Persistence and Adherence in the Management of Glaucoma

New Views on Angle Closure Glaucoma

A Summary of the Optometric Glaucoma Society's Fifth Annual Meeting

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### Table of Contents

<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>Probing the Distinguishing Features that Separate Life and Death in RGCs</td>
<td>Andrew Hartwick, OD, PhD</td>
</tr>
<tr>
<td>4</td>
<td>High Hopes for Memantine</td>
<td>William Hare, OD, PhD</td>
</tr>
<tr>
<td>5</td>
<td>Animal Models Could Improve Glaucoma Diagnosis</td>
<td>Ronald S. Harwerth, OD, PhD</td>
</tr>
<tr>
<td>6</td>
<td>Compliance: It’s Worse Than You Think</td>
<td>Harry A. Quigley, MD</td>
</tr>
<tr>
<td>7</td>
<td>Exploring Solutions to the Compliance Problem</td>
<td>David S. Friedman, MD, MPH</td>
</tr>
<tr>
<td>8</td>
<td>Four Steps to Identify Nonadherence</td>
<td>Steven R. Hahn, MD</td>
</tr>
<tr>
<td>9</td>
<td>A Plan of Attack for Better Adherence</td>
<td>Harry A. Quigley, MD</td>
</tr>
<tr>
<td>10</td>
<td>New Views on the Mechanics of Angle-Closure Glaucoma</td>
<td>Harry A. Quigley, MD</td>
</tr>
<tr>
<td>14</td>
<td>The Case for Anterior Segment OCT</td>
<td>David S. Friedman, MD, MPH</td>
</tr>
</tbody>
</table>

### Introductory Remarks

We are pleased to bring you the proceedings of the Fifth Annual Meeting of the Optometric Glaucoma Society, held in Denver, CO, on December 6, 2006. This year’s meeting honored the many contributions of Harry Quigley, MD. Professor Quigley discussed topics ranging from the correlation between the retinal nerve fiber layer and perimetry, to new thoughts on the pathogenesis of angle closure glaucoma, to a discussion of the issues surrounding patient compliance with medical therapy. Other speakers included Balwantray Chauhan, PhD, David Friedman, MD, Steven Hahn, MD, Ronald Harwerth, PhD and William Hare, OD, PhD. Our travel award recipient was Andrew Hartwick, OD, PhD.

Our speakers broke new ground and stimulated lively discussion, and we believe that these proceedings transmit much of what was exciting about this meeting. Read Dr. Harwerth’s description of his experimental glaucoma model, and how he is challenging and indeed changing conventional wisdom—right now—about the relationship between structural and functional damage in glaucoma. Who would have guessed a few years ago that detectable functional loss often occurs before detectable structural change?

Learn from Drs. Friedman and Quigley how angle-closure glaucoma (ACG) is being recognized as a global disease and how ACG may present chameleon-like signs and symptoms that can be challenging to both diagnose and treat.

In these proceedings, Drs. Friedman, Hahn and Quigley describe their research on patient compliance and adherence with prescribed medical therapy for glaucoma. How many of us knew that fewer than half of all patients who first fill a prescription for a glaucoma medication are still taking that medication 6 months later? They also explain how patient adherence can and must be continuously addressed by the clinician, how it is almost impossible for doctors to predict which of their patients are properly using their medications and the crucial importance of doctor-patient communication. Learn Dr. Hahn’s four-step approach for assessing patient adherence. We think that it will change the way you practice.

In closing I would like to thank this year’s speakers for taking time from their busy schedules to share their wisdom with us. I would also like to thank John Flanagan, Program Chair, and John McSoley, Meeting Chair, for their hard work in putting this meeting together. I would also like to thank Mike Patella, OGS secretary, for his oversight of the meeting and programs in general. Finally, I would like to thank Pfizer, Inc. and its team of Karen Fixler, Jill Burdge and Peter Zagorin for their support of the OGS and in particular, the meeting and supplement. I hope you enjoy this and find it useful.

Murray Fingeret, OD
President, Optometric Glaucoma Society
Editor, Supplement
Probing the Distinguishing Features that Separate Life and Death in RGCs
Does elevated IOP disrupt glutamate clearance in the retina?

BY ANDREW HARTWICK, OD, PhD

Nothing has the potential to change glaucoma practice patterns like data from well-designed clinical trials. However, as we all know, such trials are time-consuming and expensive. Smaller-scale in vitro research on isolated tissues or cells may lack the immediate impact of larger trials, but they do offer flexibility, low cost, rapid results—and perhaps most importantly—the ability to better control and manipulate variables in order to provide initial assessments of novel theories and treatment strategies. In my research, I have been using in vitro techniques to study the cellular basis of retinal ganglion cell (RGC) dysfunction. As an example of this line of research, here I will discuss the effects of glutamate on cultured RGCs (complete isolation from all other retinal cells) and on RGCs from dissected retinas (retinal architecture remains intact).

As the primary excitatory neurotransmitter for RGCs, glutamate is necessary for healthy retinal function, but prolonged exposure to glutamate is toxic to many neurons in the central nervous system. Strong evidence implicates a role for glutamate excitotoxicity in acute ischemic pathologies such as stroke. More controversial but intriguing nonetheless is glutamate’s potential involvement in various chronic neurodegenerative disorders like glaucoma. We sought a close-up view of what glutamate actually does to RGCs, and we specifically wanted to know what was happening to the calcium levels inside an RGC undergoing excitotoxic death. Calcium has long been thought of as a key trigger for glutamate excitotoxicity, because classic studies on cultured brain neurons showed that excitotoxic death could be completely prevented by removing calcium ions from the extracellular environment.

We monitored the amount of calcium present in cultured rat RGCs using calcium imaging techniques. Following loading with fura-2 calcium indicator dye, an RGC stimulated with glutamate exhibits a rise in the fura-2 ratio, which is indicative of a rise in internal calcium levels, that then returns to baseline as glutamate is washed away. Using this method, we determined that the vast majority of calcium influx in RGCs was mediated through NMDA-type glutamate receptors, as opposed to AMPA/kainate-type glutamate receptors.

What happens to the calcium levels in RGCs exposed to glutamate for prolonged periods? One might expect that the calcium levels might continually rise and that if a certain threshold was achieved, the cell would then die. However, our studies showed that when glutamate is introduced, there is a swift rise in calcium that then decreases slightly. Many cells then maintained relatively stable calcium levels for the duration of the treatment before returning to baseline levels with the removal of glutamate. However, in some cells, the calcium homeostasis was abruptly lost and there was a second large rise in the calcium signal during the glutamate treatment. The secondary calcium rise was irreversible. The cells showed no recovery upon glutamate washout. This phenomenon has been termed “delayed calcium deregulation,” or DCD, and it was shown to be a key indicator of glutamate excitotoxicity—those undergoing DCD died; those that did not lived. Although DCD has been described in other brain neurons, ours is its first evidence in a retinal cell.

