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July 15, 2012

REVIEW® OF OPTOMETRY

Part 1 of 2

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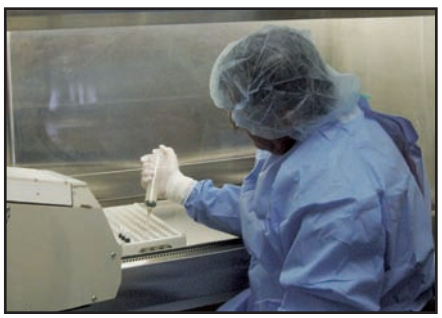

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JOSHUA MARC LAHIFF, OD

A Growing Practice Is Built on Happy Patients

Garnering new patients through **word-of-mouth referrals** is more than a great marketing strategy—it is the key to success that endures the test of time.

Successful practices see more patients

Focusing your practice on getting new patients rather than dollars per patient is a more pragmatic—and preferable—business approach. This is true for any practice. In a 2009 study of independent practices, it was reported that the practices with the greatest gross revenue see three times as many patients as the average practice. Furthermore, there is little difference in the gross revenue per exam between rural and urban settings, small and large practices.[1]

Increased patient traffic and improved exam productivity have major impacts on practice revenue. In fact, another 2009 study found that, on average, 52% of a practice's gross revenues come from exam fees alone.[2] This constitutes nearly 2/3 of gross income.[1,2]

According to Dr Josh LaHiff, practicing optometrist in Cheyenne, WY, increasing patient traffic is all about providing patients with an outstanding experience: "You want to be able to provide them [patients] with such an experience that they're going to invest more in your clinic." Dr LaHiff should know. He sees at least 30 patients per day. Which is impressive considering there are 28 other eye care professionals in Cheyenne, a town with a population of only 50,000.

Success is built on a happy patient experience

Increasing patient traffic through referrals is often as simple as delivering excellent service and the healthiest products. "To be the best, you have to use the best. If you wow the patient, treat them like gold, that's how you really generate those referrals," says Dr LaHiff.

That's why Dr LaHiff believes the most effective approach to achieving profitable, long-term patient relationships is to "do what's best for the patient, even if it may not be the most profitable option for the doctor initially, because it is what is right, and the profit will come as an annuity in return visits and the happiness of the patient."

High patient satisfaction inside and outside of the practice is the most effective catalyst for generating new patients. This can be especially true for contact lens patients. A survey of 1086 patients found that those who are happy in their contact lenses are nearly 2x more likely to recommend their eye doctor than those who are unhappy in their lenses.[2]

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The proven method to achieve satisfaction is to use a product with consistently successful results. Dr LaHiff believes that, "When you use a product that you know is going to work time and time again, it cuts your chair time down and it's easier for you, your staff, and your patients to put them in something that's comfortable."

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And happy ACUVUE® wearers refer their eye doctor: 8 out of 10 satisfied ACUVUE® OASYS® Brand Contact Lens and 9 out of 10 1-DAY ACUVUE® MOIST® Brand Contact Lens wearers would recommend their doctor to others.[2]

According to Dr LaHiff, "The more you use a superior product, the better the experience is going to be for the patient." And that's been a proven strategy for the success of his practice. ■

Joshua Marc LaHiff, OD, is a partner and practicing optometrist at Cheyenne Vision Clinic in Cheyenne, WY, and clinical instructor for the Illinois College of Optometry in Chicago, IL. He received his doctor of optometry degree with honors from the Pacific University College of Optometry in Forest Grove, OR.

Dr LaHiff is a member of numerous associations and serves as a speaker and professional consultant for several medical companies, including VISTAKON® Division of Johnson & Johnson Vision Care, Inc. He was compensated for this article.

References: 1. Management & Business Academy: Practice Profile Report, 2009. **2.** Data on file. Johnson & Johnson Vision Care, Inc. 2011-2012.

What keeps your practice growing? Referrals. From happy patients.

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*Based on percentage of satisfied contact lens patients who said they would recommend their eye doctor to others.

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VOL. 149 NO. 7 ■ JULY 15, 2012

IN THE NEWS

Older adults who take **statins** for hyperlipidemia have a significantly **lower risk** (9%) of **open-angle glaucoma**, according to a retrospective study published in the June 21 online version of *Ophthalmology*. Other reports found similar results, so the authors of this article are calling for an interventional prospective study to look further into statins' ability to prevent early glaucoma.

New hope for patients with **multiple sclerosis (MS)**: A clinical Phase III trial of oral **teriflunomide** 14mg (Genzyme/Sanofi-Aventis) **reduced relapses** by 36.3% (vs. placebo) in more than 1,000 study participants with relapsing-remitting MS, the company reported. The drug also slowed progression of disability by 31.5%. The Food and Drug Administration accepted an application for marketing approval of teriflunomide for review in October 2011.

Ronald L. Hopping, O.D., M.P.H., was installed as **President of the American Optometric Association** during Optometry's Meeting, recently held in Chicago. His installation marks the first time in the century-plus history of the AOA that a **father and son** have both served as president of the organization. His father, **Richard L. Hopping, O.D., D.O.S., D.Sc.**, was AOA President in 1971.

According to **VSP Vision Care's** first ever "Eye Health City Index," residents in these major U.S. cities have their **eyes examined** the most frequently:

- Providence, RI
- Wichita, KS
- Raleigh, NC
- Dayton, OH
- Sacramento, CA
- Birmingham, AL
- Denver
- Columbus, OH
- San Jose, CA
- Oklahoma City

Eye Care Speaks Out on 'Obamacare'

After the Supreme Court voted five to four to uphold the constitutionality of the Patient Protection and Affordable Care Act, the eye-care community immediately voiced its responses. The big unknown for optometry is whether (or to what extent) the profession will be included in state health insurance exchanges.

Photo: iStock

The American Optometric Association stated that it "anticipates that federal and state-level agencies will now increase efforts to shape state-based health insurance exchanges and implement further provisions of the sweeping new law, including the AOA-backed Harkin Amendment, Stabenow Amendment and pediatric vision care essential benefit."

The Supreme Court decision is but one speed bump in this legislation's ongoing journey. The AOA also stated, "as key health reform decisions are made in the nation's capital and in statehouses across the country in the coming weeks and months, the AOA will continue working to advance pro-access, pro-patient solutions aimed at ensuring that doctors of optometry and their patients are treated fairly under health reform and that policymakers and others fully understand the central role that optometrists play in enhanced care delivery and improved health outcomes."

David W. Parke II, M.D., CEO of the American Academy of Ophthalmology, stated in part, "The Supreme Court's ruling that the health care law is constitutional is just one chapter in a book that is still being authored. The outcome of the November elections will be another important chapter."

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* Boston lenses are prescribed 3 times more often than the closest GP competitor. Source: Survey conducted by Decision Analyst, March 2011.

O.D.s Could Help Prevent Stroke

A measurement of ocular pulse amplitude (OPA) may help eye-care providers identify patients who are at an increased risk for stroke, according to a study in the June issue of *Ophthalmology*. The researchers suggest that OPA can reliably detect carotid artery stenosis (CAS), a condition that clogs or blocks the arteries that feed the frontal portion of the brain.

A measurement of OPA is calculated by determining the difference between the systolic and diastolic blood pressures within the eye. When ocular blood flow is slowed or partially obscured by CAS, there is little difference between the pressure levels—resulting in a low OPA score.

In this study, researchers used a dynamic contour tonometer to document the OPA of 67 patients with presumed CAS. At its conclusion, the researchers determined that patients with the lowest OPA scores exhibited the most acutely blocked arteries. They subsequently performed an ultrasound on each patient to corroborate the OPA testing results and determine the severity of the blockages.

"Our results show that ocular pulse amplitude is a reliable, safe screening test for carotid artery stenosis," said lead author Pascal B. Knecht, M.D., senior researcher at the University of Zurich Eye Clinic. "We recommend further study to confirm the value of using OPA to detect and assess the severity of CAS and to define its use in stroke prevention."

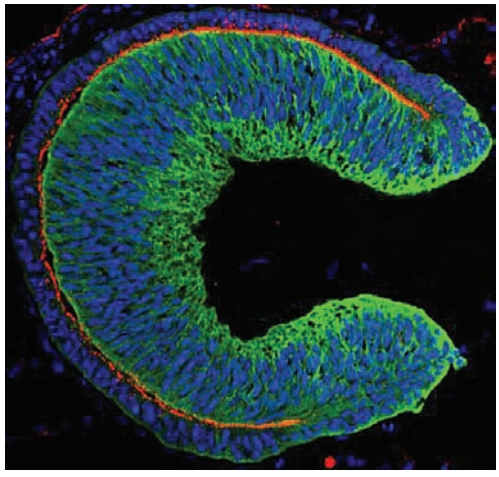
The researchers further suggested that, other than CAS, very few conditions could cause low OPA scores. Therefore, by taking a measurement of OPA, they believe that eye-care providers could efficiently determine which patients are at the greatest risk for stroke and easily rule out the presence of other diseases during a standard ocular examination.

Knecht PB, Menghini M, Bachmann LM, et al. The ocular pulse amplitude as a noninvasive parameter for carotid artery stenosis screening: a test accuracy study. Ophthalmology. 2012 Jun;119(6):1244-9.

Stem Cells May Allow Blind to See

Human embryonic stem cells (hESC) can be used to grow tissue in the form of a complete optic cup, according to a new study in the June 14 edition of *Cell Stem Cell*. In the future, transplantation of this 3D tissue could restore sight to visually impaired patients.

"Our approach opens a new avenue to the use of human stem cell-derived complex tissues for therapy, as well as for other medical studies related to pathogenesis and drug discovery," says senior study author Yoshiki Sasai of the RIKEN Center for Developmental Biology in Japan.



A human embryonic stem cell-derived optic cup generated in culture. Bright green is neural retina; off green is pigment epithelium; blue is nuclei; and red is active myosin.

The researchers optimized cell culture methods that enabled the hESC-derived cells to form the correct 3D shape and the two layers of the optic cup, including a layer of photoreceptors. Because retinal degeneration typically results from damage to these cells, this tissue could be ideal for transplantation in the future.

The same research group previously created an optic cup derived from mouse embryonic stem cells. But the hESC-derived optic cup seems to contain species-specific instructions for building an ocular structure. It's much larger in size and its multilayered tissue contains both rods and cones, which is rare in mouse ESC cultures.

Nakano T, Ando S, Takata N, et al. Self-formation of optic cups and storable stratified neural retina from human ESCs. Cell Stem Cell. 2012 June 14;10(6):771-85.

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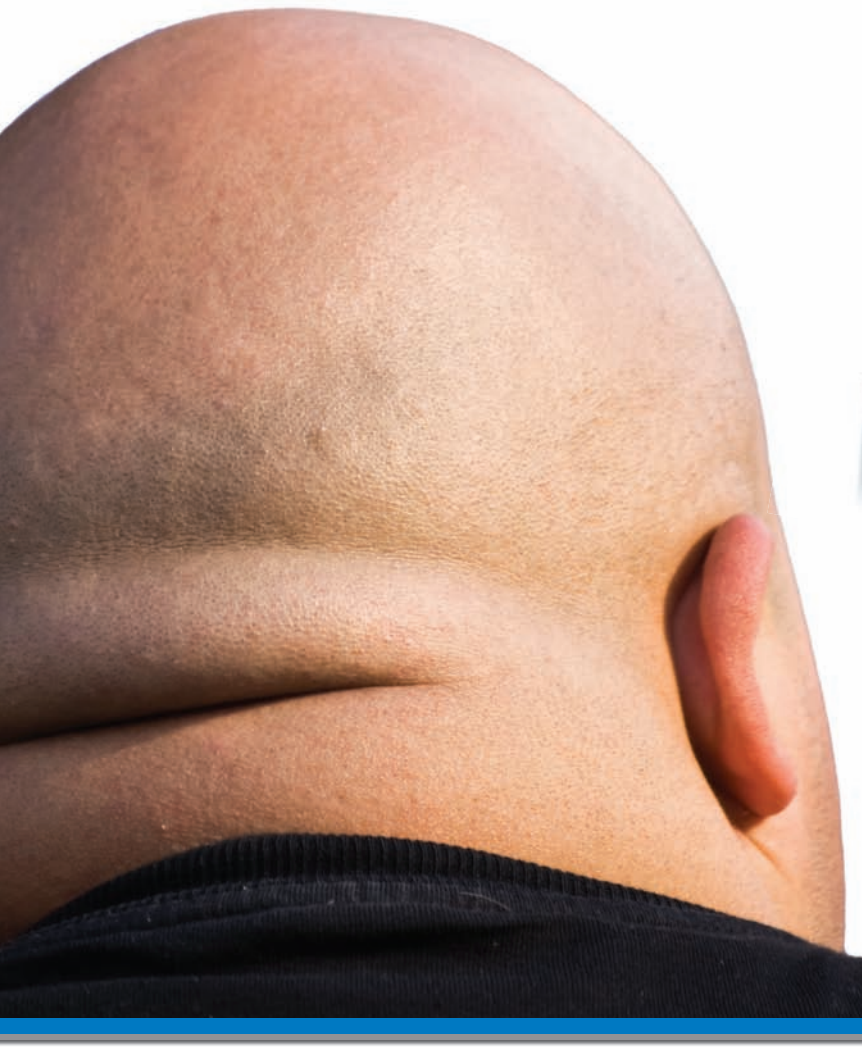

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Actos, Avandia Linked to DME

Type 2 diabetes patients who use Actos (pioglitazone, Takeda Pharmaceuticals) or Avandia (rosiglitazone, GlaxoSmithKline) to control blood glucose levels are more likely to develop diabetic macular edema (DME), according to a study in the June 11 online edition of *Archives of Internal Medicine*. In several previous studies, these drugs—broadly classified as thiazolidinediones or glitazones—have been associated with an increased risk of cardiovascular disease, bone fracture and bladder cancer.

In this retrospective study, the researchers evaluated 103,368 patients with type 2 diabetes and no trace of DME at baseline. Of this study population, 3,227 patients had a history of thiazolidinedione use.

At one-year follow-up, the researchers determined that 41 patients on thiazolidinedione therapy developed DME, compared to 227 patients from the remainder of the entire study cohort.

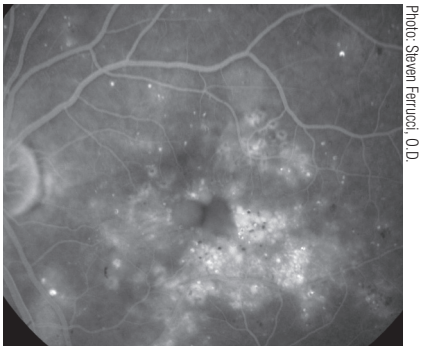

Photo: Steven Ferrucci, O.D.

The type 2 diabetes drugs Actos and Avandia may increase a patient's risk of diabetic macular edema, as seen here.

"Patients who received a thiazolidinedione were at a two- to three-fold increased risk of developing macular edema," says lead author Iskandar Idris, M.D., diabetes and endocrinology consultant at the Sherwood Forest Hospitals NHS Foundation Trust in Nottinghamshire, England. Because of this association, "More aggressive management of risk factors for macular edema should be implemented in patients who take a thiazolidinedione. In addition, routine screening for visual acuity should be performed during routine diabetes review, especially for patients who take thiazolidinediones," he adds.

The researchers concluded that larger, more detailed meta-analyses of randomized, controlled trials still would be necessary to clearly establish a risk/benefit profile of thiazolidinedione use in patients who either have or are likely to develop DME.

Idris I, Warren G, Donnelly R. Association between thiazolidinedione treatment and risk of macular edema among patients with type 2 diabetes. Arch Intern Med. 2012 Jun 11:1-7. doi: 10.1001/archinternmed.2012.1938. [Epub ahead of print]

Updates at Two Optometry Schools

• **Massachusetts College of Pharmacy and Health Sciences School of Optometry** held a ribbon-cutting ceremony on June 12 to celebrate the opening of a six-story building to house facilities for its new optometry school. The school's four-year optometry program is scheduled to begin in August, with 64 students in the inaugural class. The $10 million, 54,000-square-foot building will have an on-site optometry clinic with 24 examination rooms, each with $85,000 worth of the latest equipment, as well as laboratories, auditoriums, high-tech classrooms, offices, and meeting and study spaces. Also, the facility already houses 200 students.

• **Pennsylvania College of Optometry at Salus University** has postponed its proposed Scholars Program, an accelerated three-year degree program announced last year. "After careful consideration of the needs and expectations of the students, the University has decided to delay the launch of PCO's Scholars Program until June 2013," the college announced on its website. "[P]ortions of the Scholars Program curriculum will be piloted with eligible students who have chosen to join the Pennsylvania College of Optometry's Class of 2016."

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Important information for AIR OPTIX® AQUA (lotrafilcon B) contact lenses: For daily wear or extended wear up to 6 nights for near/far-sightedness. Risk of serious eye problems (i.e. corneal ulcer) is greater for extended wear. In rare cases, loss of vision may result. Side effects like discomfort, mild burning or stinging may occur.

References: 1. Based on a post-launch evaluation in which 88 eye care practitioners refit over 400 patients in AIR OPTIX® AQUA contact lenses. Alcon data on file, 2011. **2.** Nash W, Gabriel M, Mowrey-Mckee M. A comparison of various silicone hydrogel lenses; lipid and protein deposition as a result of daily wear. *Optom Vis Sci*. 2010;87:E-abstract 105110. **3.** Compared to HEMA contact lenses; based on the ratio of lens oxygen transmissibilities; Alcon data on file, 2010. **4.** Dumbleton K, Richter D, Woods C, et al. Compliance with contact lens replacement in Canada and the United States. *Optom Vis Sci*. 2010;87(2):131-139. **5.** Compared to 2-week replacement lenses; based on self-reported lens replacement time and third-party industry pricing information; Alcon data on file, 2012.

See product instructions for complete wear, care, and safety information.

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Eye-opening Stats in U.S. Vision Loss

The fight to prevent vision loss in America is still raging, as evident in the 2012 "Vision Problems in the U.S." report. The vision update was presented to a packed house at the Focus on Eye Health Summit held on June 20 in Washington. This year's audience included patient advocates, community-based organizations, national vision and eye health organizations, government agencies and policymakers. The Summit presented updated findings on the impact of adult eye disorders, visual impairment and blindness on the U.S. economy.

The number of people age 40 and older with vision impairment and blindness has increased 23% since 2000, according to the study conducted by researchers at Johns Hopkins University. "It's no surprise that the numbers of those affected by eye disease are continuing to climb, especially due to the aging baby boomer population," said Hugh R. Parry, president and chief executive officer of Prevent Blindness America. "What is exceptionally concerning is the dramatic spike in diabetic retinopathy cases, a consequence of the diabetes epidemic that this country is experiencing with no end in sight."

Diabetic retinopathy is the leading cause of new blindness in adults ages 20 to 74, and costs the United States about $500 million annually. So, it's no surprise that the Summit's keynote address focused on diabetes and the eye. Keynote speaker Ann Albright, Ph.D., R.D., director of the Division of Diabetes Translation at the Centers for Disease Control and Prevention, reminded the audience of how integral eye-care professionals are to the mission to eradicate diabetes. Although there is no cure for diabetic eye disease, annual eye exams for diabetes patients are essential to help slow disease progression.

As someone who has lived with type 1 diabetes for 44 years, diabetes prevention and treatment is a major part of Dr. Albright's personal and professional life. She noted that vision loss in diabetes patients was down 26% from 1997 to 2009. "We're doing some things right," Dr. Alright said. "Fewer people are having vision loss, except that the absolute number of people is so large that we're overtaking the gains that we're making in those improvements. We can't quit now. We've got a long ways to go."

Ann Albright, Ph.D., R.D., director of the CDC's Division of Diabetes Translation, gave the keynote address at this year's Focus on Eye Health Summit.

She said the approach must be two-pronged: prevention and control. Proactive measures could considerably reduce the staggering number of new cases of diabetes, while proper monitoring and control can minimize the number of complications.

Currently, one in 10 U.S. adults has diabetes, but if trends continue, one in three U.S. adults will have diabetes by 2050, she said. "We think we have an issue with diabetes now—it's going to get worse if things stay on the trajectory that we're on," Dr. Albright added. "And I think that's really the good news for all of us. It does not have to stay on the trajectory that it's on. In fact, it can't stay on the trajectory that it's on. This is unsustainable."

Eye Disease is on the Rise in America

Released by Prevent Blindness America and the National Eye Institute, the 2012 "Vision Problems in the U.S." report highlighted some alarming statistics about increases in the four most common eye diseases since 2000:

- 25% increase in late age-related macular degeneration (2,069,403 people age 50 and older)
- 19% increase in cataracts (24,409,978 million people age 40 and older)
- 22% increase in open-angle glaucoma (2,719,379 million people age 40 and older)
- 89% increase in diabetic retinopathy (7,685,237 million people ages 40 and older)

All data from the "Vision Problems in the U.S." report can now be obtained through a new searchable database housed on the Prevent Blindness America website located at www.visionproblemsus.org/index.html

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Don't Miss the Signs of Marfan Syndrome

Optometrists must become increasingly familiar with the early signs and symptoms of Marfan syndrome, according to a survey conducted by the National Marfan Foundation (NMF).

Marfan syndrome, a potentially fatal connective tissue disorder, may lead to aortic tear or rupture if not properly detected and surgically managed early on in a patient's lifetime.

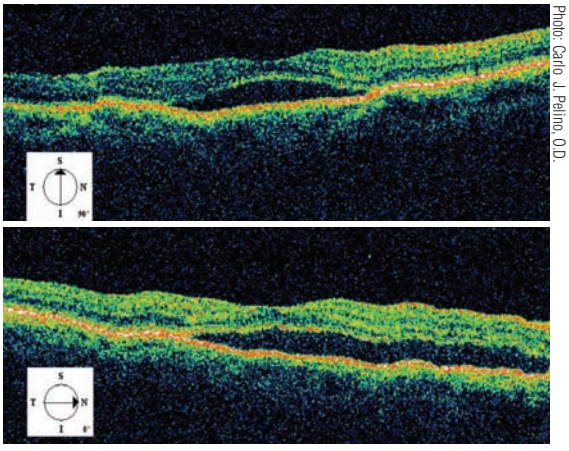

Photo: Carlo J. Pelino, O.D.

Individuals with Marfan syndrome often manifest secondary ocular complications, such as retinal detachment, as seen in this patient.

"Early diagnosis is critical so that patients can take medications to lower their heart rate and blood pressure, make lifestyle adaptations (no competitive or contact sports) and have their aorta monitored so they can have surgery before a potentially fatal tear or rupture," says Irene Maumenee, M.D., a member of NMF's professional advisory board and director of ophthalmic genetics at the University of Illinois Eye and Ear Infirmary.

To better understand the patterns of diagnosis and detection, the NMF surveyed 1,369 patients with Marfan syndrome. While 70% of respondents reported that at least one health care provider had positively identified their disease by age 20, just 4% of those polled said an optometrist was the first individual to suspect an underlying condition.

"The [associated] eye issues, which also include myopia, amblyopia, strabismus, glaucoma and retinal detachments, are often early signs of the condition and can certainly impact quality of life; however, they should also raise a red flag that something potentially more serious is going on," adds Dr. Maumenee. "Ophthalmologists and optometrists are key to early diagnosis because parents do not hesitate to bring their children in if there is a potential sight problem. I encourage eye doctors to be aware of the ocular signs of Marfan syndrome, as well as the other outward physical signs, so they can refer a patient for further evaluation, if needed." ■

Marfan Diagnostic Criteria

The NMF has developed www.marfandx.org to help doctors become more familiar with the latest diagnostic criteria for Marfan syndrome.

REVIEW OF OPTOMETRY

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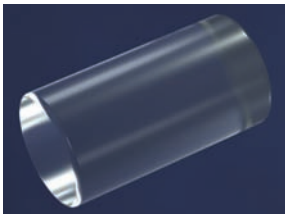

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† In most patients, one LACRISERT® placed into each eye once daily is effective in providing all-day symptom relief. Some patients may require twice-daily use for optimal results.

Please see Brief Summary of Prescribing Information on adjacent page.

Reference: 1. Koffler BH, McDonald M, Nelinson D, Improved signs and symptoms and quality of life with dry eye syndrome: hydroxypropyl cellulose ophthalmic insert patient registry. *Eye Contact Lens.* 2010;3:170-176.

Bridgewater, NJ 08807 LAC102-0512ROPT

Brief Summary of Prescribing Information

ATON PHARMA

ATON Pharma, a Division of Valeant Pharmaceuticals North America LLC
Madison, NJ 07940

Rx Only

LACRISERT® (hydroxypropyl cellulose) OPHTHALMIC INSERT

DESCRIPTION

LACRISERT® Ophthalmic Insert is a sterile, translucent, rod-shaped, water soluble, ophthalmic insert made of hydroxypropyl cellulose, for administration into the inferior cul-de-sac of the eye.

Each LACRISERT is 5 mg of hydroxypropyl cellulose. LACRISERT contains no preservatives or other ingredients. It is about 1.27 mm in diameter by about 3.5 mm long. LACRISERT is supplied in packages of 60 units, together with illustrated instructions and a special applicator for removing LACRISERT from the unit dose blister and inserting it into the eye.

INDICATIONS AND USAGE

LACRISERT is indicated in patients with moderate to severe dry eye syndromes, including keratoconjunctivitis sicca. LACRISERT is indicated especially in patients who remain symptomatic after an adequate trial of therapy with artificial tear solutions. LACRISERT is also indicated for patients with exposure keratitis, decreased corneal sensitivity, and recurrent corneal erosions.

CONTRAINDICATIONS

LACRISERT is contraindicated in patients who are hypersensitive to hydroxypropyl cellulose.

WARNINGS

Instructions for inserting and removing LACRISERT should be carefully followed.

PRECAUTIONS

General

If improperly placed, LACRISERT may result in corneal abrasion.

Information for Patients

Patients should be advised to follow the instructions for using LACRISERT which accompany the package.

Because this product may produce transient blurring of vision, patients should be instructed to exercise caution when operating hazardous machinery or driving a motor vehicle.

Drug Interactions

Application of hydroxypropyl cellulose ophthalmic inserts to the eyes of unanesthetized rabbits immediately prior to or two hours before instilling pilocarpine, proparacaine HCl (0.5%), or phenylephrine (5%) did not markedly alter the magnitude and/or duration of the miotic, local corneal anesthetic, or mydriatic activity, respectively, of these agents. Under various treatment schedules, the anti-inflammatory effect of ocularly instilled dexamethasone (0.1%) in unanesthetized rabbits with primary uveitis was not affected by the presence of hydroxypropyl cellulose inserts.

Carcinogenesis, Mutagenesis, Impairment of Fertility

Feeding of hydroxypropyl cellulose to rats at levels up to 5% of their diet produced no gross or histopathologic changes or other deleterious effects.

Pediatric Use

Safety and effectiveness in pediatric patients have not been established.

Geriatric Use

No overall differences in safety or effectiveness have been observed between elderly and younger patients.

ADVERSE REACTIONS

The following adverse reactions have been reported in patients treated with LACRISERT, but were in most instances mild and transient: transient blurring of vision, ocular discomfort or irritation, matting or stickiness of eyelashes, photophobia, hypersensitivity, edema of the eyelids, and hyperemia.

DOSAGE AND ADMINISTRATION

One LACRISERT ophthalmic insert in each eye once daily is usually sufficient to relieve the symptoms associated with moderate to severe dry eye syndromes. Individual patients may require more flexibility in the use of LACRISERT; some patients may require twice daily use for optimal results.

Clinical experience with LACRISERT indicates that in some patients several weeks may be required before satisfactory improvement of symptoms is achieved.

Issued June 2007

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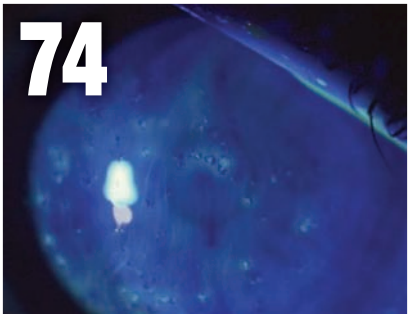

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Letters to the Editor

Yes, We Should Donate Old Glasses

The April 2012 issue of *Review of Optometry* had a disturbing news story, "Don't Donate Old Glasses, Study Says," which had many unsubstantiated statements. Reviewing the complete article ("Real Costs of Recycled Spectacles") in *Optometry and Vision Science*, it appears that *Review of Optometry* did not review the complete article and the references used to support it.[1]

One of the largest used eyeglass collection programs is part of the International Association of Lions Clubs. Its 1.35 million volunteer members in 207 countries supply free eyeglasses and eye examinations to those in need.

The sparse data included 106 pairs of eyeglasses from a small island community of Honiara, Solomon Islands, and a total of 169 pairs from optometry practices in Sydney and Canberra, Australia. The latter sample of 169 used eyeglasses appears very small for the size of the two major Australian metropolitan cities.

Statements like, "The economic imperative of valuing opportunity cost, i.e., recognizing that the volunteer labor could have been doing something else, is potentially more productive," appears arguably unfounded. Volunteers could be doing something else more productive—like working for wages or operating a hedge fund where the funds could be used to purchase new eyeglasses for those in need. However, volunteers gain psychological benefits by contributing something useful to those less fortunate who could use the glasses to support themselves by working, to feed themselves and their families, and to send their children to school.

In the United States, the present free volunteer labor force that has been active for almost 100 years doesn't appear to be in danger of subsiding. One of the largest used eyeglass collection programs is part of the International Association of Lions Clubs. Its 1.35 million volunteer members in 207 countries throughout the world (which was not included in the article) supply free used eyeglasses and eye examinations to those in need.

The authors state, "The use of recycled spectacles has development, economic, and social implications for the country or region involved. The supply of recycled spectacles does not help grow a sustainable industry in countries and communities being serviced." This statement is flawed. Free world trade allows anyone to grow a sustainable optical industry where the authorities allow them. The question is, can the communities it would service be capable of supporting them? I would argue that this not likely, because the population served would not be capable of purchasing eye examinations or Rx glasses, nor usually have enough population to support even one eye-care professional or optical.

Statements such as, "If supported by retail optical organizations, programs receiving and dispensing donated recycled spectacles also face a potential conflict of interest and therefore a possible breach of ethics as soliciting donated spectacles might be seen as an inappropriate way to generate more sales of new spectacles to donors. Furthermore, the visibly charitable work of any optical company adopting this approach may be likely to endear donors and therefore make them more likely to purchase spectacles from an organization, which is seen to be a good corporate citizen, when indeed the spectacles are largely unusable." This appears to be a paranoid statement, or a scare tactic not worthy to appear in a prestigious professional journal. They can make the unsuspecting reader believe that retail optical companies and organizations have alternative reasons to support the used eyeglasses collection program.

In conclusion, *Review of Optometry* and *Optometry and Vision Science* need to review the inaccurate statements and opinions before publishing such articles or statements. Many of them appear to be personal editorials not supported by facts or available information.

—Howard A. Levenson, O.D. (retired)
San Rafael, Calif.

David A. Wilson, Ph.D., lead author of the study, responds:

The aim of our research was to assess whether recycled spectacles were cost effective and to comment on the effects on developing sustainable capacity for eye care in areas of need. We believe that Dr. Levenson may have misunderstood or misinterpreted some of our points.

He argues that our claim that the volunteer labor could have been doing something else is unfounded. However, several organizations have used volunteer

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professionals to carry out eye tests and dispense quality brand new spectacles rather than sort through second-hand spectacles.

The supply of recycled spectacles does not help to grow a sustainable industry in countries and communities being serviced. Dr. Levenson disagrees, arguing that, "Free world trade allows anyone to grow a sustainable optical industry where the authorities allow them." While this may be technically true, few developing countries have the human resources and technical expertise needed to set up such an industry without assistance. Many organizations, understanding the importance of sustainable local refraction services, have set up functioning vision centers in several countries, supplying both ready-made and custom-made spectacles.

We believe that there is no basis for Dr. Levenson's assertion that the sample size is too small. Our sample was statistically determined to be appropriate given the expected nature of the problems and actually larger than that used by Jacqueline Ramke, M.P.H., and associates, which arrived at a similar result with respect to the low percentage of useful spectacles.[2] All spectacles were from Australian donors.

Our comment on the perception of a potential conflict of interest is also justified. We do not argue that there is any inappropriate behavior, but merely that there is a possibility of such perception. A large retailer in New Zealand was criticized by the NZAO (the optometrists' professional body) for advertising that encouraged the public to bring in their old spectacles. The NZAO saw this (whether rightly or wrongly) as a way of generating new sales for the retailer.[3]

We agree that those who donate enjoy the feeling of contributing; however, we are of the opinion that the well-being of people being served and their communities are of paramount importance. Charity makes people feel good; however, giving to a way of economically creating sustainable vision care services has a much more lasting effect.

