monkey eyes.[2] Second, a recently proposed paradigm suggests that there are 2 components of intraocular pressure-induced deformation of the lamina cribrosa, one acting on the anterior lamina cribrosa surface contributing to the posterior displacement and another, which causes canal expansion and tautening or anterior lamina cribrosa movement.[3,4] Last, we recently found a significant enlargement of the BMO in 17 eyes during the acute episode of anterior ischemic optic neuropathy, followed by a significant BMO diameter shrinking at 2 months ($P = 0.008$) with no change in the contralateral healthy unaffected eyes. Shrinking of the BMO correlated with progressive thinning of the retinal nerve fiber layer at 2 months ($\rho_{Spearman} = 0.750$; $P = 0.001$). Based on this finding, we speculated that BMO enlargement was mainly owing to optic disc swelling that distended neural canal at onset, and reversed as edema reduced.[5]

These findings suggest that the optic nerve head space is not as rigid as we might think and it may behave as a dynamic structure. Unfortunately, Wang's study lacked data about BMO diameter before and after the dark room prone provocative test. We would appreciate if the authors can give any information related to this topic. These data would be very helpful to better define associated factors and to correlate their RPE findings with changes in the diameter of the BMO.

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REPLY: We thank Rebolleda et al for their interest in our study, who proposed that the morphologic changes of the retinal pigment epithelium (RPE) seen after an acute increase in intraocular pressure (IOP) may be related to a potential increase in IOP associated expansion of Bruch's membrane opening.[1] As reported in another recent article, the increase in IOP observed in the dark room prone provocative test was associated with a widening and deepening of the optic cup, a decrease in neuroretinal rim width, and a thinning of the lamina cribrosa.[2] The diameter of Bruch's membrane opening, however, did not differ between the measurements taken at baseline and the measurements performed 2 hours later at a high IOP (1530±139 vs 1531±136 μm; $P = 0.873$). It suggests that the changes in the morphology of the RPE observed at the end of Bruch's membrane after the increase in IOP may not have been caused by a widening of Bruch's membrane opening. Also, if the Bruch's membrane opening had widened during the period of increased high IOP, one might have expected a shifting of the RPE toward the end of Bruch's membrane, or at least a constant distance between the end of the RPE and the end of Bruch's membrane. However, we observed that the distance between the RPE end and the end of Bruch's membrane enlarged during the phase of increased IOP.

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Re: Freitas-Neto et al.: Increased submacular choroidal thickness in active, isolated, extramacular toxoplasmosis

(Ophthalmology 2016;123:222-4)

TO THE EDITORS: We read with interest the report of increased submacular choroidal thickness in active isolated extramacular toxoplasmosis.[1] We commend the authors' observation of significantly diffuse changes in choroidal thickness in both

macular and extramacular reactivated ocular toxoplasmosis. Adverse drug reactions have been reported to be as high as 40% in ocular toxoplasmosis.[2] Enhanced depth imaging optical coherence tomography parameter of choroidal thickness for monitoring treatment response may allow consistency in making management decisions and act as a surrogate marker to discontinue or reinitiate treatment.

In the published optical coherence tomography images, in addition to increase submacular choroidal thickness, we noted the presence of an associated vitreous haze in the active stage of the disease, whereas there was a decrease in vitreous haze and choroidal thickness in the resolved stage.[1] Perhaps the authors can comment on the correlation of vitreous cellularity with choroidal thickness at baseline and follow-up visits in their study cohort. Additionally, were there were any associated indocyanine green angiography changes in the choroid to suggest structural changes in this layer? Based on optical coherence tomography−indocyanine green angiography−vitreous haze correlation, it would be interesting to hypothesize and possibly establish the rationale for submacular changes in the current study compared with the study by Goldberg et al.[3]

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The authors of the original article declined to reply.

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Re: Yonekawa et al.: Familial exudative vitreoretinopathy: spectral-domain optical coherence tomography of the vitreoretinal interface, retina, and choroid (Ophthalmology 2015;122:2270-7)

TO THE EDITORS: The article by Yonekawa et al[1] correlated the severity of optical coherence tomography changes with the stage of disease.

Optical coherence tomography was done in 74 eyes of 41 patients. Because 33 patients had bilateral familial exudative vitreoretinopathy, which can be asymmetrical, analyzing these eyes separately for variation between eyes may add further useful information. For example, the study mentions persistence of fetal layers of the retina at fovea.[1] Variation in such a finding in the 2 eyes with different disease stages would further consolidate the hypothesis that such finding are part of the disease process rather than just an association. Similarly, in cases of bilateral familial exudative vitreoretinopathy, if cystoid macular edema was prevalent in the eye with advanced stage and not in the eye with earlier stage, it would confirm ischemia to be the cause of cystoid macular edema and not a structural change.

We hope our thoughts add value to the study and await the authors' opinion regarding the same.

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REPLY: We thank Brijesh and Azad for their interest in our manuscript.[1] Bilateral findings are a rule for familial exudative vitreoretinopathy (FEVR). "Bilateral FEVR" is thus a misnomer; virtually all patients have bilateral abnormalities if wide-field fluorescein angiography is performed.[2] Admittedly, the findings may be highly asymmetric.[2] The correspondence asks whether eyes were analyzed separately in view of the asymmetry of disease. This was precisely the case, as described in the Results section. Our analysis revealed more pathology on spectral-domain optical coherence tomography in eyes with higher stages.[1] This was true for both persistent inner retinal layers (11% of stage 1, 47% of stage 2, 6% of stages 3−5 [lower rate for stages 3−5 because of difficulty assessing the location and qualities of the fovea]) and retinal edema (14% of stage 1, 47% of stage 2, and 65% in stages 3−5), as described in the Results section.

We respectfully disagree that correlation with advanced stage confirms or requires that ischemia is the exclusive cause of cystoid macular edema, as the authors suggest. As demonstrated in Figures 1F, 4D, and 4E of our manuscript, vitreomacular traction can cause intraretinal cystic spaces without fluorescein leakage. Vitreomacular traction can be seen in any stage owing to variable degrees of hyaloidal organization. Additionally, although peripheral ischemia-related increase in vascular permeability factors such as