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Sir,
Reply to Doyle *et al*

We would like to thank Doyle *et al* for their interest in our reported series of four cases.¹ We agree that logMAR visual acuity measurements should be encouraged especially in situations where measurements using a Snellen chart may not be sensitive enough to recognise a change in the acuity. In keeping with the standard practice we routinely record logMAR visual acuities for all patients undergoing PDT. In the text of our report we have reported the decline in visual acuity in terms of the drop in the number of letters read on the logMAR chart. This is similar to the way results were summarised in the TAP study² where reduction of visual acuity by fewer than 15 letters over a 24-month period was considered as beneficial effect of the treatment. Furthermore, although one of our patients lost only 14 letters, this happened rapidly over a 9-month period in spite of closure of the CNV which is not strictly

comparable with the beneficial outcome reported in the TAP study.

Doyle *et al* have said that nowhere in the paper is the actual logMAR visual acuity shown. We would like to draw their attention to Figures 2 and 3 in our report that clearly mention the actual logMAR visual acuity values for that case. We feel that using the reciprocal of actual logMAR values appropriately illustrates graphically in Figure 1 the steep decline in visual acuity as a downward slope. We do not think that adding a table showing logMAR visual acuities would have added to our message from this small case series that in certain cases visual acuity could decline fairly rapidly in spite of a 'successful' PDT treatment.

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Sir,
Intravitreal triamcinolone staining observation of residual undetached cortical vitreous after posterior vitreous detachment

The use of intravitreal triamcinolone to demonstrate areas of undetached vitreous¹ has gained in popularity in recent years. The authors have not clarified whether their

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 vitreoretinal relationship in eyes with retinal detachment, 'even when there is apparent PVD', is of great clinical importance. However, although triamcinolone acetonide 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 acetonide (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 exist.

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