Our major argument is that significant valuable resources are spent checking and sorting through recycled spectacles, as the vast majority of such spectacles are not useful. Whether 7% are usable (from our study) or 13% (from Ramke's study), there is still significant wastage. The argument still holds even if much less stringent criteria are used.

We have tried to adopt a rational and progressive approach to delivering vision care to those in need in a way that will endure. In this regard, we are aligned with the programs of the International Agency for Prevention of Blindness, with other similar studies and experiences, and with VOSH.[4-9] ■

—David Wilson, Ph.D., B.Ec., B.A.(Hons), Sonja Cronjé, M.Phil.(Optom), M.P.H., Kevin Frick, Ph.D., Brien Holden, Ph.D., D.Sc.

International Centre for Eyecare Education, Kensington, NSW, Australia.

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By Scott Lee, O.D.

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An Olive Branch From ASCRS

A new membership category for optometrists is a promising first step, but the society could—and should—do more.

By Jack Persico, Editor-in-Chief

Groucho Marx famously joked that he'd never want to be a member of a club that would have someone like him as a member. I wonder if any optometrists felt the same way after hearing that ASCRS was going to start letting O.D.s join—but only those employed by a board-certified ophthalmologist who's an ASCRS member.

At first blush, this move seems magnanimous. ASCRS has had a frosty relationship with optometry for years, so any signs of a thaw in relations should be welcomed. But I'll bet that employment restriction sticks in the craw of many optometrists.

Do you really want to join a club that is choosy rather than inclusive? Wouldn't you prefer it if you and *all* your optometric colleagues were welcomed and respected?

ASCRS has an opportunity to bridge the ophthalmology/optometry divide better than most organizations. Nothing in its marching orders limits its scope to ophthalmology. After all, the name of the organization is the American Society of Cataract and Refractive *Surgery*, not *Surgeons*. Obviously surgeons will take precedence in the group's efforts, as they should. But if its organizing principle is "advancing the art and science of anterior segment surgery," that gives them not only an opportunity but indeed a mandate to educate every practitioner who's involved in the delivery of eye surgery.

And like it or not, optometrists *do* participate in ophthalmic surgery. Pre-op testing, patient education, post-op care, long-term follow-up—all the work that goes into ensuring success before and after surgery can't happen without optometrists. That's especially clear when you look at the demographics of cataract and refractive patients, and the manpower disparity between optometry and ophthalmology.

What's the biggest challenge in refractive surgery? Not enough patients. What's the biggest challenge in cataract surgery? Not enough doctors. Who can help with both? You guessed it, optometrists.

If the long-standing beef ASCRS has had is O.D.s' lack of formal training—and this organization *provides* training—who better than ASCRS to correct what it perceives to be the problem? But by limiting membership to M.D.-employed optometrists, is ASCRS really directing its educational efforts to the people who need it most?

Optometrists who practice independent of ophthalmology are, by and large, going to be the ones with the bigger educational needs. By contrast, O.D.s who are employed by an ophthalmology practice already have ready access to the expertise of surgeons by dint of their practice setting. They're well educated about standards of care, the latest surgical techniques, post-op medication regimens, new IOL technology, and so on. Their techs and administrative staff are on the same wavelength, too—because they're also the surgeon's staff.

This olive branch to optometry is a promising first step. But that's all it is. By supporting optometrists of all stripes, ASCRS would be doing a service to its surgeon members, and to the public. Anything that improves care and allows surgeons to work more productively should be embraced openly and without restriction. ASCRS, don't make your newest members hide behind a pair of Groucho Marx glasses. ■

Tell us your stories!

Every manager has to deal with tricky human resources situations at some point. What are yours? For an upcoming issue this fall, we'll have an expert in staff management respond to questions from readers about hiring, firing and other nitty gritty managerial topics.

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Say Goodbye to Casual Friday

Your doctorate cannot possibly outshine your scruffy Civil War re-enactor beard and your shabby, Doritos-stained pants. **By Montgomery Vickers, O.D.**

Can we talk about grooming? Now, many of you are probably wondering, given the way I looked when you last saw me, what makes me an expert on style and beauty? OK, you got me there. On the other hand, where else you gonna find something this important in an optometric journal? I've researched this. The AOA, AOS, ABO, ARBO, AAO, ABBA and the rest of optometry has never truly addressed the importance of clean pants. Until they do, I'm all you got.

So, without further ado, here I go:

1. Who in the world cuts your hair? The "Texas Chainsaw" guy? As a child of the 1960s, it hurts me to say this, but no one wants your hair in their eyes. At least tie your freak flag back.

2. Polish your shoes. I see more nasty shoes at an optometry meeting than at my musicians' union meetings. I know you are on your feet all day but your shoes should not look like they just came off the Bataan death march, and sneakers do not help develop professional relationships with your patients. Good shoes are a must to the modern, bunion-free optometrist. I know they cost money, but so does plantar fasciitis! Trust me. Ouch.

3. Casual Fridays will not get you on the health care panel. In fact, make Friday the day you dress like Fred Astaire at the Oscars. When that frazzled, rumpled, pit-stained patient wanders in for his pressure check, he wants you to be dressed like you're fresh as a daisy and could work all weekend just for him! Dress to impress. You have to look your very best on Fridays. Unlike me. On Fridays, I'm home in my pajamas.

4. Do you own a suit? Yes, ladies, I mean you! There's a reason those actresses playing doctors on TV alternate between thousand-dollar power suits and amazing little black dresses. It's because that's what the public thinks a doctor should look like. Of course, they also think you should be dating Patrick Dempsey. But, let's be real. Surely you ladies understand that you're smarter and look better than the good ol' boys of yesteryear? Look as powerful as you really are inside!

5. You fellows aren't off the hook. Those days of slovenly belly-flopping khakis are long gone. You'd better pull your sartorial game together if you want to be respected by anyone other than your two-year-old granddaughter—the one who calls you "Sloppy Poppy." What will your patients think when they spot you in holey jeans and flip-flops at Walmart? "Hey, see that unshaven dude with the 'Bieber Fever' hat and dirty fingernails? That's my doctor! Want his office phone number?"

6. White coat at the office? I prefer that my doctors wear a white coat. At my last physical, my longtime physician was wearing some sort of Nehru shirt. I half expected him to wave crystals across my prostate. I liked it better when he had a long white lab coat with all kinds of tongue depressors and stethoscopes bulging from the 10 different pockets. His IQ went down 30 points when he walked in the room looking like George Harrison visiting the Maharishi.

Is beauty only skin deep? Does ugly go straight to the bone? Is there a reason I think my opinion matters when it comes to your grooming? Well, you're too old for your mom to dress you. Now clean up that mess in the mirror. ■

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You Can Bill for Tear Testing

Until recently, we've had no objective test for dry eye. Now you can do it, and without a lab. **By John Rumpakis, O.D., M.B.A., Clinical Coding Editor**

Managing the dry eye patient has long been a regular component of the therapeutic optometric practice. Over the years, we've been fortunate to gain significant developments in treatments and preparations that allow us to manage our patients better. But there hasn't been much innovation in diagnostic tools. We've been limited to longstanding but inexact measures such as Schirmer testing, phenol red thread testing, tear film break-up time measurement, and so forth. But lately, no truly objective, quantitative tests have appeared.

Enter TearLab.

The TearLab Osmolarity System is a device and clinical lab test that is intended to measure the osmolarity of human tears to aid in the diagnosis of patients suspected of having dry eye disease, in conjunction with other methods of clinical evaluation. TearLab is for professional in vitro diagnostic use only. In fact, it may be one of the only objective tools we have to aid us in diagnosing and managing clinically relevant dry eye.

Also, it's now reimbursable in most instances.

A Step in the Right Direction

Coding for the TearLab is not complex, but it is different because it's considered to be a clinical lab test, not a typical office procedure. The TearLab Osmolarity System is an in vitro laboratory device and, as such, testing for Medicare patients is billed under the Clinical Diagnostic Laboratory Fee Schedule. Unlike the Physician Fee Schedule, Medicare patient co-payments or deductibles do not apply to services billed under the laboratory fee schedule—covered services are 100% reimbursed. Payment by the Centers for Medicare and Medicaid Services (CMS) is the lesser of: the amount billed; the local fee for a geographic area; or a national limit.

Under the laboratory fee schedule, CMS will only reimburse providers performing laboratory tests who maintain a current certificate as required by the Clinical Laboratory Improvement Amendments (CLIA). However, as of July 2, TearLab had its CLIA status waived, thus allowing those practitioners who have CLIA waiver certification to perform this test in their offices.

How do you obtain a CLIA waiver certificate? Submit the CMS-116 application form (www.cms.gov/Regulations-and-Guidance/Legislation/CLIA/How_to_Apply_for_a_CLIA_Certificate_International_Laboratories.html). Note that state laws in California, Nevada and New York don't allow optometrists to obtain CLIA waiver certificates.

The CPT code to use is 83861 and is defined as: "Microfluidic analysis utilizing an integrated collection and analysis device, tear osmolarity." Because it is a CLIA-waived test, you should use the modifier -QW (i.e., CLIA waived) when performing it in your office.

Courtesy: TearLab Corp.

The electrical impedance osmometer takes an almost microscopic sample, just 50 nanoliters.

Additionally, it is a unilateral test, so it requires an appropriate modifier if both eyes are tested on the same day. The modifiers for this are RT and LT (right and left). So, the coding for the test looks like this if performed bilaterally:

- 83861-QW-RT
- 83861-QW-LT

The CMS reimbursement for this test nationwide is $23.40 per eye and there is no limit on the number of tests that you can run, as long as you have medical necessity established in the medical record.

Whether you believe in the clinical value of tear osmolarity or not, the development of an in-office test that allows us to diagnostically assess our patients and to evaluate our treatment protocols by objective measure is a step in the right direction.

Dr. Rumpakis has no financial affiliation with TearLab. Please send your comments to CodingAbstract@gmail.com.

1. TearLab Corp. website. Available at: www.tearlab.com/products/doctors/productinfo.htm. Accessed July 29, 2012.

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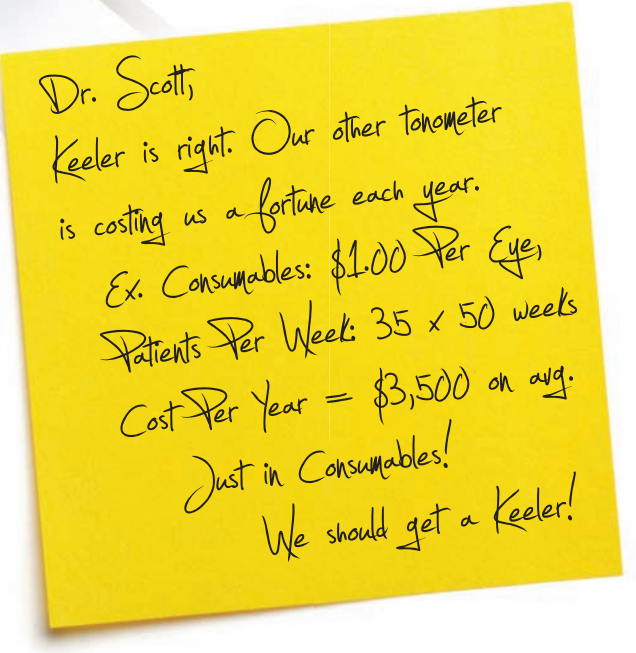

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When to Call a Compounding Pharmacist

Most medications that we require are readily available. But when they aren't, it's time to get creative. **By Jill C. Autry, R.Ph., O.D.**

The roots of medicinal compounding trace back to antiquity. In that era, copper compounds were concocted to treat headaches and a mixture of diluted snake venom was applied topically to stop bleeding.

The mixing and making of medications evolved from the first "healer," who compounded plant-based and herbal remedies around 4,000 B.C., to the multitasking professional we know today as a pharmacist. In fact, until mass drug manufacturing became commonplace in the 1950s, the neighborhood pharmacist mixed and molded almost all prescriptions made in the United States.

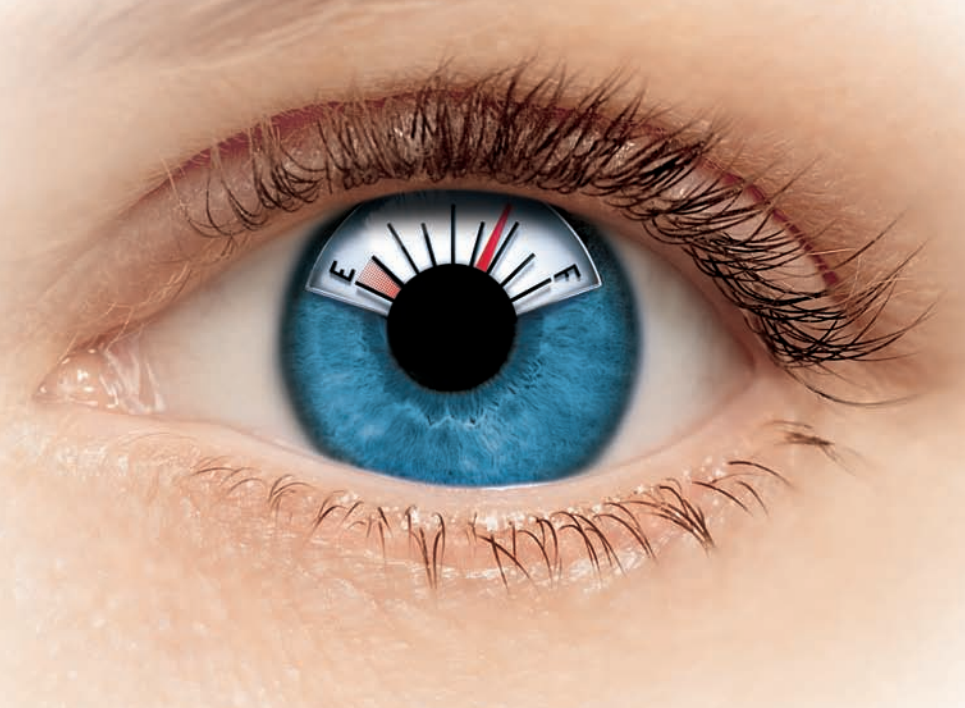
Today, although the need for compounding is far less common, there are still several situations when a compounded product may be your preferred choice. What do they offer that off-the-shelf products lack? At least four worthwhile variations:

• ***Different strength.*** The most common need for a compounded drug: The prescribed agent is not manufactured in a strength deemed necessary for the patient's condition, so the doctor needs a higher or lower concentration than what can be found in stock. For example, a terminally ill patient may need a medication delivered at half the typically prescribed strength, or a psoriasis patient may need a cream that is twice as strong as those made commercially. In such cases, the compounding pharmacist can purchase the raw materials and make the medication to match the needs of the patient.

• ***Different form.*** Another cause for calling upon a compounding pharmacist is when the prescribed medication does not come in the dosage form needed by the patient. This is common when making adult medications into suspensions for children, or for a cancer patient who cannot swallow a pill or capsule. The mixing of oral and intravenous medications into alternate dosage forms is also common in making ocular preparations, rectal

Photos: Jill Autry, O.D., R.Ph.

The neighborhood pharmacist of the 1950s mixed and molded almost all prescriptions. Today, we still need compounded drugs when manufactured ones won't do.



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• ***Different ingredients.*** Some instances require the compounding pharmacist to remove or change the manufactured formulation. Inactive ingredients, such as preservatives or buffers, may cause toxicity or allergy in susceptible individuals. In this case, the pharmacist uses the active ingredient in the dosage required but removes the offending agent from the preparation without altering the pharmacological profile of the medication.

• ***Different formulation.*** Some compounds are even formulated to ease administration or promote compliance. This is an option when two or more medications are mixed together into a single dosage form. The most common of these combinations include dermatological preparations, which are usually prescribed separately but are more effective when applied together.

In ocular disease, many of the same reasons prompt an eye care practitioner to call the local compounding pharmacist for help. This article reviews the most commonly compounded ophthalmic preparations for specific conditions, the appropriate designations for use, and helpful hints for finding the right pharmacist nearby to put it all together.

Dry Eye

In its mildest form, dry eye causes episodic symptoms of burning, tearing, foreign body sensation and intermittent blur. For these patients, artificial tears and/or environmental changes may be all they need to relieve their symptoms. For patients with moderate dry eye, treatments such as Restasis (cyclosporine 0.05%, Allergan), punctal plugs, topical steroids and doxycycline are often added.

When we exhaust these more conventional treatments for a patient with moderate to severe dry eye, we can look to additional therapeutic options that need to be compounded:

• ***Cyclosporine ophthalmic ointment.*** This ointment, applied q.h.s. in severe dry eye patients, typically is used to supplement Restasis topical emulsion. It can be formulated as a 0.1% to 2% concentration. In severely damaged, low vision and/or phthisical eyes, the ointment may be substituted for the topical cyclosporine drop b.i.d. to q.i.d. for more contact time without the concern of associated blur.

• ***Autologous serum.*** This is used in severe aqueous-deficient dry eye to provide patient-specific protein-based protection to the ocular surface. Serum and

normal tears have many of the same components, including vitamin A, various growth factors and proteins (such as lactoferrin and lysoszyme). To create the serum, the patient must make three to four blood donations a year; most clinicians ask for a 20% diluted serum to be instilled q.i.d. or more. Investigators also have tested this treatment for persistent corneal defects.[1]

• ***Albumin drops.*** Although not the preferred autologous serum-based derivative described above, albumin 5% artificial tears may be a suitable alternative tear supplement for several reasons. For one, it is easier to compound than autologous serum. Also, it avoids the need for the patient to make a blood donation. Last but not least, it's much cheaper than autologous serum.

Albumin may improve the tear film by providing mucin-like protection as well as anti-inflammatory action. Research on patients with Sjögren's syndrome found that albumin therapy inhibited the apoptotic enzyme caspase-3, and improved fluorescein and rose bengal scores in just four weeks.[2] (However, it was not statistically significant for tear break-up time or subjective symptoms.)

• ***Transdermal testosterone cream.*** Androgens play a role in dry eye through receptor activity in the lacrimal glands, the meibomian glands and the conjunctiva. Because androgen production decreases in older men and women as well as in autoimmune patients, clinicians are increasingly using topical, transdermal testosterone in a vanishing cream as a treatment for refractive dry eye in these patient populations. Various clinicians recommend a 3% to 5% concentration applied to the upper eyelids b.i.d. initially, then q.h.s.[3] Investigators also are testing compounded testosterone solution applied directly to the eye.[4]

• ***Preservative-free steroids.*** Many commercially available products for dry eye are available without preservatives, such as artificial tears and Restasis. Steroids are often an unavoidable part of our treatment regimen for dry eye, but unfortunately do not come in a preservative-free preparation.

Would your patient like a preservative-free steroid? Call your compounding pharmacist.

For patients who cannot tolerate preservatives, or if preservative-containing medications exacerbate their dry eye, the compounding pharmacist can make preservative-free products, such as 1% methylprednisolone ophthalmic drops. When necessary, other chronic medications, such as glaucoma drops or allergy treatments, can also be prepared preservative-free through compounding.

• ***Acetylcysteine solution.*** In various chronic ocular conditions—most notably severe dry eye—mucous filaments can form and attach to the cornea. This results in pain, foreign body sensation, photophobia and decreased vision. Initial treatment is to remove the filaments with forceps and, if necessary, apply bandage contact lenses. Next, aggressively treat the underlying dry eye and consider an ophthalmic solution of acetylcysteine drops. Mucomyst (acetylcysteine, Bristol-Myers Squibb) is used in patients with pulmonary conditions to reduce excess bronchial mucus. The compounding pharmacist can convert it into a 5% or 10% ophthalmic solution, which can be helpful in treating and preventing recurrences when used q.i.d.

Corneal Bacterial Keratitis

Within the first year of practice, most eye-care practitioners will encounter a bacterial keratitis that is so large, so central or so vision threatening that normal empirical treatment with topical fluoroquinolones does not meet the standard of care. The majority of these corneal ulcers are contact-lens related and, although we tend to think *Pseudomonas* in these cases, they can be caused by either gram-positive or gram-negative organisms.

In these cases, first culture the ulcer and then initiate fortified topical antibiotic therapy with one of the following:

• ***Vancomycin 25mg/ml.*** This covers a wide range of gram-positive organisms, including methicillin-resistant *Staphylococcus aureus* (MRSA). It should be alternated every half-hour or hour with a gram-negative medication, such as ceftazidime or tobramycin.

• ***Cefazolin 50mg/ml.*** Like vancomycin, this first-generation cephalosporin also covers a wide range of gram-positive organisms, but is *not* effective against MRSA. It is well tolerated and is a good choice for pregnant patients who need intense antibiotic therapy (because fluoroquinolones are contraindicated).

• ***Tobramycin 14mg/ml.*** Although generally considered a medication that is active against gram-negative species, this aminoglycoside also works well against gram-positive organisms. It is often paired with vancomycin or cefazolin for comprehensive coverage.

How to Find a Compounding Pharmacist

Find a compounding pharmacist and develop a relationship *before* a unique case occurs so you'll be prepared if—or more likely when—the patient presents to your practice.

So, how do you find one?

- Ask your local dermatologist, oncologist or local retail pharmacist where they send compound prescriptions.
- Check professional websites, such as those for the Professional Compounding Centers of America (www.pccarx.com) or the International Academy of Compounding Pharmacists (www.iacprx.org), where you can enter your city/state/zip code to find a compounding pharmacist near you.
- When you do locate one, don't forget to make sure the compounding pharmacist makes ocular preparations; many are willing to mix common creams, ointments and oral dosage forms, but may decline to make the more involved ophthalmic preparations, which must meet more stringent guidelines.

Also, it is FDA Pregnancy Category B (no known risk to the fetus), so it can be compounded for the treatment of severe corneal infections in pregnant patients.

- ***Ceftazidime 50mg/ml.*** This third-generation cephalosporin is known for outstanding gram-negative coverage. Like tobramycin, ceftazidime is paired with gram-positive vancomycin or cefazolin and is alternated every 30 minutes to an hour for initial treatment.

The aforementioned are just a few of the most common fortified antibiotics. Other choices include amikacin, gentamicin and ceftriaxone.

Amoebic Keratitis

Acanthamoeba, one of the more formidable causes of keratitis, often results in the need for corneal transplantation. The infection is almost exclusive to contact lens wearers. Its diagnosis is often delayed because it can mimic bacterial, fungal or, more commonly, herpetic keratitis. Keep in mind that the amoeba can be resistant to treatment. This is why therapy definitely requires a compounding pharmacist, because the current recommended preparations are not commercially available in the U.S. Often, treatment involves a combined approach, including the use of a biguanide (either polyhexamethylene biguanide 0.02% or chlorhexidine 0.02%) combined with a diamidine (either hexamidine 0.1% or propamidine 0.1%).[5,6]

Band Keratopathy

This ocular degeneration is characterized by a 3 o'clock to 9 o'clock band deposition of calcium across the cornea. The calcium is found just under the epithelial surface, and tends to be concentrated in the intrapalpebral area due to increased tear tonicity and evaporation in this area. Band keratopathy can occur due to a variety of etiologies, but is most often seen in chronic inflammatory conditions, both systemic and ocular. The calcium band can cause visual acuity loss as well as chronic foreign body sensation, depending on its severity.

Treatment involves an ophthalmic solution of ethylenediaminetetraacetic acid (EDTA), an effective treatment due to its chelating effect on calcium and other metal ions.[7] Therapy starts with first debriding the epithelium and then applying a 2% EDTA compounded solution to the cornea for three to five minutes. Lastly, the calcium deposits are scraped away with a spatula and then a bandage contact lens is applied. Depending on severity, multiple applications and scrapings may be necessary over time to control the keratopathy.

Intravitreal Injections

Although intravitreal injections for macular degeneration are commonplace today, compounding pharmacists have been supplying various preparations of antibiotics and steroids for intraocular injection for years.

Today, the off-label use of the anti-VEGF Avastin (bevacizumab, Genentech), a systemic cancer therapy reformulated for intraocular use, is the most commonly compounded intravitreal preparation. It continues to be prescribed in lieu of the FDA-approved Lucentis (ranibizumab, Genentech) due to the extreme cost difference between the two products. (A single Lucentis injection costs approximately $2,000 while a shot of Avastin is closer to $50.)

A 2011 outbreak of endophthalmitis cases in Avastin-treated patients was traced to a single compounding pharmacy; this serves as an object lesson on the importance of demanding strict adherence to sterility protocols from your compounding pharmacies. (*See "Compounding Pharmacists Keep it Clean," page 36.*)

A new entrant to the anti-VEGF market, Eylea (aflibercept, Regeneron Pharmaceuticals), offers comparable efficacy to Lucentis but with less frequent dosing, especially in the first year of therapy. Depending on its cost, Eylea's reduced treatment regimen and manufacturing safeguards may temper enthusiasm for reformulated Avastin used off-label.

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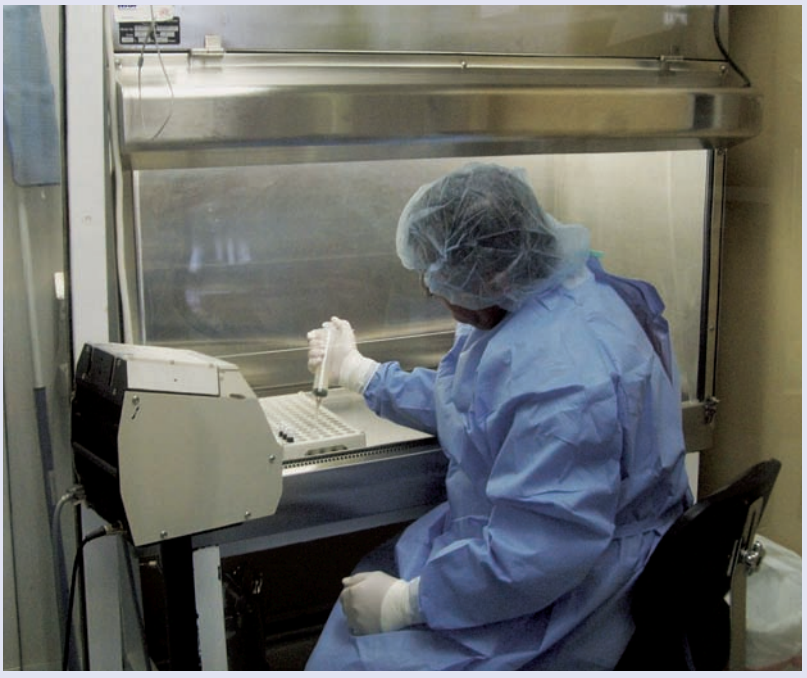

Compounding Pharmacists Keep it Clean

Despite recent reports of tainted Avastin, be assured that problems with compounded medications are rare. Ophthalmic preparations must be made under sterile conditions following the U.S. Pharmacopeia Chapter 797 guidelines. Pharmacists and technicians must use aseptic techniques to preserve sterility when preparing these products and keep current on the techniques they have learned.

Further, the medications must be prepared in a clean room inside a laminar flow hood to avoid contaminants or bacteria, and then sterilized by using a micron filter or autoclave to ensure a quality product. The clean room and laminar flow hood must be tested regularly by an outside source for bacteria and endotoxins.

Watch for the use of intravitreal injections to become even more commonplace in the future as they bypass topical administration concerns, take compliance issues out of the hands of patients, and are direct to the intended target site.

Currently, the use of anti-VEGF treatments is increasing as both on-label and off-label indications expand beyond age-related macular degeneration. For chronic disease states, such as glaucoma or diabetic retinopathy, novel intravitreal injections could someday replace and/or supplement current standard-of-care topical medications or laser treatments. For instance, a brimonidine intravitreal implant is now in two Phase II studies—one for glaucomatous optic neuropathy and one for the treatment of geographic atrophy due to AMD.[8,9]

Antifibrosis

Mitomycin-C is an antitumor antibiotic used in cancer chemotherapy. It is also commonly employed in various ophthalmological surgical procedures—such as pterygium removal, trabeculectomy and photorefractive keratectomy—to prevent vascularization, scar formation and haze.

Most physicians ask a compounding pharmacist to make a solution of 0.02% to 0.05% concentration and apply it directly to the surgical site for 20 seconds to five minutes, depending on the surgical or clinical situation.[10,11]

Corneal Collagen Crosslinking

Riboflavin (vitamin B_2) 0.1% drops are compounded and used in conjunction with an application of UV-A light during a procedure known as corneal crosslinking. Although not yet FDA approved in the United States, this technique is now being used off-label in the U.S. and is used routinely in many other countries for the treatment of keratoconus, corneal ectasia and even some cases of bacterial keratitis.

The riboflavin acts as a photosensitizer that strengthens the collagen fibers. It is applied topically five minutes before UV-A light exposure and every five minutes thereafter during the 30-minute ultraviolet light exposure.[12-14]

In-Office Compounding

In-office dilutions are off-label and anecdotal. But that doesn't mean off-label is off limits.

One of the more popular examples in eye care: diluting a sample bottle of brimonidine with artificial tears for a quick red eye remedy. Practitioners who report success with this compound most often use a ratio of two drops of brimonidine per 1ml of artificial tears, using the mixture b.i.d. until the sample is empty. Any concentration of brimonidine will work; however, samples of Alphagan P (brimonidine 0.1%, Allergan) are the most common. Six drops are placed in a 3ml sample bottle of Allergan's Optive artificial tears (because the top easily pops on and off).

Be aware that long-term use can result in rebound hyperemia, which is typical with alpha-agonists. Also,

don't use it on post-LASIK patients because it might cause slippage of the flap.[15]

Another in-office dilution: adding 10 to 20 drops of a topical anesthetic, such as proparacaine, to a sample bottle of artificial tears. This very weak amount of anesthetic is useful following refractive surgery, such as PRK, in order to provide pain relief for 24 to 48 hours.[16] Take note that this dilution should never be used for pathologic pain, such as infectious keratitis or corneal abrasions associated with contact lens wear or of unknown etiology.

In short, don't neglect to offer unconventional pharmaceutical options. These tips should make prescribing a compounded ophthalmic preparation a successful venture for both you and your patient. As we know, one size does not fit all. ■

Dr. Autry practices in a referral center in Houston. She maintains a pharmacist license, and lectures extensively on pharmaceutical and ocular disease topics. She thanks compounding pharmacists Ken Hughes, R.Ph., in Bellaire, Texas, and Tim Clark, R.Ph., in Southern Pines, N.C., for their assistance with this article.

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Consider Ortho-K For Myopia Control

Today, orthokeratology is one of most successful methods to slow or even stop myopia progression in children. **By Kenneth Daniels, O.D.**

During a pediatric eye exam, you'll often discover that the child truly cannot read the chart—even with a parent sitting there saying, "Come on, you can see that!" As you carefully address the situation and perform your refraction, you determine that the child is significantly myopic. Suddenly, you hear the child read "T, Z, V, E, C, L." Then, when you remove the phoropter, you hear him mutter the word "Wow." Now, what do you do?

First, attempt to alleviate the guilt and embarrassment that the parent is about to feel. Next, take some time to describe all the options in refractive care. While doing so, educate both the parent and child about the condition of myopia, how it progresses with increased age and how it can impact the child's performance in both school and sports. Then, be sure to review all the corrective options—from spectacles and conventional contact lenses to pharmaceutical treatments and orthokeratology (ortho-k) lenses (*see "Options for Myopia Control," page 40*). Remember to stress the potential visual benefits and financial costs associated with each corrective method. Together, you can help create the best plan to improve the child's vision.

This article chiefly will focus on the use of ortho-k lenses as a primary treatment for myopia control, an option sometimes overlooked in favor of conventional corrective lenses.

Where to Start?

First, you must reassure the child that he or she is not going blind, but instead will simply require corrective eyewear to achieve the best possible visual function and performance. Additionally, briefly summarize some of the evidence-based medicine associated with the control of myopia progression.

Confirmatory testing is critical prior to detailed planning and discussion. This should include a battery of examinations, such as cycloplegic evaluation, accommodative response and lag, phoria, accommodative convergence/accommodation ratio, intraocular pressure, corneal topography and possibly wavefront aberrometry.[1,2] Also, you may wish to screen for predictive signs of juvenile myopia, including cycloplegic refraction, spherical refractive error, axial length and corneal power.[3]

Early Myopia Development and Progression

In 1989, researchers from the Orinda Longitudinal Study of Myopia (OLSM) analyzed the relationship between normal eye growth and the development of myopia in school-age children.[4,5] In addition, the researchers investigated accommodative function, peripheral refractive error, intraocular pressure, genetic/anatomical similarities with parents, refractive error profiles of other ethnic groups and overall scholastic performance as well as DNA-based studies on the prevalence of familial trends in myopia (*see "Anatomical Influences on Refractive State," page 41*).

