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ODs and MDs forge educational initiative

The two professions look to build mutual trust and respect to improve patient outcomes

By Gretchyn M. Bailey, NCLC, FAAO
Editor in Chief, Content Channel Director

The American Academy of Optometry and the American Academy of Ophthalmology recently announced that they are planning to work together on joint educational initiatives. These initiatives are not yet planned, and the organizations anticipate that they will launch sometime in 2015.

"If we're going to move forward on this, it has to be in areas of mutual collaboration, trust, and respect between the professions," says Bernard J. Dolan, OD, MS, president of the American Academy of Optometry. "We're not naïve to think there isn't going to be conflict in some advocacy areas. We are hopeful to build a strong enough base to build positive patient outcomes. This may defuse some of the potential conflicts down the road, but I'm not naïve enough to think that they're going to disappear or never occur."

David W. Parke II, MD, chief executive officer of the American Academy of Ophthalmology, agrees. "If we're going to be totally candid, we know we're not going to agree on absolutely everything," he says. "We have a lot of history between the professions, not all of which is positive. The goal is to recognize that as the American Academy of Optometry is an educational organization, the American Academy of Ophthalmology is primarily an educational organization, and we have a tremendous positive interface."

Leadership for both organizations are aware that support from their respective memberships is important.

Says Dr. Parke: "We need to make sure that

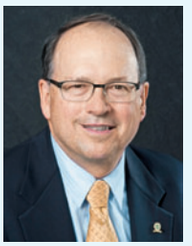
See **ODs and MDs** on page 6

Building a strong foundation

The organizations are looking for areas of mutual scientific and clinical interest that will help patients.

Hypothetical examples of such areas include:

- Keratoconus
- Ocular surface disease
- Genetic testing in eye disease



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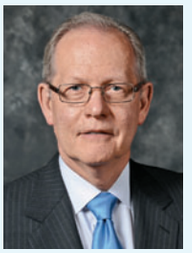

"We know we're not going to agree on absolutely everything. We have a lot of history between the professions, not all of which is positive."

—David W. Parke II, MD
Chief executive officer of the American Academy of Ophthalmology

GA Supreme Court rules in favor of optometrists

Spectera Inc. found in violation of Patient Access to Eye Care Act

By Colleen E. McCarthy
Content Specialist

The Georgia Supreme Court recently ruled in favor of a group of optometrists in a case against Spectera Inc., a vision care insurer. It is unclear how this ruling may affect other vision plans.

In 2010, Steven Wilson, OD; David Price, OD; Cynthia McMurray, OD; and Jodie Summers, OD; of Valdosta, GA, filed suit against Spectera when the company informed them that it would amend their contract by switching to an independent participating provider (IPP) agreement. Under the IPP, independent providers would be required to obtain covered materials from Spectera's laboratory, while retail providers would continue to obtain materials from any source.

Dr. Wilson filed suit against Spectera, arguing that the IPP agreement violated Georgia's Patient Access to Eye Care Act. Spectera then terminated its provider agreements with Drs. Wilson, McMurray, and Summers. Dr. Price filed a

See **GA Supreme Court** on page 6

INSIDE | **DIAGNOSTICS & IMAGING**

Demodex, AMD, more

This month's Special Section provides information on a variety of conditions helped by diagnostic and imaging technology. **SEE PAGE 18**

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References: 1. Kassan SS, Moutsopoulos HM. Clinical manifestations and early diagnosis of Sjögren syndrome. *Arch Intern Med.* 2004;164:1275-1284. **2.** Sjögren's Syndrome Foundation. Sjögren's Syndrome Foundation. 2001. Available at http://www.sjogrens.org. Accessed September 5, 2013. **3.** Liew M, Zhang M, Kim E, et al. Prevalence and predictors of Sjögren's syndrome in a prospective cohort of patients with aqueous-deficient dry eye. *Br J Ophthalmol.* 2012;96:1498-1503. **4.** Martin LS, Massafra U, Migliore A. Sjögren's syndrome: an under-diagnosed disorder. *CLI.* May 2004.

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Optometry Times is an optometry-driven publication that disseminates news and information of a clinical, socioeconomic, and political nature in a timely and accurate manner for members of the optometric community.

In partnership with our readers, we will achieve mutual success by:

- Being a forum for optometrists to communicate their clinical knowledge, insights, and discoveries.
- Providing management information that allows optometrists to enhance and expand their practices.
- Addressing political and socioeconomic issues that may either assist or hinder the optometric community, and reporting those issues and their potential outcomes to our readers.

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ODs and MDs

Continued from page 1

where we go is something that our memberships are comfortable with. Going from 0 to 60 mph is not in anyone's best interest. We are intentionally going to proceed slowly. It's our goal that this is a sustainable relationship."

According to Dr. Dolan, feedback from his organization has been overwhelmingly positive. Members of the American Academy of Ophthalmology have given modest feedback, according to Dr. Parke, with some strongly supportive and others opposed. Within both groups, some members are waiting for additional details on initiative scope and execution before lending support.

The organizations are looking for areas of mutual scientific and clinical interest that will help patients. In addition, those areas stay away from the "third rails," as Dr. Parke termed topics of disagreement between the groups. Hypothetical examples of such areas include keratoconus, ocular surface disease, and genetic testing in eye disease.

"[These examples] affect both professions, and hypothetically the two organizations can work to develop an evidence-based scientific statement to educate both member groups," says Dr. Parke. "These are examples of where you don't come close to those third rails."

Neither organization has asked other professional groups to the join the initiative.

"Up to this point, we haven't entertained having the American Optometric Association (AOA) involved," says Dr. Dolan. "The American Academy of Ophthalmology approached us to discuss education and patient outcomes. If you put the AOA in the mix, you do have one of their primary rules of the advocacy area. This is the area we're attempting to set aside. I advised the AOA leadership prior to the release of the press release, but in building trust and the relationship between the two, we haven't discussed it outside of the board of directors."

The AOA did not have a comment at this time on the proposed joint educational initiatives.

This past year, the American Society of Cataract and Refractive Surgery (ASCRS) provided education at its annual meeting to certain groups optometrists ("Certain ODs welcome to new ASCRS integrated program," February issue; "ASCRS welcomes ODs during IOMED program," May issue); however, the group is not part of this new educational initiative.

Says Dr. Parke: "ASCRS is different in many regards and not part of this initiative. The reason discussions are between the American Academy of Optometry and the American Academy of Ophthalmology is because these are, from my perspective, the two organizations in the profession that have global education as their primary missions."

ASCRS President Eric Donnenfeld, MD, says the collaboration between the two organizations is a progressive response addressing the future of eye care. "There is no question that this initiative coupled with what ASCRS is currently doing to help create a more coordinated and integrated eyecare model will lead to better healthcare delivery," he says.

A joint meeting of American Academy of Optometry and the American Academy of Ophthalmology has not been discussed; however, a joint symposium at both groups' annual meetings is on the table. The American Academy of Optometry has held joint symposia with ARVO at its past annual meetings.

Next steps for both organizations include obtaining more feedback from respective memberships and holding meetings beyond leadership. The more input members provide, the stronger the relationship will become, says Dr. Dolan.

"At the core this has to be based on the mutual interests of both organizations, based on trust, based on respect, and it has to be something which we feel can lead to a sustainable relationship," says Dr. Parke. "Those principles and a few others which underlie the culture of the relationship are going to drive how we move forward."**ODT**

GA Supreme Court

Continued from page 1

suit against the company after it failed to accept him as a provider based on his refusal to sign the IPP agreement. Those lawsuits, along with an additional suit filed by Drs. McMurray and Summers, were consolidated. The court entered a pre-trial injunction to prevent Spectera from terminating provider agreements until lawsuit was decided, and the trial court issued a permanent injunction, prohibiting Spectera from enforcing the IPP agreement.

The court denied Spectera's request to lift the injunction—making it a permanent injunction.

On appeal, the Georgia Court of Appeals agreed with the plaintiffs that the IPP agreement violated three sections of the Patient Access to Eye Care Act. The subsections violated prohibit eyecare providers (ECP) from providing services directly to their patients and restricts the licensure of an ECP's practice; and protects practices from being arbitrarily denied admittance onto an insurance company's provider panel. However, the court didn't believe the IPP agreement violated a subsection that prohibits the company from offering a more favorable contract to retail providers, which would put private-practice providers at a disadvantage, and drive patients to the retail chains.

In its appeal to the Georgia Supreme Court, Spectera argued that the court misinterpreted the Patient Access to Eye Care Act.

Spectera's attorneys argued that, "The Court of Appeals' decision misconstrues the statute ...and leads to absurd results that inevitably will raise premiums and reduce access to eye care." They further argued the ODs were not fighting for patient access, but to "preserve a lucrative revenue stream for contracted providers."

Plaintiffs' attorney argued the IPP agreement limits the ODs' function to dispensing and fitting eyeglasses and contact lenses prepared and supplied by Spectera's optical lab, and the plaintiffs would "not be able to ensure the provision of timely eye care and would not be able to ensure the provision of quality eye care to their patients covered by Spectera's plan."

The plaintiffs refuted Spectera's "lucrative income stream" claim, and said, "There is no evidence that Spectera's proposed agreement will reduce costs or maintain premiums."

The Georgia Supreme Court sustained the Appeals Court's ruling that the IPP agreement violated a subsection of the Act that allows patients to obtain services directly from the licensed ECP on the health benefit provider panel.

The Supreme Court reversed the Appeals Court's decision regarding the violation of the subsection that an vision care insurer "shall… allow each eyecare provider on a health benefit plan provider panel, without discrimination between classes of eyecare providers to furnish covered eyecare services to covered persons to the extent permitted by such provider's licensure."

The court also found that Dr. Price was justified in not signing the IPP agreement, as it violated the Act, and Spectera was wrong to deny him as a provider."

The court felt that permanently barring Spectera from terminating its contracts with Drs. Wilson, McMurray, and Summers, "goes too far," but any termination must be for legal reasons.

"The Supreme Court decision said that Spectera cannot preclude a patient from receiving any services—exams, and the services of preparing and dispensing any materials—directly from their provider," Dr. Wilson said.

Dr. Wilson said he expects other vision care insurers in the state to comply as well.

