technique is also applicable for the identification (and removal) of residual or 'undetached' posterior hyaloid membrane.

The histology, immunohistochemistry, and ultrastructure of the posterior hyaloid membrane has been extensively described,²⁻⁶ and recent research has highlighted its role in the pathogenesis of many vitreoretinal disorders, including macular holes, cellophane maculopathy, macular pucker, and vitreomacular traction syndrome.^{4,5} It has also become clear that the presence or absence of a Weiss ring is insufficient for the diagnosis of complete separation of PHM.^{4,5}

The authors allude to the possibility of 'undetached hyaloid fragments' being related to future epiretinal membrane (ERM) formation, but recent research suggests that the evidence for PHM in this role is particularly strong.⁵ Incomplete separation of the PHM, rather than the cortical gel it envelops, would explain why seven patients in the study developed ERM without any associated residual vitreous staining (with one developing severe macular pucker).

We support the authors' proposition that evaluation of the vitreo-retinal relationship in eyes with retinal detachment, 'even when there is apparent PVD', is of great clinical importance. However, although triamcinolone acetate appears helpful in the demonstration of residual cortical vitreous, it is important to recognise that residual vitreous is merely a pointer to an underlying incomplete separation of the posterior hyaloid membrane.

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Sir,
Reply to Robbie and Snead

We appreciate Robbie and Snead's comment on our article: 'Intravitreal triamcinolone staining observation of residual undetached cortical vitreous after posterior vitreous detachment'. In their comment, the authors made a distinction between posterior hyaloid membrane (PHM) and cortical gel it envelops, and questioned about whether our technique is also applicable for the identification (and removal) of residual or undetached PHM. Triamcinolone acetate (TA)-assisted identification of undetached PHM utilizes the property of TA particles to adhere to the cortical vitreous for better visualization of the posterior vitreous membrane.¹ Horio *et al*² in their study of TA-assisted internal limiting membrane (ILM) peeling found, under light microscopy, TA particles adhered to the thin layer of the residual vitreous on the ILM. This finding suggests that only a thin layer of vitreous is required for TA to stain. We assume that most, if not all, residual vitreous fragments on the retina surface after recent onset PVD should contain at least a thin layer of vitreous and thus can be stained with TA. It is not known how frequent and on what conditions would 'naked' PHM exists.

Based on their histopathological studies of PHM and epiretinal membrane (ERM),^{3,4} the authors suggest that the ERMs developed in seven cases in our series come from undetached PHM. Ultrastructural studies of ERMs found various types of cells within the membranes, including fibrous astrocytes, fibrocytes, macrophages, and even retinal pigment epithelial cells. The combination of cells in each membrane depends on clinical entities.⁵ In our study, 10 of 23 cases had macular TA staining, and none developed visible macular ERM, and those seven postoperative ERM cases did not had intraoperative TA staining in the corresponding area. Although PHM may be important in the formation of ERMs in other vitreoretinal diseases, such as stage 4

macular hole,⁶ our study results suggest that residual hyaloid may not be a necessary factor for the development of ERMs in eyes with RD.

Finally, we concur with Robbie and Snead's opinion that the presence of Weiss ring is insufficient for the diagnosis of PVD. Importantly, the actual vitreoretinal relationship in the periphery is not known after the development of Weiss ring. Still, the appearance of Weiss ring remains the most easily identifiable and reliable sign to indicate that all or part of the vitreous hyaloid has been separated from the posterior retina.^{3,4,6} Because we only used clinical examination for recruiting our cases, we specifically verified the presence of PVD by direct observation of the posterior vitreous during the initial phase of the surgery (see Materials and methods section in our article). In our conclusion, we used the phrase 'apparent PVD' to denote the inexactness of preoperative clinical examination in identifying true PVD.

We appreciate efforts put by the authors in the studies of the ultrastructures of vitreoretinal interface.^{3,4} They raised the important issue of PHM, and its role in the formation of certain ERM. We hope further studies may clarify the significance of PHM in the formation of ERM after RD, and the interaction between TA and PHM.

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Sir, Keratoconus in tuberous sclerosis

A 25-year-old Sri Lankan man presented with gradual deteriorating vision uncorrectable with spectacles. His best spectacle corrected visual acuity was 6/18 (right) and 6/9 (left), with no improvement with pinhole. Anterior segment examination showed Vogt's striae in both corneas. Fundoscopy revealed bilateral astrocytic hamartomas on the disc and nasal retinal hamartoma in the right (Figure 1b). Further examination revealed adenoma sebaceum (Figure 1a) and shagreen patch (Figure 1c).

Figure 1 Corneal topography (Orbscan).