and carefully closing the eyelid skin. Instead, the morbidity in the cutaneous approach, whether it is for blepharoplasty, orbital surgery, or eyelid rotation, relates to the orbicularis septal complex. Hematoma, inflammation, and scarring in this middle lamellar plane can result in eyelid retraction or volume deflation.

Lower eyelid entropion surgeries accomplish their anatomic goal by creating a scar between the lower eyelid retractors and the anterior tarsal fibrous tissue. This scar both transfers the force of the retractors to the anterior lamella and creates a mechanical stiffness that inhibits inward rotation of the margin. Trying to get the right amount of scar tissue is a delicate balancing act, subject to the inconsistencies of the biology of wound healing. Too little scar, and the entropion will recur. Too much scar, and overcorrection or eyelid retraction will result. Understanding the anatomy, physiology, and wound healing biology of the various available surgical manipulations provides the surgeon with an artist's palate. The accomplished surgeon individualizes each surgery based on subtle differences in anatomy, trying to accomplish just the right amount of intervention (and recognizing that undercorrection is more easily managed than overcorrection.)

Drs Erb and Dresner are likely correct that surgery to the orbicularis septal complex increases the success rate of transconjunctival entropion surgery (although comparison of the retrospective studies that they reference is not scientifically valid, and a randomized prospective study would be required to prove their point). On the other hand, aggressive treatment of the orbicularis septal complex increases the complication rate and, indeed, turns the conjunctival approach into essentially a full thickness approach, which is what we started out trying to avoid. Unnecessary surgical reduction of eyelid volume also has negative aesthetic consequences. The orbicularis septal complex is always manipulated to some extent in the process of exposing the anterior tarsus, but we suggest that debulking of orbicularis should be individualized and graded based on the degree of scar tissue needed to stabilize the margin and on the aesthetic goal of the surgery.

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Natural History of Asymptomatic Clinical Retinal Detachments

EDITOR:

WE READ THE ARTICLE BY STEVEN COHEN ON NATURAL history of asymptomatic clinical retinal detachments with interest.[1] We agree with the author that asymptomatic clinical rhegmatogenous retinal detachments (RDs) can probably be safely observed periodically for many years, though the management options depend on many factors, like patient's age, size, and location of the subretinal fluid, the causative lesion and presence of demarcation lines, as well as on patient's personality. Some patients accept self-monitoring and the need for frequent follow-up examinations, while others may not. Also, since presence or absence of symptoms is a totally subjective phenomenon, potential vision-threatening conditions might occur in some patients, depending on their observational powers and cognitive function, who are unaware of any ocular symptoms.[2]

The study did not define the presence of a partial or complete posterior vitreous detachment (PVD), which we feel might play a role in progression of the detachment. Also, since most of the cases had retinal detachments located temporally and inferiorly and were associated with demarcation lines, we feel the conclusions cannot be extrapolated to a rapidly progressive superior retinal detachment, which might lead to visual impairment sooner than later.

We agree with the author on the many potential complications that can result from a retinal detachment surgery, but these surgeries have had some impressive results over the years. Obviously, the risks and benefits of each management option have to be discussed with the patient. Also, some of these patients can be managed with the newer surgical techniques like 25-gauge vitrectomy systems.

Management of these patients with clinical retinal detachments continues to be debatable. Davis reported six (30%) out of 20 eyes with asymptomatic subclinical retinal detachments progressing to clinical retinal detachments,[3] while Byer reported 0.8% risk of progression of a subclinical retinal detachment.[4] He also recommended treating patients with subclinical retinal detachments that progress to become clinical detachments. Moreover, the progression of the retinal detachments was not associated with symptoms. The present study followed up eighteen eyes of 16 patients with asymptomatic RD over an average of 46 months; none of them became symptomatic during follow-up.

We feel that the decision to manage surgically in this subgroup of patients depends on wide range of factors. Most patients can be managed conservatively, with a thorough explanation to the patient, followed by a regular follow-up and documentation of findings. Once progression occurs, surgery may be required. With special reference to the countries where retinal specialists may not be available close to the patient, the treating physician may have to consider surgery in the first

instance, as the patient may not be able to come for follow-up on a regular basis.