In further experiments, we found that a 1-hour exposure to glutamate induced DCD in 18-28% of RGCs. Concentrations of glutamate did not seem to matter much, because we found similar levels of DCD incidence in 10, 100 and 1,000 µM exposures. Our findings suggested that it is extended exposure periods, and not brief contact with high concentrations, that triggers glutamate’s toxic effects on RGCs. The magnitude of the initial calcium spike was linked to the relative vulnera-
bility of RGCs to excitotoxicity, as RGCs that displayed larger calcium responses were more likely to undergo DCD. We therefore hypothesized that by reducing the magnitude of the glutamate-induced calcium influx, we would reduce the risk of DCD. Indeed, by including an NMDA antagonist in the treatment solution, the calcium responses were smaller and the incidence of DCD was largely prevented.

In the living retina, extracellular levels of glutamate are extremely well-regulated, ensuring that RGCs are not exposed to glutamate for long under normal physiological conditions. While the study of cultured RGCs is a useful system to understand what happens to these neurons when they are challenged with glutamate, it is not a good system in which to study why or how an RGC would be stimulated with glutamate for an extended period in vivo. We therefore imaged RGCs from whole retinas that had been dissected from rats. In these fundamentally intact retinas, glutamate is much less effective than its related agonists NMDA or kainate at inducing calcium influx in RGCs because there are transporters present that recognize and remove extracellular glutamate (NMDA and kainate are not naturally present in the retina and are not recognized by these transporters). RGCs responded to lower glutamate levels if the glutamate transporters were pharmacologically inhibited. Given the efficiency of glutamate clearance in the retina, it follows that there must be an accompanying defect in glutamate uptake in order for a chronic elevation in extracellular glutamate levels to occur. Ischemia, for example, is known to diminish transporter function, thus leading to elevated glutamate levels and subsequent excitotoxicity.

With respect to glaucoma, we were interested in utilizing our imaging technique to probe the question of whether elevated IOP causes a long-term disruption of glutamate clearance. We chronically elevated IOP in one eye of rats (by blocking aqueous outflow through hypertonic saline injections into an episcleral vein) and then dissected out the retinas and imaged RGC calcium responses to glutamate. We found no evidence for disrupted glutamate uptake in rats with IOP-related optic nerve damage, indicating that a chronic rise in extracellular glutamate levels was unlikely to be the primary cause of RGC death observed in this animal glaucoma model. Nevertheless, the ability to image RGCs from retinas that had been exposed to high IOP should prove valuable for future research on the pathogenesis of glaucomatous RGC death.

It should be noted that applications of these in vitro techniques are not limited to research on excitotoxicity; these approaches could be utilized to better characterize the sequence of changes that occur to RGCs following various insults (one example of potential relevance to glaucoma would be neurotrophin deprivation). It is my hope that, in conjunction with psychophysical studies dedicated to detecting functional changes in RGCs prior to their death, future glaucoma research will focus on the cellular and molecular mechanisms that distinguish RGC function from RGC dysfunction. The ability to identify “sick” RGCs before they die would likely not only improve glaucoma detection, but it would aid in the development of neuroprotection therapies that attempt to rescue injured neurons.

Reference:

AFTER RECEIVING HIS OD AND MSC IN VISION SCIENCE FROM THE UNIVERSITY OF WATERLOO, DR. HARTWICK COMPLETED HIS PHD IN ANATOMY & NEUROBIOLOGY AT DALHOUSE UNIVERSITY (HALIFAX, NOVA SCOTIA, CANADA). THE WORK DESCRIBED ABOVE COMPRISSES A PORTION OF HIS PHD RESEARCH AND WAS SUPPORTED BY THE CANADIAN INSTITUTES OF HEALTH RESEARCH, THE CANADIAN NATIONAL INSTITUTE FOR THE BLIND, AND BY A BAUSCH & LOMB SPONSORED EZELL FELLOWSHIP FROM THE AMERICAN OPTOMETRIC FOUNDATION.

High Hopes for Memantine
Can this neuroprotective agent stop glaucoma damage?

BY WILLIAM HARE, OD, PhD

As a research scientist at Allergan for the last 13 years, I have been involved in the preclinical evaluation of memantine, an N-methyl-D-aspartate (NMDA) type glutamatergic ion channel blocker that is currently being evaluated in a multicenter clinical trial for the prevention of glaucomatous injury to retinal neurons. This study represents the first clinical test of a neuroprotective approach to glaucoma treatment, and it is expected that preliminary results of this study will be published within the next year.

A neuroprotective therapy represents a completely novel approach for the treatment of glaucoma and may be distinguished from currently prescribed treatments that are approved for the reduction of intraocular pressure; a primary risk factor for development of glaucomatous vision loss. Although the specific mechanism(s) for glaucomatous injury is (are) unknown, considerable evidence suggests that excessive activity of NMDA-type glutamatergic ion channels may contribute significantly to neuronal injury in glaucoma as well as other forms of chronic neurodegenerative disease. Memantine treatment has been shown to be effective to reduce injury in animal models of central nervous system (CNS) disease, including experimental glaucoma, and has been approved for the treatment of Alzheimer’s disease. The rationale for memantine treatment is based not only on the considerable evidence that NMDA-type glutamatergic channels contribute significantly to neuronal injury in neurodegenerative disease including glaucoma, but also on the properties for memantine-mediated block of these ion channels.

Glutamate is the principal chemical transmitter at excitatory synapses in the CNS, including the retina. Thus, under normal conditions, glutamate is a necessary component for neuronal signaling. However, excessive activity of glutamate-gated channels can result in neuronal injury and death. There are two general classes of glutamate-gated ion channels in the CNS: the NMDA-type and the non-NMDA-type. For both classes, glutamate is the normal endogenous ligand for channel activation but they may be distinguished by their selectivity for molecules that can bind to and activate the channel. NMDA-type channels are activated selectively by NMDA while non-NMDA-types are activated selectively by either kainate or AMPA. Neurons may be injured by overactivity of either subtype. However, due to the high calcium permeability of NMDA-type channels, neurons are particularly sensitive to injury resulting from excessive activity of this channel subtype.
Memantine acts selectively to block injury associated with excessive activity of NMDA-type channels. The memantine molecule does not compete with glutamate for its binding site on the channel but instead binds to a different site within the channel to prevent the flow of calcium and sodium ions from the extracellular space to the neuronal cytoplasm. In fact, since memantine binds within the channel pore, glutamate must first bind to its receptor site to "open" the channel before memantine can enter the channel and block the pore. This "open-channel" blocking action, in combination with other properties that influence the interaction of memantine with its binding site, means that memantine will be most effective to reduce NMDA-type channel activity under pathological conditions when neurons are depolarized and extra-cellular glutamate concentrations are high but relatively less effective to block "normal" levels of NMDA channel activity. This safety profile is evident in experimental results that show efficacy for prevention of neuronal injury at memantine levels that have little or no effect on normal neuronal function.

