In reply

It is well known that several parameters, including nucleus density, axial length, and ultrasound power, have been reported to influence endothelial cell loss after phacoemulsification. It is possible that these factors distorted our consideration. The grade of nucleus density is a major factor among them, as Dr Tsai pointed out. We had already investigated whether nucleus density was significantly different between the diabetic and nondiabetic groups in our study, although we did not show the data. We classified nucleus density into 6 grades according to the Lens Opacities Classification System III and compared the number of eyes in each grade between the diabetic and nondiabetic groups using the Mann-Whitney test (Figure). As a result, the number of eyes in each grade by Lens Opacities Classification System III was not significantly different in both groups (P = .80). The distribution of grade in nucleus density was similar in both groups. From this result, we think that nucleus density did not have any influence on our conclusion in this study.

As you know, human corneal endothelial cells do not proliferate throughout life. Endothelial defects are covered by stretching, extending, and transferring the residual cells. Because of this physiological feature, corneal changes that are present 1 month after surgery may represent a temporary effect. We also think that it is important to know whether corneal endothelial impairment among patients with diabetes after cataract surgery represents a temporary effect or permanent damage. Because inflammation in the anterior chamber of an eye of a patient with diabetes may continue, the endothelial damage may worsen. Currently, we are investigating long-term corneal changes in patients with diabetes after cataract surgery.

Soichi Morikubo, MD
Yoshihiro Takamura, MD, PhD
Eri Kubo, MD, PhD
Shosai Tsuzuki, MD, PhD
Yoshio Akagi, MD, PhD

Correspondence: Dr Akagi, Department of Ophthalmology, Fukui Medical University, 23 Shimoaizuki, Matsuoka, Fukui 910-1193, Japan (akagiy@fmsrsu.fukui-med.ac.jp).

We congratulate Robin et al1 and Jampel2 on their thought-provoking editorials. Commentaries such as these will stimulate debate and further research, which, in turn, will influence medical treatment and public policy.

The major question raised in both editorials is how to incorporate the results of the Ocular Hypertension Treatment Study (OHTS) into clinical practice.3,4 The authors correctly emphasize that only a minority of ocular hypertensive individuals develop open-angle glaucoma and that even a smaller fraction develop visual impairment or blindness. On this basis, Robin et al1 conclude that there is little reason to treat individuals with ocular hypertension, and they recommend careful follow-up and initiation of treatment after glaucomatous damage is confirmed.

The original OHTS publications recommended that physicians consider treatment for ocular hypertensive individuals at moderate to high risk for developing primary open-angle glaucoma, taking into account the patient’s general health, life expectancy, and personal preferences.3,4 This approach is similar to the one discussed by Jampel2 and is also consistent with many areas of preventive medicine, such as treating elevated cholesterol levels or blood pressure.

To fully incorporate the results of OHTS into clinical practice and public policy, we must also include the values and preferences of our patients and society regarding the cost and consequences of glaucoma. Currently, investigators from the OHTS are preparing a cost-utility analysis of treating ocular hypertension that takes into consideration patient factors, such as individualized risk of developing primary open-angle glaucoma, and social factors, including the cost of blindness to society. This analysis will evaluate the cost-effectiveness of treatment for individuals at different levels of risk for developing
primary open-angle glaucoma. Robin and coworkers’ present a brief cost analysis that does not consider different levels of risk.

We are also combining the OHTS data set with the database from the European Glaucoma Prevention Study, yielding a prospective data set on more than 2700 people with ocular hypertension. This will allow us to test the OHTS risk model in an independent sample and to use the combined database to refine the risk model for the development of glaucoma. When the economic analysis and the risk model from the joint data set become available, clinicians will have useful information to make evidence-based decisions about preventive treatment in ocular hypertensive individuals.

Clearly, additional studies are needed to answer the question about the best time to initiate treatment in glaucoma. It is not clear whether treatment initiated later in the course of the disease is equally effective in preventing visual impairment.

Michael A. Kass, MD
Mae O. Gordon, PhD
Steven M. Kymes, PhD
for the Ocular Hypertension Treatment Study

Correspondence: Dr Gordon, Department of Ophthalmology and Visual Sciences, Washington University School of Medicine, 660 S Euclid Ave, Campus Box 8230, St Louis, MO 63110 (mae@vrcc.wustl.edu).

Funding/Support: This study was supported in part by grants EY09307 and EY09341 from the National Eye Institute and the National Center on Minority Health and Health Disparities, National Institutes of Health, Bethesda, Md, and Merck Research Laboratories, Whitehouse Station, NJ, and by an unrestricted grant from Research to Prevent Blindness, New York, NY.

2. Jampel HD. We should treat fewer patients with elevated intraocular pressure now that we know the results of the Ocular Hypertension Treatment Study. Arch Ophthalmol. 2004;122:378-379.