plexity, few studies have published results using the algorithm developed by Holladay, Cravy, and Koch, which represents the state-of-the-art knowledge at this time. We think that our study also outlined the benefits of such a thorough analysis. Understanding the limitations of the coupling ratio and its individual variability may help to improve results following refractive surgery. Whether the net effect (sum of the primary and secondary meridia) is truly advantageous remains to be determined.

Drs. Fenzl and Gills' comments in the second paragraph would have been more helpful if they could cite actual results from studies which have performed 5.0-mm optical clear zones in which complications occurred. Since the possible limitations of the small optical clear zone were a concern to us, we addressed this important issue in the abstract (page 65), introduction (page 66), and discussion (page 75). If surgeons choose to proceed with this surgical technique with unrestricted judgment, we can only say that the potential complications were well discussed.

In addition to the size of the optical clear zone, the length of the incision is also important. Ninety-degree incisions are not advocated due to poor wound healing and instability. Further, to attribute problems of hexagonal keratotomy, trapezoidal keratotomy, or even 5.0-mm-or-less PRK solely due to the optical zone size reflects an incomplete analysis of the problems with these procedures. Perhaps a better explanation is the abnormal topography in the central cornea, which can occur more frequently with less-than-perfect procedures located closer to the visual axis.

We acknowledge that our patient population might differ from patients who most commonly undergo astigmatic keratotomy since the level of naturally occurring astigmatism was moderate to severe, there was no prior ocular surgery, the myopia was low, and patients had poor preoperative compliance with glasses or contact lenses. Further studies evaluating the safety of small optical zone size are necessary. Additionally, we look forward to advances in incisional techniques as well as PRK to treat moderate to severe amounts of astigmatism.

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Causes of Uveitis in the General Practice of Ophthalmology

EDITOR:

IN THEIR ARTICLE, "CAUSES OF UVEITIS IN THE GENERAL Practice of Ophthalmology," by C. McCannel, G. Holland, C. Helm, P. Cornell, J. Winston, T. G. Rimmer, and the UCLA Community-Based Uveitis Study Group (Am J Ophthalmol 121:35–46, January 1996), the authors state that a cause could be assigned to 47.4% of cases and that HLA-B27–associated anterior uveitis, cytomegalovirus retinopathy, and toxoplasmosis were among the most common forms of uveitis seen. In a very similar study[1] that I did on 311 black South Africans in a nonreferral clinic setting 20 years ago, a specific diagnosis was made in only 5% of cases. The HIV virus was unknown at that time and no cases of cytomegalovirus were seen. In a study[2] of 53 black South Africans done at the same time, no association between HLA-B27 and anterior uveitis was found. Of interest is that the incidence of nematode uveitis, Vogt-Koyanagi-Harada syndrome, syphilis, and sarcoidosis is the same in both the authors' study and my study. A low incidence of sarcoidosis was found in black South Africans. In the South African study, four cases of leprosy and one of tuberculosis were seen, none of these latter two entities being noted in the authors' study, the changing patterns of disease, as noted by the authors, really being marked only by the appearance of the HIV virus. HLA-B27 would seem to be related to racial differences and not to changing disease patterns. It is interesting to note that the authors suggested limiting the numbers of tests to be done, suggesting at most screening for syphilis, HLA-B27, and sarcoidosis. In my article, I concluded that "the gleaning in terms of aetiological diagnosis obtained from non-specific blanket testing of all cases of uveitis would seem to be poor in the black South Africans.

Where positive aetiological factors were obtained, a clinical diagnosis was more often than not possible, the diagnosis being confirmed by the appropriate special tests." It would seem therefore that very little has really changed in 20 years.

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AUTHOR REPLY

DR. FREEDMAN HIGHLIGHTS SEVERAL IMPORTANT ISSUES in his letter. As the results of his study show, the causes of uveitis can vary with many factors, including geographic location and race. Causes also vary over time with the emergence of new disorders, such as HIV disease. We agree that diagnoses should be based primarily on physical findings and historical information; laboratory tests should be used to confirm or rule out disorders in the differential diagnosis generated on the basis of clinical factors. Dr. Freedman's experience emphasizes again that one must take into account factors unique to the affected population when evaluating and studying uveitis.

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Incidence of Acute Angle-Closure Glaucoma After Pharmacologic Mydriasis

EDITOR:

I READ WITH INTEREST THE PAPER ENTITLED "INCIdence of Acute Angle-Closure Glaucoma After Pharmacologic Mydriasis," by K. H. Patel, J. C. Javitt, J. M. Tielsch, D. A. Street, J. Katz, H. A. Quigley, and A. Sommer (Am J Ophthalmol 120:709–717, December 1995).

The premise of this paper is that screening can determine which patient has an occludable angle and therefore will develop angle-closure glaucoma. With regard to the 3,538 patients considered nonoccludable, the authors make the assumption that no eye had angle-closure glaucoma since the patients would unmistakably demonstrate the typical manifestations of acute angle closure which include "symptoms of vision loss, pain, nausea, vomiting, or a combination." I would bring to their attention the following case, one of several, which bears on this issue.

B.R. was a 71-year-old man with a chief complaint of having difficulty with his new glasses. Manifest refraction showed R.E.: $+0.75 \times 180 = 20/15 -3$ and L.E.: $+1.00 \times 180 = 20/20 +2$. Anterior segment examination was normal except for minimal lens changes. Initial tension (applanation) was R.E.: 20 and L.E.: 23. Fundus examination showed 60% cup R.E. and 50% cup L.E. Tension after dilatation was R.E.: 22 and L.E.: 32, at which time gonioscopy showed 20% of the trabecular meshwork L.E. The pressure was reduced to L.E.: 21 with pilocarpine and the angle was found subsequently to be open. The patient had a routine examination. Angle-closure glaucoma was diagnosed without pain, nausea, or other acute findings.

It is apparent that a patient may have angle-closure glaucoma without "vision loss, pain, nausea, vomiting, or a combination." The conclusion therefore that none of the 3,538 subjects whose pupils were dilated developed angle-closure glaucoma because they did not return with acute symptoms would seem unwarranted. One should repeat the ocular tension after dilatation in order to begin to address the question.

The second major assumption of this paper is that, based on the appearance of the anterior chamber angle, one can diagnose occludability. They equate their judgment regarding occludability with the diagnosis of angle-closure glaucoma. Of the 38 patients in this category, 28 had no pressure rise following dilatation which, at a minimum, casts doubt upon the diagnosis. The remaining ten patients had laser iridotomy. There was no indication of the pressure in these ten patients. The idea that one would perform invasive therapy for an unproven disease seems quite