It is known that both NMDA and non-NMDA type channels are expressed in retinal ganglion cells (RGCs). It is also known that RGCs are injured by exposure to high levels of exogenously applied glutamate and are particularly sensitive to exposure to NMDA. While there is no direct evidence that glutamate-mediated RGC injury is a contributing factor in glaucomatous vision loss, there is considerable indirect evidence to support this hypothesis. NMDA-type glutamatergic channels have been implicated in mechanisms for neuronal injury in chronic neurodegenerative diseases such as amyotrophic lateral sclerosis (Lou Gehrig’s disease), Parkinson’s disease and Alzheimer’s disease. There is also evidence for dysregulation of retinal glutamate buffering in animal models of experimental glaucoma. Of particular importance are results showing that treatment with selective blockers of NMDA-type channels is effective to prevent RGC injury in experimental glaucoma. Moreover, memantine treatment has been shown to be protective in a wide range of models for CNS injury, including the retina and optic nerve. Examples of animal models in which memantine treatment has been shown to be protective include: cerebral ischemia (rat), traumatic brain injury (rat), Alzheimer’s disease (mouse, rat), acute retinal ischemia (rat, rabbit), optic nerve ischemia (rabbit), traumatic optic nerve injury (rat), spontaneous glaucoma (DBA/2J mouse), and chronic experimental glaucoma (rat, monkey).

In summary, there is considerable evidence to support the notion that NMDA-type glutamatergic channels contribute to mechanisms for glaucomatous injury of RGCs and that memantine may provide a safe and effective treatment for the prevention of this injury. However, the success of any clinical trial for evaluation of a neuroprotective treatment will ultimately be limited by the natural history of the disease as well as the clinical measures used to assess treatment efficacy. Glaucoma is a slowly progressing neurodegenerative disorder and the currently accepted standard for determining the presence or progression of glaucomatous vision loss (white-on-white perimeter) does not provide a reliable measure for small changes in RGC function. Consequently, glaucoma neuroprotection trials must be of long duration and include many patients. In addition to making treatment efficacy more difficult to demonstrate, the high cost of such trials will effectively limit the number of novel treatments available to glaucoma patients. For this reason, considerable effort is being devoted to the development and validation of better measures for glaucomatous injury. These efforts can be expected to provide better tools for the diagnosis and management of glaucoma patients as well as more effective treatments for prevention of glaucomatous vision loss.

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Animal Models Could Improve Glaucoma Diagnosis
Function linked to structure in monkeys.

BY RONALD S. HARWERTH, OD, PhD

All studies of the structural and functional properties of macaque monkeys’ visual systems have demonstrated a strong similarity to those same properties in humans. At the University of Houston College of Optometry, we spend a lot of time scrutinizing glaucoma models in these animals, and the similarity to humans has led to the creation of models that reduce experimental variability, and, we believe, could develop into procedures to analyze typical patient data.

To induce experimental glaucoma in monkeys, we restrict the outflow of aqueous humor via laser scarification of the drainage angle, resulting in fairly high IOP very quickly, roughly in the 30 to 40 mmHg range. We then track the effects of glaucoma with behavioral perimetry. In a primate testing cubical equipped with a perimeter, the monkeys are trained to fixate and respond to central and peripheral visual stimuli. In due course, the animals can generate perimetric data indistinguishable from a clinical patient’s.

As one would expect, when we looked at the retinas of the monkeys with experimental glaucoma, there was a correlation between loss of visual sensitivity and loss of retinal ganglion cells. More importantly, however, we also discovered that with some relatively simple logarithmic transformations (one equation to find the intercept and another to determine the slope of the function) we could predict the amount of ganglion cell loss based solely on perimetric data.

Naturally we were interested to see if this predictive model would carry over to humans. To determine this, we consulted a 2000 study of 17 eyes from 13 glaucoma patients’ visual field data collected in the final 2 years of their lives.’ Our model proved significantly predictive. A few errors arose, but they were caused by variants in data generated outside our model, we concluded.

These experiments were so successful that we decided to try another commonly used diagnostic glaucoma measure: optical coherence tomography (OCT), which measures the thickness of the retinal nerve fiber layer. Does OCT correlate to the perimetric findings? Though our histology work on this question remains incomplete, preliminary data suggests the answer is yes. We reached this conclusion by devising a method for matching retinal nerve fiber layer thickness to ganglion cell density in equally measured sections of the retina.

The first step of this method was dividing the optic nerve head (ONH) and retinal nerve fiber layer (RNFL) into 10 equal sectors. Then the total number of axons in each RNFL sector was determined. Finally, visual field locations associated with each ONH sector were varied to determine the best agreement between ganglion cells and axons. Sure enough, a correlation between estimates of the number of gan-
Application From Monkeys to Humans
Similar retinal ganglion cell densities for monkeys and human observers result in similar visual sensitivities throughout the visual field. The field on the left was performed by a monkey and similar to the human visual field, as seen on the right.

glion cells and the number of axons was established.
Again, would it work on humans? To find out, some modifications were necessary. For example, a human eye has a longer axial length than a monkey’s, and we needed to reduce the average axon density in the RNFL of our human subjects, who were older than the monkey models. Though we are still in the early stages of this experiment, there does seem to be a significant degree of agreement between estimates of ganglion cells and axons in human subjects.
The applications of the above findings, we believe, are far-reaching and hold considerable merit. Our models link three fundamental aspects of glaucoma diagnosis: visual field sensitivity, OCT measurements and ganglion cell density—in both normal eyes and eyes with glaucomatous neuropathy. Visual field tests will remain the standard of care, but potential in everyday practice for augmentation of this test by the other two is substantial indeed.

Reference:

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Compliance: It’s Worse Than You Think
Appalling levels of non-adherence persist. Can it be helped?
HARRY A. QUIGLEY, MD

Twenty years ago, the eye-care world was stunned by a study aimed at determining how compliant glaucoma patients were in taking their drops. A novel-for-its-time computer in subjects’ bottles determined the frequency of drops used. Shocked clinicians read that patients in this study took only 80% of their prescribed doses of timolol. How disconcerting! Abundant discussion and hand wringing ensued, though little was done to address the problem. In weaker moments, we simply speculated, even hoped, that maybe the data were flawed—a few bad apples spoiling an otherwise well behaved barrel. Certainly our own patients would never be so remiss.
Since then, methods of assessing compliance have improved, but outcomes have not. In fact, given recent data, a 20% non-compliance rate is starting to look more like a best-case scenario, a cause for celebration. When one considers the effort, time, money, and scientific debate expended on developing new classes of pressure-lowering drugs—not to mention that a human being’s sight is at stake—real-world compliance rates are absolutely chilling.

Recent studies have confirmed what clinicians for years believed: lowering IOP decreases the risk of initial or progressive visual field loss. However, keep in mind that patients in these clinical trials differ considerably from the average patient population. Motivated enough to sign experimental consent forms, study subjects also receive free treatment and medication, and if they fail to show for visits, the study coordinator tracks them down and drags them in.