The OLSM researchers found that refractive errors decreased toward emmetropia at an average of

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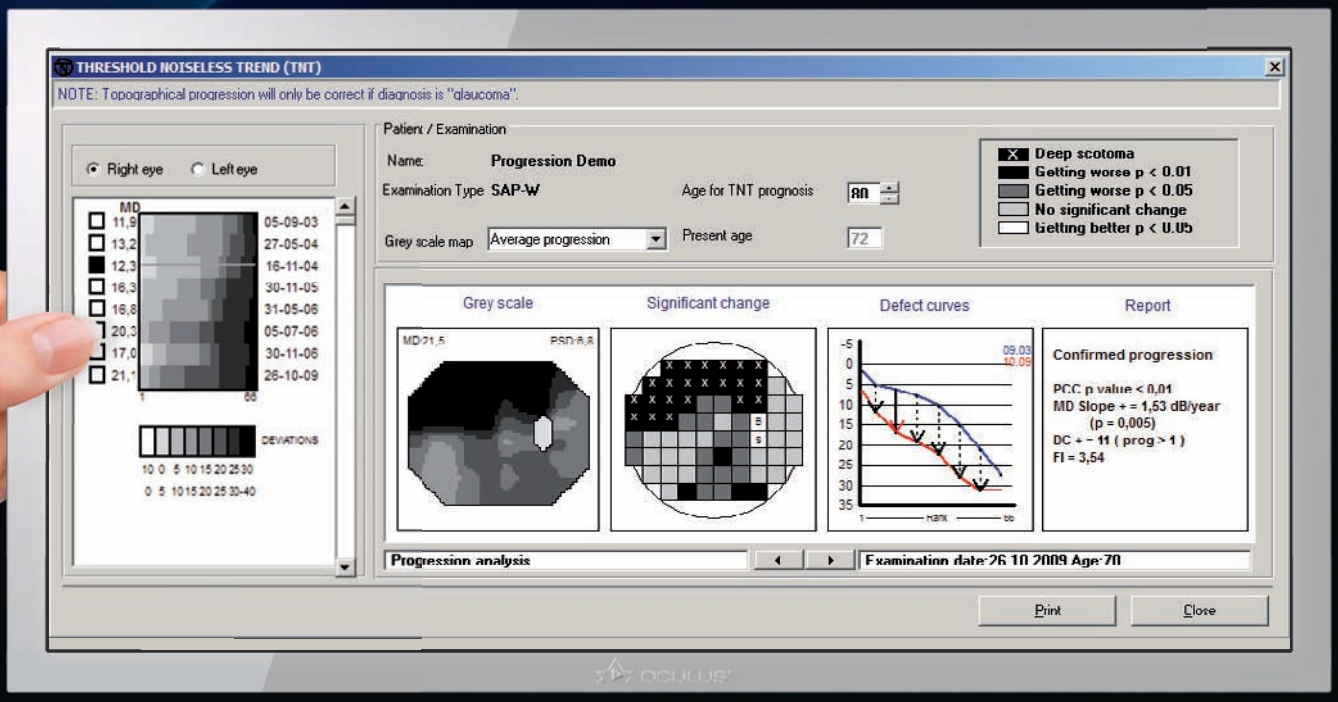

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- Pharmaceutical agents (atropine, pirenzepine, 7-methylxanthine)
- Acupuncture
- Refractive surgery (as a potential treatment option in adulthood)
- Vision therapy

+0.73D at age six to an average of +0.50D by age 12.[4] Furthermore, from ages six to 12, the vitreous chamber elongated by approximately 0.52mm and the crystalline lens power decreased by approximately 1.35D.

The Collaborative Longitudinal Evaluation of Ethnicity and Refractive Error (CLEERE) study assisted in confirming and expanding upon the data garnered from the OLSM.[6] The CLEERE researchers suggested that children who have two parents with myopia are inherently predisposed to have eyes that are shaped like those of a nearsighted person, and are also likely to become nearsighted over time. This research confirmed the presence of a genetic/anatomical relationship in myopia development and progression.

In addition to genetic predilection, there are many other factors that contribute to myopia development. These include consistently performing near-point work, education level, urban vs. rural location and the amount of time spent outdoors.[7]

Excessive near-point work and even prolonged dark exposure appears to strongly influence myopia progression.[8] Myopia occurs less frequently before children reach school age, but increases significantly during high school and college years. This pattern likely is explained by increased periods of continuous reading and studying in poor lighting conditions. For these individuals, environmental modification—including ergonomic adjustments, improved lighting, additional rest breaks and increased periods of physical activity—may decrease the rate of myopic progression.[9]

Overall, the prevalence of myopia is extremely high—not only in the United States, but also throughout the rest of the world. In the United States, at least 25% to 41% of the population has myopia.[10] Much more staggering, however, approximately 70% to 90% of individuals in some Asian countries are nearsighted.[11] It is important to mention that greater levels of nearsightedness (more than 6.00D) are associated with an increased risk of rhegmatogenous retinal detachment, glaucoma and myopic degeneration.[12]

Myopia Control With Contact Lenses

For decades, eye care practitioners have been using contact lenses to slow and/or stabilize myopia progression. However, opinions on the effectiveness of this treatment vary widely.[13]

In 1976, German researchers found that just 40% of patients age 15 to 25 years who were fitted with contact lenses experienced myopia progression, compared to 75% of patients who wore spectacle lenses during a 15-year period.[14] The researchers also studied the axial length of the eyes; however, their data was largely unreliable due to the limited technology at the time.

Then in 1990, Russian researchers published results from a five-year longitudinal study, which indicated that refraction remained unchanged in 73.2% of patients who wore contact lenses.[15] The authors suggested that the contact lenses stabilized the patients' accommodative abilities, which helped improve visual quality.[15]

Silicone gas-permeable lenses also have been used to combat myopia progression. Using an alignment fitting methodology, one study indicated that patients who wore daily silicone gas-permeable lenses exhibited an increase in myopia of 0.28D over a two-year period, compared to 0.80D in patients who wore spectacles.[16] Furthermore, the researchers found that patients experienced a significant loss of myopic control (an average of 0.76D) within four years of lens wear discontinuation. These results suggest that gas-permeable contact lenses are a very effective option for myopia control.[16]

Other Treatment Options

In addition to contact lenses, here's an overview of other common strategies for minimizing myopia progression in children.

- *Alterative/medical therapies.* Topical atropine 1%, a nonselective muscarinic antagonist, may be used to slow the progression of

myopia and ocular axial elongation. In one study, atropine effectively slowed the progression of low/moderate myopia and ocular axial elongation in Asian children.[17] In a similar study, children who received pirenzepine gel, cyclopentolate eye drops or atropine eye drops for one year showed significantly less myopia progression than children who received a placebo.[18]

Dopamine analogs also have been used to control progression. While there are no FDA-approved dopamine analogs for use as myopia treatments, both pirenzepine and 7-methylxanthine (7-MX) have been evaluated in preliminary trials.[19-21] Specifically, one European clinical trial of 7-MX showed that the agent is less effective at slowing myopia progression than topical atropine.[21]

Additionally, preliminary studies examined the safety and efficacy of pirenzepine for myopia control.[20] The results were promising, but no further testing has been conducted.

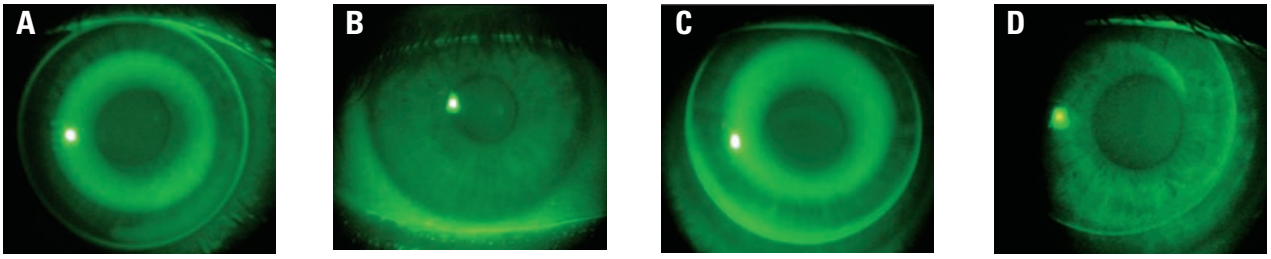
You must be wary of adverse effects when considering the use of these agents. Documented side effects include light sensitivity; pupillary dilation; increased IOP; near blur; dry mouth; hot, flushed and/or dry skin; bradycardia followed by tachycardia, palpitations and arrhythmias; rashes; fever; or even hallucinogenic response.[19-21]

Most recently, acupuncture has garnered some interest as an alternative therapy for progressive myopia.[22] Acupuncture includes the stimulation of strategic anatomical points by various methods, including needle insertion and acupressure.[22] In this instance, acupuncture needles may be inserted into specific auricular areas in order to relieve muscle spasms around the eye and improve ocular blood flow.

• ***Vision therapy.*** Accommodative factors can be adjusted via several methods of vision therapy and training. For example, vision therapy can be employed to train patients with pseudomyopia (sudden-onset, progressive myopia due to accommodative stress) to

Anatomical Influences on Refractive State

- Anterior corneal curvature
- Posterior corneal curvature
- Corneal thickness
- Refractive index
- Anterior chamber depth
- Axial length of eye
- Accommodation and convergence
- Choroidal, retinal and vitreal pressure



A) Optimally centered fit of an ortho-k lens. B) Cornea following optimally centered lens removal. Note virtually no trace of an impression ring. C) Superior-nasal decentered fit of an ortho-k lens. D) Impression ring following decentered lens removal.

relax accommodation. Additionally, several studies have shown that vision therapy can reduce or eliminate complaints of pseudomyopic shift (transient distance blur) by improving accommodative facility.[23,24]

• ***PALs.*** The Correction of Myopia Evaluation Trial (COMET)—a major study supported by the National Eye Institute—compared the effect of progressive addition lenses (PALs) vs. single-vision lenses on the progression of juvenile-onset myopia.[25] The COMET researchers determined that PALs slowed the progression of myopia significantly more effectively than single-vision lenses within the first year of wear.[25]

An Overview of Ortho-k

One additional method of myopia control, ortho-k, has become increasingly popular during the last two decades. So, what is ortho-k? In the simplest terms, it entails flattening or reshaping the anterior corneal surface in an effort to adjust the eye's refractive power.

It is not a new concept. In fact, ortho-k dates back to the 1940s. For many years, the earliest ortho-k techniques simply used "keratometry measures and clinical judgment" to determine the next step in the corneal flattening process. In fact, it was not until the early 1990s—with the advent of corneal topography and new gas-permeable materials—that orthokeratology became a more viable mainstream treatment option for refractive error correction.

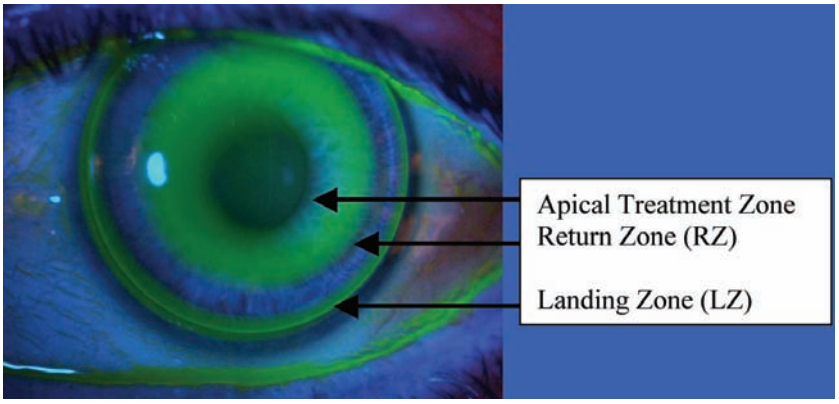
Here's how I often describe ortho-k to my patients: "The cornea is a soft tissue, and its 'skin' can be molded by the use of a rigid gas-permeable contact lens—much like orthodontic braces for your teeth. The lens reshapes the corneal surface to a specific contour, which easily can be adjusted to enhance the desired effect in safe, overnight lens wear. The process is unlike a refractive surgical procedure, which is permanent. So while the outcomes yielded by ortho-k are reversible, it allows you to function without contacts or eyewear throughout the daytime hours."

Ortho-k lenses iatrogenically induce corneal topographic sphericalization via constant pressure on the flat meridian and variable pressure on the steep meridian. A plateau is reached when the cornea becomes spherical secondary to applied uniform pressure, resulting in central flattening and peripheral steepening.[26-28]

Today, several companies manufacture ortho-k lenses. In June 2002, Paragon Vision Sciences' Corneal Refractive Therapy lens was the first ortho-k design to gain FDA approval for overnight wear. Subsequently, Euclid Systems' Emerald lens secured FDA approval in 2004 (Bausch + Lomb purchased the lens design in 2005).

Ortho-k for Myopia Control

More than a decade ago, several eye care clinicians initially suggested that ortho-k lenses potentially could be used to control or even halt myopia progression. Then, in 2004, the results from



The optimal fit and zone alignment of an ortho-k lens.

the first report on ortho-k for myopia control—the Children's Overnight Orthokeratology Investigation (COOKI) pilot study—were published.[29] COOKI researchers evaluated refractive error, visual changes and ocular health for six months in myopic children who were fit with overnight ortho-k lenses. The researchers determined that overnight ortho-k was both a safe and effective treatment for curtailing myopia progression.

In 2005, data from the Longitudinal Orthokeratology Research in Children (LORIC) study indicated that ortho-k was effective at controlling childhood myopia.[30] However, the researchers also determined that substantial anatomic variations among children can reduce the clinician's ability to accurately predict final visual outcome before starting ortho-k therapy.[30]

Results from more recent clinical trials, such as the Stabilizing Myopia by Accelerated Reshaping Technique (SMART) study and the Corneal Reshaping and Yearly Observation of Myopia (CRAYON) study, have yielded additional information regarding the safety and efficacy of ortho-k for myopia control.[31,32]

SMART, a five-year study initiated in 2009, currently is evaluating the effect of ortho-k on myopia progression in 138 patients. At one-year follow-up, subjects wearing ortho-k lenses exhibited a mean progression of 0.00D, compared to an average of 0.50D in the control group.[31]

In the two-year CRAYON study, researchers confirmed that patients who were fitted with ortho-k lenses experienced significantly less annual change in axial length and vitreous chamber depth than patients fitted with soft contact lenses.[32] These results confirmed data from previous studies by showing that ortho-k lenses slow the progression of corneal changes in myopic patients.[32]

Recently, there has been suggestion of low-level myopic orthokeratology with the use of a high-modulus silicone hydrogel lens intentionally worn in an everted position. The everted wear of a high-minus silicone hydrogel contact lens can induce alterations in corneal topography and subjective refraction. These refractive change range from plano to +1.75D sphere and +0.25D to +0.75D cylinder, but are unpredictable and vary from subject to subject.[33,34]

In one study, the mean apical topographic power change was 1.11D with slight corneal steepening in both meridians as well as 0.23mm of corneal flattening in the horizontal meridian and 0.27mm of corneal

flattening in the vertical meridian.[33] Additionally, corneal eccentricity decreased by an average of 0.65e.[34] These results suggest that this ortho-k technique may be suitable for patients with very low myopia.

Safety Concerns and Side Effects

As the aforementioned studies suggest, ortho-k is a safe procedure as long as patients are monitored properly. As with all contact lens use, the two most common side effects that occur in patients with ortho-k lenses are corneal edema and staining. Other potential side effects include pain, redness, tearing, irritation, discharge, ocular abrasion or visual distortion.[35-37] These usually are temporary conditions, especially if the lenses are removed promptly. However, clinicians must be particularly cautious to monitor for microbial lens binding of *Pseudomonas aeruginosa*.[35-37] Instruct patients to report any pain, discomfort or visual compromise immediately.

As with any contact lens or refractive procedure, induced visual aberrations and distortions can occur and should be monitored using aberrometry. Following overnight ortho-k, one study uncovered significantly lower rates of mesopic contrast as well as an increased incidence of higher-order aberrations. In another study, researchers found that higher-order aberrations—especially spherical aberration and coma—had a short-term, but significant, increase following ortho-k.[38] Fortunately, these visual effects usually are reversible within 72 hours of lens discontinuation.[38]

Other Discussion Points

• ***Residual cylinder.*** Ortho-k lenses are designed for individuals with low to moderate myopia (up to -6.00D) with or without astigmatism (up to -1.75D). But beware of higher cylindrical correction and possible residual cylinder—you may reduce the myopia but leave appreciable higher-order aberrations with uncorrected cylinder.

• ***Astigmatism.*** Even though astigmatism is addressed at low levels with traditional orthokeratology, Paragon Science is launching the dual-axis system to addresses higher cylinder corrections. In the traditional design, a lens fit on the flat meridian would de-center due to the differential between the flat and steep meridians. (Sometimes, this may be seen as an ortho-k island that is topographically similar to a LASIK island.) The dual axes have a spherical base curve with a unique peripheral system. This affords the two meridians varied depths within the return zone.[39]

• ***Full distance vs. monovision.*** In an adult patient, you can either set a goal of full-distance correction or monovision. In a full-distance correction, the patient will require reading glasses. If, however, the patient is fit with monovision and does not like the effect, simply adjust the base curve to push for a fuller distance or an intermediate distance correction.

In children, the full-distance correction will be required. However, do not abandon the concept of readers or even vision therapy.

Is Ortho-k Reversible?

As mentioned previously, the effects of ortho-k usually are temporary and reversible. This fundamental consideration often appeals to many patients who are intimidated by the lifelong effect of LASIK or PRK. Additionally, adult patients are more comfortable knowing that ortho-k lenses can be manipulated as needed to address presbyopia throughout the child's lifetime.

In order to maintain the long-term refractive effect of ortho-k, however, the patient must wear a retainer lens. Again, similar to the use of a dental retainer in orthodontia to maintain teeth alignment, a retainer lens is worn to preserve the flattening effect and prevent further myopia progression.

Overnight ortho-k lens wear compresses the corneal epithelium, forcing a reduction in central corneal thickness, without damaging the epithelial cells or the contiguous stromal tissue. Once the lens is removed, the cornea does not simply bounce back like a "top hat," but rather slowly returns to its natural, pre-treatment state. The compressed effect should persist for at least 12 to 15 hours, while the patient is awake (in some cases, the effect may last two to three days). Then, to maintain the compressed shape, the retainer lens must be placed on the eye once again during the nighttime hours.

In my clinic, we instruct the patient to wear the retainer lens each night for at least 30 days. After 30 days, we ask the patient not to wear the retainer lens for one night to determine the longevity of its effect. We explain that they may lose some of the effect throughout the second day; however, the accommodative loss will be based on subjective impression. If the patient appreciates a more rapid blur, we suggest that the retainer should be worn each night (or even every other night), as long as he or she retains comfortable and functional vision. Anecdotally, I have found that some patients have been very successful without the retainer for three full days, while others simply prefer to use

the lens each evening. No matter the case, it is best to reassure your patients that, if they miss a night of retainer wear, they will not lose the effect.

The reversibility of ortho-k's effect varies per individual; however, patients generally notice regressive changes within 24 to 72 hours. Furthermore, regression is not linear and thus not absolutely predictable. Refractive error, patient age and subjective tolerance are the determining factors of appreciable visual regression.[40] And, because of the subjective considerations, perceived regression is not always consistent with objective refractive and topographic findings. Keep in mind, however, that patients with higher levels of baseline myopia seem to be more likely to experience end-of-day regression.[40,41]

Prepare the Patient

Keep in mind that some prospective ortho-k patients never wore contact lenses before. In my office, we ask our novice patients to participate in a trial of daily disposable or silicone hydrogel lenses. This will teach the patient how to insert and remove the lenses properly as well as how to handle the lenses and care products.

Furthermore, this contact lens trial also gives us an avenue for myopia correction if, by chance, the patient is not successful with ortho-k lenses. (As a side note, our office recommends a contact lens trial during refractive surgical preparation. We prescribe lenses with the targeted refractive correction, or lesser, to facilitate visual education as well as provide a realistic illustration of the anticipated surgical outcome.)

The patient should be educated that, as with any new fit, there is lens awareness at first (the sensation often will become less noticeable after sleeping in the lenses). Also, the initial lens fitting can be enhanced with a diluted dose of proparacaine, if needed. Fortunately, ortho-k lenses are very comfortable due to their large size and contour.

Guiding Patient Expectations

The modern technology of the corneal reshaping lenses allows for immediate appreciation of the myopic reduction; however, it takes extensive training time for the cornea to hold its new shape consistently. During the first weeks, the lenses should be worn every night. Thus, it is important to establish a proper and realistic timeline for the patient. Equally important: Do not over-promise visual results.

When discussing expectations with younger

Individual Patient Needs and Considerations for Ortho-k

- Age
- Ethnicity
- Culture
- Parental influence
- Motivation (of both the child and the parents)
- Academic requirements
- Athletic requirements
- Occupation (parents) and avocations (parents and child)
- Ergonomics
- Environmental issues
- Medical issues
- Financial concerns
- Ability to manage the corrective device
- Ability to maintain proper long-term commitment to the corrective device
- Disinclination to pursue refractive surgery (parent)
- Disinclination to wear eyeglasses (child)
- Maximum refractive error: -1.00D to approximately -6.00D (low cylinder to a maximum of -0.75D)*

* Toric and dual-axis ortho-k lenses are becoming more widely available.[44]

patients, it is important to respect parental input—but not at the expense of the child. If the child is uncomfortable with the concept of contact lens wear or corneal reshaping, do not let the parent force ortho-k on the child. For ortho-k to be successful, the child (and/or parent) must genuinely believe in the corrective process and conscientiously decide to remain compliant with lens wear.

As suggested by results from the LORIC study, each patient has unique visual needs as well individualized expectations and endpoint visual goals (*see "Individual Patient Needs and Considerations for Ortho-k," above*). In the first week, ortho-k lenses could be worn throughout the day (if tolerated by the patient) to accelerate the effect. However, this step may not be necessary or desirable for all patients.

Additionally, create an informed consent contract that formally outlines the fitting method and appropriate aftercare. This contract should define all concerns and precautions of contact lens wear, lens care product prescriptions, patient responsibilities, information about the exchange of lenses and warranties, replacement policies and your contact information for emergency care. The contract also should itemize the cost of professional fees and associated materials as well as detail all financial arrangements.

On the first day of lens wear, the patient will need to be seen in the morning and afternoon to determine if the lens settled into the correct position after sleep and to monitor for regression. You should also schedule several follow-up appointments to monitor for refractive and topographic changes (*see "An Ideal Short-term Follow-up Schedule," page 49*).

After approximately one month, the patient can attempt to discontinue lens wear for 48 to 72 hours to determine the longevity of the effect and build confidence in the procedure. This step also will help the patient to decide if he or she wants to wear the retainer lens every night or every other night.

Financial Costs of Ortho-k

The cost of ortho-k is unique to each practice, because of different lens models and varied professional fees. Also, regional pricing differences occur based on localized economics and patient demand. Some offices will arrange financing through an external company or have an arrangement policy that should be detailed in the informed consent contract.

Without question, however, ortho-k is a unique practice differentiator. Not all offices offer this service, leaving a window of opportunity for an individual practice to expand on the service or even consider comanagement with area colleagues.

Keep in mind that not all patients will be successful with ortho-k, so you may wish to offer an "easy out" for unsuccessful cases that poses minimal financial risk for the patient.

Offering ortho-k in your practice is an excellent opportunity to better serve the next wave of young myopes. As with LASIK, the popularity of ortho-k has dramatically decreased over the past several years—even in geographic areas once thought to be "recession proof."[42,43] While there is little to no published literature on the key financial data associated with orthokeratology, overall demand for the procedure would most likely follow refractive surgery trends. Nonetheless, it is logical to believe that as the eye care market and global economy strengthen, so too will the interest in ortho-k. ■

Dr. Daniels is in private practice in Hopewell and Lambertville, N.J. He also is an assistant clinical professor at Salus University in

FIRST-LINE

LUMIGAN® 0.01%

(bimatoprost ophthalmic solution) 0.01%

For patients starting or changing PGA therapy

A drop with low dropout

Efficacy with low overall discontinuation rate:
8.1% (15/186) with LUMIGAN® 0.01%
and 13.4% (25/187) with LUMIGAN® 0.03%[1,2]

Indication: LUMIGAN® 0.01% and 0.03% is indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

Important Safety Information

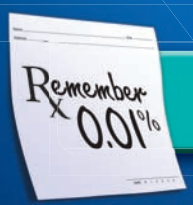
Warnings and Precautions: LUMIGAN® causes changes to pigmented tissues, mostly increased pigmentation of the iris, eyelid, and eyelashes as long as LUMIGAN® is administered. Iris color change may not be noticeable for several months to years. After discontinuation of bimatoprost, iris pigmentation is likely to be permanent, while eyelid and eyelash changes have been reported to be reversible in some patients. Patients should be informed of the possibility of increased pigmentation. The long-term effects of increased pigmentation are not known.

LUMIGAN® should be used with caution in patients with active intraocular inflammation (eg, uveitis) because the inflammation may be exacerbated. Macular edema, including cystoid macular edema, has been reported with LUMIGAN®. LUMIGAN® should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

Adverse Reactions: The most common (25%-45%) adverse event with LUMIGAN® was conjunctival hyperemia. Approximately 0.5% to 3% of patients discontinued therapy due to conjunctival hyperemia. Other common events (> 10%) included growth of eyelashes and ocular pruritus.

Please see brief Prescribing Information on adjacent page.

1. LUMIGAN® 0.01% and 0.03% Prescribing Information.
2. Katz LJ et al. *Am J Ophthalmol.* 2010;149(4):661-671.



Prescribe LUMIGAN® 0.01%

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LUMIGAN® 0.01% AND 0.03%
(bimatoprost ophthalmic solution)

INDICATIONS AND USAGE
LUMIGAN® 0.01% and 0.03% (bimatoprost ophthalmic solution) is indicated for the reduction of elevated intraocular pressure in patients with open angle glaucoma or ocular hypertension.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Pigmentation: Bimatoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid), and eyelashes. Pigmentation is expected to increase as long as bimatoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of bimatoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. The long-term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with **LUMIGAN®** 0.01% and 0.03% (bimatoprost ophthalmic solution) can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes: LUMIGAN® 0.01% and 0.03% may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intraocular Inflammation: LUMIGAN® 0.01% and 0.03% should be used with caution in patients with active intraocular inflammation (eg, uveitis) because the inflammation may be exacerbated.

Macular Edema: Macular edema, including cystoid macular edema, has been reported during treatment with bimatoprost ophthalmic solution. **LUMIGAN®** 0.01% and 0.03% should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

Angle-closure, Inflammatory, or Neovascular Glaucoma: LUMIGAN® 0.01% and 0.03% has not been evaluated for the treatment of angle-closure, inflammatory, or neovascular glaucoma.

Bacterial Keratitis: There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

Use With Contact Lenses: Contact lenses should be removed prior to instillation of **LUMIGAN®** 0.01% and 0.03% and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS
Clinical Studies Experience: Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice.

In clinical studies with bimatoprost ophthalmic solutions (0.01% or 0.03%), the most common adverse event was conjunctival hyperemia (range 25%-45%). Approximately 0.5% to 3% of patients discontinued therapy due to conjunctival hyperemia with 0.01% or 0.03% bimatoprost ophthalmic solutions. Other common events (> 10%) included growth of eyelashes and ocular pruritus.

Additional ocular adverse events (reported in 1% to 10% of patients) with bimatoprost ophthalmic solutions included ocular dryness, visual disturbance, ocular burning, foreign body sensation, eye pain, pigmentation of the periocular skin, blepharitis, cataract, superficial punctate keratitis, eyelid erythema, ocular irritation, eyelash darkening, eye discharge, tearing, photophobia, allergic conjunctivitis, asthenopia, increases in iris pigmentation, conjunctival edema, conjunctival hemorrhage, and abnormal hair growth. Intraocular inflammation, reported as iritis, was reported in less than 1% of patients.

Systemic adverse events reported in approximately 10% of patients with bimatoprost ophthalmic solutions were infections (primarily colds and upper respiratory tract infections). Other systemic adverse events (reported in 1% to 5% of patients) included headaches, abnormal liver function tests, and asthenia.

USE IN SPECIFIC POPULATIONS
Pregnancy: Pregnancy Category C.

Teratogenic effects: In embryo/fetal developmental studies in pregnant mice and rats, abortion was observed at oral doses of bimatoprost that achieved at least 33 or 97 times, respectively, the maximum intended human exposure based on blood AUC levels.

At doses at least 41 times the maximum intended human exposure based on blood AUC levels, the gestation length was reduced in the dams, the incidence of dead fetuses, late resorptions, peri- and postnatal pup mortality was increased, and pup body weights were reduced.

There are no adequate and well-controlled studies of **LUMIGAN®** 0.01% and 0.03% (bimatoprost ophthalmic solution) administration in pregnant women. Because animal reproductive studies are not always predictive of human response, **LUMIGAN®** should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers: It is not known whether **LUMIGAN®** 0.01% and 0.03% is excreted in human milk, although in animal studies, bimatoprost has been shown to be excreted in breast milk. Because many drugs are excreted in human milk, caution should be exercised when **LUMIGAN®** is administered to a nursing woman.

Pediatric Use: Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

Geriatric Use: No overall clinical differences in safety or effectiveness have been observed between elderly and other adult patients.

Hepatic Impairment: In patients with a history of liver disease or abnormal ALT, AST, and/or bilirubin at baseline, bimatoprost 0.03% had no adverse effect on liver function over 48 months.

OVERDOSAGE
No information is available on overdosage in humans. If overdose with **LUMIGAN®** 0.01% and 0.03% (bimatoprost ophthalmic solution) occurs, treatment should be symptomatic.

In oral (by gavage) mouse and rat studies, doses up to 100 mg/kg/day did not produce any toxicity. This dose expressed as mg/m^2 is at least 70 times higher than the accidental dose of one bottle of **LUMIGAN®** 0.03% for a 10-kg child.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility: Bimatoprost was not carcinogenic in either mice or rats when administered by oral gavage at doses of up to 2 mg/kg/day and 1 mg/kg/day respectively (at least 192 and 291 times the recommended human exposure based on blood AUC levels respectively) for 104 weeks.

Bimatoprost was not mutagenic or clastogenic in the Ames test, in the mouse lymphoma test, or in the *in vivo* mouse micronucleus tests.

Bimatoprost did not impair fertility in male or female rats up to doses of 0.6 mg/kg/day (at least 103 times the recommended human exposure based on blood AUC levels).

PATIENT COUNSELING INFORMATION
Potential for Pigmentation: Patients should be advised about the potential for increased brown pigmentation of the iris, which may be permanent. Patients should also be informed about the possibility of eyelid skin darkening, which may be reversible after discontinuation of **LUMIGAN®** 0.01% and 0.03% (bimatoprost ophthalmic solution).

Potential for Eyelash Changes: Patients should also be informed of the possibility of eyelash and vellus hair changes in the treated eye during treatment with **LUMIGAN®** 0.01% and 0.03%. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or vellus hairs, and/or direction of eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container: Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice: Patients should also be advised that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician's advice concerning the continued use of **LUMIGAN®** 0.01% and 0.03%.

Use with Contact Lenses: Patients should be advised that **LUMIGAN®** 0.01% and 0.03% contains benzalkonium chloride, which may be absorbed by soft contact lenses. Contact lenses should be removed prior to instillation of **LUMIGAN®** and may be reinserted 15 minutes following its administration.

Use with Other Ophthalmic Drugs: If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes between applications.

APC81RP11 based on 71807US12B.

Rx only

ALLERGAN®

An Ideal Short-term Follow-up Schedule

Generally, our office will use this follow-up schedule with all ortho-k patients.