The National Association of Vision Care Plans, which represents the managed vision care industry, filed an amicus curiae brief for the case, siding with Spectera, to ensure other vision care insurers are aware of the case.**ODT**

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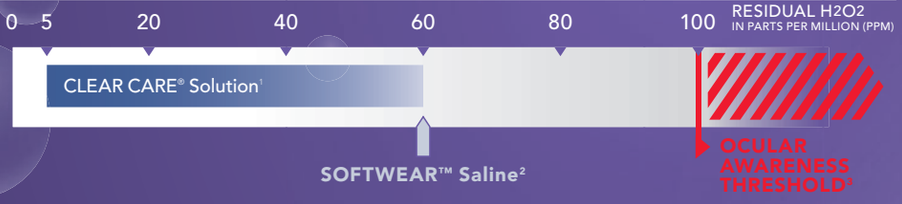

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My Christmas wish list

Ernie Bowling, OD, FAAO
Chief Optometric Editor

Remember as a child when you wrote out your Christmas wish list to Santa? You started in crayon, moved to a large, soft lead pencil and then just forgot about writing Santa as you got a little older, probably around the same time you graduated to an ink pen. I have to admit that I gave up making Christmas wish lists a long time ago. My kids, though, took care of that little chore for a lot of years. I don't think my wish lists as a child were quite as lengthy as were theirs!

Now that my children are grown and on their own, I'm thinking I want to harken back to earlier times and make a Christmas wish list. Not one for myself, as I could not ask for more, but a Christmas wish list for my colleagues and for my profession.

For my Editor in Chief, Gretchyn Bailey: I wish for you a case of 5-Hour Energy. I honestly don't know how you do what you do. Your clock has got to have more than 24 hours in a day.

For our Associate Optometric Editor Kathy Mastrota: I wish for you a stopwatch. You really need to slow down a little bit. You are a whirlwind. Or alternatively, I wish for you a southern accent so I can understand what you're saying when we're on the phone.

For the Primary Care Section of the American Academy of Optometry: I wish for us a few good Diplomates. The section can use some more working hands. It's amazing what so few people can accomplish. Mike Ohlson, Hal Bohlman, and Mike Radoiu, you are the tip of the sword. It is an honor to be associated with you.

For the inner sanctum: I wish I could see you guys more often than I do. With each passing year, I realize I'm hurling toward eternity, and I cherish the times I have with friends. I would never have gotten through optometry school without your help, support, and friendship. Mike Brown, Ray Spurlock, and Chris Teichmiller: words cannot express what you guys meant to me then, and still do.

I have a long list of wishes for my office staff, but because they think I'm Santa Claus, I guess it's up to my partner and I to make those wishes come true.

For my former writing partner Gregg Russell, I wish you a visit from the Muse and for you to start writing again. You are a talented writer and have much to offer. I miss our collaborations.

Also on the list, I would wish peace within our profession. There has been way too much bickering, divisiveness, and just general ill will in the last few years over a lot of issues. I wish for optometry to put all of that behind us and refocus on what's important: the unique opportunity we have to care for the patients who choose to grace our chair.

And to our readers, I wish health and happiness in the coming year. Everything we do at this magazine is for our readers, and I wish for you to continue to let me know how we can make this magazine even better and more helpful for your practice. Peace and prosperity throughout this holiday season and all of 2014!**ODT**

Women, leadership, and optometry

Gretchyn M. Bailey, NCLC, FAAO
Editor in Chief, Content Channel Director

The role of women in optometry has long held an important place in my heart. About 13 years ago, I was part of a fledgling women's group called DIVA (Dedication, Innovation, Vision, and Achievement), led by Drs. Carol Billups, Jan Jurkus, and Julie Ryan. Sadly, DIVA went away after several years.

A few years later, I became part of another group: Women of Vision. I have been lucky enough to be part of the leadership of this group, and I'm proud of the group's accomplishments, both in the past and under the current leadership of Dr. Louise Sclafani.

Recently, I was invited to attend a Women in Optometry meeting at Vistakon's The Vision Care Institute, the second event the company held. Vistakon gathered a group of women ODs who have been in practice fewer than 10 years. In addition to sessions on ocular comfort, astigmatism, UV, and Vistakon's latest product TruEye, plus a walk through the manufacturing and research facilities, attendees had opportunities to network and connect.

The daylong seminar kicked off with an interesting workshop using DISC (dominance, inducement, submission, and compliance) personality testing. Did you know that if everyone on your staff has the same strength as you, then you all likely have the same weakness? And, in times of stress, your greatest strength can become your weakness.

The best part of the day was the closing panel discussion of Vistakon's Women's Leadership Initiative. Moderated by Angela Deemer, the panel consisted of Drs. Sheila Hickson-Curran, Charissa Lee, Cristina Schnider, Tina McCarty, and Linda Chous. Panelists shared how they got where they are today and offered advice to attendees. I scribbled down as many pearls of wisdom as I could. I'll share a few:

"Allow yourself room for growth, both you and your practice. There are many opportunities to combine growth with seeing patients. Expect to do great things, and you will." —Linda Chous, OD

"Networking is more important than having a mentor. A mentor is just one person to help you." —Sheila Hickson-Curran, MCOptom

"Take a step out of your comfort zone. If you don't, you won't know your potential." —Charissa Lee, OD

"It always take someone else to help you, and you don't know who that will be." —Cristina Schnider, OD

Above all, panelists encouraged attendees to take on the mantle of leadership—in their local societies, their state associations, or even their churches or childrens' schools. Those in leadership will gain a lot, and it provides good networking opportunities. When you're part of leadership, said Dr. Schnider, you have the responsibility to bring others in.

"You have the power to give the gift of leadership to others," she said. "Invite someone in."

Vistakon plans to hold more Women in Optometry seminars in 2014. If you have the opportunity, go!

My colleague Sheryl Stevenson at sister publication *Ophthalmology Times* is very involved with Ophthalmic Women Leaders (OWL), a group I've not yet explored. For some time, I've wanted to check out this group. Maybe 2014… **ODT**

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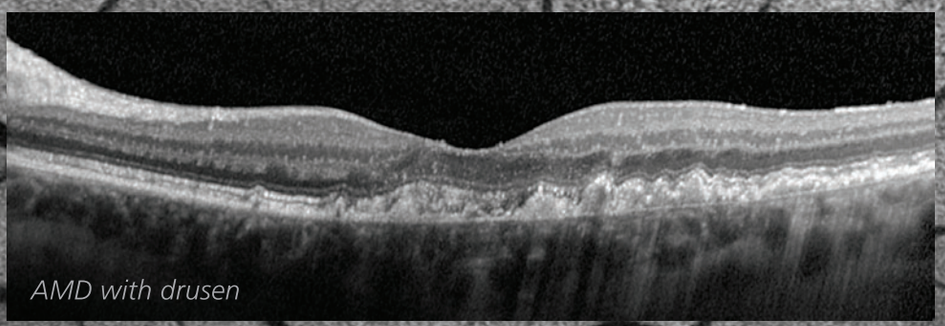

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Training doctors vs. training technical skills

Skip the why, and go straight to how

The structure of training an optometrist, or any other professional, is to start with a strong foundation of knowledge and build on that. This is designed to create a level of expertise and skill that can adapt to changes in the profession and acclimate to any situation in any system. Strong comprehension of the basics is crucial for this level of education.

How is training staff different?

We hire a new staff person not because we will need help in 4 years, but because we need help now. Because our offices are set up as a series of systems, we hire someone to run those systems. This is why technical training for staffers should wait until later.

Many people argue that giving a quick lesson on the basics is important before teaching technical component of how to accomplish any given task. This is not true. It actually makes it more difficult to learn.

Maybe it is **important** that the new employee have a basic understanding of the tests that they perform. But it is **important and urgent** that someone is doing the test soon.

In our office, most new employees begin as technicians and the first thing we "train" them in is auto-refraction and visual fields. We do this because these tasks are the easiest to learn, the hardest to mess up, and having a staffer handle those tasks can have immediate impact on the flow of the office.

Here's how:

1. Make sure the patient is comfortable. Like this....
2. Make sure the instrument is clean. Like this.....

Michael Rothschild, OD

Dr. Rothschild is director of What's Next-Leadership OD; a consultant for Alcon, Optos, and Vision Source; a member of the speakers' bureau for VSP; and a clinical researcher for CIBA Vision.

(We do break our why rule here. We explain why it is important to clean the instrument in front of the patient, or the staffer may try to improve efficiency and start cleaning between patients.)

3. Read this script and push this button.

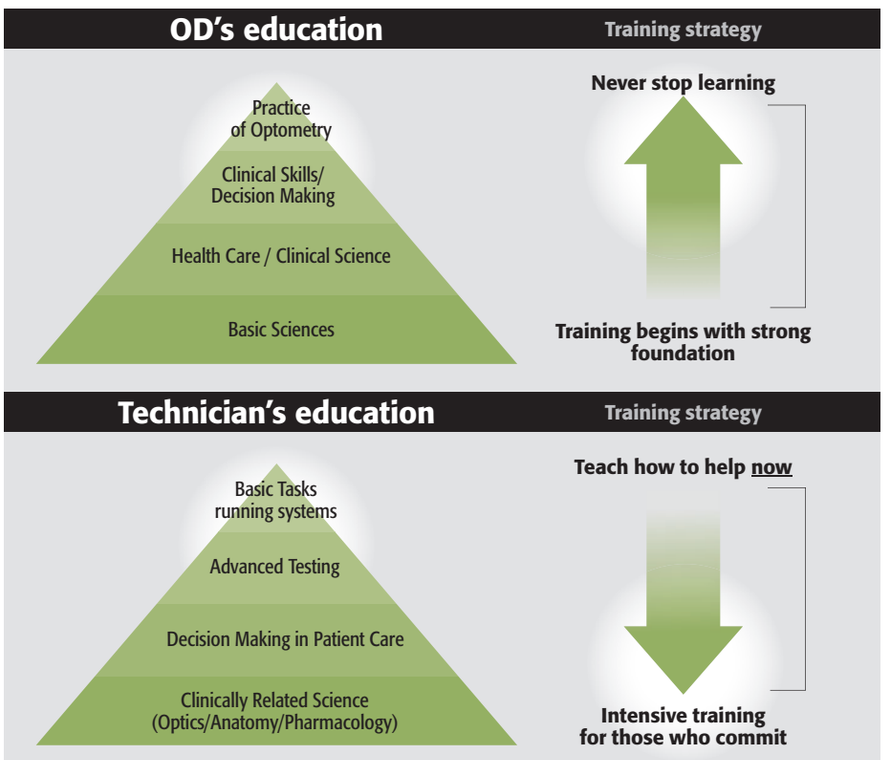
Congratulations! You are now an auto-refracting technician, and we are better because you are here.