Outside a clinical trial setting, the situation is far less ideal. Many patients discontinue medication or take their eyedrops poorly.

In discussing or studying compliance, two terms often come into play: persistence and adherence. The persistence of the patient is the amount of time between the initial prescription and the first lapse in usage, even if usage resumes after that gap. In other words, the patient went on vacation and forgot to take his or her drops. This is a stringent measurement, since almost everyone occasionally forgets to take medications.

Adherence offers a more real-world measure. An adhering patient is one who refills the prescription at every 6-month interval. Gaps in usage may occur, but overall the patient is compliant.

Since the turn of the century, much of the research occurring in compliance issues has been done on large pharmacy claim databases. For example, Medicare and Blue Cross/Blue Shield databases involving millions of patients have been mined for information, usually in the form of pharmacy refill rates.

One study found that rates of persistence during the first year of IOP-lowering treatment were as poor as 10%. An older study of seniors receiving Medicaid in New Jersey found that 25% never even filled their second prescription. Cost was unlikely a factor because no one was paying more than $1 per prescription. And, in fact, many patients continued to refill other prescriptions for non-glaucomatous conditions.

We tell ourselves, “These are not my patients,” and in a sense, this statement is correct. We never observe such levels of defaulting because we track only those patients who call back for follow-up visits. The lamentable fact is 50% of newly diagnosed glaucoma patients receive no follow up care or visits to the offices over the next 15 months. Much of the lack of persistence and adherence in glaucoma treatment is associated with failure to show up for office visits, studies show.

Furthermore, we cannot trust what patients say. In a soon-to-be-published study I am involved in, when asked, 95% of patients claim to take their drops every day. However, we know that only about 65% actually do. On the whole, doctors overestimate patient compliance, judging it to be about 80%.

Obviously, eyecare has serious problems to address. If patients cannot be trusted, can we tell which ones are adherent? Can we as eyecare professionals improve adherence? I believe the answer to these questions is yes. Achieving such goals is dealt with by other authors in this supplement.

References:

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Exploring Solutions to the Compliance Problem
Patient adherence deserves the same rigor of study as glaucoma pathology itself.

BY DAVID S. FRIEDMAN, MD, MPH

If we are honest with ourselves, we must admit that in terms of diagnosis, care and management of glaucoma, we are doing a substandard job. Chiefly, our failures are large populations of patients going undiagnosed, and those who are diagnosed frequently do not receive IOP-lowering therapies that we prescribe.

Population-based research estimates there about 2.2 million people in the United States with glaucoma, and due to the aging demographics of our country, this number will rise to 3.5 million by the year 2020. It has been repeatedly found that in more developed societies, about half of those with glaucoma go undiagnosed, with some sub-populations even less likely to be diagnosed. For example, among Hispanics in the United States, over two-thirds of glaucoma cases were undiagnosed. In the developing world the percentage undiagnosed is much higher.

Adherence to therapy among glaucoma and hypertensive patients is poor. In a retrospective database review that we conducted, 3,600 newly diagnosed glaucoma patients and 1,700 suspects all belonging to the same insurance plan were assessed over a 6-year period. The majority did not maintain adherence over follow-up, even though they had filled at least one drug prescription. Fewer than 10% continuously refilled prescriptions. Study after study both on glaucoma as well as in other chronic, asymptomatic diseases reach similar conclusions. The system to provide therapy to patients, as it exists now, is simply not working.
That is what led us to get involved in the Glaucoma Adherence and Persistence Study (GAPS), an ongoing, large-scale investigation aimed at probing deeper into noncompliance issues and patient behaviors. We have looked at four sources of data on the same set of patients: retrospective pharmacy data, chart-abstracted information, interviews with the patients and interviews with physicians caring for these patients. The following are a few preliminary observations:

The most frequently cited barrier to maintaining drug adherence was simply “forgetting to use drops” (40%). Other barriers noted were concern about side effects (16%) and running out of drops unexpectedly (16%). Only 10% of the patients we surveyed admitted to failing to take drops as directed, but the average adherence was less than 70%. For the 10% who admitted to poor adherence, concern about side effects was cited by 57%, difficulty using drops correctly was cited by 36% and cost was cited by 25%.

People who cite cost as a barrier to taking medications tend to have lower adherence rates than those who do not, even when they are insured. If a patient admits that cost is a concern (and you may have to ask the patient this question directly), you should worry about that patient’s compliance. This kind of feedback might warrant steering these patients toward less expensive medications.

A perceived risk of reduced vision also appears to have a significant positive effect on compliance. Patients who understood their disease could cause blindness were more apt to take their medications. Also, patients who researched glaucoma on their own time had better adherence rates than those who relied solely on their doctors for information about the disease.

Phone call reminders to patients’ homes also seemed to bolster patient adherence to medical regimens to a greater degree than mailed postcard reminders.

Newly diagnosed patients seem to be at higher risk for noncompliance. The second prescription fill is a point where many patients tend to drop out. Thus, it may be beneficial to schedule these patients for an extra visit or two during those crucial early months of therapy—perhaps once every 3 months instead of every 6.

In terms of diagnosing patients, broadly screening large populations has not produced great success. Many patients are identified in this fashion, but only a fraction take the next step of coming to our offices for treatment. A better strategy probably involves identifying those most likely to have glaucoma, such as family members of existing glaucoma patients. This can be easily done in the office when we are evaluating patients with glaucoma. We should also make efforts to delve back into the files of patients who have dropped out of care and to make contact with them. Such a strategy could involve improved office systems for tracking patients and phone calls to remind them of appointments. Furthermore, we might also explore ways to work with pharmacies or drug manufacturers, entities that could notify doctors and patients when prescription refills are missed.

Other possible future solutions to the difficulties of adherence include the development of new medications that are easier to administer and last longer. We know, for example, that rates of compliance among patients on prostaglandin therapy are higher than those on other classes of drugs—probably due to their easier dosing schedules. Or we could pursue the option of eschewing drugs partially or altogether by relying more on devices and procedures. Internal stent devices such as the Gold wafer shunt and Trabectome have shown promise, as have procedures such as selective laser trabeculoplasty (SLT), but further research is needed.

Many of the approaches to increasing patient adherence are empirical, with little evidence supporting the efforts we as eye doctors make. The issue of patient adherence deserves increased study. Huge questions are in great need of investigation. We need better ways to measure compliance, and new studies of techniques to improve it. What really works? Patient education? Reminders? Increased monitoring of patient behavior? The answers to these questions are just as vital as the next generation of progression software, and offer tremendous opportunity for bright young researchers with an eye toward the future.

References:

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Four Steps to Identify Nonadherence
Going beyond simple yes and no questions.