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthalmol 2005;139:777–779.
2. Greven CM, Wall AB, Slusher MM. Anatomic and visual results in asymptomatic clinical rhegmatogenous retinal detachment repaired by scleral buckling. Am J Ophthalmol 1999;128:618–620.
3. Davis MD. The natural history of retinal breaks without detachment. Arch Ophthalmol 1974;92:183–194.
4. Byer NE. Subclinical Retinal Detachment resulting from asymptomatic retinal breaks. Ophthalmology 2001;108: 1499–1504.

REPLY

TANO AND ASSOCIATES THOUGHTFULLY RAISE SEVERAL important points.

First, I did not define the presence of a partial or complete posterior vitreous detachment. Among the 18 eyes, there were no partial posterior vitreous detachments.[1] To determine the state of the vitreous, each of these patients was examined with slit-lamp biomicroscopy before and after swift ocular movements.[2]

Second, the question is raised as to what to do with "a rapidly progressive (sic) superior retinal detachment." Since no patient in my series had such a retinal detachment, I agree that one should apply the findings from my study to such an eye with reservations.

Concerning the above hypothetical case, an interesting question is: Why do some retinal detachments evoke symptoms and others do not? How could 16 patients with retinal detachments, some with a posterior margin abutting the macula, not notice peripheral vision loss?

These retinal detachments may have been asymptomatic because the posterior margin of each retinal detachment was stationary. The visual system has specialized mechanisms exclusively sensitive to motion.[3] Since the brain more easily ignores fixed visual field defects than changing visual field defects, static retinal detachments probably go unnoticed more readily than progressing retinal detachments.

Finally, the authors suggest that management of asymptomatic retinal detachments might be guided by the patient's "personality." By mentioning "personality," I assume they are concerned about patient anxiety. How we present diagnoses and management choices to our patients affects their emotional response and treatment decisions.[4] As experts, we can either alarm a patient or put him or her at ease by presenting exactly the same information differently. Most of my patients had seen an ophthalmologist who told them they were being sent to me, the retinal surgeon, for urgent surgery. On learning that they could be observed with reasonable expectations of avoiding both vision loss and surgery, they were relieved.

Interestingly, most of these 16 patients have to be schlepped (dragged) in for examinations because they did not notice any problem. The one patient whose retinal detachment progressed during the study did not want surgery even though I recommended it. She did fine.

Management of asymptomatic clinical retinal detachment should be based on published studies, provider experience, and patient preference. For asymptomatic retinal detachments, observation is a reasonable option.

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthlamol 2005;139:777–779.
2. Muakami K, Jalkh AE, Avila MP, Trempe CL, Schepens CL. Vitreous Floaters. Ophthalmology 1983;90:1271–1276.
3. Fahle M, Wehrhahn C. Motion perception in the peripheral visual field. Graefes Arch Clin Exp Ophthalmol 1991;229: 430–436.
4. Berger K. Informed consent: information or knowledge? Med Law 2003;22:743–750.

Scleral Canal Size in Patients With Optic Nerve Drusen

EDITOR:

IN THE APRIL 2005 ISSUE OF THE JOURNAL, FLOYD AND COLleagues published their observation that the mean anterior scleral canal size of eyes with optic nerve drusen (OND) is not smaller than the canal size of control eyes.[1] They compared the optical coherence tomography estimations of disk area (Carl Zeiss Meditec, Inc, Dublin, California) in 25 subjects with OND, 17 unaffected first-degree relatives, and 17 control subjects. The control subjects showed similarities in age, gender, and refractive error to the patients with OND. However, the authors did not describe the racial makeup of the patients involved in this study.

African-American patients have larger disk areas than Caucasian patients. Several papers have made this observation through Heidelberg Retinal Tomography[2] (Heidelberg Engineering, Heidelberg, Germany), video-ophthalmography[3] (Rodenstock Optic Disk Analyzer), and most convincingly, histologic evaluation of banked eyes.[4] In the study by

Dr Floyd, conceivably a larger percentage of African-Americans in the group of eyes with OND might skew the data to suggest relative disk area equality between OND eyes and control eyes. Could the authors comment on the racial distribution of the participants?

