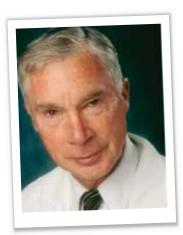
George L. Spaeth, MD, FACS

Dr. Spaeth contemplates the timing, pros, and cons of glaucoma treatment as well as the influences upon physicians' decision-making.



1. How early should glaucoma treatment begin?

Because treatment will cause symptomatology and entail a cost, before starting treatment, one must be fairly sure that a patient who has no symptoms will develop them if medical or surgical therapy is not employed. Specifically, will this individual develop either glaucomatous damage or another problem such as a retinal vein occlusion? If the patient has a narrow anterior chamber angle, one must determine the likelihood of occlusion. With an asymptomatic individual who has an open angle, the question is whether he will develop vascular occlusion or damage from primary open-angle glaucoma that will interfere with his life. Although we do not know much about the former, anecdotal evidence suggests that patients with higher IOPs are more likely to develop retinal vein occlusion. If patients have IOPs of 30 mm Hg (my own magic number) or higher, I begin pressure-lowering treatment. I am particularly concerned when such a patient also has a predisposing factor such as diabetes.

Regarding glaucoma in specific, however, the higher the person's IOP, the more likely this individual is to develop optic nerve damage and perhaps visual field loss. Nevertheless, the correlation between IOP level and subsequent disability is so poor that the former is an almost useless indicator of the latter. Theoretically, allowing early damage to occur may render the patient more prone to subsequent damage. There is, however, no evidence that the development of early disc or field damage, in itself, predisposes patients to further damage. Although patients who develop disc and field damage will probably experience continuing damage, this deterioration is due to what caused the initial damage

and is not a consequence of the presence of the early damage itself. In an often forgotten study by Shiose,¹ normal individuals who were steroid responders received treatment with topical steroids. When their IOPs rose, their optic nerves became cupped, and they developed visual field loss. At that point, the steroids were stopped, and the subjects returned to baseline, with no evidence of damage to their discs or fields. Granted, Shiose did not retest these individuals with further steroid provocation.

If a female patient has an IOP of 27 mm Hg, for example, but is asymptomatic, I will inform her that her IOP is high, that she may develop early damage to the disc or visual field, and, if so, that she may, at that point, need treatment to prevent further damage.

In my opinion, the role of a physician is to prevent patients from developing symptomatic, not asymptomatic,

FAST FACTS

- Director of the William and Anna Goldberg Glaucoma Service and Research Laboratories at Wills Eye Hospital as well as Professor of Ophthalmology at Jefferson Medical College, both in Philadelphia
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 book chapters, and 19 textbooks, including *Ophthalmic*Surgery: Principles and Practice, which has been translated into several languages
- Recipient of the AAO Senior Honor and Lifetime Achievement Awards, 1984 and 2000, respectively
- Recipient of the Herbert Newberg Peace Award, presented by the Philadelphia Chapter of the Lawyers Alliance for World Security, 1995
- A principle investigator and the director of the Disc Reading Center for the NEI's Glaucoma Laser Trial. Also a principle investigator for the NEI's Advanced Glaucoma Intervention Study and its Collaborative Initial Treatment for Glaucoma Study

damage. I may follow a patient with an IOP of 26 mm Hg for 1 year and find that he develops some damage to his optic nerve. Because he also has pancreatic cancer, I may simply ask that he return in 6 months for follow-up. If this person dies in 3 months, he has been spared the cost and anxiety of instilling glaucoma drops in his eyes. If chemotherapy leads to remission but the patient's optic nerve has deteriorated further, the patient can begin glaucoma treatment before becoming symptomatic. Recognizing the rate at which an asymptomatic patient's disease is progressing and his life expectancy facilitates appropriate treatment decisions.

2. How does a physician balance the benefits and drawbacks of glaucoma treatment?

Responsible physicians must do all they can to prevent further deterioration in patients whose glaucoma has already caused visual disability. The only method is to lower patients' IOPs or stabilize their pressures below the level at which damage is occurring. The problem is that no treatment is benign. Every 1-mm Hg decrease in IOP increases a patient's risk of complications, ranging from ocular irritation to serious systemic side effects.

I do not automatically target 12 mm Hg, a figure commonly cited at present. Consider a patient with an initial IOP of 40 mm Hg that drops to 20 mm Hg with medicinal treatment. That level may well be adequate for such an individual, especially if the damage is only moderate. I tend to be more vigorous with progressing patients who have lower levels of IOP.

3. How does the doctor/patient relationship affect glaucoma treatment?

Fifty years ago, pilocarpine was the drug physicians prescribed most frequently for glaucoma treatment. This agent effectively lowered IOP, but it constricted patients' pupils. In addition to blurring some individuals' vision, pilocarpine complicated cataract extraction for many patients by necessitating pupillary stretching. Today, almost no patients receive this drug because it is out of vogue, not because none of them would benefit from it. Pseudophakes with open angles and PCIOLs are quite happy on pilocarpine, because it increases their depth of focus. Many patients do not mind administering the drop b.i.d. or even q.i.d., and they appreciate its low cost.

My point is that, like everyone else, physicians are susceptible to environmental influences. They bring their biases, assumptions, and habits to the patient encounter. A doctor who is an excellent surgeon will probably recommend surgery to patients more readily than one who is either less skilled or who himself experi-

enced a less-than-optimal surgical result. Doctors must know who they are. If they do not recognize their own biases, they cannot factor them into their decisions and thereby provide the best care for their patients. Such self-awareness requires effort, taking some time off now and then, and talking with others.

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In many situations now, how patients care for themselves is the primary determinant of their ocular health. Physicians can encourage patients to learn how to care for themselves, and they can indicate when they think that patients could do better. I may counsel an overweight patient to begin exercising as a means of lowering his IOP instead of relying exclusively on medication. When he returns to my office a few pounds lighter, I congratulate him. The relationship developing between us increases the likelihood that the patient will care for himself better in the future.

4. What is the most important lesson you teach your fellows?

The single most important lesson is that there is no most-important lesson. Life is complex. When caring for patients, physicians must consider many factors, including who they are, who their patients are, and the fact that the world changes. Heraclitus was right: you can never put your hand into the same stream twice. Doctors must be willing to learn and change. We can all continue to examine ourselves in order to determine how we can do things a little better, be slightly more tender or caring, and become less self-aggrandizing as well as more empathetic. Our major task in life is the perfection of character.

5. How has your interest in the arts influenced your care of patients?

The arts are one way to learn about who you are, because artists teach you how to see, hear, and listen. Doctors need all of those skills in order to understand their patients and themselves.

Shiose Y, Kanda T. Quantitative analysis of the "optic cup" and its clinical application. Part II, considerations on clinical cases. *Jpn J Clin Ophthalmol*. 1974;38:367-374.