BY STEVEN R. HAHN, MD

In the daily grind of providing health care, it may be easy to forget the role we play in patients’ lives. Whether we like it or not, they view us as authority figures, even when the patient is twice our age. And authority figures render judgments. Not unlike the child who wishes to gain a parent’s approval, patients want you to judge them as “good patients.” To disobey doctor’s orders constitutes what psychologists call “socially undesirable behavior,” something most people try to avoid.

This unique psychological mindset lies at the heart of much of the misinformation we receive. The desire for approval is more compelling than the consequences of providing misinformation, which is viewed as a harmless white lie. Our patients are not immoral people or con artists looking to pull the wool over our eyes. On the contrary, the motivation for providing misinformation stems from essentially moral impulses. They genuinely want to be good patients, but are afraid of failing that test.

This article will explore ways we can harness this aspect of patient psychology and get it to work for us rather than against us.
First, let us dispense with the notion that noncompliance is anything other than a universal problem. It is not just your patients or my patients; it’s everyone’s. And not only in glaucoma, either. Studies have shown that rates of noncompliance with therapeutic regimens are similar among all patients with chronic diseases. As an internist, I deal with this problem most often in hypertension.

In a study on 19 Type I diabetic patients receiving care in a diabetes research center, microchip recorders were surreptitiously inserted into the patients’ reflectance glucose meters. These are devices used for at-home blood-sugar monitoring, sometimes as often as 7 times a day. The patients knew that every aspect of their health-related behaviors was being studied at the center, but did not know about the microchip. After 2 weeks, we compared the values they had recorded in their log books with the actual results of tests performed.

Outcomes were striking: Two-thirds of the subjects obscured clinically significant hyper- or hypoglycemia. Mean reported glucose readings were lower than the actual readings in 75% of the subjects, and 40% of the log-book values were completely made up; the test had not been performed. In short, half the patients made up half the values written in their logbooks. Alarmed at this behavior, we even observed a few of our patients in the clinic parking lot writing numbers into their log books with different colored pens to create the impression entries had been made on different days. Furthermore, the physicians, myself included, had no better than chance probability of predicting who was fabricating or obscuring data. Though it comes as a surprise to many, a lack of clinician sensitivity to noncompliance is the rule, even in patients we know well.

So, how can we alleviate this problem? To illustrate my basic tactic, a thought experiment is helpful. Let us imagine a scenario in which we have failed to do all the required reading. The professor asks, “Did you all do the reading for today’s class?” Or, instead, imagine the professor says, “Last night I was looking at the syllabus we gave you. It’s a lot of reading. I imagine it could difficult to get it all done. How much were you able to do?” Under which scenario would you feel most comfortable confessing your omission? Yes, authority figures do judge, but they can also pardon. We can increase the likelihood that patients will be able to reveal problems with adherence using four steps:

1. Begin with a direct, open-ended question: “Tell me how you are taking your medications.” You will immediately find out if the patient has an understanding sufficient to support the correct use of medication. Follow up with questions about the strategies they may be using to help them remember and organize their medications. Never ask: “Are you taking your medication?” The answer to that question is almost always “yes.”

2. Reverse the judgmental environment. Acknowledge that non-adherence is pretty much universal and in some circumstances may actually be the smart thing to do (when adverse effects occur, for example). “It’s understandable you might forget or skip your medications because...” (cost, side effects, difficult to remember, etc.). Try to anticipate barriers to compliance. For example, if the patient has hyperemia, ask if this has ever caused social embarrassment and consequent reluctance to medicate.

3. The third step, creating a collaborative problem-solving relationship, is the key step. Make the patient aware that providing you with accurate information, sharing problems with adherence, is more important than making a positive impression by insisting that there have been no problems taking medicine. Help patients understand how self-reported information will be used. You might say, “Today your pressure is too high. Before we make any changes based on today’s reading, we need to be sure you’ve been able to take the medication the way we talked about. I wouldn’t want to add medicine if today’s pressure isn’t telling us what your prescribed dose can do.”

4. Only now should you ask about adherence: “So, have you missed any drops in the past week or today?” You want to hold off asking the patient to describe their adherence until you’ve maximized the odds that they’ll be able to tell you by using the preceding three steps. The last thing you want is patients prematurely insisting they’ve been adherent—if that is not the case, they now have to confess to a cover-up in addition to failure to take medication.

Once a patient is identified as noncompliant, resist the urge to deliver a sermon on glaucoma and the risk of blindness. Experience shows that we can educate patients more effectively when we first determine what they know and don’t know. Say to the patient, “Tell me what you know about glaucoma.” Basing your comments on what the patient tells you will prevent wasting time on what he or she already knows, and will allow you to correct for misunderstandings. Then explain the risk of noncompliance. After that, initiate second round of questions aimed at assessing how much of your information is getting through. This model is called “Ask-Tell-Ask,” and you may need to repeat it several times before the message is understood.

A key driver of adherence is the patient’s understanding of what might happen (vision loss) as a result of glaucoma. Patients can learn this from you, or from knowing someone who has lost vision from an eye disease. Taking responsibility for communicating this information is critical in the long-term management of chronic illnesses like glaucoma.

References:

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A Plan of Attack for Better Adherence

Though still in its infancy, compliance research can rely on a few basic observations.

BY HARRY A. QUIGLEY, MD

A conclusion reached and demonstrated by other articles in this supplement posits that compliance among glaucoma and ocular hypertensive patients is astonishingly poor, worse even than our most pessimistic forecasts. I will not belabor this point any further, other than to say I agree with it completely and have seen the data firsthand. What I
wish to do here is address the pragmatic clinicians who treat these patients, those who recognize the magnitude of this problem and ask themselves, “What can I can do? What steps can I take to improve cooperation with therapy?”

As always in medicine, the first step is epistemology. What do we know, and how can we study it further? In terms of glaucoma compliance, we are still trying to describe the depth of the problem and its causes. To do this effectively, a fundamental requirement is the ability to measure patients’ adherence accurately. One established tool in this process is the MEMS cap bottle. These bottles are designed to hold the patient’s medicine bottles inside them. An electronic device in the cap measures how often the bottle is opened, and thus indicates how often the patient is taking the medication.

To assess adherence in topical drops, there is a device called the Travatan Dosing Aid, which reminds the patient when to dose the medication and records the time and date of dosing. We have used this device in an ongoing study and found it effective.

Another tactic is monitoring individual pharmacy refill data. When you sign up a patient for a study, he or she informs you at which pharmacy the prescription will be filled and grants permission to their pharmacist to share information with you. This is a legal and effective way to measure compliance.

Once we figure out how compliant our patient population is, how do we improve it? The first place we can look is in the mirror: what changes in clinician attitudes and behavior may improve compliance and thus improve outcomes? Here, I should caution potential researchers. There is potential for noise in our physician behavioral data. In a yet-to-be submitted study I was involved in, we looked at physician behavior and found that those who stuck most closely to preferred practice patterns—timely measurement of fields, imaging, tracking pressure, setting target pressures, etc.—were the most likely to have patients whose visual field loss progressed. At first glance, this seems like a perverse finding. Here these doctors are doing everything right, following all the established protocols, yet their patients get worse. On second thought, however, such results make sense, given the occult nature of glaucoma damage. Patients do not realize they are losing vision, and the only doctors who discover vision loss are those who look the most closely for it.