- *Day 1*. Proper lens fitting as well as insertion and removal training.
- *Day 2*. Instruct the patient to return to the clinic during the morning hours with the lenses inserted. The eye care clinician will observe the lenses for appropriate fitting, remove them, and then refit them as necessary (correcting for topography, aberrometry and manifest refraction). Then, the patient should receive additional insertion and removal training.
- *One week*. Ask the patient to present early in the morning without the lenses and then again later in the afternoon to determine if there is significant regression throughout the day (topography, aberrometry or manifest refraction). Instruct the patient to wear a retainer lens every night.
- *Two weeks*. Have the patient present in the afternoon to measure topography, aberrometry and manifest refraction. Instruct the patient to wear the retainer lens every night, EXCEPT the night before the four-week appointment.
- *Four weeks*. Ask the patient to present in the afternoon for observation as well as to discuss results, progress and the subjective opinion of the patient. Cooperatively determine an appropriate wear schedule.
- *Eight weeks*. Repeat of four-week follow-up.

Elkins Park, Pa., and an external education preceptor for New England College of Optometry in Boston. He has served on the advisory panels for Tracey Technologies, WaveTouch Technologies, Sauflon Pharmaceuticals, VMax Technology, Hydrogel Vision, SynergEyes, CooperVision, Science-Based Health and QSpex, and has participated in sponsored research for Allergan, Alcon, CooperVision, Vistakon and SynergEyes.

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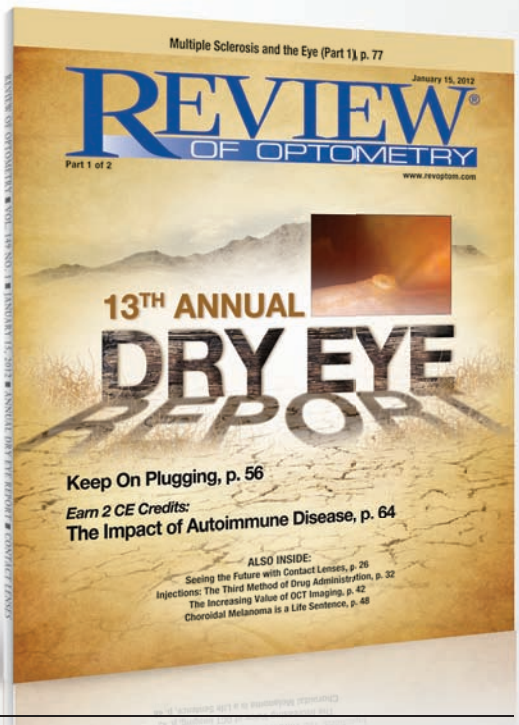

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Monthly Multifocal Pearl

Refitting the Established Monovision Patient

By Robert L. Davis, OD

We're all familiar with the saying, "If it's not broken, don't fix it," but that's not always necessarily the best policy. Take, for example, established monovision contact lens patients. Sure, they might be satisfied with their current comfort and vision, but they also may not realize what they're missing out on by not wearing multifocal contact lenses.

Maybe you're wondering if it's even possible to successfully switch an established presbyope from monovision to multifocal lenses. Based on my personal experience, it can be done—and it is a true practice-builder when you have AIR OPTIX® AQUA Multifocal contact lenses on your side. I'll explain how below.

Fitting established monovision patients into multifocal lenses will help show these patients exactly what they're missing. Monovision begins to fail as the add range increases and the discrepancy between the two eyes becomes too great to ignore the out-of-focus image. Recently, a clinical study comparing the performance of AIR OPTIX® AQUA Multifocal contact lenses to other soft lens correction options (including monovision) was conducted. The study was based on a subjective rating scale of real-world situations such as driving at night, television viewing and reading. The findings showed that study participants preferred AIR OPTIX® AQUA Multifocal lenses over monovision in 15 out of the 16 tests.[1] The rewards of switching a monovision patient to a multifocal lens are many: improved quality of vision throughout the entire visual range, binocular vision, the ability to keep a patient in a multifocal lens even as their ADD increases, no need for an ancillary pair of reading glasses, no need to suppress an eye and depth perception.

The "Holy Grail" of multifocal lens designs is finding the correct ratio of distance optics to near optics and the change from center optics to periphery optics. As more manufacturers discover new approaches to molding different multifocal configurations within the optic zone, multifocal fitting becomes more successful. And disposable options such as AIR OPTIX® AQUA Multifocal contact lenses, which have a center-near bi-aspheric design, have improved the success rate significantly.

IS A REFIT IN YOUR PATIENT'S BEST INTEREST?

First things first, it's important to discuss with patients their expectations and determine whether their current lenses are meeting these objectives. Vision, comfort and wearing time are the parameters that can cause a patient to drop out of contact lenses if their goals are not met. Ocular surface disease is the main culprit that can affect these necessary goals for contact lens success. Also considering the age and reduced tear film stability of this population necessitates being proactive and assessing ocular surface disease to provide therapies to avert these complications.

Two eyes are better than one to provide enhanced binocular full-range vision. Therefore, we should proactively try to switch monovision patients to multifocal lenses to provide an improved visual experience. Remember, patients require three visual ranges: distance, intermediate and near, but they only have two eyes to accomplish the task. With monovision, they have to compromise one of the necessary visual ranges, but with AIR OPTIX® AQUA Multifocal contact lenses, these ranges are embedded into the lens design. With three ADD ranges, we can prescribe AIR OPTIX® AQUA Multifocal lenses for any degree of presbyopia. Simply stated, multifocal lens success is dependent on meeting your patients' expectations—and practitioners need to choose a lens design that accomplishes the visual tasks of each patient.

After the in-depth patient evaluation and affirmation of a patient's needs and expectations, pull a trial lens and place it on the eye to observe fit and quality of vision. Having the patient wear a trial lens for a week helps determine whether the lens design is acceptable or not. When prescribing AIR OPTIX® AQUA Multifocal lenses, first determine the spherical equivalent distance Rx as well as the spectacle add correction. Then, choose the initial ADD (LO, MED, HI), making sure not to over-correct. Allow the lens to settle for 5 to 10 minutes while you take your patient into a real-world setting and check his vision under binocular conditions. Perform a distance over-refraction to determine how much plus he can accept at distance. It is important to maximize plus while maintaining maximum distance clarity. "Pushing plus" at distance will allow you to improve near vision without impairing distance vision. Once that's achieved, check the patient's near acuity with everyday materials, such as a magazine. Pushing plus at distance will improve near vision without impairing distance vision. When distance and near vision are satisfactory, dispense the lenses and have the patient return in 1 to 2 weeks for an evaluation. For comprehensive instructions to help patients experience clear vision at all distances, use the AIR OPTIX® AQUA Multifocal contact lenses fitting guidelines.

THE CHOICE IS CLEAR

Often, the relationship that develops through the multifocal lens fitting process spills over to supplementary spectacle lens purchases and greater referrals. Multifocal lenses not only benefit our patients, but are also a true practice-builder. I have a very high success rate fitting AIR OPTIX® AQUA Multifocal contact lenses. By offering multifocal lenses to patients, you're sending a message that you're staying current with the times. Try re-fitting some of your monovision patients with AIR OPTIX® AQUA Multifocal lenses because what they don't know and find out might hurt you.

Dr. Davis is co-founder of EyeVis Eye and Vision Research Institute. He has developed many contact lens designs and holds various contact lens patents.

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18th Annual Glaucoma Report

New Thoughts on the Newly Diagnosed Glaucoma Patient

First-line glaucoma treatment is typically a prostaglandin. Should we now offer laser trabeculoplasty instead? By Michael Chaglasian, O.D.

The biggest question in glaucoma care is: When do you start treatment?

The next biggest question: Which treatment is right for this patient?

To answer the latter, we have to consider many factors: the class, brand and concentration of medication; the cost; the frequency of dosage; and the patient's ability to comply with the treatment plan. And that only takes medication into account. What about laser therapy?

Remember, of course, that treatment is only a means to an end. Our goal is to save as much vision as possible, within the overall context of the patient's health, quality of life and life expectancy.

To that end, my usual approach for a typical newly diagnosed patient with primary open-angle glaucoma (POAG) is to begin intervention as early as possible in the course of the diagnosis, with a goal of a 30% reduction in intraocular pressure. All treatment should be individualized, of course; but as a general rule, 30% is a good goal to shoot for.

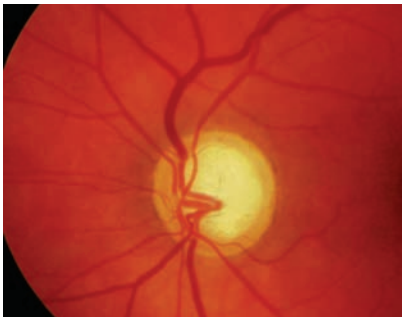
This percentage comes from a literature review of several landmark randomized clinical trials—such as the Ocular Hypertension Treatment Study, Early Manifest Glaucoma Trial, Collaborative Normal-Tension Glaucoma Study and Advanced Glaucoma Intervention Study—as the best initial target to halt and slow down further progression of the disease for a newly diagnosed patient.[1] However, target pressure is a dynamic parameter, so more advanced POAG patients may require a greater (40% to 50%) IOP reduction.

Now, what treatment can best accomplish that 30% reduction? We have some options to consider.

Medical Options

In glaucoma care, medical therapy has been the first-line treatment of choice among U.S. eye doctors because it's a conservative, relatively cost-effective therapy. (Is medicine still the go-to treatment? More on that later.)

In my practice, I place most of my newly diagnosed patients on IOP-lowering drops, typically a prostaglandin. But I also individualize care for my patients to meet our common goals and determine a treatment that is effective, safe, well tolerated and affordable for them.



This newly diagnosed patient has a large optic disc with a thin neuroretinal rim. We started her on a prostaglandin. Would a laser procedure have been preferable?

We have a number of factors to consider just within medical therapy:

• *Convenience.* Convenience may sound inconsequential when weighed against the possibility of losing sight, but we cannot discount its importance. It stands to reason that convenience facilitates compliance, which ultimately helps to preserve vision.

Of course, the most convenient option is a prostaglandin analog, which affords once-a-day therapy along with excellent IOP reduction. On the flip side, a prostaglandin can be a challenge for some patients with arthritis to instill because of the bottle's small size.

• ***Cost.*** As with convenience, cost ties into compliance. Just as patients will extend the wear of their two-week contact lenses to four weeks, they'll stretch out the use of their pricey drops by skipping every other day, or using the drug even less frequently.[2]

No recent studies compare the current costs of these medications; but, just as an example, a quick call to Walgreens (a popular pharmacy chain in my area) found that a 5ml bottle of Lumigan (bimatoprost 0.01%, Allergan) retails for $272, Travatan Z 5ml (travoprost, Alcon) is $235, and Timoptic 5ml (timolol maleate 0.25%, Aton Pharma) is $115. Bear in mind that prices vary depending on the pharmacy.

Cost is less of a concern for patients who have a pharmacy benefit plan. The copayment for a brand-name tier 2 prostaglandin can range from $25 to $45. For a generic tier 1 medication, such as timolol, the copayment is likely to be in the $5 to $15 range. Meanwhile, pharmacies in big-box stores, such as Target, Costco and Walmart, routinely offer generics for just $4. (*See "Give the Option of a Generic," below.*)

• ***Compliance.*** We know that the majority of glaucoma patients, especially newly diagnosed ones, are not compliant with their medicine at least some of the time. As many as two out of three patients who are new to ocular hypotensive therapy have a "substantial gap" in refilling their drops in the first year of therapy.[3]

To make matters worse, even existing patients incorrectly instill their drops anywhere from 66% to 90% of the time.[4,5] Along these lines, non-compliant patients include those with severe arthritis and manual dexterity problems, or with other physical or mental limitations, such as dementia or Alzheimer's, that impede their ability to properly instill an eye drop. For these patients, I consider a treatment that works every day, all day long—laser trabeculoplasty.

Laser Trabeculoplasty

Which should be our current first-line option for the newly diagnosed patient: medical therapy or laser treatment? Our clinical practice guidelines specify that medical treatment has been the traditional approach; however, our protocol recognizes that other treatments, such as laser trabeculoplasty, also offer significant benefits.[6] (Here, I'll use the term laser trabeculoplasty, or LTP, to encompass both selective laser trabeculoplasty and argon laser trabeculoplasty.)

Like medical therapy, LTP requires us to consider factors of convenience, cost and compliance:

• ***Convenience.*** LTP avoids the inconvenience and necessity of instilling glaucoma drops one or more times a day. More than this, by reducing IOP about as effectively as a prostaglandin, LTP is a good strategy for an intervention that will minimize progression.[7]

On the other hand, it's not a slam dunk, neither for effectiveness nor convenience. That's because the procedure's effect diminishes over time (about three to five years), and may require a repeat procedure and/or adjunctive medication.

• ***Cost.*** This is the jackpot question: Which is the most cost-effective method to treat our patients—ongoing medical therapy or essentially a one-time treatment with laser?

Give the Option of a Generic

Many doctors have the impression that generic ophthalmic medications are not manufactured to the same standards as brand-name drugs, and are therefore inferior. But for ophthalmic solutions at least, the generic manufacturing standards must be essentially the same.

"Currently, generic ophthalmic solutions, such as latanoprost, are expected to have the same active and inactive ingredients in the same concentrations. If they are not the same, then a study comparing the clinical bioequivalence has to be performed," explains Wiley Chambers, M.D., the FDA's Deputy Director for the Division of Transplant and Ophthalmology Products.[11]

Generic latanoprost became available in March 2011, and several manufacturers now produce it. In my experience, the price ranges from $30 to more than $70 a bottle. (At the Walgreens near me, it retails for $78.) I often recommend the brand-name medication, but I also offer my patients the option of a generic. I make sure to explain to them that their formulary plan may cover a brand-name product. I tell them I've been using these branded products for many years and I'm very familiar with their safety and efficacy. On the other hand, I explain that the generically available prostaglandin is virtually the same as the brand-name drug. Then I simply ask them their preference.

My patients are split. Some are perfectly comfortable with generics because they take generic systemic medications, so they have no qualms about taking a generic ophthalmic medication. Others just don't believe in a generic; they want the brand-name drug, and nothing else will do.

A recent article attempted to answer just that.[8] Researchers used a mathematical model to compare cost effectiveness of treating newly diagnosed open-angle glaucoma patients with a prostaglandin, LTP or observation only.

Interestingly, they found that prostaglandins and LTP are both cost-effective treatment options for this patient population. However, when patients have "optimal medication adherence," then prostaglandin therapy is a better value than LTP. Yet, as we know, patients are more likely to be noncompliant than compliant with therapy. Given this reality, the researchers concluded, LTP may be a more cost-effective alternative than prostaglandins at current prices.[8] For instance, Medicare reimburses physicians about $336 for LTP. The patient's responsibility is 20% of that, unless they have supplemental insurance. Copayment and other deductibles may also apply.

• ***Compliance.*** LTP essentially removes the issue of compliance. Indeed, it can be an effective solution when patients on medical therapy become noncompliant—especially those patients with physical or mental limitations, as mentioned above, as well as patients on two or more medications.

While compliance can be spotty for a glaucoma patient on one topical medication, it worsens when you add a second drop.[9] So, LTP plays a great role as an adjunctive therapy or second-line treatment option.

Choices at Chairside

So what does all this mean for the patient in your chair?

For me, it means that I'm going to start the vast majority of my newly diagnosed patients on medical therapy. I'm going to offer the patient the option of a generic vs. a branded product. I'm going to discuss compliance, cost and all those "real world" considerations. I'm going to follow them regularly (every three to four months) and continue to ask open-ended questions about how they're doing with their medications—cost and otherwise. Remember that patients who show progression, yet have good "in-office" IOP, might be noncompliant between visits.

And lastly, for patients who would benefit from it, I now know that laser trabeculoplasty is a safe and cost-effective alternative to medical therapy. As the recent study indicates, if my patient is not compliant with a prostaglandin, then I offer the option of LTP.[8]

Occasionally, I offer LTP as a first-line therapy—perhaps in 10% to 15% of newly diagnosed patients. They are patients who do not appear to be compliant

with medical therapy right from the start. (Other glaucoma providers may offer SLT as a first-line option. That's fine, as long as patients are given all the pros and cons of both medical and surgical options. In my opinion, I prefer to begin with a prostaglandin, because it will continue to work for years as long as the patient is reasonably compliant. I usually reserve LTP until I need it.)

What does the future hold? Unfortunately, no breakthrough glaucoma medications are on the horizon; however, investigators are working on longer-lasting drug delivery systems—via contact lenses, punctal plugs, nanoparticles and subconjunctival or intravitreal injections and depots—to overcome the compliance conundrum.

In the meantime, we must continue to talk to our patients about the importance of regular therapy. Right now, about 2.7 million American adults have glaucoma—a 22% increase since 2000—and that number is only going to grow, according to a recent report from Prevent Blindness America.[10] This calls attention to how important it is that we persist in our care of glaucoma patients, and it highlights the opportunity (and the challenge) we have in front of us. ■

Dr. Chaglasian is an associate professor at Illinois College of Optometry and chief of staff of the Illinois Eye Institute, in Chicago.

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18th Annual Glaucoma Report

Optic Neuropathies:

Glaucomatous vs. Non-glaucomatous

While these conditions have overlapping clinical features, distinguishing one from the other is vital to chart the appropriate treatment and follow-up plan.

By Julie K. Hutchinson, O.D., Andrew S. Gurwood, O.D., and Marc D. Myers, O.D.

Glaucomatous optic neuropathy is the most commonly acquired optic neuropathy encountered in clinical practice.[1] While it has clinical features that overlap with non-glaucomatous optic neuropathies—including the presence of vision loss, visual field (VF) loss and optic disc cupping—there are distinct differences in conditions. Non-glaucomatous optic nerve disorders must be differentiated from their glaucomatous brethren because their underlying pathophysiological mechanisms are often part of systemic disease processes that have the potential to impact mortality.

Characteristic signs of irreversible retinal ganglion cell destruction include optic nerve cupping, optic disc sinking, optic disc pallor as well as focal and diffuse retinal nerve fiber layer (RNFL) loss. While these are commonly observed as part of the disease process in glaucoma, they are not exclusive signs of the disease. Other diseases or congenital conditions may exhibit these findings in either the absence of glaucoma or in tandem with it.

Thus, it is crucial for clinicians to determine the context of a discovered optic neuropathy so that they can consider an appropriate work-up that will lead to suitable intervention and follow-up. Accurate and timely management of impending, worsening or developing conditions can only be instituted when the correct diagnosis is uncovered and the underlying causes are revealed.

Glaucomatous Optic Neuropathy

Glaucoma is traditionally defined as a progressive optic neuropathy with accompanying characteristic optic nerve and visual field changes.[1-8] It is classically diagnosed by the presence of a progressive optic nerve cupping with concurrent progressive VF loss.[1-8] Diagnosis is aided by the presence of risk factors such as elevated intraocular pressure (IOP), positive family history, predisposed race, advanced age and thin central corneal thickness.[1-8]

There are three mechanistic theories to explain why glaucomatous

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Goal Statement: Glaucomatous optic neuropathy is the most commonly acquired optic neuropathy encountered in clinical practice. While it has clinical features that overlap with non-glaucomatous optic neuropathies—including the presence of vision loss, visual field (VF) loss and optic disc cupping—there are distinct differences. Thus, it is crucial for clinicians to determine the context of a discovered optic neuropathy so that they can consider an appropriate work-up that will lead to suitable intervention and follow-up.
Faculty/Editorial Board: Julie K. Hutchinson, O.D., Andrew S. Gurwood, O.D., and Marc D. Myers, O.D.
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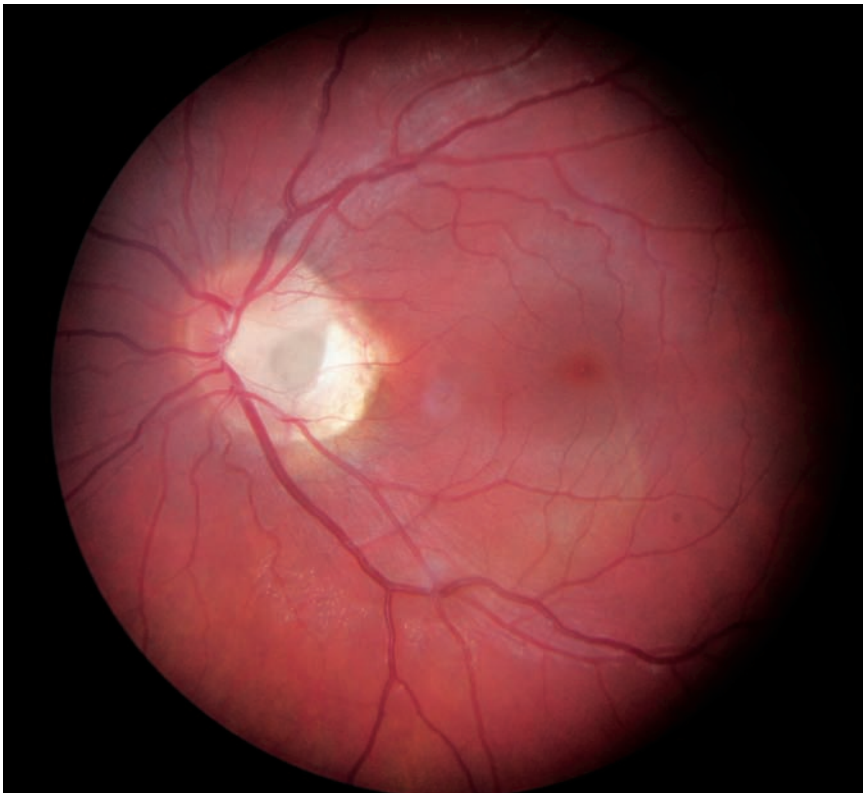
optic neuropathy develops:[9-29]

• ***The pressure-dependent biomechanical mechanism.***[9-14] Here, "high" IOP causes the lamina cribrosa to physically deform and bow, which results in pinching of the axons and supportive tissues, leading to ganglion cell death and tissue remodeling.[30-32] "High" IOP also creates an environment of increased resistance to perfusion, which can produce axonal death by inducing poor axoplasmic and vascular flow.[30-32]

• ***The vascular/autoregulatory mechanism.***[20-26] This process is defined by vascular insufficiency or malfunctioning autoregulatory mechanisms at the level of the optic nerve.[33] Poor perfusion induces ganglion cell or supportive tissue death.[20-26] This mechanism most likely explains why glaucomatous processes are seen at "normal" IOPs.

• ***Linkage to genetic factors that coerce preprogrammed cell death (apoptosis).***[27-30,34-39] The classic clinical finding seen in most glaucoma patients is a level of IOP inconsistent with the health of the optic nerve. While increased IOP has traditionally been associated with the disease process, the literature has lobbied that it be considered a single risk factor among several risk factors, rather than diagnostic for the disease.[40-41]

In diagnosing glaucoma, other clinical features that must be consistently detected with evidence of repeatability and reliability include spared central visual acuity in early-stage disease, spared central visual field with intact color vision in early- and moderate-stage disease, optic disc elongation with disc notching and evidence of RNFL atrophy or dropout providing the anatomic correlation for the non-vertical-respecting visual field loss. Also, an afferent pupillary defect (APD) may be present, depending on the relative asymmetry of optic nerve damage between the two eyes in glaucoma.



Optic pits are depressions in the optic disc that result from herniation of neuroectoderm tissue during development. Although they may occur anywhere on the disc, they often are observed in the temporal quadrant and give the impression of a large disc.

Glaucomatous VF defects traditionally correlate to the focal areas of optic nerve compromise. As the retinal ganglion cell axons undergo alterations, the regions of photoreceptors they subserve lose their connection with the visual pathway, creating the recognizable patterns of functional deficit seen in glaucoma. Because the lamina cribrosa is anatomically weaker in its vertical regions, the nerve tissues that course those locations often succumb to the pathology during the beginning phases, creating pathognomonic patterns in synchrony with the distribution of the affected nerve fibers.[50-56] Early glaucomatous VF defects exhibit classic patterns of loss corresponding to the specific nerve bundles affected. In most cases, the earliest field defects are superior or inferior arcuate defects occurring secondary to loss of the optic nerve's corresponding portions. As a result, the corresponding VF defects present as relative, arcuate nasal scotomas (nasal step and Bjerrum scotoma).

With progressive optic disc damage, an arcuate defect will emerge in the affected eye having no respect for the virtual vertical field dividing line, permitting the loss to arc from the nasal field to the temporal blind spot.[56] The earliest field defects in glaucoma are small, shallow fluctuating scotomas due to highly variable retinal sensitivity, leading early field tests to be noisy and difficult to replicate. Eventually, the field loss becomes persistent in early and mid-level disease.

In most instances of open-angle glaucoma, bilateral, advancing disease inevitably presents as relative

Glaucomatous Optic Neuropathy

Characteristics often noted when observing the glaucomatous optic disc include:[42-49]

- Neuroretinal rim tissue that does not respect the "ISNT" rule.
- Notching of the rim.
- Verticalization of the optic cup.
- An acquired optic pit.
- Baring of a circumlinear vessel.
- Vessel bayoneting at the optic rim (indicating bean-pot cupping).
- Nasalization of vessels.
- Disc hemorrhage (Drance hemorrhage).
- Abnormally large or atypical pattern of peripapillary atrophy (beta zone atrophy).
- Nerve not exhibiting rim pallor.

biarcuate defects. As the disease progresses, the field defects coalesce, creating a "tunnel" field in both eyes.[56] In glaucoma, because the ganglion cell axons lose function gradually as their injury progresses, the measurement of the loss can be variable until the end stage. This, along with peculiarities created by automated testing, creates a scenario where ganglion cells may be taxed in differing patterns, which gives the appearance of VF defects that seemingly come and go or change position from measurement to measurement.

Inflammatory Optic Neuropathy

Inflammatory optic neuropathy may be caused by a number of underlying conditions, including systemic infection, vaccination or autoimmune disease.[57] The most commonly associated cause of inflammatory optic neuropathy is demyelinating optic neuritis secondary to multiple sclerosis.[57,58] A complex cascade of inflammatory events begins peripherally in the body, induces the activation of T lymphocytes, and serves as the peripheral trigger.[59] The activated cells cross the blood-brain barrier and gain access to the central nervous system.[59] Antigens cause the activation of more inflammatory mediators, microglia, lymphocytes and cytokines.[59] The initial inflammatory cascade leads to phagocytosis of the myelin sheath.[60] As a result, fibrotic tissue is laid down in place of missing myelin; as this substitutive tissue lacks the saltatory conduction properties of myelin, the nerve impulse is interrupted.[59-60] As the disease progresses, further demyelination occurs, which exaggerates axonal damage and culminates in retinal ganglion cell death.[60]

Patients with optic neuritis typically present with ocular pain (exaggerated upon eye movement), noticeable changes in color vision in the affected eye and variable unilateral loss of vision, ranging from 20/30 to no light perception.[58,61-64] This is often accompanied by an APD, the magnitude of which depends on the degree of visual disability. Light disturbances in the field of vision known as phosphenes may also be reported during rapid eye movements (saccades). Dyschromatopsia as well as color desaturation and depressed contrast sensitivity are also often noted in these patients.[58,61,62]

Affected eyes present with a depressed field having a variety patterns ranging from cecocentral scotomas to dense central scotomas.[61-66] As the eye naturally recovers over the course of the event, in most cases VF defects resolve as axonal damage becomes limited to localized nerve bundles.[65] Interestingly, patients often exhibit non-specific field deficits in the fellow, unaffected eye.[65]

In patients presenting with acute optic neuritis, the optic nerve's clinical appearance ranges from normal to edematous (papillitis). Only one-third of patients manifest diffuse optic disc edema.[67] Disc hemorrhages are possible but less common in optic neuritis.[67] Segmental or diffuse optic disc pallor represents axonal infarction. Cases that present without the telltale optic disc signs are diagnosed based on the patient's history, demographics (young, typically female, white, age 20 to 40) and the constellation of clinical signs and symptoms. The nomenclature is often referred to as retrobulbar optic neuritis. Demylinating optic neuropathy is confirmed definitively through magnetic resonance imaging (MRI) and cerebrospinal fluid analysis.[1,67,68]

Optic disc cupping and peripapillary tissue remodeling are plausible outcomes of demyelinating optic neuropathy.[68]

The Optic Neuritis Treatment Trial (ONTT), a multicenter study of optic neuritis patients, delineated treatment options.[58,62,69] The study helped to point out that the previous treatment of oral steroids alone

Demyelinating Optic Neuropathy

Demyelinating optic neuropathy can be distinguished from glaucomatous optic neuropathy by these characteristics:

- IOP is typically within normal range.
- VF defects occur in the central or cecocentral portion of the visual field.
- Visual fields often demonstrate a history of improvement correlating to recovery from the acute event.
- Consistent with death of ganglion axons from recurrent episodes, there will often be dyschromatopsia, red color desaturation defects, brightness desaturation defects and an APD that is not consistent with the appearance of the disc's cupping or severity of the visual field.
- Typically asymmetric.
- Age and demographics do not match the demographics of the typical glaucoma patient.

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INDICATIONS AND USAGE

ALPHAGAN® P (brimonidine tartrate ophthalmic solution) 0.1% or 0.15% is an alpha-adrenergic receptor agonist indicated for the reduction of elevated intraocular pressure (IOP) in patients with open-angle glaucoma or ocular hypertension.

IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS

ALPHAGAN® P is contraindicated in neonates and infants (under the age of 2 years) and in patients who have exhibited a hypersensitivity reaction to any component of this medication in the past.

WARNINGS AND PRECAUTIONS

ALPHAGAN® P may potentiate syndromes associated with vascular insufficiency. Use with caution in patients with depression, cerebral or coronary insufficiency, Raynaud's phenomenon, orthostatic hypotension, or thromboangiitis obliterans.

ALPHAGAN® P had minimal effect on blood pressure. Caution should be exercised in treating patients with severe cardiovascular disease.

There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

ADVERSE REACTIONS

ALPHAGAN® P adverse reactions (10% to 20%) included allergic conjunctivitis, conjunctival hyperemia, and eye pruritus. Adverse reactions (5% to 9%) included burning sensation, conjunctival folliculosis, hypertension, ocular allergic reaction, oral dryness, and visual disturbance.

DRUG INTERACTIONS

Antihypertensives/Cardiac Glycosides: Because ALPHAGAN® P may reduce blood pressure, caution in using drugs such as antihypertensives and/or cardiac glycosides with ALPHAGAN® P is advised.

CNS Depressants: Although specific drug interaction studies have not been conducted with ALPHAGAN® P, the possibility of an additive or potentiating effect with CNS depressants (alcohol, barbiturates, opiates, sedatives, or anesthetics) should be considered.

Tricyclic Antidepressants: Tricyclic antidepressants have been reported to blunt the hypotensive effect of systemic clonidine. It is not known whether the concurrent use of these agents with ALPHAGAN® P in humans can lead to resulting interference with the IOP lowering effect. Caution is advised in patients taking tricyclic antidepressants which can affect the metabolism and uptake of circulating amines.

Monoamine Oxidase Inhibitors: Monoamine oxidase (MAO) inhibitors may theoretically interfere with the metabolism of brimonidine and potentially result in an increased systemic side-effect such as hypotension. Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

Please see brief prescribing information on adjacent page.

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Please see **ALPHAGAN® P** package insert for full prescribing information.

INDICATIONS AND USAGE

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CONTRAINDICATIONS

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Hypersensitivity Reactions
ALPHAGAN® P is contraindicated in patients who have exhibited a hypersensitivity reaction to any component of this medication in the past.

WARNINGS AND PRECAUTIONS

Potentiation of Vascular Insufficiency
ALPHAGAN® P may potentiate syndromes associated with vascular insufficiency.

ALPHAGAN® P should be used with caution in patients with depression, cerebral or coronary insufficiency, Raynaud's phenomenon, orthostatic hypotension, or thromboangiitis obliterans.

Severe Cardiovascular Disease
Although brimonidine tartrate ophthalmic solution had minimal effect on the blood pressure of patients in clinical studies, caution should be exercised in treating patients with severe cardiovascular disease.

Contamination of Topical Ophthalmic Products After Use
There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface (see **PATIENT COUNSELING INFORMATION**).

ADVERSE REACTIONS

Clinical Studies Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice.

Adverse reactions occurring in approximately 10-20% of the subjects receiving brimonidine ophthalmic solution (0.1-0.2%) included: allergic conjunctivitis, conjunctival hyperemia, and eye pruritus. Adverse reactions occurring in approximately 5-9% included: burning sensation, conjunctival folliculosis, hypertension, ocular allergic reaction, oral dryness, and visual disturbance.