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REFERENCES

1. Floyd MS, Katz BJ, Digre KB. Measurement of the scleral canal using optical coherence tomography in patients with optic nerve drusen. Am J Ophthalmol 2005;139:664–669.
2. Girkin CA, McGwin G Jr, Xie A, Deleon-Ortega J. Differences in optic disk topography between black and white normal subjects. Ophthalmology 2005;112:33–39.
3. Chi T, Ritch R, Stickler D, et al. Racial differences in optic nerve head parameters. Arch Ophthalmol 1989;107:836–839.
4. Quigley HA, Brown AE, Morrison JD, Drance SM. The size and shape of the optic disk in normal human eyes. Arch Ophthalmol 1990;108:51–57.

Measurement of the Scleral Canal Using Optical Coherence Tomography in Patients With Optic Nerve Drusen

EDITOR:

I SINCERELY APPRECIATE DR LEE'S INTEREST IN OUR MANUscript. Dr Lee has pointed out that black patients have different optic nerve topographies compared with whites. Optic nerve drusen is almost exclusively a disease of whites.[1] To eliminate race as a possible cause for differences between our subject groups, only whites were recruited for our study. For this reason, race could not have affected our results.

Sincerely,

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REFERENCE

1. Rosenberg MA, Savino PJ, Glaser JS. A clinical analysis of pseudopapilledema. I. Population, laterality, acuity, refractive error, ophthalmoscopic characteristics, and coincident disease. Arch Ophthalmol 1979;97:65–70.

Natural History of Asymptomatic Clinical Retinal Detachments

EDITOR:

THE RECENT REPORT BY COHEN[1] CONFIRMS PREVIOUS OBSERvations, highlighted by Brod[2] and Byer[3] that not all rhegmatogenous retinal detachments progress to visual loss. A common feature in Cohen's series was that the eyes were asymptomatic and, during a period of observation ranging from 12 to 72 months, none of the eyes required surgery.

My concern with the report is on the word asymptomatic. While Cohen's patients no doubt were asymptomatic, my own experience has been that patients can present with rhegmatogenous retinal detachment involving the macula and be unaware of their visual loss, remaining apparently symptom free.[4] While Cohen did exclude patients with symptomatic retinal detachment in their fellow eye, it is not uncommon for patients presenting with bilateral retinal detachments to be symptomatic in only one eye. Furthermore, the association of poor macular function may mask progressive visual loss in an evolving retinal detachment.

I would suggest the notion that the opposing forces of reattachment and detachment are in equilibrium for patients with asymptomatic retinal detachment is not valid. Instead I believe it more likely that the presence or absence of symptoms is an epiphenomenon in both progressive and nonprogressive retinal detachments.

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthalmol 2005;139:777–779.
2. Brod RD, Flynn HW, Jr Lightman DA. Asymptomatic rhegmatogenous retinal detachments. Arch Ophthalmol 1995;113:1030–1032.
3. Byer NE. Subclinical retinal detachment resulting from asymptomatic retinal breaks: prognosis for progression and regression. Ophthalmology 2001;108:1499–1503; discussion 1503.
4. Polkinghorne PJ, Craig JP. Analysis of symptoms associated with rhegmatogenous retinal detachments. Clin Experiment Ophthalmol 2004;32:603–606.

REPLY

I AGREE WITH DR POLKINGHORNE THAT "NOT ALL RETINAL detachments progress to visual loss." My prospective, long-term natural history study was undertaken to determine if asymptomatic clinical retinal detachments that were followed for several years progress to visual loss. None did.[1]

Dr Polkinghorne questions the relevance of the asymptomatic status of these patients to their outcome. Evidence suggests that the absence of symptoms in these patients was associated with their lack of progressing visual loss. My study included eighteen eyes with asymptomatic clinical retinal detachments. The absence of symptoms was the only inclusion criterion for my prospective consecutive study. With the exclusion of eyes that had undergone recent intraocular surgery and eyes with fellow eye retinal

detachments, eighteen eyes is a good number with this uncommon problem.