Obviously, patient attitudes and behaviors need to be assessed as well. An area open for study here is cost factors. Would making drugs cheaper improve compliance? What if we gave medication away for free?

Reminders need to be assessed. Do telephone calls or mailings to the home improve adherence? In-office education might be a factor here as well. Doctors, staff and pre-recorded videos might be employed to reach patients and change behavior.

Another big research question: does adherence depend on the stage of the disease? We have found in our studies a significant drop-off in patient compliance early in the disease among patients who have little or no visual field loss, often around the time of the second prescription fill. Traditionally, we follow up on newly diagnosed patients every 6 months to a year to assess visual field loss and/or optic disc damage. We might consider scheduling these patients more frequently—say once every three months—for assessment of levels of adherence, in addition to structure and function.

For those who lack the time or resources to engage in clinical trials, an easy-to-implement step might be a simple questionnaire that the patient can fill out in the waiting room. Answers to simple questions can provide clues to patient adherence. For example:

• Does all of what you know about glaucoma come from your doctor? An answer of yes to this question indicates a patient at risk for noncompliance. We have found a significant link between gleaned information solely form the practitioner and decreased adherence.

• Do you believe reduced vision results from failing to take medication? You might be surprised at how many patients answer no to this question. Perhaps they consider “glaucoma” a conspiracy hatched by pharmaceutical companies to sell medicine. Obviously these patients are at risk for noncompliance.

• Do you have trouble paying for medications? Cost concerns are linked to noncompliance, we have found.

• Do you forget drops when traveling? A yes answer is a red flag.

• Do your drops cause stinging and burning? Surprisingly, an answer of no is indicative of noncompliance. The drops do not sting because they are not being used.

• Do you take 100% of your drops all the time, or do you miss every once in a while? A patient who admits to small amounts of noncompliance is often not taking the drops at all.

• Are you more likely to take your drops on the day of your office visit? A yes is indicative of noncompliance.

Other red flags for noncompliance include the patient who often misses office visits and patients who were treated by you or another practitioner, began therapy, but then dropped out for several years and are now back to restart treatment anew. A patient who has dropped out once is very likely to do so again.

Finally, patients with a non-white, non-European racial background are more likely to be noncompliant. Whether this results from poor patient-doctor communication, cultural and/or socioeconomic dynamics or other factors, we do not know. But there appears to be a link. This is a significant concern because non-white patients are most at risk for developing glaucoma.

These are just a few suggestions to get us started. Above all, we must realize as a profession that patient noncompliance needs to be addressed if we ever hope to reduce rates of vision loss caused by this disease. The best drugs in the world will fail completely if they do nothing but sit on a shelf.

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OGS Honoree Lecture
New Views on the Mechanics of Angle-Closure Glaucoma
A thought-provoking theory from an eminent physician.

BY HARRY A. QUIGLEY, MD

Most of the information in this article is unproven and speculative. And a lot of it suggests that what is contained in our textbooks is dead wrong. But such is the nature of our medical knowledge; we know that
many of the presently accepted concepts will be found incorrect by future investigation. Indeed, it is only in the last 10 years or so that we have produced an agreed-upon and clear definition of what glaucoma actually is. Where I teach, we are fond of telling residents, "Half of what I'm teaching you is wrong. We just don't know which half."

Angle-closure glaucoma is less prevalent than open-angle glaucoma. Worldwide, two of three cases of glaucoma are open-angle; one of three is angle-closure. Yet angle-closure glaucoma puts the patient at greater risk for blindness. Half the glaucoma patients in the world who are blind became that way as a result of angle-closure glaucoma. And with glaucoma having recently displaced trachoma as the second leading cause of blindness in the world, this disease is receiving increased research attention.

I want to offer a novel theory on how we might better predict who should receive surgery to prevent angle-closure glaucoma. But first I would like to debunk some commonly held myths about this disease.

Myths

Myth #1: One gonioscopic grading system is better than another. Glaucoma sub-specialists trained in different parts of the country are taught different grading systems, and other systems exist outside the United States. There is much debate about which is best, but the truth is each system is nothing more than a good idea proposed by a particular individual at a point in time, and none has ever been validated as better than the rest.

Myth #2: The majority of patients with angle-closure glaucoma have had, or are in eminent danger of having, an acute attack. This is what prompts iridotomies, the fear of an attack about to occur. The actual proportion of patients who appear narrow and have some disease and go on to have an acute attack is only 20%. That means 80% of these patients have a silent, asymptomatic disease that slowly causes vision loss (albeit more rapidly than open-angle glaucoma). True, acute attacks are impressive and scary, but they are far less common than we think.

Myth #3: After iridotomy, many primary angle closure eyes continue to have a "creeping" form of closure of the angle. A recent study of eyes that had received iridotomies after acute angle closure attacks in fellow eyes found this not to be the case. Follow-up revealed that only 2.5% of fellow eyes went on to develop glaucoma. Even after 6 years, only 6.5% more went on to develop glaucoma. Yes, there is some creeping that occurs, but the percentage is relatively small.

Myth #4: African-Americans rarely develop angle-closure glaucoma. Population-based studies show that those of African descent develop angle-closure glaucoma at the same rate as those of European descent.

Myth #5: Rates of angle closure in Asian countries are higher than in Europe and North America. The Japanese have a rate of primary angle-closure glaucoma similar to that of Europe, while China and countries in South Asia have a much higher rate.

Myth #6: The Chinese have higher rates of primary angle closure because they have smaller eyes. Yes, those with smaller eyes are predisposed to the disease. Yes, there are higher rates of the disease in China. But no, the Chinese do not have smaller eyes than the rest of the world; in fact, if anything, eye sizes in China are slightly bigger. Hence, Chinese persons must have another risk factor that makes the disease more likely. What is it? More on that later.

Myth #7: After iridotomy, all primary angle closure eyes are cured. Certainly iridotomy relieves pupillary block as the iris falls back away from the cornea after the procedure. This leads to dramatic improvement in the anterior chamber resting anatomy. However, about 30% of eyes after iridotomy do not deepen, which is apparent with gonioscopic examination. Angles still look narrow and the trabecular meshwork is difficult to view. We refer to this as the “plateau iris configuration.” But it is not always a major problem. Most patients do not demonstrate a high pressure after dilation, and they do not develop further disease. But a very small proportion, when dilated, go back into an acute attack. Why this occurs is a mystery. It may relate to the position of the ciliary body, or the configuration of the iris insertion into the base of the angle.

Myth #8: Mydriatic provocative tests:
- tell us a lot about whom to treat;
- are worthless.

