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REPLY

WE THANK THE CORRESPONDENT FOR HER COMMENT ON our recent publication reporting that total posterior vitreous detachment (PVD) by ultrasound was associated with 3 times as many cases of dry age-related macular degeneration (AMD) as cases with wet AMD (69% vs 21%; $P = .002$). An attached posterior vitreous cortex by optical coherence tomography (OCT) was associated with 4 times as many cases of wet AMD as dry AMD (38% vs 10%; $P = .008$). We agree with the proposal that one of the plausible mechanisms by which PVD may be salubrious and vitreomacular adhesion may be deleterious is that oxygenation of the macula is enhanced in the absence of an attached posterior vitreous. Indeed, in our original publication, the third sentence of the fifth paragraph of the discussion states that: "*The presence of the posterior vitreous cortex attached to the macula could prevent oxygen and perhaps nutrients from diffusing from the ciliary body into the macula. The resulting state of relative ischemia could promote choroidal neovascularization via the action of VEGF.*"

There are reasons, however, to suspect this is not the only explanation. A recent study (Binder et al, IOVS 2009;50: ARVO E-abstract 5245) has shown that in most cases of exudative AMD with vitreomacular adhesion, there is a positive correlation between the site of vitreous adhesion to the macula and underlying choroidal neovascularization (CNV). In 72% of these eyes, traction of the posterior vitreous cortex onto the macula was visible and the traction lines were directed towards the CNV, as visualized in 3-D and longitudinal OCT images (Figure). Vitreous was adherent to the fovea in 52.4% of cases and was juxtafoveal in 47.6%. In 93% of the juxtafoveal adhesions, a retinal angiomatous proliferation (RAP) was present, and the area of the adhesion corresponded with the localization of the "hot spot" on ICG angiography.

Thus, vitreomacular traction may directly induce mechanical effects that result in CNV. Traction, which is likely exacerbated by something as simple as ocular saccades, can cause inflammation and contribute to neovascularization. It is also plausible that the physical effects of vitreomacular traction alter macular anatomy and permit the migration and proliferation of vasogenic cells under the influence of ischemia-induced growth factors and cytokines. Furthermore, as described in our original publications,[1,2] the presence of an attached posterior vitreous cortex may prevent the egress of these cytokines, increasing their local concentrations and potency.

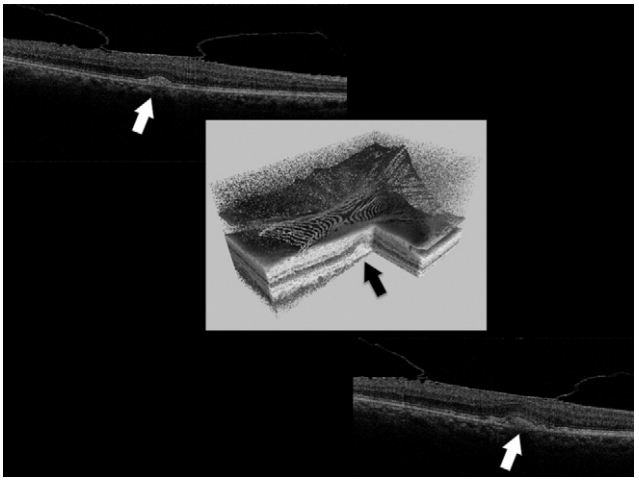

FIGURE. Longitudinal and 3-D optical coherence tomography (Carl Zeiss Meditec Cirrus HD-OCT; Dublin, California, USA) scans demonstrating anomalous posterior vitreous detachment[5] with persistent attachment to the macula. The site of vitreoretinal adhesion is directly overlying the location of the choroidal neovascular complex (arrows).

As previously described,[1,2] the phenomenon of choroidal neovascularization is likely multi-factorial. We thank the correspondent for underscoring the possible role of ischemia as it relates to the state of vitreous, a consideration that was in fact mentioned in our original publication. It is likely, however, that ischemia and/or inflammation-induced cytokine activity may be necessary, but not sufficient, to cause the observed events in exudative AMD. Vitreomacular traction and its subsequent effects may indeed be a critical component in the development of choroidal neovascularization. If so, a new avenue of therapeutics emerges as pharmacologic vitreolysis[3,4] may provide an important adjunct to anti-VEGF treatments.

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Prospective Comparison of Two Suturing Techniques of Amniotic Membrane Transplantation for Symptomatic Bullous Keratopathy

EDITOR:

WE READ WITH GREAT INTEREST THE ARTICLE ENTITLED "Prospective comparison of two suturing techniques of amniotic membrane transplantation for symptomatic bullous keratopathy" by Altiparmak and associates.[1] The authors concluded that modified amniotic membrane transplantation (AMT) suturing technique has a similar epithelialization rate to standard AMT suturing to the cornea.

The donor of amniotic membrane should be screened to exclude risk of transmissible infections such as human immunodeficiency virus (HIV), hepatitis B virus, hepatitis C virus, and *Treponema pallidum* infections. However, the authors did not mention any screening test in their article. We believe that the authors had excluded the donors with positive serologic tests. Transmission of these infections to recipient patients may cause legal issues.

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REFERENCE

1. Altiparmak UE, Oflu Y, Yildiz EH, et al. Prospective comparison of two suturing techniques of amniotic membrane transplantation for symptomatic bullous keratopathy. Am J Ophthalmol 2009;147:442–446.

REPLY

WE THANK SAKARYA AND ASSOCIATES REGARDING THEIR question about screening of amniotic membrane donors. Indeed, it is a very important issue that should not be neglected, both ethically and from a medicolegal standpoint.

Before harvesting amniotic membrane, informed consent should be taken from the donor, and donor anonymity must be maintained.

In our practice, donors are screened for human immunodeficiency virus (HIV) type 1 and 2, hepatitis B virus (HBV), hepatitis C virus (HCV), and syphilis, as recommended by various authors.[1–4] Since certain pathogens are more frequently encountered in different geographic areas, screening for such agents may also be added to the standard protocol. The serological test records should be maintained for 11 years post transplantation. Review of the records may become necessary if HIV or any slow virus infections develop in the recipient(s), assuming that these would develop in not later than 10 years.[5] Nevertheless, readers of the manuscript are strongly recommended to follow the current literature for donor screening, because of the changes in screening methods and their sensitivity/specificity.

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Spontaneous Corneal Clearance Despite Graft Detachment after Descemet Membrane Endothelial Keratoplasty

EDITOR:

WE CONGRATULATE BALACHANDRAN AND ASSOCIATES on their excellent article regarding spontaneous corneal clearance despite graft detachment after Descemet membrane endothelial keratoplasty.[1] They raise interesting questions and the possibility that Fuchs dystrophy may be treatable by either just stripping off the central Descemet membrane (DM) alone or by combining that with the mere insertion of a scrolled-up donor graft of DM and endothelium into the anterior chamber without subsequent positioning. They report low but gradually increasing cell counts in the recipient between 3 and 6 to 9 months. It has now been a year and a half since those