To put this concept into perspective, consider these two points:

- To understand basic refraction, you have to first understand basic ocular anatomy and basic optics. *(If new employees don't understand, you never know.)*
- To operate an auto-refractor for the 2:00 patient, you need to know only how to push a button. *(If they don't understand, you will know very soon.)*

Employees are eager to make a difference in the practice, and optics is not easy for someone who might have been selling shampoo last week. Resist the very natural urge to talk about "why" we do the test, and get straight to "how." The "**why**" will come soon enough.**ODT**

Note: *Our first day of training is Day 3 on the job. Our first two days are reserved for Orientation which focuses on our mission, our values and the basics of the practice.*



These graphics give a general representation of an OD's education (top), building up from a strong foundation, and a technician's education (bottom), with a strong understanding of the foundations of the care they give. The strategy for training technicians follows the same basic structure as that of training optometrists; the difference is in the direction in which the concepts are taught. (Figures courtesy of Mike Rothschild, OD)

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As an optometrist who spent 33 years in private practice, I understand the needs of you and your patients. I know the rigors and challenges of independent practice and I am committed to making SynergEyes a company that is here for you. I look forward to bringing you the new SynergEyes: ***the leading global contact lens company for the independent eye care professional.***

I am genuinely interested in hearing from you. Please visit **http://SynergEyes.com/Partner/** to share your thoughts on how we can be a true partner to the independent ECP.

Very Sincerely,

James K. Kirchner, O.D.

President & CEO

SynergEyes

Vitreoretinal traction leads to a macular hole

Third in a series of retinal vignettes.

A 51 year-old female presented with recent-onset "distorted" vision in the right eye. The duration was described as 3 days and specifically, she used the terms "fragmented" and "pulsing" to describe her symptoms. She did say that she experienced slight headache just prior to the vision change. At the visit, there was no report of pain. Her medical history is significant for rheumatoid arthritis (RA). She was oriented as to person, place, and time.

Visual acuity was correctable to 20/60 (OD) and 20/25 (OS) with -4.00-0.75x039 and -5.75-0.25x150, respectively. Except for a cyst on the lower temporal lid and a mild papillary bulbar conjunctival response in each eye, the anterior segment was unremarkable. The IOP was 13 mm Hg @ 10:38 AM, in each eye.

Dilated fundus examination revealed clear lenses in each eye. Evaluation of the optic nerve head showed a distinct scleral ring with well-perfused rim tissue and an estimated C/D ratio of 0.5 in each eye. The macula in the right eye was significant for full-thickness macular hole with associated vitreomacular traction (Figure 1). The retinal vasculature was intact, and the periphery showed no signs of pre-disposing conditions to retinal detachement in either eye. The left macula was uninvolved (Figure 2).

An optical coherehnce (OCT) study was ordered and showed considerable traction at the macula of the right eye consistent with clincial obercvation (Figures 3A-C).

A retinal consultation was obtained. Given the recent onset of symptoms, the patient was offered the option of being re-evaluated in one month or sooner if symptomatic vision decrease occurred. In addition, a surgical option of intravitreal ocriplasmin (Jetrea, Thrombogenics) injeciton was reserved for the case of non-spontaneous resolution in the context of further visual acuity reduction.

Discussion

This case illustrates vitreomacular traction (VMT) causing macular hole formation. Recently two developments in the realm of VMT have emerged. First, development of a classification scheme for VMT based on OCT.[1] Briefly, vitreomacular *adhesion* is perifoveal separation of the vitreous with remaining vitreomacular attachment but preserved foveal tissue structure as seen on OCT. This situation is recognized as part of the normal course of vitreous aging but has the potential to lead to patholgoical consequences. Vitreomacular *traction* represents anatomic disruption of the fovea secondary to posterior vitreous detachment and may include pseudocysts, macular schisis, cystoid macular edema, and subretinal fluid. The foveal disruption can be quantitated from OCT measurements as greater (broad) or less than 1500 µm (focal). Finally, full-thickness macualr hole (FTMH) is a foveal lesion with interruptoin of all retinal layers from the internal limiting membrane to the retinal prgment epithelium. Subclassifications of FTMH are reported, as well.[1]

By Leo Semes, OD, FAAO,
Dr. Semes is a professor of optometry at the University of Alabama-Birmingham. He is a founding member of the Optometric Glaucoma Society and a founding fellow of the Optometric Retina Society.

By Audrey Thompson Otto, OD
Dr. Otto is the family practice resident at the UAB School of Optometry; she received her Doctor of Optometry degree there in 2013.

The clinical classification of macular hole would categorize the present case as a Stage 1B macular hole, according to the revised biomicroscopic staging scheme of Gass.[2] In a spectral-domain characterization, there is foveal pseudocyst with outer retinal separation and evidence of perifoveal PVD.[3]

Next, the FDA approval of ocriplasmin intravitreal injection based on a clinical trial conducted in Belgium.[4] The recommendation in this case illustrates the results from that clinical trial in which about 15% of 188 patients in the sham-treated group experienced spontaneous release of VMT, while about 28% of the 464 patients administered ocriplasmin had release of their VMT. Interestingly in each group, the vast majority of resolution occurred within the first month following intervention of initial observation.[4]

The receommendation to the patient appears to be perfectly consistent with

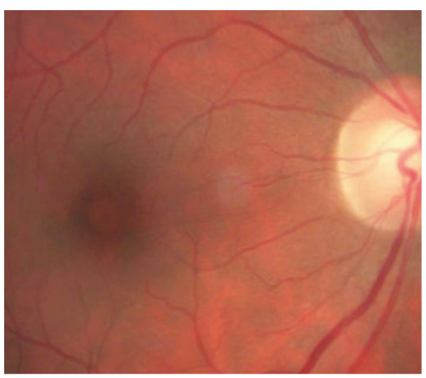

Figure 1. Fundus photo of the right eye showing disrupted macular region with specific distortion of foveal tissue.

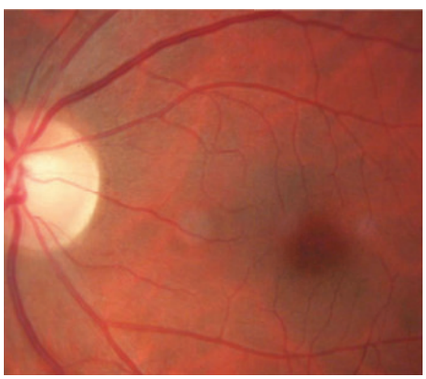

Figure 2. By comparison, the left macula appears normal. (Images courtesy of Leo Semes, OD, FAAO)

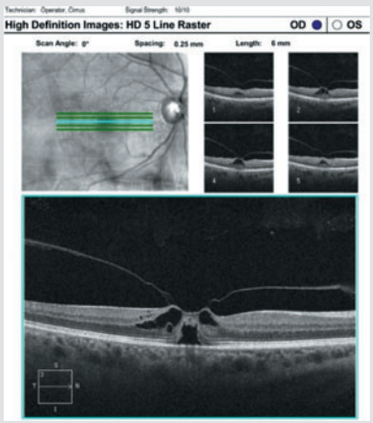

Figure 3A. Horizontal 5-Line raster SD-OCT showing vitreomacular traction (as defined by Reference 1) and full-thickness macular hole (FTMH).

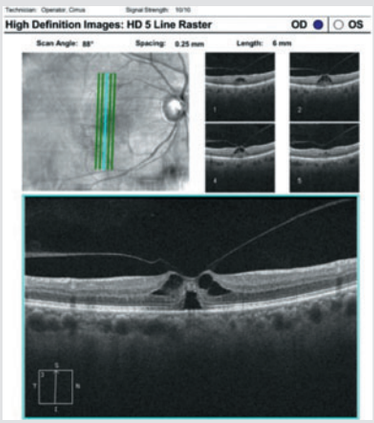

Figure 3B. Vertical 5-Line raster SD-OCT.

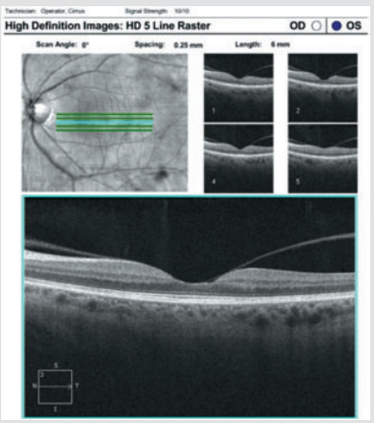

Figure 3C. Vitreomacular adhesion is evident in the scan from the left eye. Note that all cross-sections show two points of VMT. Visual field results from the right and left eyes as described in the text.

the guidance from interpretation of the study results.**ODT**

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7 pearls to guide glaucoma treatment

It's a tough diagnosis to make…and take away once made

We hear about structure vs. function in glaucoma. We hear that structural damage should correspond with functional damage (as evidenced by visual field studies).[1] We go to weekend lectures and see tidy cases of inferior neuroretinal rim notches with corresponding superior nasal step defects. We say, "That makes sense. That's how I would treat that patient."

By Benjamin P. Casella, OD, FAAO
Dr. Casella, a 2007 graduate of University of Alabama at Birmingham School of Optometry, practices in Augusta, GA , with his father in his grandfather's practice. He is a member of Allergan's speakers' bureau.

I get back to my office on Monday and sit down before glaucoma suspects who are not able to undergo reliable visual fields, have cataracts or other confounding variables affecting my evaluation of their optic nerves, can't lean over to get in front of the OCT, are poor historians, don't know their medications, etc. Then I'm left with diagnostic information that makes, at best, very little sense. I'm not saying structure and function don't always agree. I'm saying that my *testing* of them doesn't always agree.

I like having answers for my patients when they ask me if they have glaucoma. Often I just don't know if a patient truly has glaucoma or not (and, if present, whether I need to do anything about it). Glaucoma can be a tough diagnosis to make—and tough to take away once made. I've made use of a few pearls that have helped me to keep calm in the face of the beast that *is* glaucoma.

1. Keep it as simple as you can.

Making a case for a glaucoma diagnosis is complicated, but a fundamental approach can help. I start with stereoscopic optic nerve evaluation to help me to go through my mental flowchart of glaucoma care.

2. Look at the optic nerves without thinking about IOPs.

I can't do it either, but it hammers home a good point that we label people glaucoma suspects because of their optic nerves, not based solely on IOPs.

3. Patients can have as many diseases as they pleases.

Hypertension, hypotension, migraine headaches, etc., can affect, at least transiently, blood flow to the optic nerve.