The table represents the location for the amount of angle closure found throughout the world. Europe refers to European-derived individuals. 86% of angle-closure glaucoma is in Asia.
Mydriatic provocative tests have at least a 30% false negative rate. So if you are prepared to do a laser peripheral iridotomy on a patient, then it is probably OK to perform this test, because the patient may in fact be positive and you may have to do an iridotomy on the spot. But if you are not prepared for an immediate laser peripheral iridotomy, I think it reasonable to avoid this test or to send the patient to a sub-specialist.

Myth #9: Another cause of failure to cure angle-closure by iridotomy is ciliary block or malignant glaucoma—and this is due to “misdirected aqueous.” The theory of misdirected aqueous simply does not make sense, and I will explain why later in the article.

When Is Treatment Necessary?

The truth is, we have no idea who to treat. If you were to gonioscope 100 folks in a village in China, you would find that 10% have narrow angles. Of those 10%, only 1 in 10 will ever develop primary angle-closure or primary angle-closure glaucoma (Stated another way the prevalence of narrow angles is 10%, and the prevalence of angle-closure glaucoma is 1%). And the other nine will live their whole lives with no glaucomatous damage. We want avoid performing iridotomies on all 50 million Chinese people with narrow angles. Iridotomy hastens cataract development, among other negative side effects.

Why does one person develop angle closure and the others not do not? One reason is gonioscopic examination does not provide enough information to separate those with disease. Gonioscopy is vital, but it is not enough. For example, if you look at a patient’s angle with the lights turned on in the room, and then look at that same patient with the lights turned off, you will notice that the patient’s angle appears more open with the lights on.

First, this indicates that in performing gonioscopy, when you shine a light through the pupil, an artificial opening of the angle occurs. Hence, you should try to illuminate only the meshwork and not the pupil during gonioscopy, especially in patients with narrow angles.

Secondly, this indicates that the anatomy judged by gonioscopy can change minute to minute, moment to moment. Gonioscopy offers a static view of a process that is dynamic and that likely involves a physiological component. There is something else happening inside these eyes, but we do not know what it is.

I believe there is a physiological component to angle-closure because of a recent study that compared angle-closure eyes and control eyes. The study looked at fellow eyes of patients who had an acute attack in the first eye, before iridotomy was performed on the fellow eye. Researchers observed the fellow eyes in lighted conditions, in the dark, and also after treatment with pilocarpine. Then, they compared them with control patients who had never had angle closure in either eye, but who had the same anatomic length and the similar gonioscopic appearance. They found those who had developed the disease opened less in the light and opened less with pilocarpine, by a statistically significant amount. Hence, the resting appearance of the eye tells the practitioner little of what happened. One must do something to provoke the eye into an active or a physiological behavior. Put another way, when angle-closure eyes were compared to normal eyes of the same small size, the angle-closure eyes behaved differently—and this difference is the key, not simply their size or narrowness.

Aqueous Flow

Aqueous moves from behind the iris to the front of the iris. It moves through an area where the iris and the lens are so near each other that even ultrasonic biomicroscopy cannot measure the space. But we know there is a space there because the aqueous does pass through. It may flow episodically, but it does flow.

The resolution of ultrasonic biomicroscopy is about 25-50 microns. So if we cannot resolve the iris lens channel by ultrasonic biomicroscopy, that means that space must be narrower than 25 microns. We became interested in what effect this very narrow channel would have on fluid flow and resistance. One of our engineers generated equations predicting fluid pressure on channels as narrow as 15 microns, and even 5 microns—plausible measurements in these eyes. In very narrow channels, the resistance to fluid movement is such that the pressure differential behind the iris compared to the front of the iris can be as high as 6 to 8 mmHg. Of course, in all eyes there is always a slight difference in pressure between the back of the iris and the front;
otherwise, water could not move. But in normal eyes this difference is probably very minimal. Eyes with very narrow iris-lens channels develop a substantial pressure difference between these two areas.

What does all this mean to us in everyday clinical practice? It means that when we perform tonometry on the surface of the eye, we could be getting a reading that is 8 mmHg lower than that being experienced by the optic nerve.

If all this is true, that leads us to the conclusion that most eyes have pupillary block; it is only a matter of determining how much. Furthermore, the degree of pupillary block may not be discernable by how narrow the angle appears.

Obviously other pressure increases that occur in the back of the eye could worsen this situation. Which brings us to the phenomenon of what used to be called malignant glaucoma but is now called ciliary block glaucoma. This is a condition in which there is a high intraocular pressure (IOP) along with a flat anterior chamber. Those who have seen this condition describe it as “the vitreous has become very compressed, and lakes of aqueous can be seen behind it.” The vitreous humor is compressed and situated in a forward position.

The vitreous humor is a gel. In most adults, it is posteriorly detached, and fluid collects in a compartment behind the vitreous gel. Water, if it is going to pass out of the eye, must pass through this gel. If the pressure differential changes across the vitreous gel, it also impacts the fluid conductivity. The higher the pressure differential, the lower the fluid conductivity. Thus the vitreous, in a sense, collapses like an accordion.

Thus, we see patients whose vitreous is coming forward and the lens is coming forward until the anterior chamber is flat and IOP is soaring. We call this malignant glaucoma or ciliary block glaucoma, but perhaps we should call it vitreous block glaucoma, because it occurs due to a property in the vitreous humor. If what I state here is true, then in addition to having a narrow anterior chamber angle, poor vitreous fluid conductivity could represent a second risk factor for angle-closure glaucoma.

This is why the theory of “aqueous misdirection” makes no sense to me. If fluid can move back through the vitreous, it would move forward just as easily. In order for this theory to work, there would have to be some kind of “one-way valve” within the eye that prevented aqueous from moving forward—and such a mechanism simply does not exist.

Poor vitreous fluid conductivity is a problem in smaller eyes because the fluid cannot get into the posterior and anterior chambers where the lens is, and it cannot get into where the ciliary body is because the vitreous is crushed up against it. The only place fluid can get through is in the doughnut-shaped area that is outside the lens and inside the ciliary body. Small eyes, by their nature, have half the area for forward fluid conductivity movement out of the vitreous gel. Even in the absence of poor vitreous fluid conductivity, such eyes will have difficulty equalizing posterior and anterior pressure differentials.

**Choroidal Expansion**

So, you may ask, how does IOP become elevated behind the vitreous gel? We know patients with ciliary block glaucoma have a high incidence of choroidal detachments. I would suggest that we think of these cases not as choroidal detachments but as choroidal expansions.

Anyone who performs cataract surgeries or trabeculectomies knows that in a certain percentage of cases, upon making an incision in the front of the eye, everything seems to shoot forward at you. The iris prolapses and the lens moves forward. We refer to this as “positive pressure.” By making an incision, the surgeon has lowered pressure substantially in the anterior chamber, thus creating a larger pressure differential. Surgeons who operate on angle-closure eyes know to watch for a higher incidence of positive pressure in these cases. Flat anterior chambers following trabeculectomy are more common in eyes with angle-closure glaucoma. This is an established fact you can read in any textbook.