Adverse reactions occurring in approximately 1-4% of the subjects receiving brimonidine ophthalmic solution (0.1-0.2%) included: abnormal taste, allergic reaction, asthenia, blepharitis, blepharoconjunctivitis, blurred vision, bronchitis, cataract, conjunctival edema, conjunctival hemorrhage, conjunctivitis, cough, dizziness, dyspepsia, dyspnea, epiphora, eye discharge, eye dryness, eye irritation, eye pain, eyelid edema, eyelid erythema, fatigue, flu syndrome, follicular conjunctivitis, foreign body sensation, gastrointestinal disorder, headache, hypercholesterolemia, hypotension, infection (primarily colds and respiratory infections), insomnia, keratitis, lid disorder, pharyngitis, photophobia, rash, rhinitis, sinus infection, sinusitis, somnolence, stinging, superficial punctate keratopathy, tearing, visual field defect, vitreous detachment, vitreous disorder, vitreous floaters, and worsened visual acuity.

The following reactions were reported in less than 1% of subjects: corneal erosion, hordeolum, nasal dryness, and taste perversion.

Postmarketing Experience
The following reactions have been identified during postmarketing use of brimonidine tartrate ophthalmic solutions in clinical practice. Because they are reported voluntarily from a population of unknown size, estimates of frequency cannot be made. The reactions, which have been chosen for inclusion due to either their seriousness, frequency of reporting, possible causal connection to brimonidine tartrate ophthalmic solutions, or a combination of these factors, include: bradycardia, depression, hypersensitivity, iritis, keratoconjunctivitis sicca, miosis, nausea, skin reactions (including erythema, eyelid pruritus, rash, and vasodilation), syncope, and tachycardia. Apnea, bradycardia, coma, hypotension, hypothermia, hypotonia, lethargy, pallor, respiratory depression, and somnolence have been reported in infants receiving brimonidine tartrate ophthalmic solutions.

DRUG INTERACTIONS

Antihypertensives/Cardiac Glycosides
Because **ALPHAGAN® P** may reduce blood pressure, caution in using drugs such as antihypertensives and/or cardiac glycosides with **ALPHAGAN® P** is advised.

CNS Depressants
Although specific drug interaction studies have not been conducted with **ALPHAGAN® P**, the possibility of an additive or potentiating effect with CNS depressants (alcohol, barbiturates, opiates, sedatives, or anesthetics) should be considered.

Tricyclic Antidepressants
Tricyclic antidepressants have been reported to blunt the hypotensive effect of systemic clonidine. It is not known whether the concurrent use of these agents with **ALPHAGAN® P** in humans can lead to resulting interference with the IOP lowering effect. Caution is advised in patients taking tricyclic antidepressants which can affect the metabolism and uptake of circulating amines.

Monoamine Oxidase Inhibitors
Monoamine oxidase (MAO) inhibitors may theoretically interfere with the metabolism of brimonidine and potentially result in an increased systemic side-effect such as hypotension. Caution is advised in patients taking MAO inhibitors which can affect the metabolism and uptake of circulating amines.

USE IN SPECIFIC POPULATIONS

Pregnancy
Pregnancy Category B: Teratogenicity studies have been performed in animals.

Brimonidine tartrate was not teratogenic when given orally during gestation days 6 through 15 in rats and days 6 through 18 in rabbits. The highest doses of brimonidine tartrate in rats (2.5 mg /kg/day) and rabbits (5.0 mg/kg/day) achieved AUC exposure values 360- and 20-fold higher, or 260- and 15-fold higher, respectively, than similar values estimated in humans treated with **ALPHAGAN® P** 0.1% or 0.15%, 1 drop in both eyes three times daily.

There are no adequate and well-controlled studies in pregnant women; however, in animal studies, brimonidine crossed the placenta and entered into the fetal circulation to a limited extent. Because animal reproduction studies are not always predictive of human response, **ALPHAGAN® P** should be used during pregnancy only if the potential benefit to the mother justifies the potential risk to the fetus.

Nursing Mothers
It is not known whether brimonidine tartrate is excreted in human milk, although in animal studies, brimonidine tartrate has been shown to be excreted in breast milk. Because of the potential for serious adverse reactions from **ALPHAGAN® P** in nursing infants, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

Pediatric Use
ALPHAGAN® P is contraindicated in children under the age of 2 years (see **CONTRAINDICATIONS**). During postmarketing surveillance, apnea, bradycardia, coma, hypotension, hypothermia, hypotonia, lethargy, pallor, respiratory depression, and somnolence have been reported in infants receiving brimonidine. The safety and effectiveness of brimonidine tartrate have not been studied in children below the age of 2 years.

In a well-controlled clinical study conducted in pediatric glaucoma patients (ages 2 to 7 years) the most commonly observed adverse reactions with brimonidine tartrate ophthalmic solution 0.2% dosed three times daily were somnolence (50-83% in patients ages 2 to 6 years) and decreased alertness. In pediatric patients 7 years of age (>20 kg), somnolence appears to occur less frequently (25%). Approximately 16% of patients on brimonidine tartrate ophthalmic solution discontinued from the study due to somnolence.

Geriatric Use
No overall differences in safety or effectiveness have been observed between elderly and other adult patients.

Special Populations
ALPHAGAN® P has not been studied in patients with hepatic impairment.

ALPHAGAN® P has not been studied in patients with renal impairment. The effect of dialysis on brimonidine pharmacokinetics in patients with renal failure is not known.

OVERDOSAGE

Very limited information exists on accidental ingestion of brimonidine in adults; the only adverse reaction reported to date has been hypotension. Symptoms of brimonidine overdose have been reported in neonates, infants, and children receiving **ALPHAGAN® P** as part of medical treatment of congenital glaucoma or by accidental oral ingestion (see **USE IN SPECIFIC POPULATIONS**). Treatment of an oral overdose includes supportive and symptomatic therapy; a patent airway should be maintained.

NONCLINICAL TOXICOLOGY

Carcinogenesis, Mutagenesis, Impairment of Fertility
No compound-related carcinogenic effects were observed in either mice or rats following a 21-month and 24-month study, respectively. In these studies, dietary administration of brimonidine tartrate at doses up to 2.5 mg/kg/day in mice and 1 mg/kg/day in rats achieved 150 and 120 times or 90 and 80 times, respectively, the plasma C_{max} drug concentration in humans treated with one drop of **ALPHAGAN® P** 0.1% or 0.15% into both eyes 3 times per day, the recommended daily human dose.

Brimonidine tartrate was not mutagenic or clastogenic in a series of in vitro and in vivo studies including the Ames bacterial reversion test, chromosomal aberration assay in Chinese Hamster Ovary (CHO) cells, and three in vivo studies in CD-1 mice: a host-mediated assay, cytogenetic study, and dominant lethal assay.

Reproduction and fertility studies in rats with brimonidine tartrate demonstrated no adverse effect on male or female fertility at doses which achieve up to approximately 125 and 90 times the systemic exposure following the maximum recommended human ophthalmic dose of **ALPHAGAN® P** 0.1% or 0.15%, respectively.

PATIENT COUNSELING INFORMATION

Patients should be instructed that ocular solutions, if handled improperly or if the tip of the dispensing container contacts the eye or surrounding structures, can become contaminated by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions (see **WARNINGS AND PRECAUTIONS**). Always replace the cap after using. If solution changes color or becomes cloudy, do not use. Do not use the product after the expiration date marked on the bottle.

Patients also should be advised that if they have ocular surgery or develop an intercurrent ocular condition (e.g., trauma or infection), they should immediately seek their physician's advice concerning the continued use of the present multidose container.

If more than one topical ophthalmic drug is being used, the drugs should be administered at least five minutes apart.

As with other similar medications, **ALPHAGAN® P** may cause fatigue and/or drowsiness in some patients. Patients who engage in hazardous activities should be cautioned of the potential for a decrease in mental alertness.

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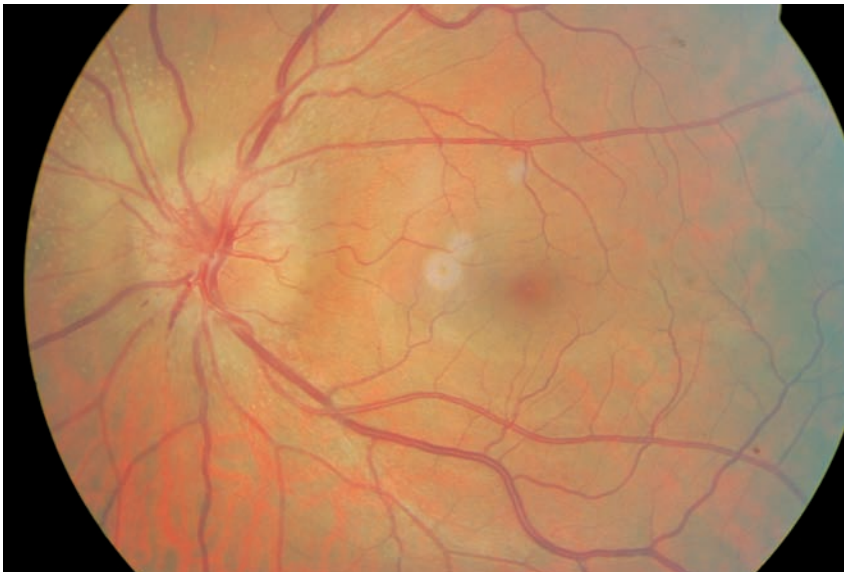
was contraindicated, recommending intravenous methylprednisone, administered over the course of three days followed by an 11-day course of oral prednisone.[58,62,69] The study also concluded that monitoring—while leading to increased recovery time—was essentially equivalent to the intervention arm of the study.[58,62,69]

Compressive Optic Neuropathy

Compressive optic neuropathy results from mechanical mass effect secondary to tumors and non-neoplastic lesions (i.e., retrobulbar hemorrhage, aneurysm, mucocele, orbital apex syndrome), which impinge on intraorbital or intracranial structures of the visual pathway.[70-71] Intracranial tumors such as sphenoid wing meningioma, pituitary adenoma, craniopharyngioma, meningioma and masses secondary to metastases to the orbit or anterior intracranial cavity may cause unilateral or bilateral optic nerve compression. Optic nerve tumors, such as optic nerve sheath meningioma and optic nerve glioma, also may result in unilateral or bilateral optic nerve compromise.[70-71]

Patients who develop compressive optic neuropathy have no specific demographic predilection to age or gender. In the case of traumatic orbital apex syndrome or traumatic retrobulbar hemorrhage, a provocative mechanical force will be uncovered in the history. In the case of space-occupying lesions (SOLs), history reveals a slowly progressive process with changes or fluctuations in visual acuity or missing visual field that occur over several months to years.[70-72] In cases where there is sudden expansion of an SOL, acute changes in field and acuity may occur as a result of novel vascular compression causing sudden infarction of the optic nerve.



NAION is caused by a lack of optic nerve perfusion or embolic disease that affects the arteries supplying the optic nerve. Fundoscopic examination in the acute phase of cases reveals mild to severe optic nerve swelling.

Along with acuity changes, patients also may report changes in color vision and/or diplopia. Double vision results from interruption of any of the cranial nerves innervating the extraocular muscles (CN III, IV, VI). Compression may occur at the orbital apex, in the cavernous sinus or via mechanical restriction of the extraocular muscles (with or without proptosis) secondary to orbital tumor expansion or thyroid myopathy.[70,71]

VF defects associated with compressive lesions vary depending on their location.[70,71] Retrobulbar

Compressive Optic Neuropathy

Compressive optic neuropathy can be distinguished from glaucomatous optic neuropathy by these characteristics:

- Acute vision loss secondary compressive optic neuropathy has the potential to be marked (20/100 or worse).
- IOP typically is within normal range unless altered by an intraorbital mass (mass effect).
- In cases where the mass effect evolves inside the orbit, there may be proptosis with poor retropulsion, lid retraction, EOM restriction or choroidal folds.
- VF defects occur in the central or cecocentral portion of the visual field.
- The visual fields often demonstrate a steep depth with respect to virtual vertical hemianopic line.
- Compressive lesions often induce disc pallor.
- Dyschromatopsia, red color desaturation defects, brightness desaturation defects and an APD that is inconsistent with the appearance of the disc's cupping or visual field severity.
- Unilateral compressive optic neuropathy is asymmetric compared to open-angle glaucoma and lesions evolving from the chiasm or behind induce congruous or incongruous homonymous VF defects that respect the neuroanatomical architecture (quadrant or hemianopic defects).
- The age and demographics of compressive optic neuropathy often do not match the demographics of the typical glaucoma patient.

optic nerve compression commonly produces central, cecocentral and paracentral defects. Compression in the optic chiasm region can produce junctional (the affected eye has a deep central scotoma with an afferent defect and the fellow eye has a temporal defect) and bitemporal hemianopsia. Optic tract lesions produce incongruous homonymous hemianopias.[66,70,71] In cases of unilateral or asymmetric optic nerve compression, an afferent defect may be noted. Orbital and eyelid signs may include proptosis, resistance to retropulsion, enophthalmos, ptosis or lid retraction.

The optic disc's appearance may vary greatly depending on the lesion's magnitude.[70,71] Note that early in the compressive disease process, the optic disc may appear normal. Other optic disc findings may include atrophic changes (most often due to chronic compression), optic disc edema and the presence of optociliary shunt vessels.[70,71]

Optic disc cupping and tissue remodeling are a plausible outcome of compressive optic neuropathy.[73,74]

Accurately diagnosing and promptly treating the underlying cause of the compression is vital to preserve the patient's vision and medical health. Surgical and medical treatments directed at managing the underlying etiology may result in recovery of acuity, fields and symptoms.[70,71]

Anterior Ischemic Optic Neuropathy

Aside from glaucoma, anterior ischemic optic neuropathy (AION) is the most common cause of optic nerve-related permanent vision loss in adults.[75-79] The two forms of anterior ischemic optic neuropathy are non-arteritic (NAION) and arteritic (AAION). The main cause of AAION is infarction of the short posterior ciliary arteries due to giant cell arteritis (GCA), when large multinucleated monocytes infiltrate the small- and medium-sized arteries, causing obliteration of their lumen.[75-80] Some research suggests that transient hypoperfusion or non-perfusion of the optic nerve is the predominant cause of NAION, while atherosclerosis along with other systemic diseases like diabetes mellitus or hypertension are merely risk factors.[33,75-79,107-108]

AION patients typically are more than 40 years of age, and the condition does not demonstrate a particular bias toward gender (slight predilection for males over females). Patients present with sudden unilateral visual acuity and/or visual field loss that occurred upon awakening. Patients may present with scalp or jaw pain, which may be interpreted by the patient as eye pain or may result in eye pain that is referred. Depending upon the anatomical involvement (cranial nerves III, IV, VI), diplopia is possible; however, global systemic neurologic symptoms are absent.[75-80]

Key diagnostic signs include acuity that may range from normal to variably decreased, variably altered color vision, presence of a relative APD in the setting of no proptosis, no ptosis, normal ocular motilities, and normal corneal and facial sensation.[75-80]

Fundoscopic examination in the acute phase of AION cases reveals mild to severe optic nerve swelling. Depending on the timing of the observation, the disc may appear hyperemic or pale, with sectorial findings in less severe cases and diffuse characteristics in a more severely damaged papilla. Retinal hemorrhages may be present at the optic disc margins with cotton-wool spots suggesting an arteritic etiology.[75-80] The uninvolved eye commonly presents with a "crowded" optic nerve head with an absent or small cup.[81,82] Medical history may include one or more vasculopathic risk factors such as hypertension, hypercholesterol-

Anterior Ischemic Optic Neuropathy

AION can be distinguished from glaucomatous optic neuropathy by these characteristics:

- IOP typically is either within normal range or lower normal range secondary to reduced aqueous production because of poor perfusion to the ciliary body.
- Optic disc cupping seen in AAION does not involve the peripapillary zones.
- VF defects seen in ischemic optic neuropathy occur in the central or cecocentral visual field and usually present with a complete altitudinal pattern respecting the virtual horizontal meridian because of a propensity to be sectorial rather than diffusely damaging.
- Visual fields in AION cases often present attitudinally and can easily mimic the arcuate defects seen in glaucoma. The telltale signs of AION fields are that they often demonstrate a steep depth, they are very repeatable and consistent, they do not worsen or improve, and because of the anatomy they affect, they have a distinct respect for the horizontal meridian.
- Ischemic optic neuropathy produces disc pallor.
- The cupping in AAION exhibits less cup volume and less cup depth compared to that seen in open-angle glaucoma upon scanning laser ophthalmoscopy evaluation.[84]
- Ischemic optic neuropathy often produces dyschromatopsia, red color desaturation defects, brightness desaturation defects and an APD immediately after its detection that is not consistent with the disc's cupping or visual field severity.
- Unilateral ischemic optic neuropathy is asymmetric compared to open-angle glaucoma and in many cases presents with some form of head pain (eye pain, jaw claudication, earache, scalp tenderness, etc.) and possibly diplopia.

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emia, diabetes or smoking.[75-79]

Optic disc cupping and tissue remodeling are a plausible outcome of ischemic optic neuropathy.[83-86]

Acute management for any suspected ischemic optic neuropathy includes prompt laboratory and diagnostic studies (erythrocyte sedimentation rate, C-reactive protein, and platelets) to not only differentiate AAION from NAION, but to identify systemic etiologies that warrant immediate medical intervention.[75-80]

The initial treatment for AAION includes high-dose (80mg to 100mg p.o. q.d.) prednisone or hospitalization to begin intravenous methylprednisolone followed by a course of oral prednisone.

There is no one perfect test for diagnosing GCA. The laboratory work-up includes complete blood count with differential and platelets, liver function studies, erythrocyte sedimentation rate and C-reactive protein. Suspicious results are followed up with temporal artery biopsy (TAB), while management is immediately initiated.[75,76,78,79]

While the TAB is not foolproof (false positives and false negatives occur), the constellation of signs and symptoms dictate the treatment protocols. Occult GCA is subtler, because its eye problems occur in the absence of systemic signs and symptoms. Expert evaluation by neurology or neuro-ophthalmology is critical.

When there is no suspicion of GCA, the initial treatment of NAION should focus on medical management of potential vasculopathic etiologies. The main goal of therapy is to control the suspected disease process and to ultimately protect the unaffected eye and reduce the risk of cerebrovascular accident and myocardial infarction.[75-79]

In cases of AAION, recovery of visual acuity and/or visual field is rare.[75-80] There are reports describing NAION patients who recover some visual acuity and some visual field; however, the recovery is often small and not approaching the pre-loss function.[75]

In both AAION and NAION, the disease's natural course over several months brings about resolution of the disc swelling, eventual resultant disc pallor with the potential for papillary disc and tissue remodeling that results in what appears to be disc cupping.[75-79]

Infiltrative Optic Neuropathy

Infiltrative optic neuropathy can result from systemic infection, systemic lupus erythematosus, blood-borne cancers and metastatic disease; however, it is most commonly found in association with sarcoidosis.[87-97]

While neuroimaging may be used to confirm the presence of inflammation or an SOL, it is not specific in cases of infiltrative optic neuropathy. Infiltration can be difficult to confirm without biopsy and the ability to directly observe the histological characteristics of the pathological cells.[87-92]

The sequence of events that occurs in optic nerve infiltration varies. Generally, it is believed that the bloodstream carries pathologic cells to the meninges via the perivascular and subarachnoid space.[93] Once present in meningeal tissue, these cells have the capability to invade the cranial nerves.[89,93] Infiltration of optic nerve tissue along with accompanying impairment to its blood supply lead to infarction and loss of axons.[93]

The demographics of infiltrative neuropathy varies widely by age, race, gender and is subject to the particular demographics of underlying disease.[87-97] Clinically, these patients often experience a reduction in visual acuity, color vision and contrast sensitivity that is typically painless, unilateral or asymmetric and variable in its nadir.[87-97] An APD is often present.[88,91,94] Associated ocular inflammation (iritis, vitritis, pars planitis, intermediate uveitis) can be present depending on the etiology.[94] The optic disc is often diffusely edematous.[87-97]

In cases producing granulomatous infiltration, such as sarcoidosis, the optic nerve may accumulate numerous discrete granulomata, creating a cauliflower-like appearance.[88,94] However, because infiltrative events may occur anywhere along the length of the optic nerve, it is not always possible to distinguish

Infiltrative Optic Neuropathy

Infiltrative optic neuropathy can be distinguished from glaucomatous optic neuropathy by these characteristics:

- IOP typically is within normal range, barring the presence of an accompanying anterior chamber inflammatory event that might produce a secondary open-angle uveitic glaucoma in the acute stages or secondary synechial angle closure glaucoma secondary to chronic disease.
- VF defects vary widely and are interpreted as diffuse, lacking the classic pattern seen in primary open-angle glaucoma.
- Visual fields can improve with pharmaceutical intervention.
- Consistent with optic nerve infarction, there will often be dyschromatopsia, red color desaturation defects, brightness desaturation defects and an APD.
- Infiltrative optic neuropathy is often unilateral with a unilateral diffusely edematous optic disc in the acute stages that inevitably becomes pale over time.
- Discs and nerves with significant infiltration may create interrupted venous egress, causing the filling of optociliary shunt vessels, a clinical sign of pathology independent of glaucoma.
- The patient's age and demographics do not match the typical glaucoma patient.

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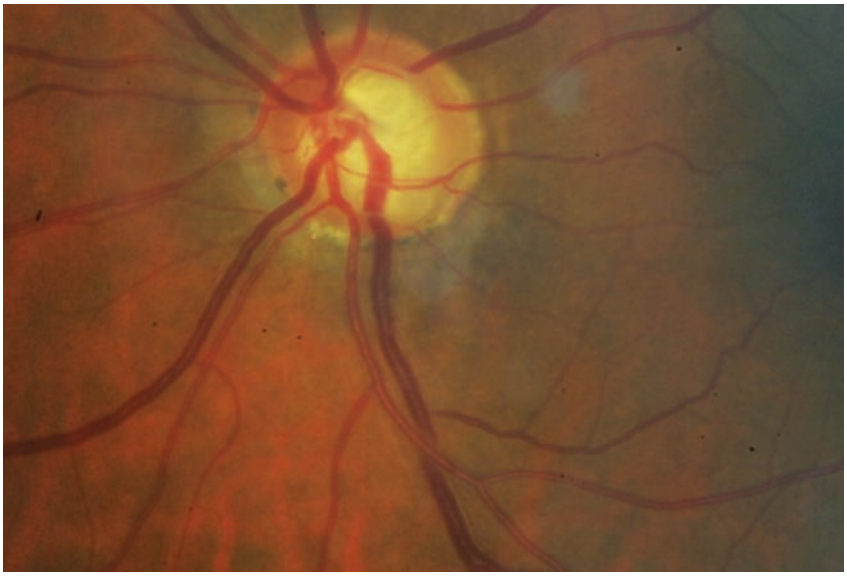
infiltrative etiology based on disc observation alone.

Optociliary shunts may develop at the optic disc to serve as a second drainage route in cases where infiltrative, compressive or neoplasmic compression reduces ocular vascular egress.[88] The presence of optociliary shunts predicts a poor visual outcome and definitively suggests a pathological process independent of glaucoma.[88]

The pattern of VF defects seen in these cases is variable, ranging from diffuse, non-specific defects to amaurosis.[89,91,94] Visual field improvement is possible in some cases with timely therapeutic intervention.[89,91]



Glaucoma traditionally is defined as a progressive optic neuropathy with accompanying characteristic optic nerve and VF changes. It is classically diagnosed by the presence of a progressive optic nerve cupping with concurrent progressive VF loss.

Congenital Optic Disc Anomalies

Congenital optic nerve anomalies can appear pseudo-glaucomatous. Congenital anomalies that may mimic glaucomatous disease include megalopapillae, tilted discs and optic pit. Each of these entities has distinguishing characteristics that allow differentiation from glaucomatous nerves.

Megalopapilla is similar to its glaucomatous disc counterpart in that it is defined as having a disc volume greater than 2.5mm^2 with increased cupping.[98,99] Two variants of megalopapillae exist:[99]

- Type 1, which is bilateral with large round or oval cupping where the rim tissue obeys the ISNT rule but may appear paler than the average healthy nerve.
- Type 2, which is unilateral with a displaced round cup.

What distinguishes megalopapilla from optic discs that incur cupping from glaucomatous optic neuropathy is that megalopapilla discs display a normal visual field (some may exhibit an enlarged blind spot), have a normal rim area, have a normal cup volume and have a normal RNFL, as measured by scanning laser technology.[99,100] It should be noted that skewed findings can result from interpretation of scanning laser topography, as the Moorfields regression analysis on the Heidelberg Retinal Tomograph is calibrated to discs ranging from 1.2 mm^2 to 2.8mm^2.[99]

Tilted optic discs, colobomatous discs and discs exhibiting conus from axial myopia can also mimic glaucomatous discs. Scanning laser ophthalmoscopy-optical coherence tomography (OCT) has shown tilted discs to have thicker-than-average temporal nerve fiber layer tissue.[101,102] Interestingly, there is no actual rotation of the disc in tilted disc cases—the name is a misnomer. The tilted nature appears secondary to abnormal scleral canal morphology where there is an absence of fibers inferiorly and an excess of fibers superiorly.[103]

Additionally, due to the optic disc's counterclockwise rotation, the superior nerve fiber layer tissue's peak thickness appears to be dislocated temporally.[101-102] Helpful clues to discriminate myopic conus discs from glaucomatous discs include high myopia and increased axial length (detectable on A-scan echography).[104]

Optic disc colobomas also can mimic glaucomatous optic neuropathy. They appear as enlarged, excavated discs with reduced nerve fiber volume in the vertical pole where the coloboma is present.[100] A distinguishing characteristic of disc coloboma that can help differentiate it from a glaucomatous disc is that there may be other colobomas present in the iris or lens. Involvement of neighboring tissues (including the choroid or retina) and the presence of microphthalmia are also indicators of coloboma.[100]

Disc cupping with a concomitant presentation of macular retinoschisis or serous macular retinal detachment is inconsistent with glaucomatous disease.[105] Records indicating the presence of this anomaly from birth should provide definitive proof that the unusual appearance is not secondary to glaucomatous processes.

Optic pits are depressions in the

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Congenital Optic Disc Anomalies

These characteristics distinguish congenital optic disc anomalies from glaucomatous optic neuropathy:

- IOP typically is within normal range.
- VF defects vary widely and may mimic those seen in glaucoma; however, they are often deep defects that neither show improvement or worsening over time and are consistently repeatable.
- Visual fields (as well as the disc appearance), for the most part, remain consistent over time, unless a clinical complication (retinoschisis, serous retinal detachment) develops.
- Anomalies do not have a specific demographic predilection.

optic disc that result from herniation of neuroectoderm tissue during development.[100] They may occur anywhere on the disc but are often observed in the optic disc's temporal quadrant and give the impression of a large disc.[100]

Optic pits are associated with variable visual field defects. Associated paracentral arcuate deficits can mimic those seen in glaucoma.[100] Optic pits increase the risk of vision loss from complications, which include macular schisis or serous retinal detachment.[100,106]

They can be differentiated from the glaucomatous cupping process by their location, depth, lack of change over time, and associated complications that are inconsistent with glaucomatous disease, such as retinoschisis and serous macular detachment.

It is imperative to note that the presence of an optic disc anomaly does not preclude the development of glaucomatous disease. Reliable and repeatable changes detected in diagnostic testing may indicate the development of an optic neuropathy.

Differentiating non-glaucomatous optic nerve disorders from glaucomatous disease can save significant time and money that would have been spent managing a condition that is neither there nor developing. It can also save many lives. In cases where optic disc changes evolve secondary to non-glaucomatous processes, pathophysiological mechanisms may be budding, producing consequences that are systemic with the potential to impact other organ systems or mortality. ■

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OSC QUIZ

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1. What is the most commonly acquired optic neuropathy in practice?
a. Ischemic optic neuropathy.
b. Demyelinating (multiple sclerosis) optic neuropathy.
c. Glaucomatous optic neuropathy.
d. Compressive optic neuropathy.

2. Which of the following is not a characteristic finding of glaucoma?
a. Focal nerve fiber layer defects.
b. Optic disc cupping.
c. Optic disc pallor.
d. Optic disc notching.

3. Which is considered to be the least important risk factor associated with developing glaucoma?
a. White race.
b. Elevated intraocular pressure.
c. Thinner central corneal thickness.
d. Aunt with glaucoma.

4. Which is considered a possible mechanistic theory to explain glaucomatous optic neuropathy?
a. Apoptosis.
b. Physical deformation.
c. Increased blood flow.
d. Vasospasm.

5. Glaucomatous discs may have all of the following characteristics except:
a. Bayonetting.
b. Alpha zone atrophy.
c. Drance hemorrhage.
d. Verticalization.

6. Which of these tests may help differentiate glaucomatous from non-glaucomatous optic neuropathy?
a. Automated visual field testing.
b. Color testing.
c. Confrontation field testing.
d. Pupil testing.

7. What is the ONTT-recommended treatment protocol for inflammatory optic neuropathy associated with multiple sclerosis?
a. Oral prednisone only.
b. IV methylprednisone only.
c. Oral prednisone, then IV methylprednisone.
d. IV methylprednisone, then oral prednisone.

8. Retrobulbar optic nerve compression may manifest with which of the following visual field defects:
a. Cecocentral, nasal, paracentral.
b. Nasal, central, paracentral.
c. Cecocentral, central, paracentral.
d. Cecocentral, nasal, central, paracentral.

9. Which best characterizes the typical non-arteritic anterior ischemic optic neuropathy (NAION) patient?
a. 40-year-old female with 0.05/0.05 CDR.
b. 40-year-old male with 0.05/0.05 CDR.
c. 70-year-old female with 0.1/0.1 CDR.
d. 65-year-old male with 0.1/0.1 CDR.

10. All of the following lab tests are appropriate to rule out arteritic ischemic optic neuropathy except:
a. Platelets.
b. C-reactive protein (CRP).
c. Angiotensin-converting enzyme (ACE).
d. Erythrocyte sedimentation rate (ESR).

11. What is the most common underlying etiology of infiltrative optic neuropathy?
a. Leukemia.
b. Lymphoma.
c. Sarcoidosis.
d. Breast cancer.

12. Which of the following may be true about a patient with optociliary shunts?
a. Infiltrative etiology with a good prognosis.
b. Glaucomatous etiology with a poor prognosis.
c. Compressive etiology with a good prognosis.
d. Neoplastic etiology with a poor prognosis.

13. Which optic nerve characteristics may help distinguish glaucoma from megalopapilla?
a. Disc area of 2.5mm^2.
b. Disc area of 2.9mm^2.
c. Large round cupping.
d. Large oval cupping.

14. The presence of which additional clinical finding might aid in discriminating a congenital anomaly from glaucomatous optic neuropathy?
a. Lens coloboma.
b. Optociliary shunts.
c. Collateral vessels.
d. Nerve fiber layer dropout.

15. What is a possible mechanism to explain a patient with optic neuropathy and diplopia, both the result of the same underlying etiology?
a. Compression of cranial nerve II.
b. Infiltration of cranial nerve III.
c. Compression of cranial nerve IV.
d. Vascular insufficiency of cranial nerve VI.

16. A 35-year-old female patient presents with sudden onset of pain and vision loss with a well-perfused optic nerve with distinct margins. Of the following choices, which is most appropriate diagnosis?
a. Posterior optic neuritis.
b. Anterior optic neuritis.
c. Retinal detachment.
d. Malingering.

OSC QUIZ

17. Which visual field defect is most consistent with advanced glaucoma?
a. Biarcuate.
b. Dense nasal step.
c. Altitudinal.
d. Central scotoma.

18. Suspected ischemic optic neuropathy must trigger a workup to rule out what life-threatening disease?
a. Multiple sclerosis.
b. Elevated blood pressure.
c. Giant cell arteritis.
d. Viagra use.

19. Which is not a feature consistent with infiltrative optic neuropathy?
a. Granulomata.
b. Vitritis.
c. Optociliary shunts.
d. Oligoclonal IgG bands.

20. What is a documented sight-threatening complication of optic pit?
a. It increases the risk of glaucomatous damage.
b. It is associated with an increased risk of retinal detachment.
c. It changes rapidly.
d. It most often occurs inferiorly and can mimic cupping.

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7.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
8.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
9.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
10.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
11.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
12.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
13.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
14.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
15.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
16.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
17.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
18.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
19.	Ⓐ	Ⓑ	Ⓒ	Ⓓ
20.	Ⓐ	Ⓑ	Ⓒ	Ⓓ

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The Finer Points of Thygeson's

What are the latest treatment options for a patient with Thygeson's superficial punctate keratitis? An antiviral, perhaps? **Edited by Paul C. Ajamian, O.D.**

Q A 27-year-old white female presented with a two-week history of irritation and foreign body sensation in the left eye. But the slit lamp appearance shows that the eye is white and quiet. What am I dealing with and how should I treat it? Is comanagement with a corneal specialist necessary?

