thesia. The authors suggest that ocular anesthesia is better provided by the surgeon who ultimately must take responsibility for the care, management, and outcome of the patient. In their survey, however, several comments indicated that where formal training was provided, complications were exceedingly rare.

Although many anesthesiologists may have a lack of knowledge of orbital anatomy and axial eye length and may have had no formal training in ocular anesthesia during their residency, they can be trained adequately in these areas if they are willing to learn. Most ophthalmologists begin administering retrobulbar blocks during their first year of residency (in many programs, this is the only procedure first-year residents are allowed to perform). Of course, there is usually an attending physician or senior resident standing by their side and "training" them.

In our practice, anesthesiologists currently perform peribulbar blocks on all patients undergoing intraocular and strabismus surgeries. Several years ago, we were approached by a group of 15 anesthesiologists who expressed interest in providing the anesthesia at our ambulatory surgical center. We hired them under the condition that only five would rotate through our office, and that these five would learn to administer ocular anesthesia, in addition to sedating and monitoring the patients. Of these five board-certified anesthesiologists, only one had training during her residency in performing peribulbar anesthesia.

These anesthesiologists received an informal course on ocular anatomy, axial length, and potential complications resulting from peribulbar injections. In addition, each anesthesiologist was observed and assisted while performing peribulbar and occasionally retrobulbar anesthesia until we were comfortable with their technique on our patients (for whom we ultimately are responsible).

In the last 2 years, these anesthesiologists have performed blocks on more than 1500 patients without, to our knowledge, a single perforation, and our surgical center subsequently has operated much more efficiently. Although we were skeptical initially, as most ophthalmic surgeons probably are, we now believe that motivated anesthesiologists with adequate "post-graduate" training in ocular anesthesia can provide safe, reliable, and efficient peribulbar injections.

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### Author's reply

Dear Editor:

I am pleased that Drs. Zeiter were interested in our recent publication. I am also happy to know they reached the same conclusions that we had derived from our survey. Specifically, if training is to be provided to nonophthalmologists, it should be done by the surgeon or surgical group. They have shown that very admirably in their letter. They undertook a program to provide training to specific anesthesiologists and, once assured of their competency, allowed them to continue with this technique. They remarked that in 1500 patients, there had not been a perforation in the last 2 years after their training program. Because the primary focus within an ophthalmology department is the training of ophthalmology residents, anesthesiology residents become a competing factor in providing adequate training for all residents. Also, a nonophthalmologist may have very brief training interaction in ocular anesthesia, making it difficult to ensure competency over a longitudinal basis. However, ophthalmology residents perform "blocks" each year; therefore, they continually are re-evaluated in aspects of that training, and one can better ensure competency.

After our survey, there have been other articles published regarding the safety of nonophthalmologists administering ocular anesthesia. It was not the intent of our publication to comment on the safety or efficacy of nonophthalmologists performing this procedure; rather it was to assert that this training is difficult to provide in an ophthalmology residency training program. To ensure competency and allow hospital privileges based on perceived training, one needs to have continual re-evaluation and assurance that competency has been obtained. This is difficult to do under current residency training conditions, as was evidenced by our survey.

The Zeiters' approach to training anesthesiologists within their institution and then following them carefully is the most appropriate course of action if one desires to have nonophthalmologists administer anesthesia. One has to keep in mind that although their results have been very successful, in the most recent publications the single greatest risk factor for globe perforation has been nonophthalmologist administration of ocular anesthesia, as noted in our article.

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## Recurrence of Macular Holes

Dear Editor:

I read with interest the article by Duker and associates entitled, "Late Re-opening of Macular Holes after Initially Successful Treatment with Vitreous Surgery" (*Ophthalmology 1994;101:1373–8*). I also have observed late recurrence of macular hole after initially successful closure of full-thickness macular hole after surgery and presented two cases at the Western Association for Vitreoretinal Education meeting in July 1993.

I also have observed late recurrence of macular hole in an eye with an impending macular hole, which initially resolved after surgical intervention with significant improvement in vision.

An 89-year-old woman had a sudden onset of decreased vision in the right eye for 2 weeks. On initial evaluation, visual acuity was 20/200 ($-0.50$ $+0.75 \times 16$) with a 250-$\mu$m yellow ring and central macular cyst, consistent with a stage IB macular hole.[1] The left eye had chronic loss of vision related to a pigment epithelial detachment, with visual acuity of 20/80 and metamorphopsia. Both eyes were pseudophakic with posterior chamber implants. After 2 months without change, a pars plana vitrectomy, peeling of the posterior hyaloid, and injection of 18% $C_3F_8$

were performed. Six months postoperatively, visual acuity improved to 20/30 with resolution of the macular cyst and yellow ring. One month later, recurrent blurred vision developed, with visual acuity of 20/50. A temporal epiretinal membrane with oblong macular cyst measuring 100 × 150 μm (vertical × horizontal) and surrounding yellow ring was noted. Three months later and 11 months postoperatively, a 400-μm full-thickness macular hole was noted, with visual acuity of 20/100. The patient elected not to pursue further surgery.