Now here is the essence of my theory: I posit that the mechanism by which this occurs is an expansion of the choroid.

Let us imagine that the choroid is normally 200 microns thick. Now let us imagine that it doubles in thickness to 400 microns in a short period of time. It would then take up enough volume to push the lens-iris diaphragm forward. In fact, according to my calculations, in a shallow-chambered eye even a choroidal expansion of 20% would displace an amount of fluid equal to the total volume in the anterior chamber. Thus a 20% choroidal expansion would be enough to flatten the anterior chamber of that eye.

This expansion is probably extravascular. It does not occur in the blood vessels but by an increase in vascular permeability, which is either spontaneous or drug induced. A rapid expansion of the choroid causes an increase in the release of proteins into the extracellular space, and these proteins maintain the expansion even in the presence of elevated IOP. These proteins can create an osmotic pressure of at least 25 mmHg. And small eyes have a thick sclera and a smaller surface area, making it more difficult for fluid to escape.

I believe choroidal expansion is part of the primary mechanism of angle closure. Angle closure occurs in patients whose choroid has a greater capacity to expand than the normal population.

To sum up my theory then, primary angle closure is an abnormality related to small-eyed persons whose positional angle structure appears narrow via gonioscopic examination, but who do not develop the disease until some physiologic reactions associated with responsiveness in the structures in the iris-lens channel occur. This physiologic component might be differential fluid conductivity in the vitreous chamber, and/or vascular permeability in the choroid, and/or reactions to light, perhaps leading to a more active choroidal expansion under certain kinds of stimulatory phenomena.

Thus, I propose this disease occurs because of four different phenomena that may occur in the eye: pupillary block, plateau iris, vitreous block (malignant or ciliary block) glaucoma and an expanding choroid.

What this means is that if a patient has a small eye with poor vitreous flow, or a small eye with a high tendency for choroidal expansion, glaucoma is likely to develop. If the patient has all three of these characteristics, he or she would present a narrow-chambered eye that has malignant glaucoma. If the eye was not small, but had poor vitreous flow and choroidal expansion, the patient might also present malignant glaucoma. We know this occurs. Persons with big, myopic eyes can develop ciliary block or malignant glaucoma.

**Practical Implications**

If what I have written here is correct, the next logical step would be
the development of a provocative test of potential choroidal expansiveness. We need to find out who expands by 50% and who expands by only 10%. This will not be an easy thing to provoke or to measure, but it could be extremely useful.

Once we have such a test, a subsequent longitudinal study should reveal that potential for choroidal expansion is a risk factor for angle closure. And then this test will become useful in determining who among patients with narrow anterior chambers would benefit most from an iridotomy.

References:

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The Case for Anterior Segment OCT
It will not eliminate gonioscopy, but could be used as an angle screening test.

BY DAVID S. FRIEDMAN, MD, MPH

The current standard of care for assessing the anterior chamber angle is gonioscopy. Unfortunately, gonioscopy is not a popular test among practitioners. It is difficult to learn to perform effectively, is subjective and is somewhat uncomfortable for patients. Even after years of practice, the most diligent and well-meaning clinician may still be doing it wrong. The result is practitioners avoid it more than they should. Gonioscopy ought to be a part of the work-up on every single patient suspected of glaucoma, and should be performed periodically on otherwise normal patients, but surveys indicate this test simply is not done in many cases.

Thus, the field is open for a diagnostic test to come along and offer better efficacy and ease of implementation. There are three candidates in use today: Ultrasound biomicroscopy (UBM); Scheimpflug imaging and anterior segment OCT, or AS-OCT.

I have experience using all these devices. We recently compared gonioscopy to AS-OCT in patients attending a glaucoma clinic in Singapore. AS-OCT identified more patients as having closed angles than gonioscopy. Whether AS-OCT is more accurate or gonioscopy is more accurate will require long-term follow-up studies. The bottom line, however, is that AS-OCT in this study did not miss many cases of closed angles on gonioscopy. Given that AS-OCT is non-contact and much faster to implement, this test has potential as a pre-screener, selecting patients who need to move on to gonioscopy.

We looked at 203 subjects with primary angle closure, primary open-angle glaucoma, ocular hypertension or cataract from clinics in Singapore. We used AS-OCT to image nasal, temporal and inferior angles. Spaeth classification was used to grade gonioscopic angle width for each gonioscopic quadrant in low lighting conditions.

A closed angle in one or more of the inferior, nasal and temporal quadrants was found in 152 eyes (44%) with gonioscopy and 228 eyes (68%) with AS-OCT. AS-OCT identified one or more quadrants as closed in 85 eyes that looked open in all four quadrants on gonioscopy. Angle closure in one or more quadrants was detected by AS-OCT in 142 patients (71%) and by gonioscopy in 99 patients (50%).

So, again, as data above indicate, AS-OCT could be effective as the pre-screen and gonioscopy as the backup. There are a couple of caveats, however. The AS-OCT cannot visualize the ciliary body, so if ciliary body anatomy ever becomes a predictor for someone who will experience acute attacks, the device will be of no help in this respect. Also, as of now, no automated software exists for the device, although that obviously may change in the future.

Still, based on our results, AS-OCT is a device deserving additional study that may well play an important role in the management of glaucoma in the future.

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About the Optometric Glaucoma Society

The Optometric Glaucoma Society (OGS) was formed 6 years ago with a mission to promote excellence in the care of patients with glaucoma through professional education and scientific investigation. The major objectives are to: promote education of the membership and other healthcare providers related to all forms of glaucoma; promote the acquisition of new knowledge about glaucoma, in part through the development of clinical research within optometry that is related to glaucoma; facilitate the dissemination of information about glaucoma to healthcare providers and the public and, establish collaborative relationships with other related organizations.

There are 64 members in the OGS, coming from several countries around the world. The OGS is equally divided between clinicians and scientists, with ODs, PhDs and MDs making up our membership. The OGS is a member of the Association of International Glaucoma Societies (AIGS), with seven members on the faculty of the 2007 World Glaucoma Congress. Additional information including membership information and the application may be found on the OGS website at www.optometricglaucomasociety.org.

The OGS is involved in several programs that are related to improving optometric glaucoma education. We publish on a quarterly basis an electronic journal that is free of charge and available to anyone who wishes to subscribe. Individuals may sign up at the OGS website. We also publish a glaucoma handbook, produced yearly that is a review of glaucoma diagnosis and management. This is also available for free and distributed to 35,000 optometrists. The OGS held a glaucoma educators forum this past December that was chaired by Tom Lewis, OD, PhD. In addition, the OGS will be holding a glaucoma residency program next fall in which one new graduate from each school going on to a residency program will participate. And finally, the OGS in collaboration with the American Optometric Foundation is funding an Ezell Fellowship in Glaucoma, which is intended to enhance the opportunities for post-graduate optometric glaucoma research.

The Group Photograph from the 2006 Optometric Glaucoma Society Annual Meeting, Denver, CO.