A "Based on the symptoms and clinical appearance, this patient most likely has Thygeson's superficial punctate keratitis (TSPK)," says Sheila Morris, O.D., currently a resident at Omni Eye Center, in Atlanta.

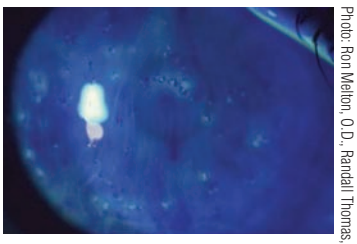

Photo: Ron Melton, O.D., Randall Thomas, O.D.

Thygeson's SPK is notable for small, central epithelial opacities.

As with this patient's presentation, most individuals with TSPK have a history of irritation, foreign body sensation, photophobia and tearing, Dr. Morris says. These symptoms are typically bilateral and asymmetric, but may be unilateral, as it was in this patient.

Slit lamp examination reveals a white, quiet eye with small, central, corneal epithelial opacities, she says. TSPK lesions are round, granular, white-gray, and stain faintly with fluorescein. A subepithelial haze may be present under these opacities, while the stroma and endothelium are uninvolved.

Differential diagnoses include:

• ***Herpes simplex virus (HSV) keratitis.*** "HSV patients typically have a single, red, painful eye, with either a diffuse mild epithelial haze or the standard dendritic ulceration," Dr. Morris says. "Corneal sensitivity may be reduced in HSV, but not in TSPK."

• ***Sterile corneal infiltrates.*** "Sterile infiltrates are more likely peripheral, and have an intact epithelium," she says.

• ***Standard SPK.*** "The lesions found in standard SPK are much smaller, more diffuse and stain brightly with fluorescein," she says.

No specific etiology of TSPK has yet been found. "A viral cause has been postulated, but PCR studies have shown no viral involvement," Dr. Morris says.[1] "An immunological component has also been theorized because of TSPK's clinical response to steroids."

Studies have shown a possible genetic link to the HLA-DR3 antigen, which is also associated with Graves' disease, multiple sclerosis and celiac disease.[2] This suggests that the antigen could be affecting the immune status of patients with TSPK.

TSPK usually responds to treatment, but with the disease's many remissions and exacerbations, it can be an extended, time-consuming process. For mild cases, frequent use of artificial tears may relieve symptoms. But many patients require a mild topical steroid, dosed q.i.d. A gradual taper over weeks to months is necessary to prevent immediate recurrence. If these measures don't resolve the problem, try a stronger steroid q.i.d. Monitor patients on steroids every week or every other week to check IOP.

The next step is to try a bandage soft contact lens. "Referral to a corneal specialist is only required in severe cases," Dr. Morris says.

If studies determine that TSPK does indeed have a viral etiology, then topical antivirals would likely help resolve the signs and symptoms.[3] "We did in fact start this patient on Zirgan (ganciclovir gel, Bausch + Lomb) five times a day" Dr. Morris says. "After two weeks, her corneal epithelium was almost completely healed with only a few faint areas of haze remaining. We decreased Zirgan to t.i.d. and added Pred Forte (prednisolone acetate 1%, Allergan) q.i.d., and will see her again in two weeks. Zirgan has been known anecdotally to have an effect on adenoviral conjunctivitis, so it will be interesting to see what happens as we taper it."

Lastly, educate patients that TSPK is a recurrent disease that may reoccur after several months to many years. "Most importantly, reassure patients that TSPK usually resolves without any long-term effect on vision," Dr. Morris says. ■

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Infiltrative Keratitis and Gram-Negative Bacterial Resistance to PQ-Aldox Lens Care Products

The rate of infiltrative keratitis especially with daily wear silicone hydrogel lenses has been reported with greater frequency.[1-4] Infiltrative keratitis is associated with several factors[1-8] including lens care solutions,[9,10] lens type,[1,3] smoking,[5] and bacterial bioburden.[5-8] **Contact lens associated infiltrative keratitis (CLAIK) has been reported at higher rates in particular with polyquaternium (PQ)-Aldox (myristamidopropyl dimethylamine) based Multi-Purpose Solutions (MPS).[1-5,9]**

Notably, CLAIK has repeatedly been associated with one PQ-Aldox MPS, Opti-Free RepleniSH in independent studies.[1-4] In one report, this solution was being used in 71% of CLAIK cases.[3] **Importantly, there has been no demonstrated correlation between transient, solution related corneal staining and inflammatory keratitis.[11]**

Low levels of lens case contamination may occur with any MPS or peroxide system in asymptomatic patients, but gram-negative contamination was reported highest with Opti-Free RepleniSH.[12] Recent scientific findings in patients using lens care solutions with CLAIK, demonstrate case contamination with certain gram-negative clinical isolates, the predominant species being *Stenotrophomonas maltophilia* and *Achromobacter*.[13] These gram-negative bacteria have also been cultured in the lens cases of patients using PQ-Aldox MPS.[12,14] **Additional research has shown that these clinical isolates are resistant to a PQ-Aldox MPS and can re-grow during storage in PQ-Aldox MPS in as few as 6 days.[14-16]** Non-Aldox PQ-based MPS, such as those containing PHMB, and peroxide lens care solutions have demonstrated excellent biocidal efficacy against these same clinical isolates.[14-17] Table 1 presents biocidal efficacy against clinical isolates of *Achromobacter* and *Stenotrophomonas* when exposed to PQ-Aldox MPSs and a PHMB-PQ MPS.[18] Lens care solutions that are ineffective against these clinical isolates may be prone to case contamination and CLAIK may result directly from these bacteria and/or their endotoxins being repeatedly exposed to the ocular surface.[14]

Log unit reduction		
	Achromobacter*	Stenotrophomonas*
Biotrue® MPS (PHMB-PQ)	2.9	3.5
OPTI-FREE PureMoist (PQ-Aldox)	0.1	1.2
OPTI-FREE RepleniSH (PQ-Aldox)	0.0	1.3
OPTI-FREE Express (PQ-Aldox)	0.2	1.2

Table 1. MPS Biocidal Efficacy Against Achromobacter and Stenotrophomonas Clinical Isolates Associated with CLAIK[18]

Further investigation is warranted to understand the causality between infiltrative keratitis events and the use of PQ-Aldox MPS products. **The inefficacy of PQ-Aldox MPS against clinical isolates cultured from CLAIK patients should be considered by eye care practitioners in recommending lens care systems for their patients.**

CLAIK has the potential of creating a significant economic burden on patients[19] and may contribute to patients choosing to stop wearing lenses. **Switching patients to MPS products with broad antimicrobial efficacy and proven biocompatibility, along with recommending appropriate lens and lens case cleaning regimens,[6] may help to prevent CLAIK, minimize risk for future recurrence[20] or contact lens drop out.**

Biotrue® MPS from Bausch + Lomb has proven biocompatibility and also demonstrates excellent disinfection efficacy compared to competitive multi-purpose solutions,[21,22] even against clinical isolates such as *Stenotrophomonas* and *Achromobacter, which are known to be associated with corneal infiltrative keratitis.*

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A Peripheral Look at BMS

Rare and often asymptomatic, Brown-McLean syndrome is a corneal edema that involves the peripheral 2mm to 3mm of the cornea. **Edited by Joseph P. Shovlin, O.D.**

Q I recently saw a patient with a peculiar bilateral peripheral corneal edema and a clinical history that included a past intracapsular surgery for cataracts (now pseudophakic). I sent the patient for a corneal consult and the diagnosis was Brown-McLean syndrome. What is this entity and what causes it? Are there any effective treatments?

A First described in 1969, Brown-McLean syndrome (BMS) is a clinical entity of peripheral circumferential corneal edema that usually starts in the inferior cornea and is often associated with orange-brown pigmentation at the level of Descemet's membrane.[1] Because the central cornea is usually spared, vision is often normal.

This condition is frequently asymptomatic, but the most common presenting symptom is foreign body sensation from bulla formation and, in some instances, pain due to ruptured bullae.[1] The cause of BMS is still unknown, but researchers suggest it may develop in patients who have a genetic predisposition when their eyes are exposed to certain conditions, such as the insertion of an anterior chamber lens.[2]

"It is most commonly seen after intracapsular cataract extraction, although it can be seen after any intraocular procedure, and occasionally in patients who have never had surgery," says Thomas S. Boland, M.D., of Northeastern Eye Institute in Scranton, Pa.

BMS has been linked to several lens surgeries, including extracapsular lens extraction, phacoemulsification, pars plana lensectomy and vitrectomy.[1]

The onset of this condition usually ranges from six to 16 years after the time of procedure.[2] "Due to the decline of intracapsular surgery over the last 20 to 30 years, it is rarely seen these days," Dr. Boland says. "It can usually be treated conservatively with lubrication, hyperosmotics and monitoring for infection in the case of ruptured bullae." In some cases, anterior stromal puncture or a bandage contact lens have been suggested as alternative treatments to control severe foreign-body sensation.[3]

It's important to educate patients with BMS about the signs and symptoms of corneal surface problems, particularly corneal ulceration secondary to contact lens wear, and to perform regular follow-up to monitor for any disease progression. Some clinicians have found confocal microscopy to be a convenient and informative means of documenting BMS as well as a useful tool in follow-up.[4] ■

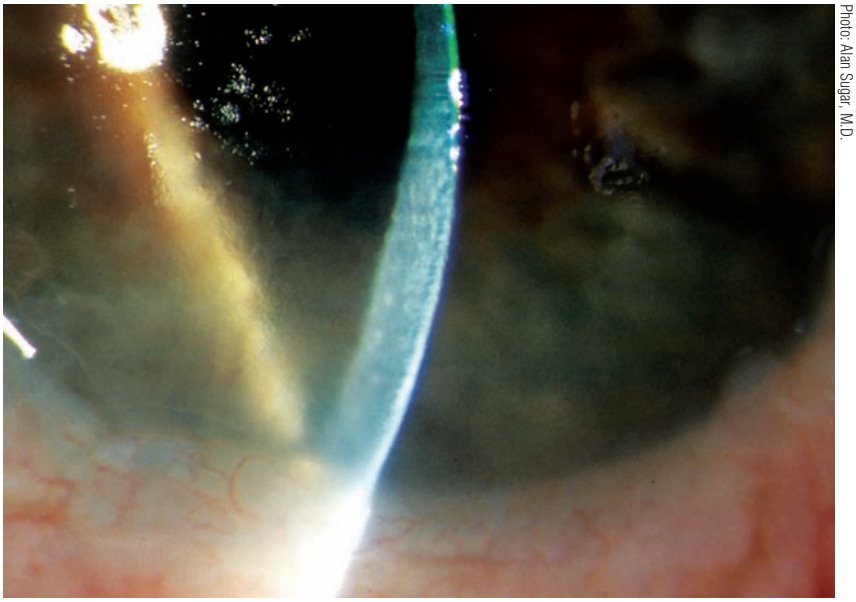

Photo: Alan Sugar, M.D.

Brown-McLean peripheral corneal edema in an eye with long-standing aphakia.

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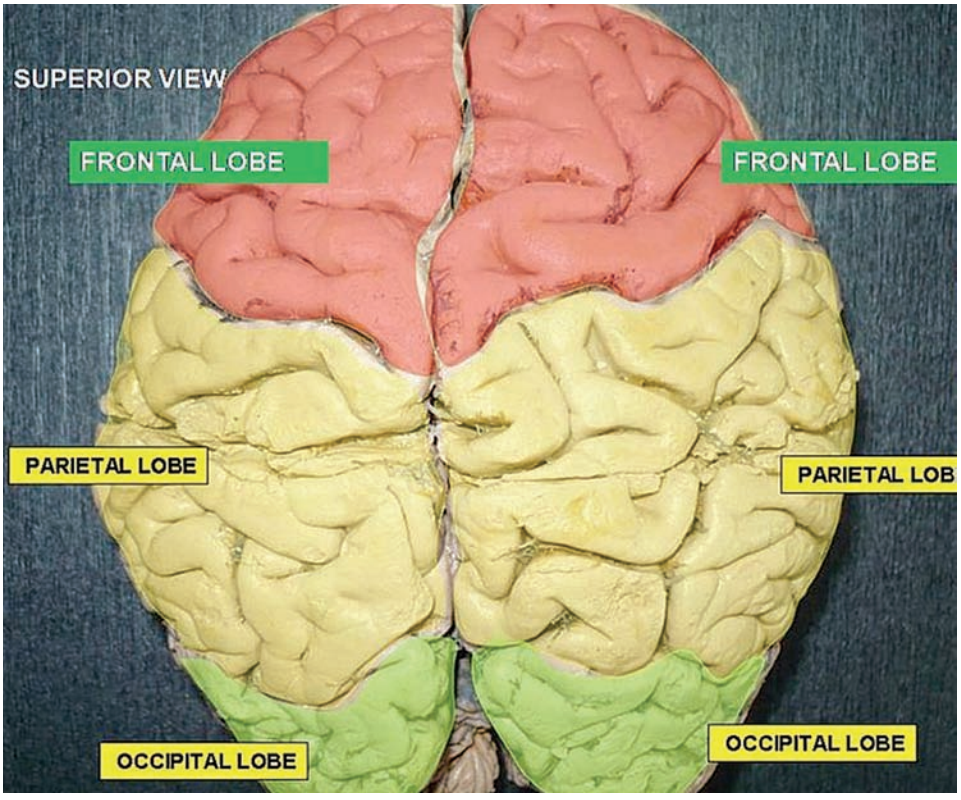
Beyond the Eye

Macular pigments (Part 2): Xanthophylls may protect other bodily tissues and systems as well as the retina. **By Joseph Pizzimenti, O.D., and Carlo Pelino, O.D.**

In our nutrition-based efforts to preserve our patients' macular health and enhance visual performance, optometrists also may be supporting systemic wellness. The biological properties of xanthophylls lutein and zeaxanthin as well as emerging epidemiological evidence in relation to chronic and degenerative diseases have triggered interest in these carotenoids as potentially beneficial for general health.[1,2]

Like other carotenoids, they are not produced by the body and must be obtained through diet (*see Foods High in Xanthophylls, page 80*).[1,3,4] While the highest concentrations of lutein and zeaxanthin are found in central neural regions, such as the retina (as macular pigment) and the brain's frontal and occipital cortical regions, they are distributed throughout tissue in the entire body (*see Xanthophylls in Human Tissue, page 80*).[1]



The brain's frontal cortex has high concentrations of xanthophylls.

Part 1 of this three-part series (*"Add Color to Your Diet," May 2012*) gave an overview of how xanthophylls protect the retina and enhance visual function. In this installment, we'll take a look at xanthophylls beyond the eye, reviewing a number of studies that investigate their protective role in skin damage and various types of cancer.

Skin Damage

In the eye, lutein and zeaxanthin comprise macular pigment, which acts as a filter to absorb blue light. Macular pigment has been shown to act as a potent antioxidant, quenching free radicals associated with the pathogenesis of age-related macular degeneration.[3,4] Recent research suggests that their roles are not exclusively protective to the eye, but to the skin as well.

As a consequence of dietary intake, lutein and zeaxanthin are found in the skin.[5] Just as the lutein and zeaxanthin found in the macula protect the retina against exposure to damaging visible wavelengths of light, the lutein and zeaxanthin present in skin cells play a role in protecting the skin from environmental damage. Research suggests that the presence of lutein and zeaxanthin in the skin may be important in helping to maintain its health and proper function.

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A 2007 double-blind, placebo-controlled study investigated oral, topical or combined (oral and topical) applications of lutein and zeaxanthin on the skin.[6]

Results indicated that separate topical or oral administration of lutein and zeaxanthin reduced lipid peroxidation and increased skin elasticity, superficial skin lipids and skin hydration.[6] Applying the topical preparation while simultaneously taking the oral supplement increased the benefits obtained for each of the measured parameters.[6]

Cancer Risk

Some studies have suggested xanthophylls may play a potential protective role against carcinogenesis in many ways, including selective modulation of apoptosis, inhibition of angiogenesis, enhancement of gap junctional intercellular communication, induction of cell differentiation, prevention of oxidative damage and modulation of the immune system.[7,8]

A number of observational studies have found these xanthophylls may help reduce the risk of certain types of cancer, particularly those of the breast and lung.[8]

• ***Breast cancer.*** During the 14-year follow-up of the Nurses Health Study—a prospective study of more than 83,000 female nurses ages 34 to 59 at baseline—2,697 developed invasive breast cancer.[9] Pre-diagnostic intake of lutein plus zeaxanthin was inversely associated with breast cancer risk in pre-menopausal women (784) but not in post-menopausal women (1,913).[9]

Researchers found a similar relationship when investigating pre-menopausal breast cancer risk and intake of nutrients in a case-control study of 297 cases and 311 control subjects.[10] A reduction in pre-menopausal breast cancer risk was associated with a high intake of lutein plus zeaxanthin during the two years prior to dietary interview.[10]

In a study of 270 case-control pairs nested within a prospective cohort study with up to nine-year follow-up, breast cancer risk was doubled among subjects with pre-diagnostic serum lutein at the lowest quartile, compared to those at the highest quartile.[11]

Korean researchers reported the serum concentration of lutein plus zeaxanthin was inversely associated with breast cancer risk, while studies in human mammary cells and in animal models also support a protective role of xanthophylls.[8,12]

Conversely, a large case-cohort analysis (1,452 cases, 5,239 controls) of women enrolled in the Canadian National Breast Screening Study who completed a self-administered dietary questionnaire exhibited different results. The research analysts found no association between dietary intakes of lutein plus zeaxanthin at baseline and breast cancer risk during the 11-year follow-up in the overall study population.[13]

Xanthophylls in Human Tissue

- Eye
  - -Lens
  - -Retina
- Blood serum
- Skin
  - -Epidermis
  - -Dermis
- Cervix
- Brain
  - -Frontal cortex
  - -Occipital cortex
- Breast

Foods High in Xanthophylls

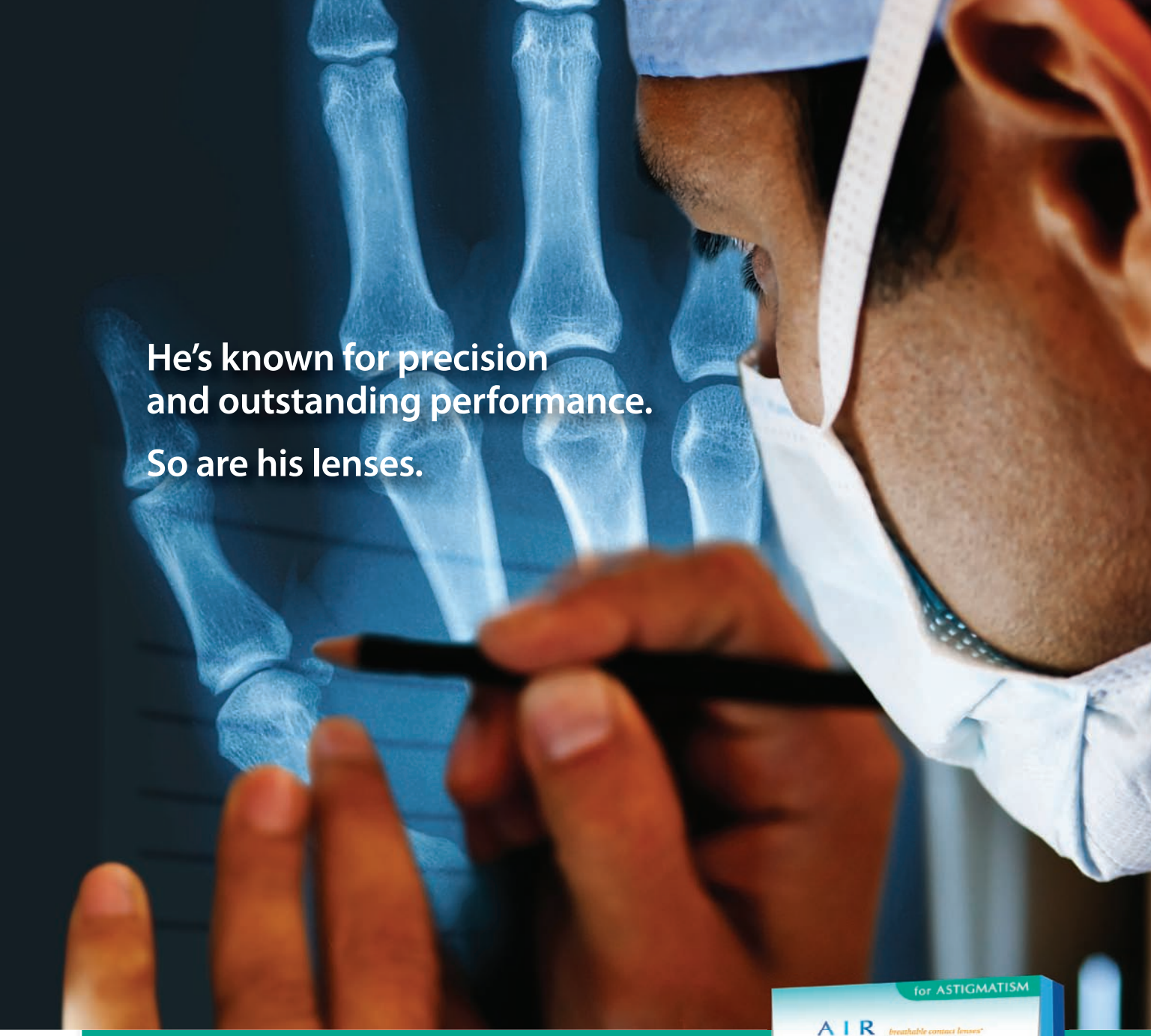
- Collards, frozen, chopped, cooked, boiled, drained, without salt
- Collards, cooked, boiled, drained, without salt
- Kale, frozen, cooked, boiled, drained, without salt
- Kale, cooked, boiled, drained, without salt
- Spinach, canned, drained solids
- Spinach, cooked, boiled, drained, without salt
- Spinach frozen, chopped or leaf, cooked, boiled, drained, without salt
- Turnip greens, frozen, cooked, boiled, drained, without salt
- Turnip greens, cooked, boiled, drained, without salt

Source: Release 18 of the USDA National Nutrient Database for Standard Reference

• ***Lung cancer.*** A prospective study of male smokers in Finland in the Alpha-Tocopherol, Beta-Carotene Cancer Prevention Study investigated the association of xanthophylls and lung cancer risk. Of 27,084 male smokers who completed a dietary questionnaire at baseline, 1,644 developed lung cancer during the 14-year follow-up.[14] Men in the highest quintile of lutein plus zeaxanthin intake at baseline had a 17% lower risk of lung cancer compared to men in the lowest quintile.[14]

• ***Colon cancer.*** Other studies have found inverse associations of the xanthophylls with precancerous lesions in the colon and rectum. The concentration of serum zeaxanthin—but not of lutein or other carotenoids—was lower in 59 patients with adenomatous colorectal polyp than in a healthy control group.[15]

• ***Ovarian cancer.*** Dietary intake of lutein plus zeaxanthin has been inversely associated with ovarian cancer risk. A case-control



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study of 1,031 Italian patients with confirmed incident epithelial ovarian cancer and 2,411 patients admitted to area hospitals for acute, non-neoplastic diseases (controls) showed that those in the highest quintile of lutein plus zeaxanthin intake had a risk of developing ovarian cancer that was 40% lower than those in the lowest quintile of intake.[16]

The literature discussed here is just a sampling of the growing body of data that suggests lutein and zeaxanthin may help prevent several systemic diseases and conditions. Randomized trials of diets high in carotenoid-rich vegetables and fruits are needed to confirm these results.

Keep an eye out for the final part of this series, which will appear in the September 2012 issue. There, we'll discuss how xanthophylls factor into heart disease, stroke, diabetes, and brain and cognitive impairment. ■

Drs. Pelino and Pizzimenti have no proprietary interest in any instrument, food product, vitamin or supplement. Dr. Pizzimenti serves on the scientific advisory board for ZeaVision.

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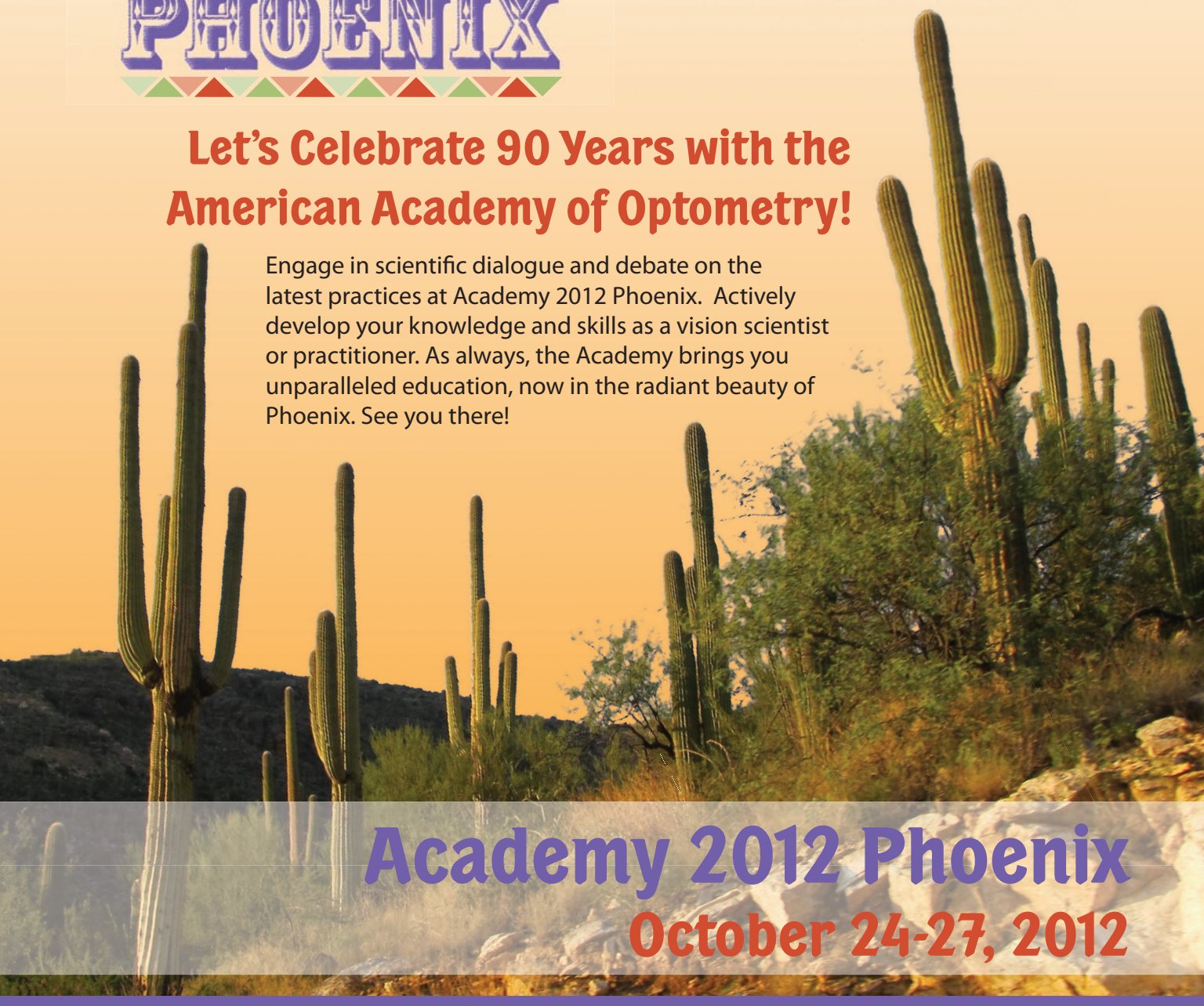

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Is This Metastasis?

This patient with a history of colon cancer presented with multiple lesions O.U. Did the cancer spread to her eyes? **By Mark T. Dunbar, O.D.**

A 61-year-old white female with a history of colon adenocarcinoma was referred for evaluation of "multiple white lesions" in both eyes. She had no significant ocular history, but was concerned about the possibility of metastasis from her history of colon cancer. At the time, she was undergoing chemotherapy and Avastin (bevacizumab, Genentech/Roche) therapy.

On examination, her best-corrected visual acuity measured 20/20 O.U. Confrontation fields were full to careful finger counting O.U. Extraocular motilities were full O.U. Her pupils were equal, round and reactive to light, without evidence of afferent defect.

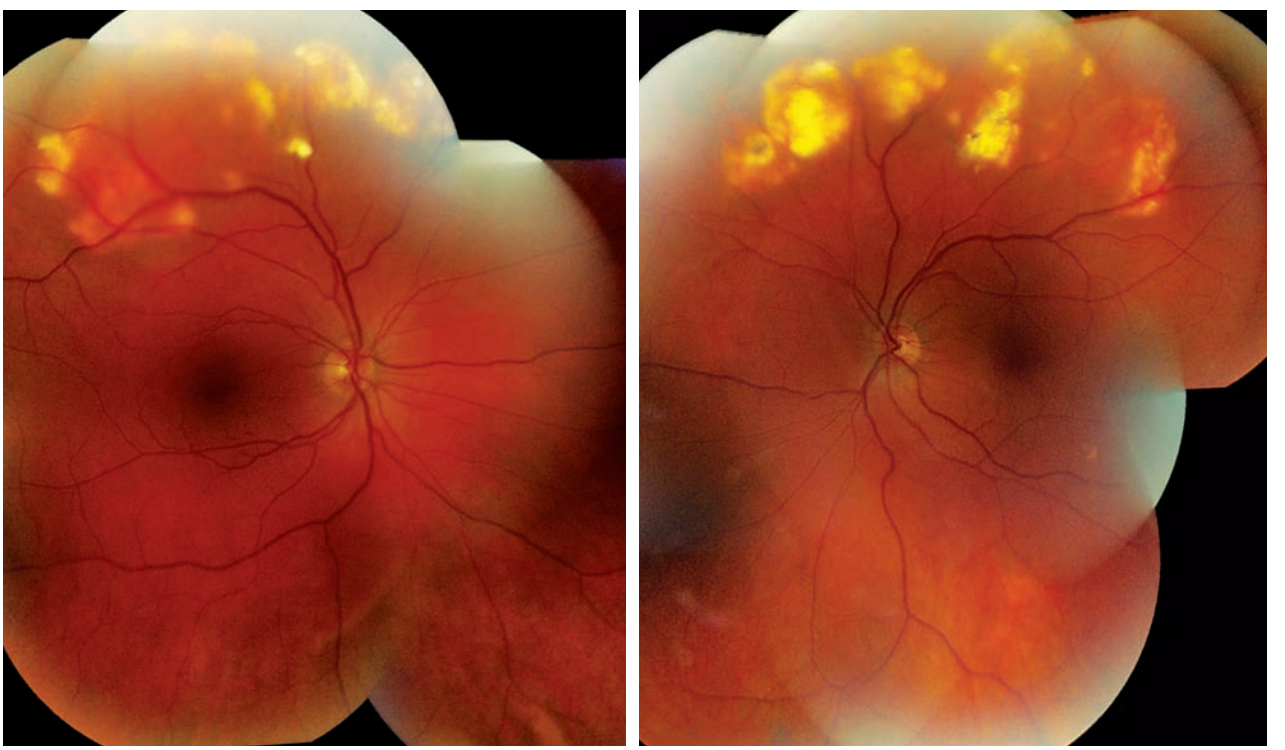
The anterior segment evaluation was unremarkable. Her intraocular pressure measured 16mm Hg O.U. Dilated fundus exam revealed small cups with good rim coloration and perfusion in both eyes.

The vessels and maculae were unremarkable O.U. The peripheral retina examination revealed the changes seen in figures 1 and 2. Additionally, we performed fluorescein angiography (FA) imaging (*figures 3 and 4*).

Take the Retina Quiz

1. Which additional tests would be most helpful in establishing a diagnosis?
   a. Standardized ultrasound.
   b. Optical coherence tomography.
   c. MRI.
   d. Visual fields.

2. What is the correct diagnosis in this case?
   a. Metastatic carcinoma to the choroid.
   b. Coats' disease.
   c. Choroidal osteoma.
   d. Sclerochoroidal calcification.



1, 2. The peripheral retinal images show peculiar white lesions in both eyes (O.D. left, O.S. right). Are these a cause for concern?

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3. How should this patient be managed?
a. Plaque radiotherapy.
b. Systemic chemotherapy.
c. Observation.
d. Enucleation.

