Dear editor: Stitt et al. [1] recently presented two cases of histopathologic analysis following PRP laser therapy for proliferative diabetic retinopathy (PDR). There was no mention of whether the new vessels, which were presumably noted prior to laser therapy, “regressed” following treatment. If so, was fluorescein angiography performed to confirm this? More importantly, if there was “regression”, what were the histopathologic features of these “regressed” structures?

One of the hypotheses explaining the beneficial effect of PRP therapy contends that laser therapy induces posterior vitreous detachment (PVD). Thereafter, any new vessels that arise or persist will be “abortive” [2] in the sense that they are not likely to bleed or be a nidus for traction upon the retina. In one study [3] the incidence of PVD following PRP was 53%, compared to 7% in controls (P<0.02).

Apart from not mentioning this as one of the potential effects of PRP therapy, the authors regrettably show no photomicrographs displaying the vitreoretinal interface. As it is probable, if not certain, that the histologic sections obtained in this study [1] included the inner retina, could the authors address whether or not vitreous cortex was found attached in the area of laser treatment?

References

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Thomas A. Gardiner

Reply

Dear editor: In response to the comments of Dr. Sebag we wish to point out that, as the main focus of our paper concerned the microanatomical legacy of panretinal photocoagulation at the chorio-retinal interface, we chose not to discuss the changes at the vitreous surface. However, we can confirm that on gross examination of the specimens the posterior vitreous did appear degenerate and that electron microscopy revealed no attachment between the internal limiting membrane of the retina and vitreous collagen fibres. It may also be of interest that although no vitreous haemorrhage or traction detachment had occurred in the 6-month period following photocoagulation, in at least one of the eyes the fronds of new vessels at the periphery of the macula showed no evidence of regression. Electron microscopy of these vessels revealed a well-preserved endothelium perfused by a plasma-like substance and red blood cells. The vessels were enmeshed in a fine collagen matrix which was continuous with the breach in the internal limiting membrane from which the vessels were derived. At the editor’s discretion we are willing to provide several additional micrographs to illustrate the above points.

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