4. Glaucoma is only one optic neuropathy.

A lot can happen to a ganglion cell on its way to its first synapse, which doesn't occur until it gets all the way to the lateral geniculate nucleus in the midbrain—1,000 miles for you and me. Many optic neuropathies may not be progressive at all. Yes, I know this directly contradicts the first pearl.

5. Repeat the visual field study.

Reliable and repeatable visual fields are important when determining the presence and rate of progression. Serial visual fields and OCTs can be a great way to get a feel for how a patient is going to behave in the future. Getting paid for that is a whole other subject.

6. You've got time on your side.

Establishing a diagnosis of glaucoma and deciding how to proceed can take time, but that's OK. Glaucoma is typically a slow disease, and it should take time to establish diagnosis and progression. You've got time to collect data and make sense of it.

7. It's OK to say "I don't know"

If a patient asks me, "Do I have glaucoma?" and I'm not sure, I'm honest. I may say, "I'm suspicious, and that's why we've done these baseline tests. However, I can't say that you *have* glaucoma. I want to repeat these tests again in a few months. If the results change, we should start treatment."

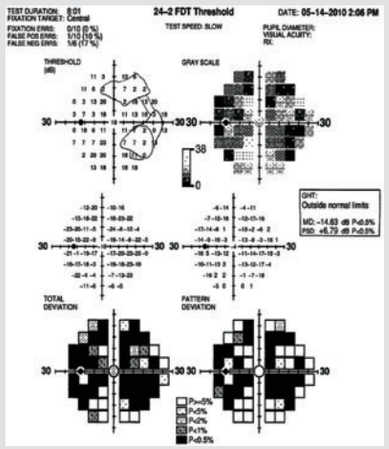

Figure 1. I couldn't make sense out of this FDT visual field study.

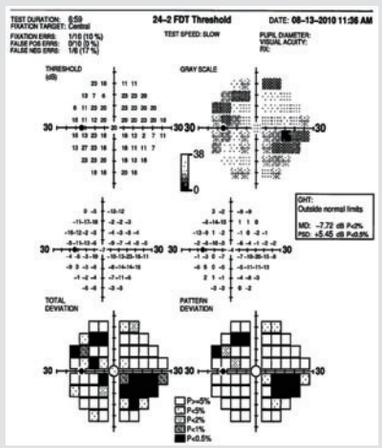

Figure 2. I brought the patient back in for another visual field, and this time I saw an inferior nasal defect. (Photos courtesy Benjamin P. Casella, OD, FAAO)

Taking a second look

In my naiveté, I tried to make some sense out of a patient's FDT visual field (see Figures 1 and 2). Bringing the patient back to repeat the study yielded an inferior nasal defect that I was more "comfortable" with.**ODT**

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New technologies to improve global eye health

Innovations on the horizon may reduce the incidence of refractive error

By Brien Holden, PhD

Statistics show that vision impairment is on the rise. In fact, over 600 million people worldwide suffer from uncorrected refractive error.[1,2] Two of the most common and rapidly increasing refractive conditions are myopia and presbyopia.

In 2010, nearly 1.4 billion people were impacted by myopia worldwide, and this number is expected to increase to 2.5 billion by 2020, with a significant number living in urban areas of Asia. In Singapore, approximately 70% of college graduates have myopia,[3] and in China, studies have shown that as many at 78% of 15 year olds living in urban areas of southern China children have the condition.[4] As a result of the world's aging population and increased life expectancy, presbyopia is also growing and is estimated to affect 1.5 billion people by 2050.[2]

Among all of the important public health priorities, vision impairment is one of the top causes of loss of wellbeing—ranking just below HIV/AIDS.[5] If left untreated, conditions like myopia and presbyopia can have an immense effect on people's performance at school, employment opportunities and, ultimately, quality of life.[6,7]

Take-Home Message

Approximately 2.5 billion people will be affected by myopia by 2050, while another 1.5 billion people will be affected by presbyopia. Through a series of collaborations, the Brien Holden Vision Institute is developing technology to slow the progression of myopia, treat cataracts, and detect and treat blinding eye diseases.

The correction of myopia and presbyopia is not only a humanitarian imperative; there is also a heavy economic cost to the community, particularly resulting from reduced productivity. The global economic burden of vision impairment ranks among cancer, dementia, and arthritis,[8] with the cost of global lost productivity resulting from uncorrected refractive error estimated at US$202 billion.[9,10] By contrast, the cost of correcting all global uncorrected refractive error is estimated between US$20 billion and US$28 billion.[10]

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and

US$28 billion.[10]

Slowing the progression of myopia

While glasses can help correct the problem of poor vision, they don't treat or prevent it. For a condition such as myopia, they are especially ineffective because they aren't able to slow or halt the progression to high levels of myopia, which significantly increases the risk of developing potentially blinding conditions including myopic macular degeneration (MMD), retinal damage, retinal detachment, glaucoma, and cataract.

Specifically, evidence is mounting that MMD will become the major cause of blindness in Asia and a growing threat for many other countries where the prevalence of myopia is on the rise. In fact, a meta analysis conducted in 2006 found that the impact of myopia may be greater than it seems and that a better understanding of the risks of the condition by eyecare practitioners may help facilitate the screening and management of myopia-related ocular complications.[11]

Data from Asia also provides some understanding of the magnitude of this emerging threat. A recent study found that MMD is now the leading cause of blindness in Jing-An District, Shanghai, China, with rates of blindness increasing from 114 out of 100,000 people in 2003 to 166 out of 100,000 people in 2009.[12] Additionally, in the U.S. there is clear evidence that myopia is on the rise, but worryingly, there is little knowledge about the potential impact on levels of blindness.[13,14]

Research and subsequent action are essential to combat the health concerns associated with this increasing prevalence of eye conditions. In the area of myopia, researchers around the world are looking at different approaches to treat the condition by reducing its rate of development and preventing its progression to the higher levels which significantly increase the risk of blindness. In the

See **Global eye health** on page 16

Global eye health

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field of presbyopia and cataract, a range of surgical treatments such as an intraocular lens (IOL) is being pursued.

Brien Holden Vision Institute is continuously looking at ways of reducing the incidence of refractive error in addition to employing the normal corrective measure, glasses. This is currently being achieved through a variety of projects, including the Myopia Control Program, Accommodating Gel Project, and the Intelligent Retinal Camera Project.

Myopia Control Program

The rate of increase in myopia can be slowed by an optical intervention based on bringing the peripheral focus closer to, on, or in front of the retina.[15] In collaboration with the University of Houston, the Brien Holden Vision Institute is working to translate this technology into contact lenses and spectacle treatments.

Through the program, several spectacle lenses were designed and developed in collaboration with industry partner Carl Zeiss Vision. These lenses were then assessed in a 1-year trial conducted at the Zhongshan Ophthalmic Center, Guangzhou, China. The results of the trial showed a reduction in myopia progression by 30% in children between the ages of 6 and 12 years old (with at least one myopic parent) and led to the development of the first myopia control spectacle lens. Results from this trial and subsequent studies have also generated invaluable information that has been used in designing and testing the next generation myopia control spectacle lenses. Contact lens studies have been even more rewarding with a steady 40% reduction in rate of progress of myopia using lenses testing the Earl Smith strategy of reducing peripheral hyperopia. Such a reduction would result in far less children developing high myopia with its attendant risks.

Accommodating Gel Project

Cataracts are presently the leading cause of blindness and the second largest cause of vision impairment in the world. In 2010, there were an estimated 24 million Americans 40 years old and over with cataracts and this number is predicted to increase to approximately 50 million by 2050.[16]

Through the Accommodating Gel project, Adventus Technology, Inc., as subsidiary of the Brien Holden Vision Institute, and collaborators in the Vision Cooperative Research Centre (Vision CRC) are developing a clear gel-lens system that will replace old presbyopic or cataractous lenses of the eye. Existing technology doesn't restore the eye's natural ability to focus, but the Accommodating Gel technology could potentially provide an alternative for lens replacement in cataract surgery procedures, during which the opaque lens is removed and replaced by a synthetic IOL.

> Vision impairment is one of the top causes of loss of wellbeing—ranking just below HIV/AIDS.
>
> Brien Holden, PhD

Intelligent Retinal Camera Project

Through stimulus funding provided by the Australian Government's Cooperative Research Centre Program, Brien Holden Vision Institute and collaborators in the Vision CRC based in Sydney are developing imaging technology for a breakthrough retinal camera that will enable real-time detection and assessment of common blinding eye disease and general health disorders.

The intelligent retinal camera is intended to be able to accurately and rapidly detect and eventually diagnose sight-threatening conditions such as diabetic retinopathy and glaucoma. Additionally, it is being designed for use in the most extreme environments, so that it can be operated by technical support staff in remote and under-served locations worldwide. The camera will take high-resolution, multispectral images. The camera is being designed to provide fixed-baseline stereoscopic images, which would allow clinicians to see the shape and depth of features within the eye and monitor changes in those features over time.

Additional information on the global impact of avoidable blindness and the research being conducted by Brien Holden Vision Institute can be found in "A Vision for All to See: A report on global eye health and vision care."**ODT**

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Author Info

Brien Holden is a world leader in vision science, eye health, and elimination of vision impairment and avoidable blindness around the world. His contributions extend across research, education, public health, and social enterprise. Brien has been awarded an Order of Australia Medal for his work in eye health and vision science; the Schwab Social Entrepreneur Award for Africa 2010 at the regional World Economic Forum; and seven honorary doctorates from universities worldwide. He has generated over $1 billion in research, education, and humanitarian funds over the last 40 years.

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Diagnosing Demodex

Five clinical cues and two diagnostic techniques confirm mite presence

By Scott Hauswirth, OD, FAAO

Over the past 10 years, we have witnessed a striking transformation in the way we as eye physicians approach ocular surface disease. We have seen definitions tighten and become more descriptive to better indicate what we should understand about the pathophysiologic disease process of each subcategory. And while the movement toward a more detailed understanding of meibomian gland dysfunction, dry eye, and blepharitis has brought us great advances, the truth is that due to the close proximity and continual interaction of the system as a whole, there is much overlap among these disease entities, and most patients have more than one of these processes occurring simultaneously.

Take-Home Message

Minimizing Demodex mite populations on the lids contributes toward a pro-inflammatory environment of the lids. Eliminating parasitic overpopulation in the lids and lashes decreases inflammation and provides a healthier environment for the ocular surface to function at its best.