This case demonstrates that late recurrence is a potential complication of even successfully resolved stage I macular holes in eyes that clinically never had a full-thickness macular break. This late complication may be best labeled "late recurrence," rather than "late re-opening," which emphasizes that this complication can develop in eyes with previously resolved impending or stage I macular holes.[1]

I would like to know if the authors have observed late recurrences in any stage I macular holes that resolved either by natural history or after surgery. The mechanism of late recurrence may be similar in both impending and full-thickness macular holes. In the patient described above an epiretinal membrane developed temporal to the fovea, which could have resulted in recurrent foveal traction, as the authors suggest. This finding implies that epiretinal membranes can result in enough traction on the fovea to result in full-thickness macular hole, even without residual attached posterior cortical vitreous in rare instances. Smiddy[2] also has noted macular hole development after surgical peeling of epiretinal membrane over the macula. Thus, macular holes can develop in certain situations in which the posterior cortical vitreous has been removed from the macula, either surgically or by natural posterior vitreous detachment.[2]

Alternative theories for late recurrence include recurrent vitreofoveal traction from residual cortical vitreous traction. This may be related to possible splitting of the cortical vitreous during attempted removal,[3] or dehiscence of the posterior hyaloid around the macula resulting in the development of a complete peripheral vitreous detachment with unrecognized residual vitreomacular attachments (personal observation).

Intraretinal traction or tension is also a possible factor. The retina has inherent biomechanical properties.[4] If the fovea is already in a mechanically weakened state from either previous traction, or after surgical closure of the macular hole, then intraretinal tension or traction also may be a factor in late recurrence of macular holes. This possibility is supported by the observation in most macular holes that the operculum often is much smaller than the macular hole, suggesting that the retina may be under some chronic intraretinal tension, which causes enlargement of the retinal defect after its initial development.

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### Author's reply

Dear Editor:

I appreciate Dr. Kokame's interest in late re-opening of macular holes. I have not personally observed a stage I hole becoming a full-thickness hole after initial resolution either spontaneously or with surgery. This is an important observation, however, and may influence our approach to treating stage I holes.

Dr. Kokame's theory concerning intraretinal tractional forces is intriguing. With further histopathologic studies, perhaps we will know which of these theories concerning the etiology is correct.

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## Transconjunctival Blepharoplasty

Dear Editor:

We read with interest the article by Doxonas entitled, "Minimally Invasive Lower Eyelid Blepharoplasty" (*Ophthalmology 1994;101:1327–32*), describing a technique for transconjunctival lower blepharoplasty through "two small buttonhole incisions." Transcutaneous blepharoplasty invariably results in some degree of lateral lower eyelid retraction, creating an undesired surgical look.[1] We compliment the author on his efforts to simplify the transconjunctival blepharoplasty. At the same time, we wish to stress that the transconjunctival blepharoplasty must not be trivialized. This may occur when viewing it as a "minimally" invasive procedure. We have three significant concerns.

First, it is essential for the surgeon to understand the conceptual purpose of lower blepharoplasty. Although not explicitly stated by the author, the goal of blepharoplasty is the sculpting of the eyelid to achieve a desired appearance without compromising structure and function. The transconjunctival approach to the lower eyelid avoids damaging the orbital septum which must be violated when performing a transcutaneous lower blepharoplasty. Disrupting the orbital septum results in middle lamellar cicatrix and contraction and is the cause of postblepharoplasty lower eyelid retraction, round eye, and sceral show. Eyelid sculpting generally is accomplished by the graded resection of orbital fat and, when necessary, redundant lower eyelid anterior lamella (skin and orbicularis).

Second, while lower eyelid fat can be removed through buttonholes in the conjunctiva, it is important to appreciate the benefit of wide exposure in the sculpting of lower eyelid fat. With an "open-sky" transconjunctival technique, all of the fat pockets of the eyelid are readily laid out, and the inferior oblique muscle is exposed.[2] This exposure minimizes risk to the oblique by identifying its location. Fat then is sculpted by selective resection to obtain the desired lower eyelid contour. No traction on the