Author Response: Macular Migration Toward the Optic Disc After Inner Limiting Membrane Peeling for Diabetic Macular Edema

We thank Jonas¹ for their interest in our article.² They raised a question about whether the shortening of the papillofoveal distance after inner limiting membrane (ILM) peeling for diabetic macular edema (DME) depends on the shift of the neural retina on the RPE sheet or the sliding of Bruch’s membrane on the spongy choroid.

Several forces or membranes might contribute to the globe morphologies and deformation. As discussed in the article, we hypothesized that there are endogenous forces in the retinal parenchyma.² Retinal nerve fibers contain an actomyosin system, which may produce contractile forces moving toward the optic disc. The zigzag pathway of the nerve fibers after ILM peeling or the fragile ischemic retina in the temporal subfield could also result in shortening of the papillofoveal distance. Jonas et al. suggested that the exogenous effects that wall tension induced by scleral remodeling or enlargement might stretch the inner structures, including the retina, Bruch’s membrane, and the choroid in high myopia.³ Intraocular pressure also might affect such structures by wall tension, according to LaPlace’s law. Such forces might to some extent be counteracted by the ILM, a rigid membrane, which would tether the retinal parenchyma, and stretch Bruch’s membrane with elastic fibers.⁴

Regarding the main question raised by Jonas,¹ we evaluated the changes in the parapapillary regions after ILM peeling for DME. We measured the horizontal length from the edge of the sclera (probably corresponding to the disc margin) and the end of Bruch’s membrane on Spectralis optical coherence tomography (OCT) images in 41 eyes with peripapillary atrophy (PPA), but failed to find shortening of the membrane (P = 0.906). It seems that PPA on OCT images (from the scleral edge to the end of RPE on horizontal section) or color photographs (the areas relative to the vertical optic disc diameter) was not changed after surgery (P = 0.906 and P = 0.941, respectively). Further, we did not observe lesions on the horizontal OCT images corresponding to macular Bruch’s membrane defects after surgery. These data suggested that ILM peeling did not affect the sliding of Bruch’s membrane,³ although it remains ill-defined whether these structures on clinical examination correspond to the histologic ones. We could not evaluate the choroidal thickness, because we obtained spectral-domain OCT images using normal methods, but not enhanced-depth imaging or swept-source OCT, both of which would have enabled us to measure the thickness.

We then considered the second suggestion that all layers in the neural retina slide on the RPE sheet. We found that the retinal vasculature around the fovea shifted toward the optic disc, whereas photocoagulation scars within the vascular arcade did not move in eight eyes that underwent ILM peeling. This discrepancy means that the inner layers shifted toward the optic disc, which is supported by the negative correlation of papillofoveal shortening to the inner thickness, but not the outer thickness in the temporal subfield after ILM peeling.² The article by Jonas et al. showed that the outer retinal layers slid toward the restored RPE and choroid, compared to the inner layers, in the area of a macular Bruch’s membrane defect.³ In addition, ganglion cells are physiologically displaced from photoreceptor cells in the macula. These also suggested that horizontal sliding of either the inner or outer retinal layers can occur independently.

Although we did not measure the forces in the individual layers in the eye, structural changes after ILM peeling for DME suggested that the contractile properties in the inner retinal layers at least partly contribute to macular migration. We recommend that researchers consider the various forces from the innermost to the outermost segments of the eyeball and the properties in membranes when the structural changes are delineated.

Munemitsu Yoshikawa
Tomoaki Murakami

From the Department of Ophthalmology and Visual Sciences, Kyoto University Graduate School of Medicine, Kyoto, Japan. E-mail: mutomo@kuhp.kyoto-u.ac.jp

References


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