Enter another "new" (yet surprisingly old) twist: the presence of a small ectoparasite which has been—for the most part—ignored for many decades: Demodex. This small creature is something we were briefly educated about in school; however, chances are high that we never actually witnessed our attendings diagnose or present to us a patient with mites or even saw a photo of what they looked like. Yet prevalence studies indicate that by age 70, virtually 100% of our patients carry significant numbers of these mites in their lashes.[1]

How and why have we been missing this for so long? Recently, much more attention has been given to this tiny creature and the contributory role it may play in just about every form of ocular surface disease—pterygium,[2] ocular rosacea, allergy, and meibomian gland dysfunction (MGD),[3] to name but a few. On the opposite side of the argument is that a large number of patients present without significant symptoms, with the thought process being that it is an entity that can be dismissed. As a clinician who has a passion for ocular surface disease, the fact we may have been overlooking a potential piece of the ocular surface puzzle is amazing, demonstrating to me that there is still much opportunity to learn how all of these entities interact.

I have been baffled at the sheer number of patients in my clinic who have large infestations of this tiny mite. I have been pleased when we are able to minimize their level of

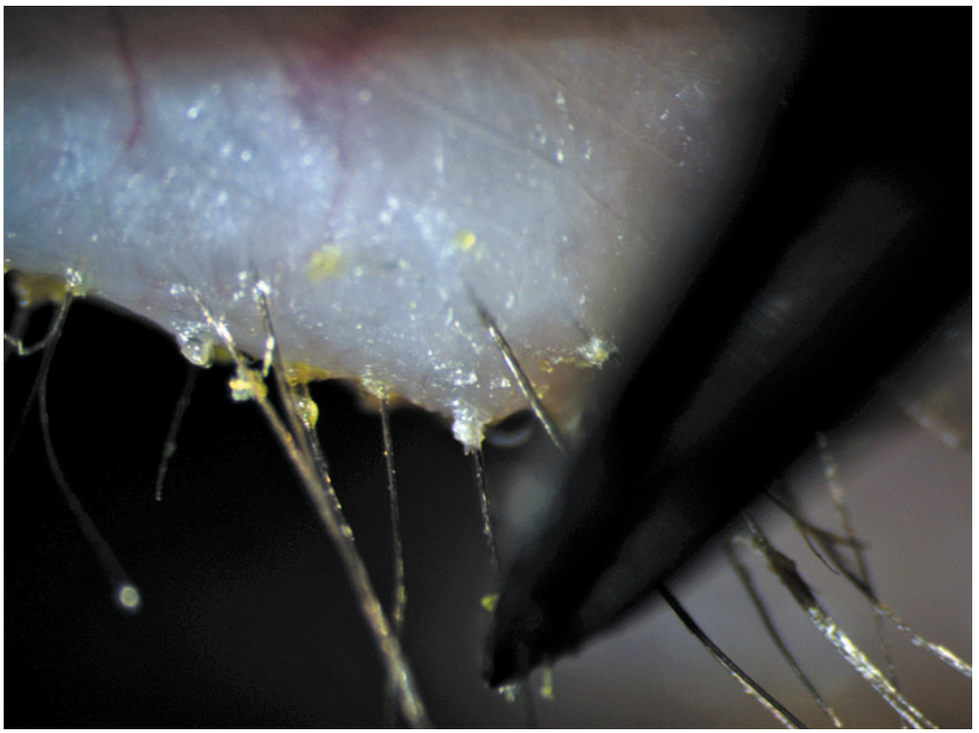

Figure 1. Gently pull a lash to begin rotating. Note the three mite tails visible as the smoothness of the CD is broken.

Figure 2. Lid with Demodex blepharitis. Note the fragility and thinning of the lashes. Almost all lashes have presence of CD.

Finding the mite

Look for these 5 clinical clues to alert you to the presence of Demodex overpopulation, then choose one of these 2 diagnostic techniques to confirm mite presence.

- **Clinical clues**
- **Cylindrical dandruff**
- **Alterations of the skin surrounding the lash follicle**
- **Changes in lash appearance**
- **Lid hyperemia/telangiectasia**

Patient history/associations with other disease
Diagnostic techniques

- **Lashe pilation**
- **Lash rotation**

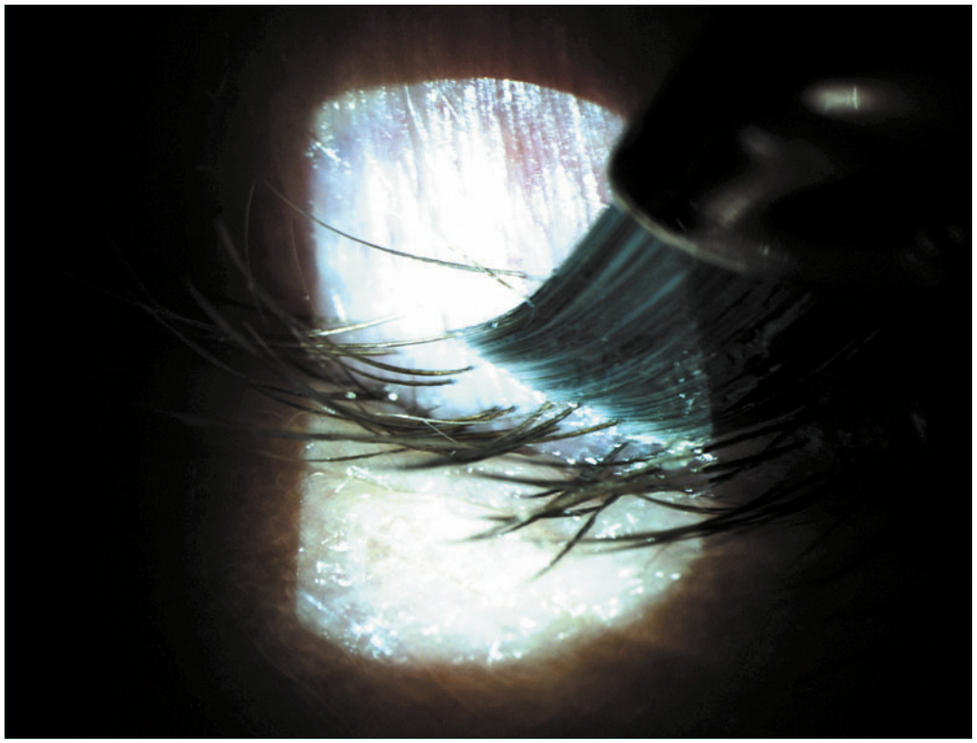

Figure 3. Tea tree oil application with an Ocusoft brush. (Photos courtesy Scott G. Hauswirth, OD, FAAO)

infestation, and often their ocular surface health indicators, both symptoms and objective signs, seem to improve as well. Like many ocular surface patients, Demodex patients often present without symptoms, most likely due to damage to the nerves that conduct signals or simple adaptation to the constant irritation. Just as in dry eye and MGD, symptoms alone are not necessarily the best means to diagnose the problem, and lack of symptoms shouldn't preclude these patients from treatment.

5 clinical clues

How do we best diagnose the presence of Demodex? There are several clinical signs that can alert us to the presence of Demodex in the lashes, and if we are aware of these clues, we can then perform one of two simple examination techniques that will confirm the presence of the mites and help us determine the best course of action.

1 Cylindrical dandruff (CD). This is the most obvious clinical sign and is the best indicator for mite infestation. Cylindrical dandruff is visible at the slit lamp, and it is different than the scaly, scurf-like debris seen with traditional anterior blepharitis. CD is a type of debris that tends to appear more waxy, similar to dripping candle wax. It forms a sheath on the lower portion of the lash as the lash emerges from the follicle and can be a millimeter or longer in length. It tends to be most pronounced on the lashes of the upper lid, but it may be found on the lashes of the lower lid as well. When present, it is nearly pathognomonic for Demodex infestation.[4]

2 Alterations of the skin surrounding the lash follicle. The skin near the opening of the follicle becomes distended and rises up, looking much like a pyramid. Also, there may be a greasy, oily appearance to the skin surrounding the follicle.[1]

3 Changes in lash appearance. In longstanding infestations, the lashes become thin and brittle, or they may begin to lose their color. Occasionally we may see misdirection and loss of lashes,[5] which likely is the result of longstanding infestation and the effects of inflammatory damage to the structure of the lash follicle.

4 Lid hyperemia/telangiectasia. Because the presence of mites is associated with increases in interleukins and different types of pro-inflammatory cytokines,[6] we can see increases in levels of lid inflammation as well. This may lead to increased vascularization along the lid margin.

5 Patient history/associations with other disease. Prevalence of Demodex increases with age. Systemically, we see a very strong association in patients with acne rosacea, seborrheic dermatitis, and other forms of inflammatory skin conditions,[7] as well as diabetes.[8,9] There is also an association symptomatically with allergic symptoms such as itching.[10]

Confirming mite presence

Those are the clinical signs that can alert us to the presence of Demodex overpopulation. However, each of these items is not truly diagnostic in its own right—with the possible exception of cylindrical dandruff. Confirmation of the presence of mites, as well as establishing a means of categorizing the degree of mite infestation, can be done through one of two simple steps:

- **Lash epilation.** This method involves removal of a lash from the lid along with any associated debris/cylindrical dandruff and viewing the complex under a light microscope. Lash selection is important because the number of mites may vary between adjacent follicles. Choose lashes which have a fair amount of CD visible at the base, and I tend to choose those lashes which are slightly thinned relative to the average lash. Many mites (mostly dead, but some live) may also be found within the CD debris, so it can be productive to look at that as well. Lash epilation is relatively easy to do, but there is a good possibility that several mites may be left behind in the follicle. However, most studies involving Demodex utilize this method, typically examin-

See **Demodex** on page 20

Demodex

Continued from page 19

ing between two and four different lashes per lid.[11]

- **Lash rotation.** This method, first described by Mastrota,[12] involves using a forceps to pinch a lash a few millimeters above the base of the follicle. Keep the lash follicle as the centerpoint, and use a combination of a gentle pull and rotation of the lash around the follicle like the hands of a clock. After a few turns, oily debris and mite tails will begin to emerge out of the follicle. This debris can also be transferred to a slide and examined under a light microscope, but is easy to visualize under high magnification at the slit lamp (25-40x). The technique of lash rotation is slightly more difficult to master; however, in a busy clinic it is more efficient to perform than lash epilation, and is my preferred method. The major drawback is if a lot of debris is exuded from the follicle, it may be more difficult to visualize all of the mites to get an accurate count.

When cylindrical dandruff is present, it is nearly pathognomonic for Demodex infestation.