4. What's the prognosis for this patient?
a. Excellent.
b. Guarded.
c. Poor.
d. Too early to tell.

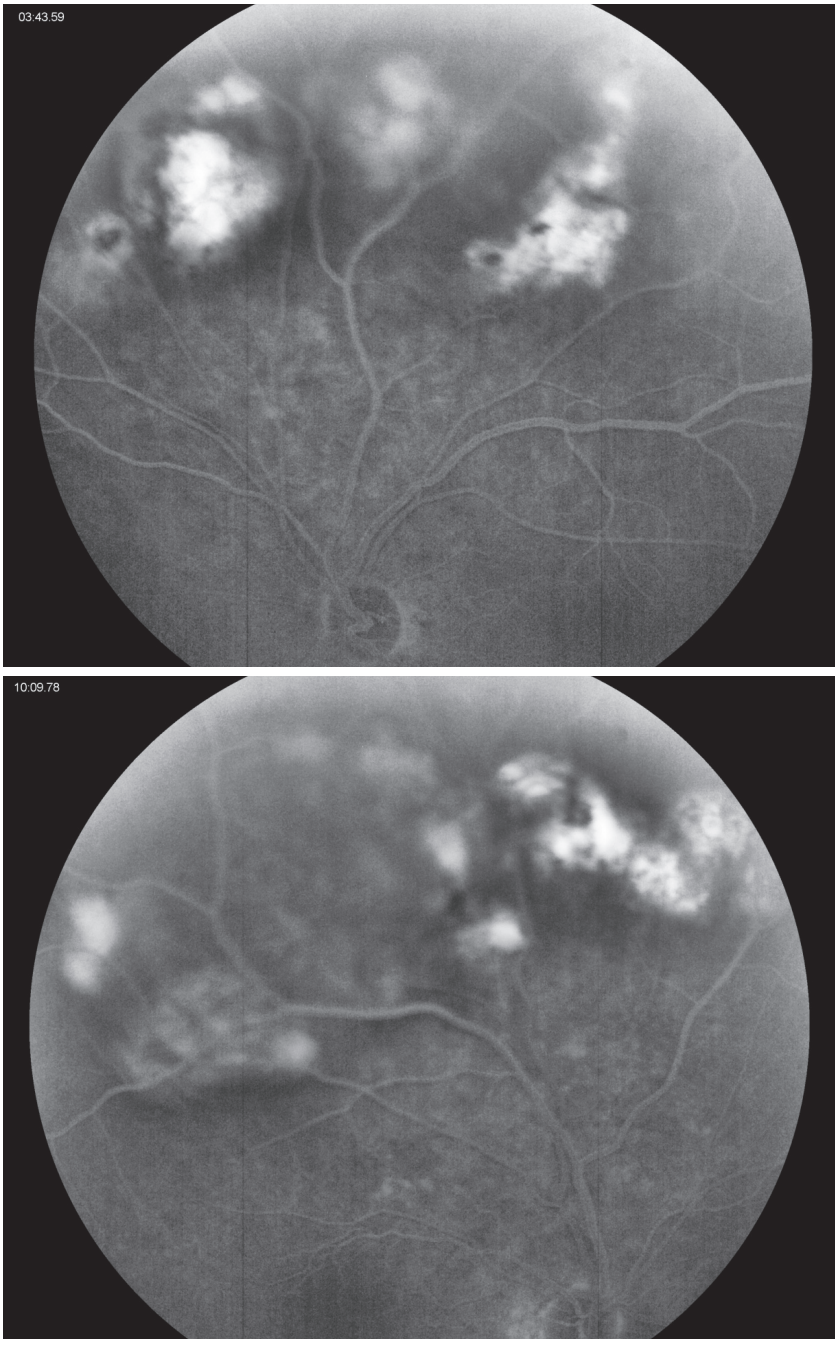
For answers, go to page 114.

Discussion

Our patient has sclerochoroidal calcification—a rare condition characterized by the presence of irregular, yellow-white subretinal placoid lesions usually seen in the superotemporal mid-periphery of the fundus. The presentation commonly is misdiagnosed as a malignant tumor.

Sclerochoroidal calcification typically is idiopathic and often is documented as an incidental finding in elderly patients (although it can be associated with hyperparathyroidism, vitamin D intoxication, sarcoidosis, hypophosphatemia or chronic renal failure).[1,2] The calcifications are believed to be deposited at the insertion sites of the extraocular oblique muscles.[1] Nonetheless, the condition remains a poorly recognized and understood entity.

Because sclerochoroidal calcification exhibits a very characteristic clinical appearance and anatomical location, the diagnosis should be easy to make—so long as the clinician is aware of the condition and maintains a heightened level of suspicion. Additionally, FA and ultrasound can help confirm the diagnosis.



3, 4. Late-phase fluorescein angiography shows staining and hyperfluorescence of the lesions (O.D. top, O.S. bottom).

In the early phases of the angiogram, FA will reveal autofluorescence of the lesions with the red-free filter and mild-to-moderate hypofluorescence.

In the later phases, the lesions will show increased hyperfluorescence as well as persistent late staining and mottling around the lesion that corresponds to areas of RPE atrophy. This is exactly what occurred in our patient.

The ultrasound finding—also

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typical of sclerochoroidal calcification—shows high reflectivity of the lesion's anterior surface as well as intense echodense placoid lesions at the scleral-choroidal interface with orbital shadowing.[2] According to one study, sclerochoroidal calcification chiefly involves the choroid; however, the sclera also may be involved.[2] In this case, the lesions had a maximum thickness of 2.2mm.

Given their creamy white appearance, sclerochoroidal calcifications often are mistaken for choroidal osteomas—a benign ossifying tumor comprised of mature bone that forms within the choroid.

Osteomas commonly are located immediately adjacent to or around the optic nerve and can extend to the macula. They are yellow-white to orange-red in color (similar to a sclerochoroidal calcification), and will exhibit high reflectivity and posterior shadowing on ultrasound due to their bony configurations. In fact, sclerochoroidal calcifications initially were believed to be choroidal osteomas that presented in older patients. Subsequent histological studies revealed the identifiable differences between these lesions.

Lastly, choroidal metastasis must be ruled out, because these lesions also tend to exhibit a creamy white appearance and often present bilaterally. With our patient's history of colon cancer, this was a legitimate concern. Fortunately, cancerous tumors are easily differentiated on ultrasound. Choroidal metastasis shows medium to high reflectivity, but lacks the shadowing of sclerochoroidal calcifications.

We explained the findings to our patient, and she was relieved to learn that the retinal changes were benign and unrelated to her history of colon cancer.

At both three- and six-month follow-up, we detected no signs of progression. We will continue to follow her every six months for a year, then annually thereafter. ■

Thanks to former Bascom Palmer optometry resident Melanie Denton, O.D., of Burnsville, N.C., for contributing this case.

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POAG & OSD: Double Trouble

Don't overlook the potential problem of ocular surface disease when treating primary open-angle glaucoma. **By Alan G. Kabat, O.D., and Joseph W. Sowka, O.D.**

A 60-year-old man recently was referred for glaucoma management. The diagnosis was very straightforward, and the severity was only moderate. However, treatment was a challenge.

The patient had a history of intolerance to numerous glaucoma medications due to severe dry eye. He had tried several common glaucoma medications, but suffered greatly with each formulation.

After each trial, he presented with significant redness and irritation. His tear film break-up time (TFBUT) was reduced to two to three seconds, and there was diffuse punctate staining across each cornea. The situation improved a little when he wasn't using the medications; however, his symptoms came back with a vengeance when he used any medication preserved with benzalkonium chloride (BAK).

Several issues arise when we start to think about glaucoma management in the context of ocular surface disease (OSD). Common symptoms of OSD include variable and intermittent blurred vision, itching, burning, stinging, grittiness, redness, irritation, dryness and excessive tearing. We now understand that glaucoma therapy may exacerbate or even cause OSD. Clinicians who manage glaucoma patients with early or pre-existing OSD must act to improve their patients' ocular surface health and thereby enhance their quality of life.

OSD in Glaucoma Patients

Using the Ocular Surface Disease Index to account for patients' subjective comfort and visual difficulties, one study of 630 patients indicated that OSD occurred in 48.4% of those using topical agents for glaucoma.[1] Of these, 21.3% of patients reported mild symptoms, 13.3% reported moderate symptoms and 13.8% reported severe symptoms.[1] Patients on multiple medications reported even higher scores.

In another study of 101 subjects who received topical glaucoma therapy, the authors documented that 59% of patients experienced OSD symptoms.[2] Additionally, Schirmer testing revealed that 61% of patients exhibited decreased tear production in at least one eye. Corneal and conjunctival lissamine green staining showed epitheliopathy in 22%, and TFBUT was abnormal in 78% of patients.[2] Further, a severe decrease in tear quality was found in 65% of patients.

Reduced TFBUT values result in redness, itching and discomfort, and are often indicative of chronic inflammation. Note that each additional BAK-containing medication was associated with twice the risk of abnormal lissamine green staining.[2]

Time to BAK Off

When managing glaucoma, we must be concerned not only with a glaucoma agent's ability to lower IOP, but also whether it's preserved with BAK. To be sure, BAK is an effective preservative. And at this time, most medications are still preserved in multi-use bottles. Likely, this is the most cost-effective option today. But, there may come a time soon when all topical ophthalmic medications are packaged in preservative-free unit doses.

Furthermore, we must be aware of all other topical preparations that our patients use, including over-the-counter medications for dry eye or allergy management. So, when you reach for an artificial tear to manage patients with OSD, be sure to select a BAK-free product to avoid worsening existing inflammation and to more effectively alleviate OSD symptoms.[3]

BAK-Free Glaucoma Drugs

Fortunately, glaucoma patients with concurrent OSD now have several available BAK-free IOP-lowering drugs. Among beta blockers, there is Timoptic in Ocudose (timolol maleate, Aton Pharma/Valeant Pharmaceuticals). Another option is the alpha agonist Alphagan P (brimonidine 0.01%, Allergan), which is preserved with "non-disruptive" Purite.

Travatan Z (travoprost, Alcon) employs an alternative preservative system, Sofzia, that breaks into non-toxic components on the ocular surface.

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Tafluprost Appears More Tolerable

In early clinical trials, Anton Hommer, M.D., and associates examined the efficacy and tolerability of tafluprost in 544 patients. They found that tafluprost was an effective, well tolerated and safe medication in a patient population with unsatisfactory IOP control and/or poor tolerability issues with their former medication(s).[7]

In another study of tolerability and efficacy, researchers found that switching treatment to tafluprost in patients who were already being treated with another PGA resulted in a statistically significant improvement of all symptoms evaluated—including stinging/burning/irritation, itching, foreign body sensation, tearing and dryness sensation. In these eyes, it is important to note that there was no documented difference in IOP between the previous PGA and tafluprost. However, in naïve eyes with either ocular hypertension or glaucoma that underwent tafluprost treatment as initial primary therapy, there was a 22% to 30% reduction in IOP.[8]

In a third study, tafluprost was better tolerated than BAK-containing latanoprost, exhibiting lower tear osmolarity levels while maintaining effective IOP control.[9] Another research team documented that the overall IOP-lowering effect of tafluprost was similar to that demonstrated by travoprost, latanoprost or bimatoprost in patients who were intolerant to the latter three PGAs.[10] However, when each PGA was individually compared with tafluprost, bimatoprost seemed to provide an additional statistically significant IOP-lowering effect.[10]

Another study indicated that travoprost 0.004% monotherapy produced lower diurnal IOP than tafluprost 0.0015% in patients with primary open-angle glaucoma or ocular hypertension, while exhibiting a similar safety profile.[11]

Finally, in an investigation of the tolerability and IOP-reducing effect of preservative-free tafluprost in patients who previously exhibited ocular surface side effects during latanoprost therapy, researchers saw that tafluprost maintained IOP at the same level as latanoprost, but was better tolerated.[12] Also, patients who received preservative-free tafluprost reported improved quality of life and increased overall satisfaction.[12]

received FDA approval in February. Zioptan is available in unit-dose vials, and has demonstrated good efficacy and tolerability in patients who are being treated for glaucoma and ocular hypertension (*see "Tafluprost Appears More Tolerable," page 93*).[4-6]

While current headlines may be dominated by the recent arrival of Zioptan, we also should mention that there is now a preservative-free fixed combination agent, Cosopt PF (dorzolamide hydrochloride 2%/timolol maleate 0.5%, Merck). Like Zioptan, Cosopt PF is supplied in unit-dose vials.

OSD is a pervasive condition that exists in many of our glaucoma patients. Patient comfort and visual function are of fundamental concern, ultimately influencing adherence to therapy, quality of life and personal satisfaction. Glaucoma patients with OSD benefit from less exposure to BAK-preserved anti-glaucoma drugs and tear preparations. Proper counseling is imperative.

Managing OSD in our glaucoma patients is not a short-term issue; rather it requires a long-term view that is focused upon the overall health of the eye. ■

Drs. Sowka and Kabat have no direct financial interest in any of the products mentioned.

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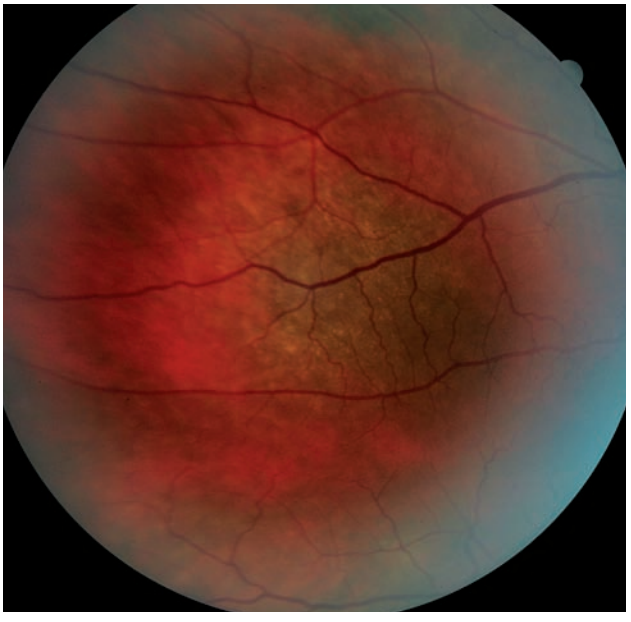
When it's Not a Nevus

Knowing the difference between choroidal nevi and melanomas can save your patient's life. **By Diana L. Shechtman, O.D., and Paul M. Karpecki, O.D.**

A 55-year-old white female presented with no ocular or visual complaints. During the dilated fundus examination, we observed a choroidal lesion. The lesion measured 4.0 disc diameters (DD) in size, appeared flat, and was associated with numerous overlying drusen deposits (*figure 1*). We noted no other pertinent findings.

A 42-year-old white female presented for an annual comprehensive eye exam. Dilated fundus examination revealed a 2.5DD choroidal lesion. The lesion appeared elevated and had an overlying orange pigmentation (*figure 2*). Optical coherence tomography revealed the presence of subretinal fluid. We detected no associated drusen or halo nevus.

Although both patients exhibit somewhat similar choroidal lesions, one presentation has the potential to be cancerous. But how can you effectively determine which lesion that is?



1. Fundus photograph of our first patient. What do you notice?

Nevi vs. Melanomas

Choroidal nevi and choroidal melanomas have many overlapping features, which poses a diagnostic dilemma. Yet, several distinguishable characteristics may be noted in both lesions.

• ***Choroidal nevi*** are relatively common, and are documented in 5% to 10% of the American population.[1] They are flat, green or slate gray choroidal lesions that typically measure less than 3.0DD, yet the size and thickness of choroidal nevi vary widely. Associated findings include serous retinal pigment detachment, choroidal neovascularization pigmentary changes and drusen formation.[2]

• ***Choroidal melanomas*** are the most common ocular malignancy. They appear as darkly pigmented lesions and typically present with some degree of elevated thickness. Choroidal melanomas are classified as small (<10mm in diameter and <3mm thickness), medium (10mm to 15mm in diameter and 3mm to 5mm in thickness) or large (>15mm in diameter and >5mm in thickness).[3] Features may include lipofuscin and associated serous fluid.[4] Documented evidence of short-term growth seems to be a pathognomonic sign of a choroidal melanoma.[5] And although some studies have shown progressive enlargement of choroidal nevi, malignant features are absent.[6] Whether melanomas arise from preexisting nevi or are completely separate entities is somewhat debatable.

Small Melanoma Characteristics

Typically, patients with larger, thicker melanomas have worse long-term prognoses than individuals with smaller lesions. Several meta-analyses indicate that patients with choroidal tumors exhibit a five-year mortality rate that ranges from 15% to 50%, depending upon lesion size and thickness.[4,7]

Therefore, early detection of choroidal melamomas is crucial to minimize the risk of metastatic disease. A prompt diagnosis and timely referral for proper management will increase the patient's overall chances for survival dramatically.[7]

Because histological confirmation of a choroidal melanoma is not feasible, we chiefly rely on clinical characteristics of growth to determine the risk of metastasis. Within the last 15 years, Carol L. Shields, M.D., and associates conducted two retrospective medical reviews of patients with presumed small choroidal

melanomas.[4,8] In both studies, the authors' primary goal was to identify specific features associated with small choroidal melanoma that could predict lesion growth and potential metastasis.

In the first study, the researchers evaluated the records of approximately 1,300 patients who were followed regularly from 1970 to 1990.[4] They determined that the average diameter of the subjects' choroidal lesions was 5mm, with thicknesses ranging from flat to 3mm. All patients underwent standardized testing, including dilated fundus examination and ultrasonography.

At the study's conclusion, the authors identified five common traits associated with small choroidal melanomas:[4]

- Thickness (>2mm)
- Fluid (retinal)
- Symptomatic (photopsia, decreased visual acuity, metamorphopsia, etc.)
- Orange pigment (lipofuscin)
- Margin near the nerve (within 3mm)

These features may be remembered more easily using the mnemonic "*To Find a Small Ocular Melanoma*."

In the second study, Dr. Shields and associates sought to determine whether additional clinical characteristics might be indicative of a risk of malignancy.[8] This retrospective medical review included nearly 2,500 patients followed from 1974 to 2006. At the study's completion, the researchers documented the presence of two additional features:[8]

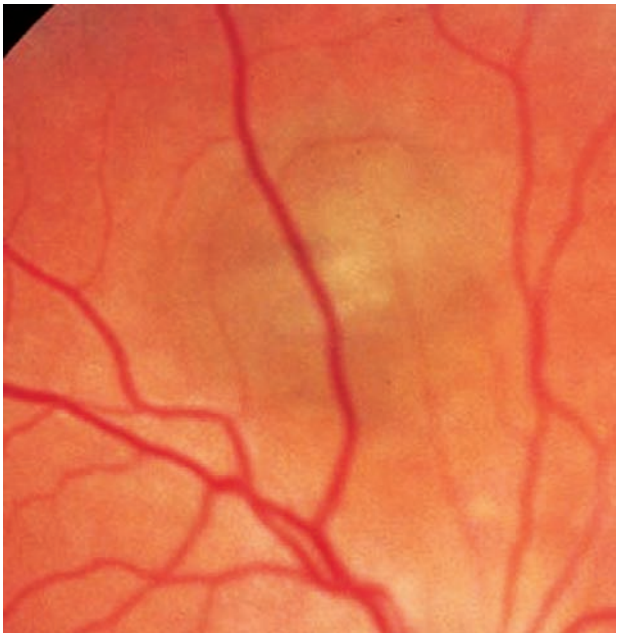
- Ultrasonography hollowness
- Halo nevus

This discovery adds to the mnemonic: "*To Find a Small Ocular Melanoma, Use Helpful Hints*."

The researchers noted that patients with three or more of the traits had a 50% higher risk of melanoma growth.[8] Further, they determined that patients with three to four traits were 15% to 20% more likely to experience metastasis; those with one to two factors were at a 5% higher risk of metastasis; and individuals with zero risk factors had less than a 1% chance of metastasis.[8]

Accordingly, a management protocol was developed for these small choroidal melanomas, based on the number of documented traits:

• 0: Patients should be followed on an annual basis.
• 1-2: Patients should be followed every four to six months to evaluate both short- and long-term lesion growth.
• 3 or more: Patients should be referred to a specialist for further management and treatment.



2. Fundus image of our second patient. How does this presentation differ from the findings observed in our first patient?

Back to Our Patients

Given the overall clinical features of our patients introduced above, we diagnosed the first individual with a large choroidal nevus and the second individual with a small choroidal melanoma. Malignant features in the second case included lesion thickness as well as the presence of orange lipofuscin and associated serous fluid.

Our diagnostic accuracy when encountering small choroidal melanomas continues to improve. Early recognition of high-risk characteristics helps the clinician make a prompt referral, which ultimately translates into a better prognosis for the patient. ■

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Contact Lenses

Biotrue OneDay Contact Lenses

The FDA recently approved Bausch + Lomb's Biotrue OneDay, a premium daily disposable contact lens. Made from a new material called HyperGel (nesofilcon A), the lens offers high water content and delivers more oxygen than a traditional hydrogel lens while maintaining the comfort of conventional hydrogels, the company says.

Bausch + Lomb created Biotrue OneDay to be the first daily disposable lens with three bio-inspired features: (1) the lens contains 78% water, the same as the cornea, (2) it delivers the oxygen level needed by the open eye to maintain healthy, white eyes, and (3) the anterior surface of the lens is designed to mimic the tear film's lipid layer to prevent dehydration.

B+L says the lens maintains virtually the same moisture level as the natural eye, even after 16 hours of wear, to help Biotrue OneDay lenses retain their shape and provide clear vision throughout the day. The lens also helps protect against UV-A and UV-B exposure.

Visit www.bausch.com.

Ophthalmic Lenses

Zeiss Progressive Individual 2

The new Zeiss Progressive Individual 2 features proprietary EyeFit and center-of-rotation evaluation (CORE) technologies that are designed to improve visual performance and allow eye care professionals to meet the needs of patients more precisely. EyeFit technology can personalize the lens based on the patient's visual activity profile, enhancing the near or intermediate/dynamic performance of the lens if needed based on the patient's visual priorities, while maintaining outstanding all-distance vision performance, the company says.

The CORE algorithm customizes Zeiss Progressive Individual 2 for the patient's center-of-rotation distance. The manufacturer says CORE technology is based on its own extensive research and calculates each

SIMULATED FIELDS OF USABLE VISION

(Continued on page 100)

Frames

Fendi Maserati-inspired Sunglasses

Experience the beauty of Rome through the Whispered Grand Tour, as Fendi partners with Maserati to create a sunglass style that complements a luxurious lifestyle. The lightweight FS5262L is hand crafted with supple genuine leather and fine metal in Italy. This style is available in gray with gunmetal temples and green lenses, as well as yellow with gold metal temples and brown lenses.

The yellow Fendi logo on the lens adds a pop of color to both of these aviators. The classic Maserati trident logo is featured on the temple tips in silver. The polarized lenses are made of mineral glass and feature an anti-glare coating, designed to ensure the best possible experience both on and off the road.

Visit www.marchon.com or www.fendi.com.

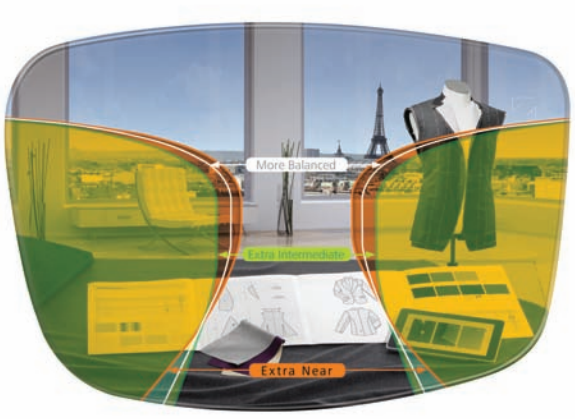
Baby Genius Sunscape Eyewear

Sunscape Eyewear has signed a two-year licensing agreement to manufacture and distribute Baby Genius-themed eyewear for boys and girls from infant to age four. Products will include sunglasses, eyewear and eyewear accessories, such as cords and cases, that feature popular Baby Genius characters.

The initial line of Baby Genius products from Sunscape will debut in time for the 2012 holiday season, and will be distributed to large national mass retailers including Walmart, Toys 'R' Us, Babies 'R' Us and Target.

Visit www.isunscape.com.

wearer's center of rotation to within 1mm for 99% of wearers. The lenses feature an extended Rx range, allowing add powers up to +4.00 and cylinder out to -6.00, and can be decentered up to 5mm per eye for maximum cutout.



Zeiss Progressive Individual 2's EyeFit technology provides three different zone balances to choose from, which include:

- Balanced (white line).
- Enhanced Near (orange line).
- Enhanced Intermediate (green line).

Visit www.zeiss.com.

Intraocular Lens

enVista

Bausch + Lomb received FDA approval for enVista, which the company describes as the first FDA-approved, glistening-free, single-piece hydrophobic acrylic intraocular lens in the United States.

Glistenings are fluid-filled microvacuoles that can form within an IOL and can be found in some hydrophobic acrylic IOLs.

Literature reports indicate that glistenings may present an aesthetic issue following cataract surgery and can also potentially impact visual function, including visual acuity and contrast sensitivity, due to a portion of light coming into the eye being scattered, the company says.

The enVista lens is designed to minimize posterior capsular opacification, a common post-surgical complication of IOLs that causes vision to become clouded after surgery.

enVista received CE Mark approval in the European Union in September 2011 and is currently pending approvals worldwide. Full commercial release of the enVista IOL in the United States is planned upon FDA clearance of its supporting insertion system.

Visit www.bausch.com.

Nutritional Supplement

Eye Vitamin Leader Introduces New Product

ZeaVision introduced EyePromise Zeaxanthin + Lutein Macular Pigment Formula to its portfolio of EyePromise eye vitamins.

This formula features all-natural ingredients, including 10mg of lutein and 10mg of dietary zeaxanthin, which is the highest amount available in any eye vitamin brand, the company says.

More than 25 million doses of EyePromise have been safely consumed, and the new formula provides an additional high-quality eye vitamin option to support macular health.

Visit www.zeavision.com. ■

Frames

MMJ316S

MMJ315S

Marc Jacobs "I Love Stripes" Collection

Marc by Marc Jacobs presents its new "I Love Stripes" collection of sunglasses and optical frames, produced and distributed by the Safilo Group. Featuring acetate and metal styles, the brand's signature stripes provide eye-catching detail to the temples. From rectangular silhouettes for the men's offerings to refined cat-eye shapes for the women's line, the collection is chic and wearable, the company says.

Drawing inspiration from downtown New York in the 1980s, the new wave shapes are now more colorful. Plays on transparency, matte finishes and bold color combinations create modern styles. The iconic stripes are interpreted in two versions: a finer stripe available in two-tone models (MMJ316/s, MMJ525, MMJ534) and a wide stripe featuring exclusive multilayer acetate pastel tones (MMJ315/s, MMJ530, MMJ531, MMJ533).

Visit www.safilo.com.

Agathitas from Agatha Ruiz de la Prada

Agatha Ruiz de la Prada presents her new collection of Agathitas—frames for babies and children up to age three. It includes eight new colors, which are available in two sizes (O38 and O41).

Manufactured in Italy, the frames are made from a light, flexible and 100% hypoallergenic material with double silicone injection inside of the bridge. The temples also feature the double silicone injection on their interior, as well as an integrated hinge and adjustable temple tips.

All models are accompanied by a colorful case with the designer's hallmark style and a new adjustable elastic cord that attaches to the earpieces with a convenient click system that can be adjusted for a secure fit.

Visit www.grupoptim.com.

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Meetings + Conferences

August 2012

■ **3-4.** *Summer Education Event.* Blue Harbor Resort, Sheboygan, Wis. Hosted by: Wisconsin Optometric Association. Call (800) 678-5357 or visit www.woa-eyes.org.

■ **3-5.** *32nd Annual Educational Retreat 2012.* South Seas Island Resort, Sanibel, Fla. Hosted by: Southwest Florida Optometric Association Inc. CE hours: 12. Contact Brad Middaugh, O.D., at (239) 481-7799 or swfoa@att.net. Visit www.swfoa.com.

■ **10-11.** *Key West Educational Conference.* Key West, Fla. Hosted by: The Foundation for Ocular Health. Contact Gloria Ayan at gayan@araneye.com or call (305) 491-3747.

■ **19.** *Orlando Super Sunday #1.* Orlando Campus, NOVA Southeastern University, Orlando, Fla. CE hours: 8. E-mail oceaa@nova.edu or visit optometry.nova.edu/ce/supersunday.

■ **23-25.** *Idaho Optometric Physicians Association Annual Congress.* The Grove Hotel, Boise, Idaho. Contact Randy Andregg, O.D., at randregg@frontiernet.net or (208) 461-0001. Visit idaho.aoa.org.

■ **23-26.** *105th SCOPA Annual Meeting.* Myrtle Beach Marriott Resort & Spa at Grande Dunes, Myrtle Beach, S.C. Hosted by: South Carolina Optometric Physicians Association. CE hours: 20. Visit southcarolina.aoa.org.

■ **24-26.** *UABSO Continuing Education Alumni Reunion Weekend.* Hill University Center, University of Alabama at Birmingham. Hosted by: UAB School of Optometry. CE hours: 18. Contact Candie Bratton at (205) 934-5701 or uabsoce@uab.edu. Visit www.uab.edu/optometry.

■ **25.** *San Antonio Ophthalmic Symposium.* Westin Riverwalk Hotel, San Antonio. CE hours: 7. Visit www.revophth.com/saos2012.

September 2012

■ **5-8.** *International Vision Expo & Conference West 2012.* Sands Expo & Convention Center, Las Vegas. Call (800) 811-7151 or visit www.visionexpowest.com.

■ **6-9.** *72nd Annual Middle Atlantic Optometric Congress.* Doubletree Hotel and Convention Center, Pittsburgh/Monroeville, Pa. Under the auspices of: The Optometric Extension Program Foundation & the Western Pennsylvania Optometric Society. CE hours: 12. E-mail Barry Cohen, O.D., at barryc51@gmail.com.

■ **6-10.** *The Art and Science of Optometric Care: A Behavioral Perspective.* Grand Rapids, Mich. Held by: The Optometric Extension Program Foundation. CE hours: 35. E-mail Theresa Krejci at TheresaKrejciOEP@verizon.net or visit www.oepf.org.

■ **7-9.** *43rd Annual Colorado Vision Training Conference.* YMCA of the Rockies. Estes Park, Colo. Contact Jamie Anderson, O.D., F.C.O.V.D., at (303) 683-4466 or drjamieanderson@gmail.com.

■ **8-9.** *Primary Eye Care Update.* Northeastern State University, Oklahoma College of Optometry, Tahlequah, Okla. CME hours: 10. Contact Ashley Beason Manes at beason01@nsuok.edu or (918) 444-4033. Visit www.optometry.nsuok.edu.

■ **8-9.** *Fall Conference 2012.* Terry Auditorium, Nova Southeastern University, Fort Lauderdale, Fla. E-mail oceaa@nova.edu or visit http://optometry.nova.edu/ce/index.html.

■ **9-10.** *Northeast Congress.* Westford Regency Inn & Conference Center, Westford, Mass. CE hours: 12. Contact Kathleen A. Prucnal, O.D., at (978) 597-5227 or drkaprucnal@msn.com.

■ **12-15.** *Envision Conference.* Hilton St. Louis at the Ballpark, St. Louis. E-mail info@envisionconference.org or call (316) 440-1530. Visit www.envisionconference.org.

■ **13-14.** *South Dakota Optometry Society Fall Conference.* Hilton Garden Inn, Sioux Falls, S.D. Call (605) 224-8199 or e-mail deb.mortenson@pie.midco.net. Visit www.sdeyes.org.

■ **14-16.** *SWCO 2012.* InterContinental Hotel, Dallas. Sponsored by: Southwest Council of Optometry. Contact Niki Bedell, M.P.H., at (713) 743-1856 or nbedell2@uh.edu. Visit www.swco.org.

■ **14-16.** *VOA Annual Meeting.* Basin Harbor Club, Vergennes, Vt. Hosted by: Vermont Optometric Association. CE hours: 17. Contact David J. DiMarco, O.D., at (802) 524-9561 or djd@nveyecare.net. Visit www.vtoptometrists.org.

■ **14-16, 18-20.** *CE in Italy: Florence and/or Castiglion Florentino, Tuscany.* To register for one or both programs, contact James L. Fanelli, O.D., at (910) 452-7225 or jamesfanelli@CEinItaly.com. Visit www.CEinItaly.com.

■ **21-23.** *New Technology and Treatments in Vision Care.* California. Hosted by: *Review of Optometry.* Meeting chair: Paul Karpecki, O.D. CE hours: 15. Contact Lois DiDomenico at ReviewMeetings@jobson.com or (866) 658-1772. Visit www.revoptom.com/conferences.