Second, I contend that based on how the visual system functions, nonprogressing retinal detachments tend to be asymptomatic and progressing retinal detachments tend to be symptomatic.

People cannot pay attention to all of their senses all of the time. We have the ability to focus our attention on what interests us and ignore all else. Yet, even when fully captivated by a dazzling journal article, we withdraw our hand from a flame. Similarly, visual perception allows us to easily ignore stationary visual field defects. Thus, the reader can trudge through this letter, word after word, sentence after sentence, unable to perceive the ever-present map of his or her retinal blood vessels on this page. Similarly, when reading, a physician can easily disregard a stack of medical charts in plain view, but will likely look up when a coworker silently moves one.

When a cat stalks a mouse, it does so very slowly and barely moves, to avoid being noticed. All of this suggests that stationary visual field defects are more easily ignored than progressing visual field defects.

Unless we work contrary to the Preferred Practice Patterns for retinal detachment,[2] which recommend urgent surgery for symptomatic retinal detachment, we cannot study symptomatic retinal detachments to see if they progress. My article helps physicians when deciding which retinal detachments may not progress to vision loss. Asymptomatic clinical retinal detachments can probably be safely observed.

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthalmol 2005;139:777–779.
2. American Academy of Ophthalmology Retina Panel Preferred Practice Patterns Committee. Retinal detachment. San Francisco, CA: American Academy of Ophthalmology (AAO);1990;1–17.

Natural History of Asymptomatic Clinical Retinal Detachments

EDITOR:

IN THE ARTICLE BY COHEN,[1] THE AUTHOR REPORTED ON the natural history of asymptomatic clinical retinal detachments. While we do not want to over treat asymptomatic retinal breaks, we also do not want to under treat those lesions that are at risk of developing into clinical retinal detachment. In this report, none of the 18 asymptomatic retinal detachments developed into full blown retinal detachment. However, there are certain characteristics that were unique in this group of patients.

1. None of the studied patients had developed further posterior vitreous detachment during the study period.
2. All had temporal or inferior detachment.
3. Presence of demarcation line indicating the chronicity of the detachment.
4. Severe myopia appears not to be a major risk factor in the study group.

As mentioned by the author, retinal detachment occurs when there is an imbalance distribution of forces across the retina. The state of equilibrium in this group of patients was hypothetically contributed, at least partly, by the four characteristics mentioned above. However, while the last three characteristics would not change over the course of time, further posterior vitreous detachment could occur and tip the balance. In highly myopic eyes, not only is the retina more prone to develop retinal degeneration and breaks, but premature posterior vitreous detachment also occurs more frequently.[2] This is particularly important in patients undergoing intraocular operations like cataract extraction and especially when complications like posterior capsular tear occurs.

While there are risks associated with retinal detachment surgery, and pneumatic retinopexy is not practical for inferior detachment, a few rows of barrier laser will provide additional support to an otherwise asymptomatic retinal detachment at minimal risk. Such barrier laser could have even stopped the 4-mm progression in the patient mentioned in this study.

Perhaps, the clinical decision on the management of asymptomatically retinal detachment should not only depend on the issues discussed above, but also the accessibility of subspecialist service in a short period of time. In view of the relatively high prevalence of high myopia in some parts of the world[3] and the associated higher risk of retinal detachment, a large-scale prospective study to evaluate whether or not asymptomatic retinal breaks in patients with high myopia should be treated seems warranted. Should these services not be readily available, we may want to have a lower threshold for prophylactic treatment.

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REFERENCES

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthalmol 2005;139:777–779.
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Natural History of Asymptomatic Clinical Retinal Detachments

EDITOR:

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REFERENCES

1. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthalmol 2005;139:777–779.

2. Morita H, Funata M, Tokoro T. A clinical study of the development of posterior vitreous detachment in high myopia. Retina 1995;15:117–124.
3. Ho T-C, Shih Y-F, Lin LLK, et al. Prevalence of retinal detachment and peripheral retinal degenerations in myopic college students in Taiwan. Proceedings of the 5th International Conference Myopia, MIRF 1994;161–164.