While minimizing Demodex mite populations on the lids will not likely solve all forms of ocular surface disease for our patients, decreasing their ability to contribute toward a pro-inflammatory environment of the lids would logically carry benefit for patients and aid in any concurrent treatments. As clinicians, our primary goal should be to identify pathology and provide treatment, helping ensure that our patient's visual systems continue to function at the highest level possible for as long as we can keep it there. Eliminating parasitic overpopulation in the lids and lashes decreases inflammation and provides a healthier environment for the ocular surface to function at its best.**ODT**

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New technology may lead to earlier AMD Diagnosis

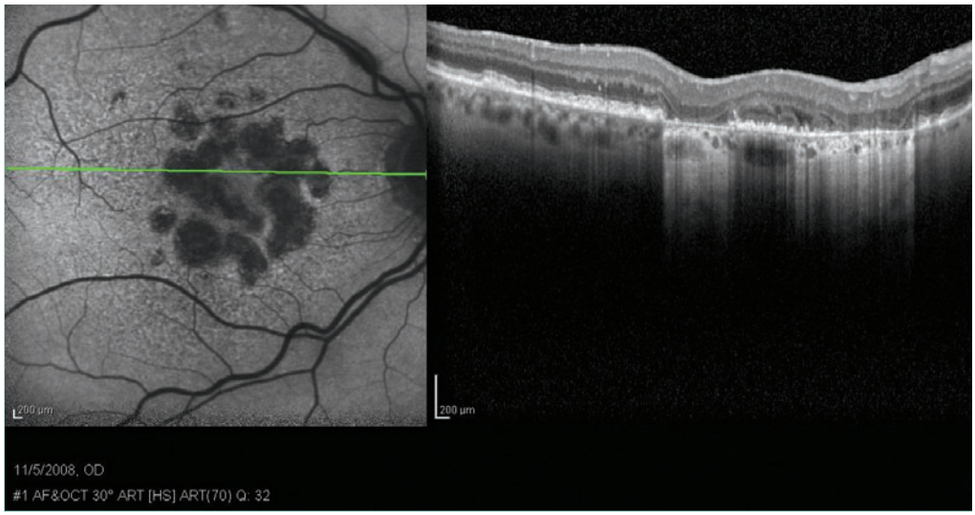
These devices may aid in early detection

By Steven Ferrucci, OD, FAAO

Great advances have been made over the last decade in the treatment of age-related macular degeneration (AMD) since the advent of Macugen (pegaptanib, Eyetech, Pfizer) in 2004. As most clinicians are aware, the current mainstay of treatment for wet or neovascular AMD is serial injections of anti-vascular endothelial growth factor (VEGF) agents, most notably Lucentis (ranibizumab, Genentech), Avastin (bevacizumab, Genentech), and Eylea (aflibercept, Regeneron), the newest FDA-approved agent.

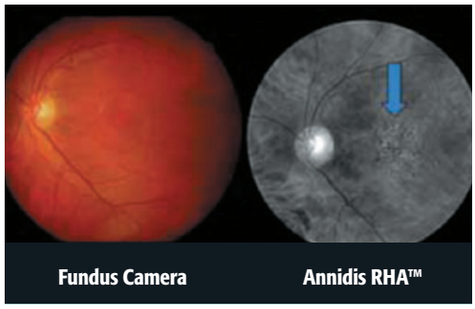
Take-Home Message

While there have been advances in the treatment of AMD, the methods to detect and diagnose AMD have remained the same, with the exception of OCT. But new technology—including the Adapt Dx, multi-spectral imaging, and fundus autofluorescence—may allow for earlier detection.



Fundus autofluorescence (FAF) of a patient with geographic atrophy, OD. (Photo courtesy Heidelberg Engineering)

The Adapt Dx screening test is non-invasive and can be completed in 5 m inutes. (Photo courtesy of Maculogix)



RPE atrophy is easily revealed by MSI in early AMD. (Photo courtesy of Annidis)

However, despite advances in treatment of AMD, the diagnosis methods to detect and diagnose AMD have remained relatively the same over the same period, with the obvious exception of OCT. Fortunately, new technology may soon allow us to detect AMD sooner than our current screening methods.

Dark adaptation

The Adapt Dx (Maculogix) is based on the functional test of dark adaptation—that is, the transition from being light-adapted to being dark adapted. Specifically, it measures the rate of recovery of scotopic sensitivity after photobleaching. Several studies at University of Alabama at Birmingham (UAB) have determined that dark adaptation is impaired with AMD.[1] Further, there is a marked deterioration of dark adaptation speed as the disease progresses.[2]

According to this same study, dark adaptation is impaired up to 4 years before AMD is clinically relevant, with mean recovery time of AMD patients twice that of age matched normal adults. In an unpublished study conducted at Penn State University, the instrument was shown to have excellent sensitivity and specificity, 88% and 100% respectively. A second study at Penn State University revealed even better sensitivity of 91%.

The instrument itself is approximately the same size of a Humphrey Visual Filed Analyzer. Patients are exposed to a brief camera flash, and then instructed to respond when they detect a progressively dimmer spot of light presented near the macula. The screening test in non-invasive and can be completed within 5 minutes or less by a technician. If results are found to be abnormal, a more extensive threshold test may be performed. There is currently an established CPT code for dark adaptometry (92284) that has a reimbursement between $60 and $80.

However, it is important to note that the AdaptDx is cleared for sale only as a dark adaptometer by the FDA—not approved as a test for AMD. It is currently under investigation as a diagnostic test for AMD, but as of this time is not cleared by the FDA.[3]

Multi-spectral imaging

Multi-spectral imaging (MSI) is an emerging technology that uses various wavelengths in order to better evaluate the retina and choroid for changes. Essentially, multiple monochromatic wavelengths from 550nm to 780 nm are used to visualize the retina in spectral slices, all the way from the internal limiting membrane (ILM) to the choroid. In general, the shorter wavelengths are used to visualize the more anterior surfaces,

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AMD

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while the longer wavelengths are used to reflect the deeper structures, including the choroid. It is thought that by examining the choroid, RPE, and deeper tissues, the clinician may be able to visualize changes in the retinal pigment epithelium (RPE(representing early AMD prior to it becoming clinically apparent.[4] This earlier diagnoses may prompt the clinician to counsel patients earlier or more aggressively regarding diet, vitamin supplements, or other modifiable risk factors for AMD.

One company's system, the Annidis RHA, also adds additional features to its commercially available unit. It has additional imaging that can be used to highlight oxygenated and deoxygenated hemoglobin, which may relate to the metabolic activity of the retina. The MSI technology is also available under the name Multi Color as a cSLO through Heidelberg.

At a glance

Dark adaptation

- Dark adaptation is impaired up to 4 years before AMD is clinically relevant
- Non-AMD patients adampt, on average, twice as fast as AMD patients
- Adapt Dx shows 91% sensitivity, according to an unpublished Penn State University study

Multi-spectral imaging

- Multiple monochromatic wavelengths from 550nm to 780 nm visualize the retina in spectral slices
- Images show the internal limiting membrane (ILM) to the choroid
- Examining deeper tissues may show retinal pigment epithelium (RPE) changes prior to AMD becoming clinically apparent
- Annidis RHA shows deoxygenated hemoglobin, which may relate to the metabolic activity of the retina

Fundus autofluorescence (FAF)

- This non-invasive technique uses properties of lipofuscin to analyze retinal heath
- FAF documents geographic atrophy (GA) development and progression
- Hyperfluorescent borders surrounding a GA lesion may be the best predictor for advancement

However, despite advances in treatment of AMD, the diagnosis methods to detect and diagnose AMD have remained relatively the same over the same period, with the obvious exception of OCT. Fortunately, new technology may soon allow us to detect AMD sooner than our current screening methods.

Fundus autofluorescence

Fundus autofluorescence (FAF) is a non-invasive technique which uses the properties of lipofuscin to help analyze the heath of the retina. Lipsofuscin is a byproduct of damaged outer segment photoreceptors that accumulates in the RPE with age and with certain retinal disease. Hence, it serves a s a precursor to retinal damage and eventually death.[5] When exposed to short to medium wavelength visual light, liposfuscin autofluoresce.

The images obtained by fundus autofluorescence appear somewhat similar to images associated with more traditional flourescein angiography but without needing to inject any dye. Increased areas of lipofuscin appear as bright, hyperfuorescent areas, whereas the absence of lipofuscin appears dark, or hyofluorescent.

Fundus autofluorescence imaging can be obtained in two ways: either with confocal scanning laser ophthalmoscope (cSLO) or by filter based cameras. Instruments such as the Daytona by Optos, or under the trade name Blue Peak through Heidelberg, capture retinal FAF using a low-energy laser that excites the lipofuscin. Filter based cameras such at the Canon CX-1 uses a excite filter with high energy white flash to get the same results.

Currently, FAF is probably most useful in AMD for documenting the development and progression of geographic atrophy (GA).[6]The exact borders of the GA can be much more readily identified than by clinical appearance or standard color photography, perhaps giving a more accurate assessment of the true extent of the atrophy. Further, some studies have indicated that hyperfluorescent borders surrounding a GA lesion may be the best predictor for advancement.[6]

Early diagnosis of AMD will be paramount as the population continues to age. Earlier disease detection may mean earlier intervention, such as alteration of risk factors such as smoking, poor diet, and obesity. It may also mean starting patients sooner on vitamin therapy in hopes of preventing the decline to advanced AMD. It may also mean more frequent monitoring for patients found to have AMD by these new diagnostic measures, in hopes of detecting advancing AMD or worse yet, conversion to wet AMD. Ultimately, these new techniques and equipment will hopefully help our patients maintain better vision and a better quality of life as they age.**ODT**

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Identifying Sjögren's syndrome in the dry eye populace

Emerging data, tests, and treatments target a long-ignored autoimmune disease.

By Kelly Nichols, OD, MPH, PhD, FAAO, DiplPH

Often, patients who present to their optometrist's office with Sjögren's syndrome are diagnosed with late-stage dry eye disease. Most optometrists expect that those who have Sjögren's syndrome have already been diagnosed and are at the end of the spectrum in the disease stage. Until now, there has been little effort for early detection and diagnosis of Sjögren's syndrome, but recent clinical data, treatment approaches, and a new panel test incorporating proprietary biomarkers designed for early detection of Sjögren's syndrome place optometrists in a meaningful position to identify Sjögren's syndrome earlier and initiate management.