■ **23.** *CPOS CE Forum XVI.* The Hotel Hershey, Hershey, Pa. Hosted by: Central Pennsylvania Optometric Society. Featured speakers: Ron Melton, O.D., and Randall Thomas, O.D., M.P.H. CE hours: 6. E-mail Mary Good, O.D., at cposrsvp@gmail.com.

■ **27-30.** *GWCO Congress 2012.* Oregon Convention Center, Portland. Hosted by: Great Western Council of Optometry. CE hours: 59. Visit http://www.gwco.org/Congress.html.

■ **27-30.** *2012 WOA Convention and Annual Meeting.* Kalahari Resort, Wisconsin Dells, Wis. Hosted by: Wisconsin Optometric Association. CE hours: 22. Visit www.woa-eyes.org.

■ **28-30.** *Illinois Optometric Association Annual Convention.* Crowne Plaza Hotel, Springfield, Ill. Call (800) 933-7289 or visit www.ioaweb.org.

■ **30-Oct 2.** *NDOA 109th Annual Congress & Exhibition.* Ramkota Hotel, Bismarck, N.D. Hosted by: North Dakota Optometric Association. Call (877) 637-2026 or e-mail ndoaz@btinet.net. Visit www.ndeyecare.com.

October 2012

■ **4-7.** *EastWest Eye Conference.* Cleveland Convention Center, Cleveland. Hosted by: Ohio Optometric Association. Call (800) 999-4939 or e-mail info@ooa.org. Visit www.eastwesteye.org.

■ **6-7.** *PSS 2012: 2nd Annual Forum on Ocular Disease.* The Castle Hotel & Resort, Orlando, Fla. Hosted by: PSS EyeCare. CE hours: 18. Call (203) 415-3087 or e-mail education@psseyecare.com. Visit www.psseyecare.com.

■ **10-11.** *44th Annual Fall Seminar.* The Lansing Center, Lansing, Mich. Hosted by: Michigan Optometric Association. Contact Amy Possavino at amy@themoa.org or (517) 482-0616. Visit www.themoa.org.

■ **12.** *HVOS Fall Seminar.* The Grandview, Poughkeepsie, N.Y. Hosted by: Hudson Valley Optometric Society. Contact Robert Greenbaum, O.D., at robertgreenbaum58@gmail.com or (845) 473-0220. Visit www.hvos.org.

■ **12-13.** *Northwoods Education Events.* Black Bear Lodge, St. Germain, Wis. Hosted by: Wisconsin Optometric Association. E-mail joleenwoaoffice@tds.net or (800) 678-5357. Visit www.woa-eyes.org.

■ **13-14.** *Fall Conference.* Lansdowne Resort, Leesburg, Va. Hosted by: Virginia Optometric Association. Call (804) 643-0309 or visit www.thevoa.org.

■ **16-20.** *COVD 42nd Annual Meeting.* Omni Fort Worth Hotel, Fort Worth, Texas. Hosted by: College of Optometrists in Vision Development. Contact info@covd.org or (330) 995-0718. Visit www.cvod.org.

■ **24-27.** *Academy 2012 Phoenix.* Phoenix Convention Center. Hosted by: American Academy of Optometry. Visit www.aaopt.org/meetings/academy2012.

■ **27-28.** *VOSH/International Annual Meeting.* Marriott Renaissance Phoenix Downtown Hotel. Hosted by: VOSH/International. Contact Harry I. Zeltzer, O.D., at vosh@vosh.org. Visit vosh-california.org/voshinter/annual12.html.

November 2012

■ **8-11.** *Monterey Symposium.* Monterey Marriott Hotel & Conference Center, Monterey, Calif. Hosted by: California Optometric Association. Call Will Curtis at (916) 266-5037 or e-mail wcurtis@coavision.org. Visit www.coavision.org.

■ **9-10.** *C.E. Charleston.* Doubletree Charleston Historic District, Charleston, S.C. Hosted by: Pacific University College of Optometry. CE hours: 12. Call Jeanne Oliver at (503) 352-2740 or e-mail jeanne@pacific.edu. Visit www.pacificu.edu/optometry.ce.

■ **9-11.** *FCO International 23rd Annual Educational Conference.* Abe Martin Lodge, Brown County State Park, Nashville, Ind. Hosted by: Fellowship of Christian Optometrists. Visit www.fcoint.org/services/annualConference.html.

To list your meeting, contact:
Colleen Mullarkey, Senior Editor
E-mail: cmullarkey@jobson.com
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Azopt®

(brinzolamide ophthalmic suspension) 1%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

1 INDICATIONS AND USAGE

AZOPT® (brinzolamide ophthalmic suspension) 1% is a carbonic anhydrase inhibitor indicated in the treatment of elevated intraocular pressure in patients with ocular hypertension or open-angle glaucoma.

2 DOSAGE AND ADMINISTRATION

The recommended dose is one drop of AZOPT® (brinzolamide ophthalmic suspension) 1% in the affected eye(s) three times daily. If more than one topical ophthalmic drug is being used, the drugs should be administered at least ten (10) minutes apart.

3 DOSAGE FORMS AND STRENGTHS

Solution containing 10 mg/mL brinzolamide.

4 CONTRAINDICATIONS

AZOPT® (brinzolamide ophthalmic suspension) 1% is contraindicated in patients who are hypersensitive to any component of this product.

5 WARNINGS AND PRECAUTIONS

5.1 Sulfonamide Hypersensitivity Reactions

AZOPT® (brinzolamide ophthalmic suspension) 1% is a sulfonamide and although administered topically it is absorbed systemically. Therefore, the same types of adverse reactions that are attributable to sulfonamides may occur with topical administration of AZOPT® (brinzolamide ophthalmic suspension) 1%. Fatalities have occurred, although rarely, due to severe reactions to sulfonamides including Stevens-Johnson syndrome, toxic epidermal necrolysis, fulminant hepatic necrosis, agranulocytosis, aplastic anemia, and other blood dyscrasias. Sensitization may recur when a sulfonamide is re-administered irrespective of the route of administration.
If signs of serious reactions or hypersensitivity occur, discontinue the use of this preparation.

5.2 Corneal Endothelium

Carbonic anhydrase activity has been observed in both the cytoplasm and around the plasma membranes of the corneal endothelium. There is an increased potential for developing corneal edema in patients with low endothelial cell counts. Caution should be used when prescribing AZOPT® (brinzolamide ophthalmic suspension) 1% to this group of patients.

5.3 Severe Renal Impairment

AZOPT® (brinzolamide ophthalmic suspension) 1% has not been studied in patients with severe renal impairment (CrCl < 30 mL/min). Because AZOPT® (brinzolamide ophthalmic suspension) 1% and its metabolite are excreted predominantly by the kidney, AZOPT® (brinzolamide ophthalmic suspension) 1% is not recommended in such patients.

5.4 Acute Angle-Closure Glaucoma

The management of patients with acute angle-closure glaucoma requires therapeutic interventions in addition to ocular hypotensive agents. AZOPT® (brinzolamide ophthalmic suspension) 1% has not been studied in patients with acute angle-closure glaucoma.

5.5 Contact Lens Wear

The preservative in AZOPT® (brinzolamide ophthalmic suspension) 1%, benzalkonium chloride, may be absorbed by soft contact lenses. Contact lenses should be removed during instillation of AZOPT® (brinzolamide ophthalmic suspension) 1%, but may be reinserted 15 minutes after instillation.

6 ADVERSE REACTIONS

6.1 Clinical Studies Experience

Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to the rates in the clinical studies of another drug and may not reflect the rates observed in practice.

In clinical studies of AZOPT® (brinzolamide ophthalmic suspension) 1%, the most frequently reported adverse events reported in 5-10% of patients were blurred vision and bitter, sour or unusual taste. Adverse events occurring in 1-5% of patients were blepharitis, dermatitis, dry eye, foreign body sensation, headache, hyperemia, ocular discharge, ocular discomfort, ocular keratitis, ocular pain, ocular pruritus and rhinitis.

The following adverse reactions were reported at an incidence below 1%: allergic reactions, alopecia, chest pain, conjunctivitis, diarrhea, diplopia, dizziness, dry mouth, dyspnea, dyspepsia, eye fatigue, hypertonia, keratoconjunctivitis, keratopathy, kidney pain, lid margin crusting or sticky sensation, nausea, pharyngitis, tearing and urticaria.

7 DRUG INTERACTIONS

7.1 Oral Carbonic Anhydrase Inhibitors

There is a potential for an additive effect on the known systemic effects of carbonic anhydrase inhibition in patients receiving an oral carbonic anhydrase inhibitor and AZOPT® (brinzolamide ophthalmic suspension) 1%. The concomitant administration of AZOPT® (brinzolamide ophthalmic suspension) 1% and oral carbonic anhydrase inhibitors is not recommended.

7.2 High-Dose Salicylate Therapy

Carbonic anhydrase inhibitors may produce acid-base and electrolyte alterations. These alterations were not reported in the clinical trials with brinzolamide. However, in patients treated with oral carbonic anhydrase inhibitors, rare instances of acid-base alterations have occurred with high-dose salicylate therapy. Therefore, the potential for such drug interactions should be considered in patients receiving AZOPT® (brinzolamide ophthalmic suspension) 1%.

8 USE IN SPECIFIC POPULATIONS

8.1 Pregnancy

Pregnancy Category C: Developmental toxicity studies with brinzolamide in rabbits at oral doses of 1, 3, and 6 mg/kg/day (20, 62, and 125 times the recommended human ophthalmic dose) produced maternal toxicity at 6 mg/kg/day and a significant increase in the number of fetal variations, such as accessory skull bones, which was only slightly higher than the historic value at 1 and 6 mg/kg. In rats, statistically decreased body weights of fetuses from dams receiving oral doses of 18 mg/kg/day (375 times the recommended human ophthalmic dose) during gestation were proportional to the reduced maternal weight gain, with no statistically significant effects on organ or tissue development. Increases in unossified sternebrae, reduced ossification of the skull, and unossified hyoid that occurred at 6 and 18 mg/kg were not statistically significant. No treatment-related malformations were seen. Following oral administration of ^{14}C-brinzolamide to pregnant rats, radioactivity was found to cross the placenta and was present in the fetal tissues and blood.

There are no adequate and well-controlled studies in pregnant women. AZOPT® (brinzolamide ophthalmic suspension) 1% should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus.

8.3 Nursing Mothers

In a study of brinzolamide in lactating rats, decreases in body weight gain in offspring at an oral dose of 15 mg/kg/day (312 times the recommended human ophthalmic dose) were seen during lactation. No other effects were observed. However, following oral administration of ^{14}C-brinzolamide to lactating rats, radioactivity was found in milk at concentrations below those in the blood and plasma.

It is not known whether this drug is excreted in human milk. Because many drugs are excreted in human milk and because of the potential for serious adverse reactions in nursing infants from AZOPT® (brinzolamide ophthalmic suspension) 1%, a decision should be made whether to discontinue nursing or to discontinue the drug, taking into account the importance of the drug to the mother.

8.4 Pediatric Use

A three-month controlled clinical study was conducted in which AZOPT® (brinzolamide ophthalmic suspension) 1% was dosed only twice a day in pediatric patients 4 weeks to 5 years of age. Patients were not required to discontinue their IOP-lowering medication(s) until initiation of monotherapy with AZOPT®. IOP-lowering efficacy was not demonstrated in this study in which the mean decrease in elevated IOP was between 0 and 2 mmHg. Five out of
32 patients demonstrated an increase in corneal diameter of one millimeter.

8.5 Geriatric Use

No overall differences in safety or effectiveness have been observed between elderly and younger patients.

10 OVERDOSAGE

Although no human data are available, electrolyte imbalance, development of an acidotic state, and possible nervous system effects may occur following oral administration of an overdose. Serum electrolyte levels (particularly potassium) and blood pH levels should be monitored.

13 NONCLINICAL TOXICOLOGY

13.1 Carcinogenesis, Mutagenesis, Impairment of Fertility

Carcinogenicity data on brinzolamide are not available. The following tests for mutagenic potential were negative: (1) *in vivo* mouse micronucleus assay; (2) *in vivo* sister chromatid exchange assay; and (3) Ames *E. coli* test. The *in vitro* mouse lymphoma forward mutation assay was negative in the absence of activation, but positive in the presence of microsomal activation. In reproduction studies of brinzolamide in rats, there were no adverse effects on the fertility or reproductive capacity of males or females at doses up to 18 mg/kg/day (375 times the recommended human ophthalmic dose).

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TRAVATAN Z®

(travoprost ophthalmic solution) 0.004%

BRIEF SUMMARY OF PRESCRIBING INFORMATION

INDICATIONS AND USAGE
TRAVATAN Z® (travoprost ophthalmic solution) 0.004% is indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

DOSAGE AND ADMINISTRATION
The recommended dosage is one drop in the affected eye(s) once daily in the evening.
TRAVATAN Z® (travoprost ophthalmic solution) should not be administered more than once daily since it has been shown that more frequent administration of prostaglandin analogs may decrease the intraocular pressure lowering effect.

Reduction of the intraocular pressure starts approximately 2 hours after the first administration with maximum effect reached after 12 hours.

TRAVATAN Z® Solution may be used concomitantly with other topical ophthalmic drug products to lower intraocular pressure. If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes apart.

CONTRAINDICATIONS
None

WARNINGS AND PRECAUTIONS
Pigmentation
Travoprost ophthalmic solution has been reported to cause changes to pigmented tissues. The most frequently reported changes have been increased pigmentation of the iris, periorbital tissue (eyelid) and eyelashes. Pigmentation is expected to increase as long as travoprost is administered. The pigmentation change is due to increased melanin content in the melanocytes rather than to an increase in the number of melanocytes. After discontinuation of travoprost, pigmentation of the iris is likely to be permanent, while pigmentation of the periorbital tissue and eyelash changes have been reported to be reversible in some patients. Patients who receive treatment should be informed of the possibility of increased pigmentation. The long term effects of increased pigmentation are not known.

Iris color change may not be noticeable for several months to years. Typically, the brown pigmentation around the pupil spreads concentrically towards the periphery of the iris and the entire iris or parts of the iris become more brownish. Neither nevi nor freckles of the iris appear to be affected by treatment. While treatment with TRAVATAN Z® (travoprost ophthalmic solution) 0.004% can be continued in patients who develop noticeably increased iris pigmentation, these patients should be examined regularly.

Eyelash Changes
TRAVATAN Z® Solution may gradually change eyelashes and vellus hair in the treated eye. These changes include increased length, thickness, and number of lashes. Eyelash changes are usually reversible upon discontinuation of treatment.

Intraocular Inflammation
TRAVATAN Z® Solution should be used with caution in patients with active intraocular inflammation (e.g., uveitis) because the inflammation may be exacerbated.

Macular Edema
Macular edema, including cystoid macular edema, has been reported during treatment with travoprost ophthalmic solution. TRAVATAN Z® Solution should be used with caution in aphakic patients, in pseudophakic patients with a torn posterior lens capsule, or in patients with known risk factors for macular edema.

Angle-closure, Inflammatory or Neovascular Glaucoma
TRAVATAN Z® Solution has not been evaluated for the treatment of angle-closure, inflammatory or neovascular glaucoma.

Bacterial Keratitis
There have been reports of bacterial keratitis associated with the use of multiple-dose containers of topical ophthalmic products. These containers had been inadvertently contaminated by patients who, in most cases, had a concurrent corneal disease or a disruption of the ocular epithelial surface.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

ADVERSE REACTIONS
Clinical Studies Experience
Because clinical studies are conducted under widely varying conditions, adverse reaction rates observed in the clinical studies of a drug cannot be directly compared to rates in the clinical studies of another drug and may not reflect the rates observed in practice. The most common adverse reaction observed in controlled clinical studies with TRAVATAN® (travoprost ophthalmic solution) 0.004% and TRAVATAN Z® (travoprost ophthalmic solution) 0.004% was ocular hyperemia which was reported in 30 to 50% of patients. Up to 3% of patients discontinued therapy due to conjunctival hyperemia. Ocular adverse reactions reported at an incidence of 5 to 10% in these clinical studies included decreased visual acuity, eye discomfort, foreign body sensation, pain and pruritus. Ocular adverse reactions reported at an incidence of 1 to 4% in clinical studies with TRAVATAN® or TRAVATAN Z® Solutions included abnormal vision, blepharitis, blurred vision, cataract, conjunctivitis, corneal staining, dry eye, iris discoloration, keratitis, lid margin crusting, ocular inflammation, photophobia, subconjunctival hemorrhage and tearing.

Nonocular adverse reactions reported at an incidence of 1 to 5% in these clinical studies were allergy, angina pectoris, anxiety, arthritis, back pain, bradycardia, bronchitis, chest pain, cold/flu syndrome, depression, dyspepsia, gastrointestinal disorder, headache, hypercholesterolemia, hypertension, hypotension, infection, pain, prostate disorder, sinusitis, urinary incontinence and urinary tract infections.

In postmarketing use with prostaglandin analogs, periorbital and lid changes including deepening of the eyelid sulcus have been observed.

USE IN SPECIFIC POPULATIONS
Pregnancy
Pregnancy Category C
Teratogenic effects: Travoprost was teratogenic in rats, at an intravenous (IV) dose up to 10 mcg/kg/day (250 times the maximal recommended human ocular dose (MRHOD), evidenced by an increase in the incidence of skeletal malformations as well as external and visceral malformations, such as fused sternebrae, domed head and hydrocephaly. Travoprost was not teratogenic in rats at IV doses up to 3 mcg/kg/day (75 times the MRHOD), or in mice at subcutaneous doses up to 1 mcg/kg/day (25 times the MRHOD). Travoprost produced an increase in post-implantation losses and a decrease in fetal viability in rats at IV doses > 3 mcg/kg/day (75 times the MRHOD) and in mice at subcutaneous doses > 0.3 mcg/kg/day (7.5 times the MRHOD).

In the offspring of female rats that received travoprost subcutaneously from Day 7 of pregnancy to lactation Day 21 at doses of ≥ 0.12 mcg/kg/day (3 times the MRHOD), the incidence of postnatal mortality was increased, and neonatal body weight gain was decreased. Neonatal development was also affected, evidenced by delayed eye opening, pinna detachment and preputial separation, and by decreased motor activity.

There are no adequate and well-controlled studies of TRAVATAN Z® (travoprost ophthalmic solution) 0.004% administration in pregnant women. Because animal reproductive studies are not always predictive of human response, TRAVATAN Z® Solution should be administered during pregnancy only if the potential benefit justifies the potential risk to the fetus.

Nursing Mothers
A study in lactating rats demonstrated that radiolabeled travoprost and/or its metabolites were excreted in milk. It is not known whether this drug or its metabolites are excreted in human milk. Because many drugs are excreted in human milk, caution should be exercised when TRAVATAN Z® Solution is administered to a nursing woman.

Pediatric Use
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

Geriatric Use
No overall clinical differences in safety or effectiveness have been observed between elderly and other adult patients.

Hepatic and Renal Impairment
Travoprost ophthalmic solution 0.004% has been studied in patients with hepatic impairment and also in patients with renal impairment. No clinically relevant changes in hematology, blood chemistry, or urinalysis laboratory data were observed in these patients.

NONCLINICAL TOXICOLOGY
Carcinogenesis, Mutagenesis, Impairment of Fertility
Two-year carcinogenicity studies in mice and rats at subcutaneous doses of 10, 30, or 100 mcg/kg/day did not show any evidence of carcinogenic potential. However, at 100 mcg/kg/day, male rats were only treated for 82 weeks, and the maximum tolerated dose (MTD) was not reached in the mouse study. The high dose (100 mcg/kg) corresponds to exposure levels over 400 times the human exposure at the maximum recommended human ocular dose (MRHOD) of 0.04 mcg/kg, based on plasma active drug levels. Travoprost was not mutagenic in the Ames test, mouse micronucleus test or rat chromosome aberration assay. A slight increase in the mutant frequency was observed in one of two mouse lymphoma assays in the presence of rat S-9 activation enzymes.

Travoprost did not affect mating or fertility indices in male or female rats at subcutaneous doses up to 10 mcg/kg/day [250 times the maximum recommended human ocular dose of 0.04 mcg/kg/day on a mcg/kg basis (MRHOD)]. At 10 mcg/kg/day, the mean number of corpora lutea was reduced, and the post-implantation losses were increased. These effects were not observed at 3 mcg/kg/day (75 times the MRHOD).

PATIENT COUNSELING INFORMATION
Potential for Pigmentation
Patients should be advised about the potential for increased brown pigmentation of the iris, which may be permanent. Patients should also be informed about the possibility of eyelid skin darkening, which may be reversible after discontinuation of TRAVATAN Z® (travoprost ophthalmic solution) 0.004%.

Potential for Eyelash Changes
Patients should also be informed of the possibility of eyelash and vellus hair changes in the treated eye during treatment with TRAVATAN Z® Solution. These changes may result in a disparity between eyes in length, thickness, pigmentation, number of eyelashes or vellus hairs, and/or direction of eyelash growth. Eyelash changes are usually reversible upon discontinuation of treatment.

Handling the Container
Patients should be instructed to avoid allowing the tip of the dispensing container to contact the eye, surrounding structures, fingers, or any other surface in order to avoid contamination of the solution by common bacteria known to cause ocular infections. Serious damage to the eye and subsequent loss of vision may result from using contaminated solutions.

When to Seek Physician Advice
Patients should also be advised that if they develop an intercurrent ocular condition (e.g., trauma or infection), have ocular surgery, or develop any ocular reactions, particularly conjunctivitis and eyelid reactions, they should immediately seek their physician's advice concerning the continued use of TRAVATAN Z® Solution.

Use with Contact Lenses
Contact lenses should be removed prior to instillation of TRAVATAN Z® Solution and may be reinserted 15 minutes following its administration.

Use with Other Ophthalmic Drugs
If more than one topical ophthalmic drug is being used, the drugs should be administered at least five (5) minutes between applications.

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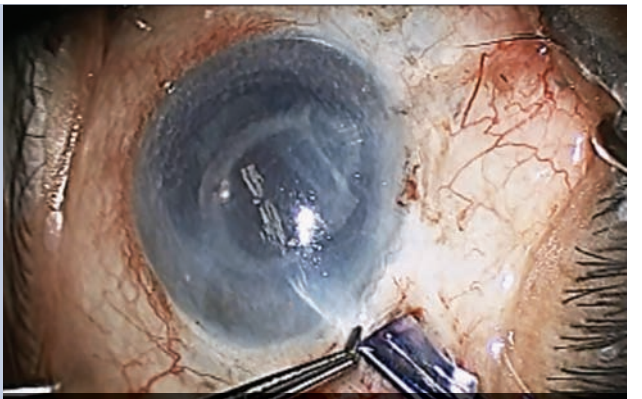
4/12 TRV12070JAD

DSAEK

Descemet's stripping automated endothelial keratoplasty has changed the landscape of corneal transplant surgery—improving outcomes, hastening recovery and reducing complications.

By Derek N. Cunningham, O.D., and Walter O. Whitley, O.D., M.B.A.



DSAEK is performed through a 5mm incision, substantially less invasive than the 360° incision created in PK.

Go to www.revoptom.com to see video footage of this innovative corneal transplant procedure.

On The Web ›› View a narrated video of DSAEK performed on an endothelial dystrophy patient.

Optometrists play a key role in diagnosing and managing guttata and Fuchs' dystrophy. Previously, when conventional treatments (e.g., sodium chloride drops, aggressive ocular surface disease therapy, heat therapy) couldn't adequately manage the condition, our only surgical option was a penetrating keratoplasty. Fortunately, corneal transplant surgery has been evolving continuously from a full-thickness procedure to several partial-thickness surgeries—chief among them Descemet's stripping automated endothelial keratoplasty (DSAEK). It has revolutionized surgical intervention for endothelial dysfunction because it leaves the anterior cornea structurally intact, allows faster healing and VA recovery, reduces graft rejection risk, lessens post-op astigmatism and allows for stronger wound integrity.

DSAEK is a sutureless transplant of the posterior cornea that replaces only the diseased portion with a donor graft. This 45-minute procedure can be performed alone or in combination with cataract surgery.

The endothelium is stripped and scored around the peripheral stroma to promote adhesion of the donor tissue. Next, the surgeon prepares the donor tissue, which is measured, centered and trephinated to ensure proper sizing. The donor graft is created by either manual dissection using a microkeratome (DSEK) or automated dissection with a femtosecond laser (DSAEK). The graft—typically about 150µm thick—consists of posterior stroma, Descemet's membrane and corneal endothelium.

Viscoelastic is placed on the donor endothelium to protect it during insertion. The donor tissue is folded and inserted using specialty forceps to minimize insult to the delicate tissue. The surgeon centers the graft with an injection of balanced saline, then fills the entire anterior chamber with sterile air to aid graft attachment. Additional surgical measures taken include peripheral iridectomies and properly sized air tamponades to prevent pupillary block glaucoma. Once the graft is stabilized, the wound is sealed and closed, and a pressure patch is secured.

On the one-day post-op exam, vision is approximately <20/200. IOP is measured and the peripheral iridectomy is checked for patency. Mild to moderate corneal edema may be present. The anterior chamber should be filled with 30% air. Patients are encouraged to remain supine for several days to promote graft adherence. The follow-up schedule typically is one day, one week, then monthly to ensure proper healing and recovery. At six months, the majority of patients see >20/40.

DSAEK has been a promising development in corneal surgery, and the procedure is constantly evolving. Some surgeons are starting to use thinner grafts (Descemet's membrane endothelial keratoplasty, or DMEK), which improve visual outcomes but require a more technically challenging procedure. In the meantime, DSAEK is the current standard for endothelial disease. As optometrists, it is important for us to understand the latest surgical procedures so we can educate and treat our patients accordingly. ■

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Diagnostic **Quiz**

Blunt Trauma Drama

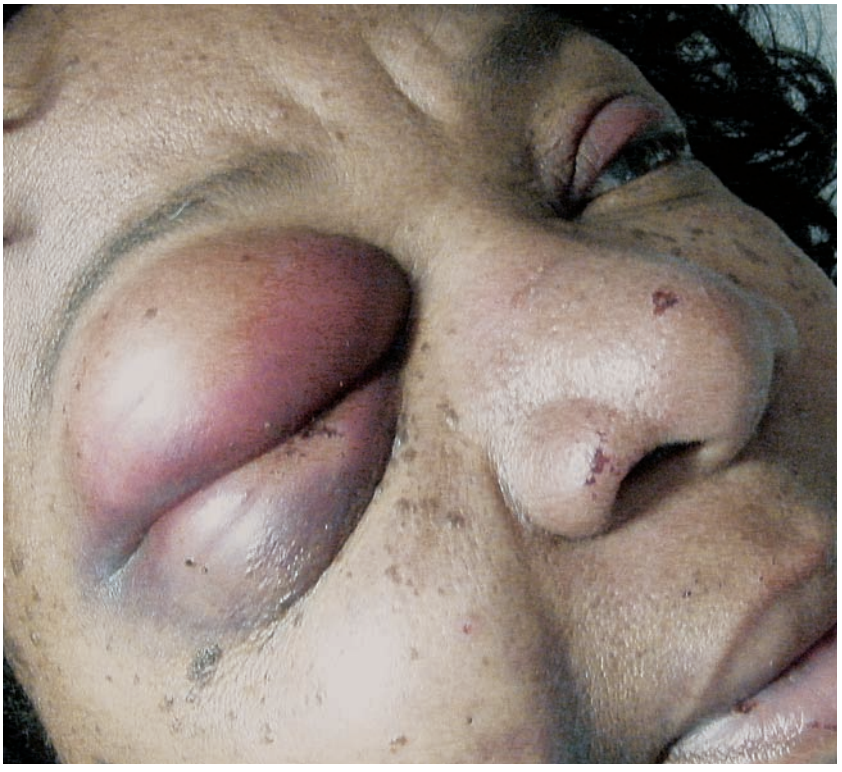
By Andrew S. Gurwood, O.D.

History

A 57-year-old black female was admitted to the hospital after being struck by an automobile. The intensive care unit requested an ocular consult, which was prompted by marked swelling of her right eye that had persisted for three days.

Her chart revealed the presence of an orbital floor fracture without dislocation or apparent entrapment. The patient explained that she could not see because she was unable to open her eye. However, she did not complain of exceptional pain, and reported discomfort only upon upward gaze.

Her systemic history was significant for hypertension, for which she was properly medicated. Her ocular history was significant for bilateral cataracts. She had no known allergies.



External examination of our patient showed evidence of substantial bruising.

Diagnostic Data

Her best-corrected visual acuity measured 20/20 O.U. at distance and near. External examination revealed ecchymosis of the right eye, with a palpable soft edema and painful area on the inferior temporal orbital rim.

There was no evidence of afferent pupillary defect or visual field involvement. The anterior segment findings were normal. Her intraocular pressure measured 14mm Hg O.U. Dilated fundoscopy was within normal limits O.U.

Your Diagnosis

How would you approach this case? Does this patient require any additional tests? What is your diagnosis? How would you manage this patient? What's the likely prognosis?

To find out, visit www.revoptom.com. Click on the cover icon for this month's issue, and then click "Diagnostic Quiz" under the table of contents. ■

Retina Quiz Answers *(from page 84):* 1) a; 2) d; 3) c; 4) a.

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When night falls, IOP rises.[1-3]

AZOPT® Suspension, an adjunctive partner to a PGA that has IOP-lowering efficacy all day and all night[4]

INDICATIONS AND USAGE
AZOPT® Brinzolamide Ophthalmic Suspension 1% is a carbonic anhydrase inhibitor indicated in the treatment of elevated intraocular pressure in patients with ocular hypertension or open-angle glaucoma.

DOSAGE AND ADMINISTRATION
- Instill one drop in the affected eye(s) three times daily
- If more than one topical ophthalmic drug is being used, the drugs should be administered at least ten (10) minutes apart

IMPORTANT SAFETY INFORMATION

CONTRAINDICATIONS
- Hypersensitivity to any component of this product

WARNINGS AND PRECAUTIONS
- Sulfonamide hypersensitivity reactions
- Corneal edema may occur in patients with low endothelial cell counts

ADVERSE REACTIONS
Most common adverse reactions are blurred vision and bitter, sour or unusual taste.

Before prescribing AZOPT® Suspension, please read full prescribing information on adjacent page.

References:
1. Liu JHK, Weinreb RN. Monitoring intraocular pressure for 24 h. *Br J Ophthalmol*. doi:10.1136/bjo.2010.199737. **2.** Bagga H, Liu JH, Weinreb RN. Intraocular pressure measurements throughout the 24 h. *Curr Opin Ophthalmol*. 2009;20(2):79-83. **3.** Liu JHK, Zhang X, Kripke DF, Weinreb RN. Twenty-four-hour intraocular pressure pattern associated with early glaucomatous changes. *Invest Ophthalmol Vis Sci*. 2003;44(4):1586-1590. **4.** Liu JHK, Medeiros FA, Slight JR, Weinreb RN. Comparing diurnal and nocturnal effects of brinzolamide and timolol on intraocular pressure in patients receiving latanoprost monotherapy. *Ophthalmology*. 2009;116(3):449-454.

Azopt® (brinzolamide ophthalmic suspension) 1%

Alcon®

7 STUDIES
1,563 PATIENTS
1 POOLED RESULT

30% SUSTAINED

IOP lowering for up to a full day[1]

FOR ALL YOUR PATIENTS NEEDING A PGA CONSIDER
BAK-FREE TRAVATAN Z® SOLUTION

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INDICATIONS AND USAGE:
TRAVATAN Z® Solution is a prostaglandin analog indicated for the reduction of elevated intraocular pressure in patients with open-angle glaucoma or ocular hypertension.

Dosage and Administration:
One drop in the affected eye(s) once daily in the evening.

IMPORTANT SAFETY INFORMATION

Warnings and Precautions:
Pigmentation: Pigmentation of the iris, periorbital tissue (eyelid) and eyelashes can occur. Iris pigmentation likely to be permanent.

Eyelash Changes: Gradual change to eyelashes including increased length, thickness and number of lashes. Usually reversible.

Adverse Reactions:
Most common adverse reaction (30% to 50%) is conjunctival hyperemia.

Use In Specific Populations:
Use in pediatric patients below the age of 16 years is not recommended because of potential safety concerns related to increased pigmentation following long-term chronic use.

For additional information please refer to the accompanying brief summary of prescribing information on adjacent page.

Reference:
1. Dubiner HB, Noecker R. Sustained intraocular pressure reduction throughout the day with travoprost ophthalmic solution 0.004%. *Clin Ophthalmol.* 2012;6:525-531.

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TRAVATAN Z®
(travoprost ophthalmic solution) 0.004%