at whatever surface it is, whether it's the conjunctiva, the iris, the trabecular meshwork, they can feel that a little bit more.

In regular SLT, we will lower our energy. With DSLT, we're still early in the experience, so if you don't lower it, they may feel it more in those darker pigmented individuals.

**Audience Member:**
Okay, thank you.

**Dr. Whitley:**
Well, thank you for your question, and thank you all for being here so much.

In the end, the main thing is do something, address the surface, consider SLT and DSLT first line. We all do within our clinic. Thank you all very much. Have a great rest of your Vision Expo.