About Sjögren's syndrome

Sjögren's syndrome is a systemic autoimmune disease affecting lacrimal and salivary glands characterized by dry eye and dry mouth. It is also systemically associated with other autoimmune conditions, such as lupus and rheumatoid arthritis, and common conditions, such as fatigue and memory loss. Multiple symptoms often result in a patient presenting to several different specialists for each symptom without receiving an explanation. Because of the non-specific nature of its systemic parts, Sjögren's syndrome is often overlooked. The mean time of diagnosis from clinical presentation to diagnosis is 4.7 years, yet Sjögren's syndrome is one of the most common autoimmune diseases.[1]

Dry eye is a common presentation in up to 93% of Sjögren's patients, and it is one of the most common reasons patients present to an eyecare practitioner.[2] Dry eye disease is a localized autoimmune disease that attacks the lacrimal and salivary glands, rendering them incapable of producing enough tears to lubricate the eyes. The same lymphocytic infiltration of the lacrimal gland occurs in late-stage Sjögren's syndrome. The earlier stages of Sjögren's syndrome appear as routine dry eye because there is an evaporative component and an aqueous deficient component, but the aqueous deficient component is not prominent enough to be classified as Sjögren's syndrome using traditional modes of screening. As a result, approximately 3 million patients with Sjögren's remain undiagnosed because they are not exhibiting the very severe characteristics that are expected in an advanced Sjögren's patient.

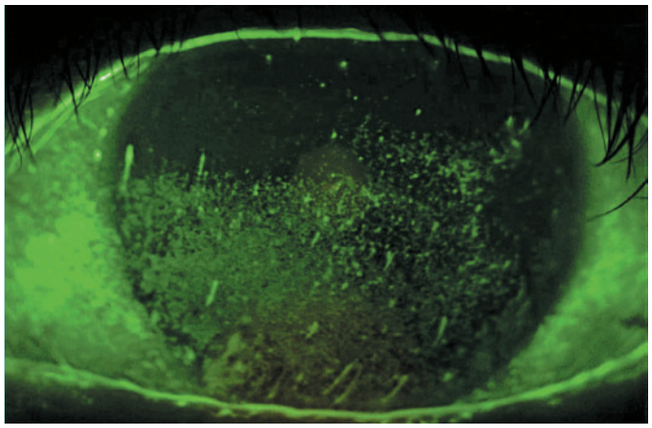

Figure 1. A a severe Sjögren's patient with filamentary keratitis. (Photo courtesy of Kelly Nichols, OD, MPH, PhD, FAAO, DiplPH)

New approaches for diagnosing Sjögren's syndrome

The criteria for diagnosing Sjögren's syndrome have changed 12 times since 1965. Current guidelines for diagnosing Sjögren's syndrome include fluorescein staining of the cornea, Schirmer's test, phenol red thread test, and family history because Sjögren's patients often have a family history of other autoimmune conditions. Although these tests have a place in dry eye evaluation, there is a need for a more consistent and comprehensive protocol for identifying Sjögren's patients.

> Multiple symptoms often result in a patient presenting to several different specialists for each symptom without receiving an explanation.

A recent classification in 2012 by the American College of Rheumatology suggested using symptom biomarkers—Sjögren-specific antibody A (SS-A or Ro), Sjögren-specific antibody B (SS-B or La), rheumatoid factor (RF), and antinuclear antibody (ANA)—along with a salivary gland biopsy and ocular stain. SS-A and SS-B are commonly performed in Sjögren's patients, but 20- 30% of those with Sjögren's Syndrome never test positive for the biomarkers, even those who have been diagnosed with Sjögren's with a salivary gland biopsy. ANA and RF are often performed when a clinician is diagnosing an autoimmune or inflammatory condition, but both are nonspecific to Sjögren's syndrome, so although they provide some additional information, they do not confirm a diagnosis of Sjögren's syndrome. Even when SS-A, SS-B, ANA, and RF are performed together, they produce a specificity of only 40-60%.[3] None of these tests confirm or rule out a Sjögren's diagnosis and are unlikely to detect the disease until an advanced stage.

A new panel identifies traditional biomarkers as well as three novel antibodies in Sjögren's syndrome—salivary gland protein-1 (SP-1), carbonic anhydrase-6 (CA-6), and parotid secretory protein (PSP)—that have been shown in research studies to diagnose Sjögren's earlier. Sensitivity and specificity is in the 90th percentile, and approximately 50% of early and new cases are identified.[4]

New approaches for treating Sjögren's syndrome

The Sjögren's Syndrome Foundation is creating a treatment algorithm for the management of patients with Sjögren's syndrome that are diagnosed across all stages of severity. This is a joint approach for eyecare professionals, dental care professionals, and rheumatolo-

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Sjögren's

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gists to promote collective management of Sjögren's patients, so that patients are managed from all subspecialties treating the most common areas affected—eyes, mouth, and joints. Establishing a referral network for rheumatologists requires improved communication between rheumatologists and optometrists. These two medical communities are well suited to have excellent relations with patient management.

Conclusion

Diagnosing Sjögren's syndrome earlier provides opportunity for the patient to receive systemic treatments that reduce inflammation and overall fatigue, allowing the body to function normally for a longer period of time. This creates an opportunity for optometrists to be more aggressive in initiating monitoring to stave off long-term effects. Furthermore, identifying Sjögren's patients earlier advances the role of the optometrist as a front-line medical practitioner and positions the optometrists as a medical manager. A laboratory test for Sjögren's syndrome empowers the clinician to make a diagnosis of a condition that is life-long and can be threatening. Whether the test result is positive or negative, optometrists should consider the best way in which to counsel patients. There should be a certain amount of care and preparation in the communication that occurs after the test is performed. It will require the thoughtfulness that is characteristic of optometrists.**ODT**

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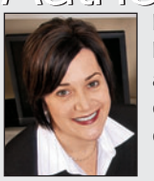

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Emerging therapies for Sjögren's syndrome make way for collaboration

There are opportunities for partnership with rheumatologists

By Julian L. Ambrus, Jr, MD

Scientists are researching whether blocking certain types of mediators at the earlier stages of Sjögren's syndrome may lead to halting the disease process or preventing downstream complications from occurring. There are specific therapies undergoing clinical research for treating Sjögren's, such as B-cell, T-cell, and cytokine targeted therapy.

A roadblock for these studies is that they are being performed on patients who meet American College of Rheumatology (ACR) criteria based on having symptoms—biomarkers SS-A (Ro), SS-B (La), ANA, RF; lymphocytes in their lip biopsies; and clinical manifestations of their disease. As a result, patients in clinical trials are in the late stages of Sjögren's syndrome, so their salivary and lachrymal glands have already failed. For example, Rituxan (rituximab, Genentech), a B-cell targeted therapy, underwent US clinical trials that concluded that it was ineffective. In France, clinical trials were done on the same therapy with positive results, perhaps because study participants were in the earlier stages of disease. A similar result appeared with Orencia (abatacept, Bristol-Myers Squibb), a T-cell targeted therapy, in the Netherlands. Clinicians found that ACR criteria patients with late-stage disease did not respond at all, but those with earlier disease responded well.

In my experience, for some rheumatoid arthritis patients developing early Sjögren's syndrome, the Sjögren's seems to fade when we prescribe etanercept (Enbrel, Amgen) for arthritis. My lab has performed research on animal models that have revealed that blocking lymphotoxin prevents progression of Sjögren's. When using a lymphotoxin antibody, the disease is prevented from manifesting. There is optimism for clinicians as we learn that early Sjögren's patients have a different pathophysiology than the accepted paradigms; therefore, it becomes critical to diagnose patients earlier. When patients are diagnosed earlier, our approaches to therapy, some of which have failed in the past, may turn out to be successful because they are being initiated when the glands are still amenable to being fixed as opposed to when they are already dead and scarred.

When a patient with Sjögren's syndrome is referred to my practice, my colleagues and I first look for hypergammaglobulinemia by doing a serum protein electrophoresis. We then image the lungs to rule out lymphocytic pneumonia. We examine the kidneys with urinalysis, blood studies, and ultrasound, if necessary. We determine our next steps depending on the findings of these analyses. If the patient has interstitial lung disease, we prescribe cyclophosphamide (Cytoxan, Bristol-Myers Squibb); if the patient is positive for renal tubular acidosis, we prescribe bicarbonate and potassium; and if the patient is confirmed to have glomerular disease in the kidney, we may prescribe a cytotoxic drug.

There is immense value in establishing a referral network with eyecare professionals who are the front-line practitioners in identifying patients with Sjögren's. Patients are examined from a systemic point of view, and we can set up populations earlier in order to study which medications are useful. Collaboration allows us to examine patients from various aspects to ensure they receive appropriate care.

In a recent study conducted in my lab, eyecare professionals referred 28 patients who had unexplained dry eyes. Of those patients, 50% had antibodies to the new SP1 autoantibody, and 30% had antibodies for SSA, SSB, ANA, RF. Only 5 out of 28 patients were identified to have dry eye based on Schirmer's test and who did not have these autoantibodies. The ideal management of Sjögren's patients involves a specialized approach where optometrists, ophthalmologists, rheumatologists, and immunologists can collaborate to determine how to best treat patients.**ODT**

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Apple induces motion sickness with iOS 7

For some users, the OS upgrade is a bad apple.

By Whitney Hauser, OD

As of May 2013, 56% of American adults have a smartphone. That represents over 170 million people. Of those cell phone owners, and 29% describe their cell phone as "something they can't imagine living without." Smartphone ownership has increased by 20% over the last 2 years.[1]

Pervasive use of smart devices doesn't present without challenges. They are great for checking the weather and connecting with friends on social media. Yet, they have also been blamed for increased anxiety, acne, back and neck pain, sleep problems, car accidents, and even brain cancer.[2]

Enter iOS 7. With much fanfare, Apple released its latest software upgrade in September. Apple claimed that iOS7 makes the things you do every day even easier, faster, and more enjoyable.[3] Shortly after many iPhone users downloaded the new platform, they did not report a more enjoyable experience. In fact, they reported dizziness, headaches, and nausea.[4]

Take-Home Message

Apple's latest mobile operating system iOS 7 has induced motion sickness in some users. The software uses layered parallax to organize data on a series of planes that move relative to one another to cause the effect. Mention to your iPhone-using patients that the effect can be disabled in preference settings.

iSickness

So, how can a smart phone make someone sick? Motion sickness. Motion sickness is caused by a conflict between what is visualized vs. what is perceived by the vestibular system, a complex group of nerves and fluid-filled membranes that provide the individual with a sense of gravity and motion.[5] Motion sickness can present in three distinct forms. Normal motion sickness is caused when motion is felt but not visualized (commonly, car-sickness or sea-sickness). Simulation sickness, or visually induced motion sickness (VIMS), is perceived when motion is seen but not felt (commonly with high-speed video games or virtual reality). The final type of motion sickness is a combined form.[6,7]

iOS 7 causes motion sickness in some users.
(Photos courtesy of Apple)

The dispute between the two systems causes symptoms of dizziness, fatigue, nausea, and in some cases vomiting. The area postrema (AP) located on the medulla oblongata at the caudal end of the fourth ventricle has been identified as a chemoreceptor trigger for vomiting.[8] The AP is triggered because the brain believes that it is hallucinating during the conflict between the visual world and the motion felt. Vomiting developed through evolution as a form of self-preservation. For example, someone eats poisonous berries, and hallucinates. The body vomits to eliminate the offending agent.[5] The brain cannot distinguish between poisonous berries and simulation sickness induced by iOS 7 software.