REPLY

CHENG, YUEN, AND LAM ASK: WHY NOT LET LASER DEMARcate the asymptomatic clinical retinal detachments? Laser demarcation and observation are both reasonable alternatives to surgery.[1,2] The best course is unknown. My study excluded all patients with fellow eye retinal detachments, subclinical retinal detachments, symptomatic retinal detachments, and retinal detachments in eyes with recent surgery.[3] Recruitment of an adequate number of comparable patients with asymptomatic clinical retinal detachments for a randomized trial comparing laser demarcation to observation would be difficult.

Cheng and associates also ask: What about patients presenting with asymptomatic clinical retinal detachment in parts of the world with few retina specialists? There is one ophthalmologist per 10,000 people in the United States; one per 100,000 in India, and one per one million in sub-Saharan Africa.[4] In Florida, most people live within fifteen miles of a retina specialist. When managing a patient who has a condition that may progress and who may not be able to return for timely treatment, physicians must balance the risk of treatment with the risk of observation, subsequent progression, and delayed treatment. Published studies provide guidelines for managing patients and need to be considered in concert with physician experience and patient preference. Timely treatment of symptomatic retinal detachment is critical. Therefore, if a patient has a retinal detachment that might become symptomatic, and if that patient may not be able to present for timely treatment, then the benefit of more definitive therapy might be worth the risk.

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REFERENCES

1. Greenberg PB, Baumal CR. Laser therapy for rhegmatogenous retinal detachment. Curr Opin Ophthalmol 2001;12:171–174.
2. Vrabec TR, Baumal CR. Demarcation laser photocoagulation of selected macula-sparing rhegmatogenous retinal detachments. Ophthalmology 2000;107:1063–1067.
3. Cohen SM. Natural history of asymptomatic clinical retinal detachments. Am J Ophthlamol 2005;139:777–779.
4. Spivey B. The opportunity for international ophthalmology in treating blindness. Tr Am Ophth Soc 2001;99:73–77.

Postoperative Corneal Swelling Correlates Strongly to Corneal Endothelial Cell Loss After Phacoemulsification Cataract Surgery

EDITOR:

IN THE ARTICLE "POSTOPERATIVE CORNEAL SWELLING Correlates Strongly to Corneal Endothelial Cell Loss After Phacoemulsification Cataract Surgery" by Lundberg and associates,[1] the authors concluded that the degree of permanent corneal endothelial damage was reflected in the degree of early postoperative corneal swelling.

Despite significant univariate correlations found between pachymetry change in day 1 and nucleus color with central cell loss, only nucleus color remained statistically significant after multiple regression analysis (Table 1).[1] The lack of independent predictability of pachymetry change for central cell loss suggests possible confounding effect of nucleus color. Dense nucleus has been identified as a major risk factor for corneal endothelial cell loss.[2,3] In the current study, ten consecutive patients were allocated into each of the three groups according to the degrees of corneal swelling at the first postoperative day. It was noted that there was a trend for increasing nucleus color in groups with more postoperative corneal swelling.[1] Therefore, it would be appropriate to stratify nucleus color before estimation for any correlation between pachymetry change and corneal cell loss.

We congratulate the valuable work from Lundberg and his associates, and hope that our understanding of postoperative corneal endothelial cell loss would be enhanced by further discussion.

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REFERENCES

1. Lundberg B, Jonsson M, Behndig A. Postoperative corneal swelling correlates strongly to corneal endothelial cell loss after phacoemulsification cataract surgery. Am J Ophthalmol 2005;139:1035–1041.
2. Bourne RR, Minassian DC, Dart JK, Rosen P, Kaushal S, Wingate N. Effect of cataract surgery on the corneal endothelium: modern phacoemulsification compared with extracapsular cataract surgery. Ophthalmology 2004;111:679–685.
3. Hayashi K, Hayashi H, Nakao F, Hayashi F. Risk factors for corneal endothelial injury during phacoemulsification. J Cataract Refract Surg 1996;22:1079–1084.