Apple whiz kids make the brain hallucinate by using the parallax effect, the observable position of an object at any given distance dependent on the viewer's viewpoint. The visual system employs parallax to quickly assess distances between objects in space, providing depth cues.[9] Parallax scrolling was used in animation as early as the 1940s to simulate motion against a background.[10] Apple upped the ante with its iOS 7 software upgrade by incorporating layers of parallax that respond to the iPhone's internal micro-electro-mechanical (MEM) gyroscope. The gyroscope measures angular velocity. Originally used in navigation of ships and aircraft, smart devices exploit the modern nanotechnology gyroscopes to orient the phone or tablet in space. The internal gyroscope can perceive rotation along three axes. The gyroscope works in concert with an accelerometer to determine the device's position in space. The software then uses layered parallax to organize data on a series of planes that move relative to one another, providing the snazzy 3-D motion effect.[9]

The perceived motion is an amusing innovation for some users, but others find it to be troublesome at best and nauseating at worst. The good news is that there's an easy fix. To disable the parallax effect, go to "Settings" and choose "General," followed by "Accessibility." Scroll down to "Reduce Motion" and toggle from OFF to ON. The parallax effect will be minimized but not eliminated.[11] This adjustment will likely provide more enjoyable iPhone for the motion sickness sufferer.**ODT**

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Radio

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he says he learned how to handle chatty patients, act as referee between couples or parents and their children, and encourage shy patients to respond to his questions. Musicians, he says, are no different than his patients, so he applied the same skills during live shows.

Still, not every radio interview went as planned. He points to an independent Canadian band composed of identical twin sisters. Dr. Twelker planned on asking them about their relationship because they had earned a reputation of arguing with each other while on tour.

"They got into an argument in the middle of the radio interview about the origins of a song they wrote," he recalls. "It was a friendly argument, but for a period of time, I completely lost control of the interview."

By far, his worst interview was with a 19-year-old singer-songwriter. Before going on the air, he reminded her that the interview would be live. "We don't have a bleep button."

But some habits are hard to break.

When Dr. Twelker asked the young musician about her musical influences, she mentioned Stevie Nicks from Fleetwood Mac , then added, "But I like just about anything that doesn't sound like s---."

The musician realized her mistake by the expression on Dr. Twelker's face, but by then it was too late. Fortunately, Dr. Twelker had learned how to handle such situations. Simply ignore it, keep talking, and never—ever—apologize.

The interview continued. Then the young woman played a song and "dropped the F bomb," he says, adding that when she finished singing, he ended the interview and played a recorded track.

By far, Dr. Twelker's worst interview was with a 19-year-old singer-songwriter. Before going on the air, he reminded her that the interview would be live. 'We don't have a bleep button.' But some habits are hard to break. The musician realized her mistake by the expression on Dr. Twelker's face, but by then it was too late.

"I thought she was going to faint," he says, after realizing she repeated the same mistake. "She kept fanning herself and asked for a cold towel to put on her forehead."

So many bands, so little time

As Dr. Twelker's fan base grew, so did his reputation for introducing local talent. Every week, his inbox at the radio station was overstuffed with invitations to CD or album release parties. He attended three to four each week, acting as KXCI's face in the community. It didn't take long before he was burned out.

"I reached a point in my life where I didn't have the energy to do it at that level anymore," he says. He has since trained his replacement and fills in as host when needed.

Looking back, he says the radio shows were challenging, exciting, and sometimes intimidating.

"Your interviews are being broadcast to hundreds or thousands of people," he says. "Still, if you love music, there's no better way to promote artistic expression in your community."**ODT**

Radio shows are challenging, exciting, and sometimes intimidating, says Dr. Twelker.

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DISPENSARY

Eye doctor hosts **community radio programs** to **promote musicians**

Dr. Dan introduces community to new talent

By Carol Patton

Many people in Tucson, AZ, recognize Dan Twelker, OD, PhD, not by his face, but by his voice.

Dr. Twelker is a local celebrity of sorts. For about 6 years, he hosted two radio programs on KXCI, a community radio station, featuring interviews with up-and-coming musicians and live performances.

After moving to Tucson in 2002, he discovered a thriving music scene, much to his surprise. Although he's not a professional musician, Dr. Twelker enjoyed playing the guitar and writing and singing folk songs.

"I decided to become a part of that scene," he says. "I started playing my guitar for more than 2 hours a day, created 12 songs, played and recorded them and put them on an album called *Recycled Love*.

While his debut CD hasn't quite made Billboard's top 10, he says success comes in many forms. "Just creating and recording the songs, that's success right there," he says, adding that so far, he's distributed a few hundred CDs.

Meanwhile, a friend told him about KXCI, which invited local volunteers to host 2-hour weekly shows that focused on a wide variety of music genres, such as blues, rock, and jazz, and referred him to a 3-week DJ course. Dr. Twelker enrolled, studied the broadcast rules and regulations set by the Federal Communications Commission (FCC), and received helpful tips for interviewing guests, producing an entertaining program, speaking clearly into a microphone, and operating the mixing board, CD players, turntables, and other equipment.

Dan Twelker, OD, PhD, hosted programs on a community radio station, featuring interviews with up-and-coming musicians and live performances.

> Dr. Twelker's first program aired every Tuesday evening for roughly 3 years. He called himself Dr. Dan, selecting the same name his patients used.

Live from Tucson

In 2005, Dr. Twelker's first program—"The Road Show"—aired every Tuesday evening for roughly 3 years. He called himself Dr. Dan, selecting the same name his patients used. By 2008, he was ready for a change and began hosting, "Locals Only," a 2-hour show on Monday evenings that promoted local musicians.

He says his communication skills as an optometrist helped him conduct radio interviews. As an eye doctor,

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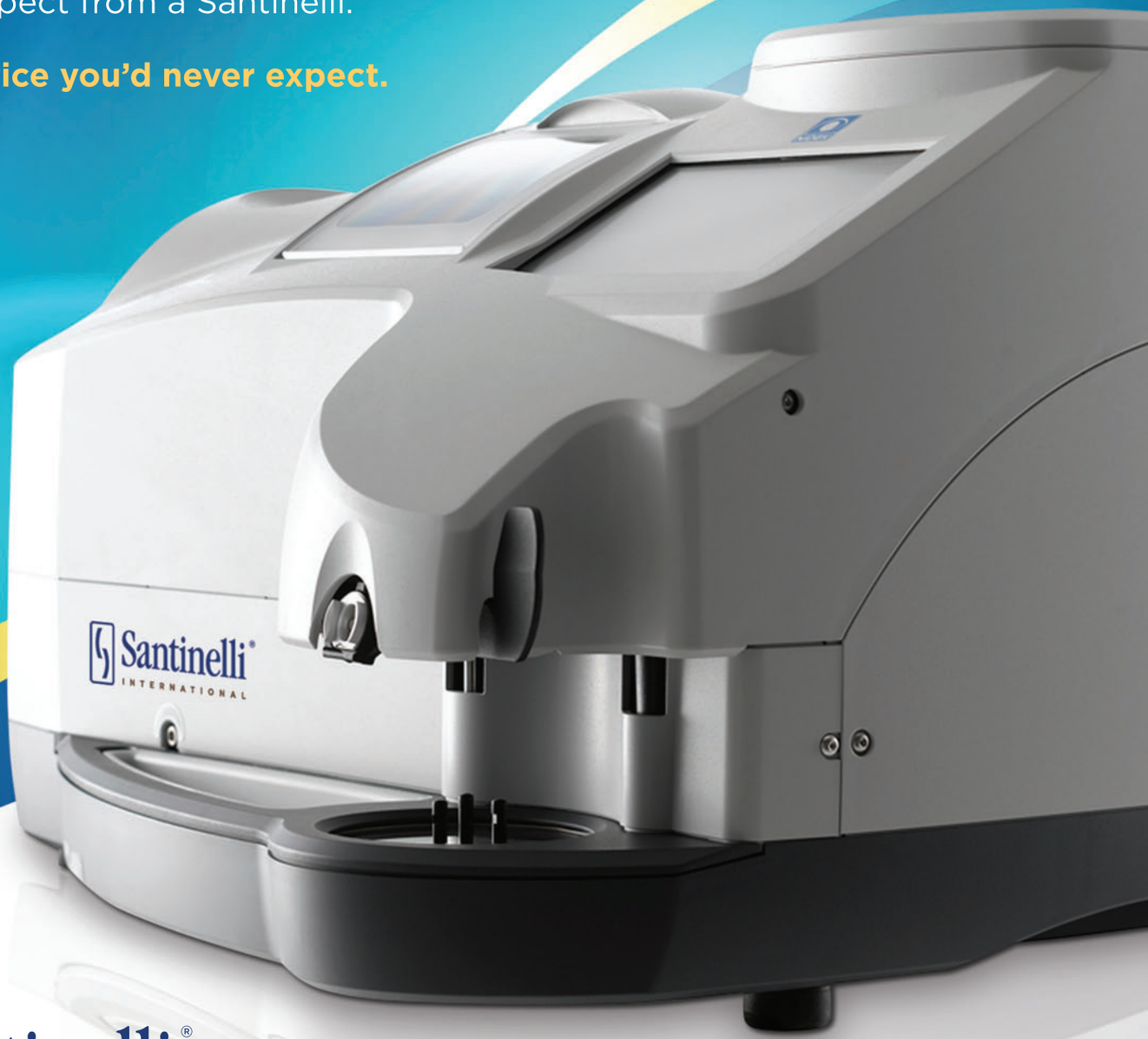

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