antibacterial activity from Oxybuprocaine® in the presence of P. aeruginosa and S. marcescens (MIC > 2000 mg/l). However, a MIC was determined for all the other microbes, from 250 mg/l for M. catarrhalis up to 2000 mg/l for S. aureus and S. epidermidis. Finally, a MIC was determined for all the microbes in the presence of Tetracaine®. These MIC were systematically lower than with Oxybuprocaine®, ranging from 312 mg/l for C. macginleyi and M. catarrhalis up to 2500 mg/l for P. aeruginosa.

The MIC results are summarized in Table 1.

This is the first study to evaluate the antibacterial activity of Fluorescein®. Our MIC results for the local anaesthetics were similar to those of previous studies (Kleinfeld & Ellis 1966; Labetouille et al. 2002; Pelosini et al. 2009). It should be noted that in clinical practice, Fluorescein® is very often used in combination with a local anaesthetic (Daru- gar et al. 2011). An additional analysis was therefore carried out using the combinations of Fluorescein®-Oxybuprocaine® and Fluorescein®-Tetracaine® (Table 1). No interaction was detected for the Fluorescein®-Tetracaine® combination. However, the Fluorescein®-Oxybuprocaine® combination appeared to have a synergistic effect on Gram-positive cocci (Staphylococci and Pneumococci).

Finally, with any cases of severe CA, the patient’s eye should be routinely rinsed with sterile saline solution before the patient’s eye should be routinely rinsed with sterile saline solution before the patient’s eye should be routinely rinsed with sterile saline solution before the patient’s eye should be routinely rinsed with sterile saline solution before the patient’s eye should be routinely rinsed with sterile saline solution before the patient’s eye should be routinely rinsed with sterile saline solution before.
In 2016, 3/26 (12%) patients with pVCR developed a redetachment compared to 1/65 (2%) patients without pVCR. In each of the three cases with pVCR and redetachment, pVCR were not completely removed during the first operation and a causative PVR membrane was identified during the second operation. No PVR had developed in the redetachment case without pVCR.

Differences between 2016 and 2018 could be attributed to more extensive and targeted staining (better detection) and more effective and safer removal of pVCR in 2018, using a new technique for pVCR removal, Vitreous Wiping (Van Overdam et al. 2018). The findings of this preliminary study support the theory that pVCR are more prevalent than previously thought (not only in highly myopic or diabetic eyes), that pVCR play a role in PVR development, and that visualization and removal of pVCR can improve the surgical outcome and reduce retinal redetachment rates.

Certainly, larger prospective studies are required to further support and confirm these findings, but the author suggests that a ‘missing link’ in the pathophysiology of PVD and PVR has been revealed: VCR over the (mid)peripheral retina (peripheral membrane), which can act like a scaffold for fibrocellular proliferation, in which RPE cells from the macula (posterior membrane), which may result in a macular hole or macular pucker (depending on membrane thickness, presence of hyalocytes and vitreopapillary adhesion). In this updated version, the diagram is completed by adding that not only full, but also partial thickness PVD can be revealed: VCR over the (mid)peripheral retina (peripheral membrane), which may result in a macular hole or macular pucker (depending on membrane thickness, presence of hyalocytes and vitreopapillary adhesion). In this updated version, the diagram is completed by adding that not only full, but also partial thickness PVD can lead to peripheral and posterior traction, but most importantly by adding the ‘missing link’: anomalous PVD with vitreoschisis can lead to VCR over the (mid)peripheral retina (peripheral membrane), which can act like a scaffold for fibrocellular proliferation, in which RPE cells from retinal tears and hyalocytes in VCR, together with previously identified PVR risk factors, conspire to form PVR membranes.

Fig. 1. Updated, redesigned version of the schematic diagram of anomalous PVD (Sebag et al. 2014), including the ‘missing link’ (indicated in red). The original version showed that PVD can be full thickness (without VCR) or partial thickness (vitreoschisis with VCR). Full thickness PVD may lead to peripheral traction (which may result in a retinal tear and detachment), posterior traction (which may result in vitreomacular or vitreopapillary traction/adhesion and macular hole) or no traction (without vitreoretinopathy). Partial thickness PVD may lead to VCR over the macula (posterior membrane), which may result in a macular hole or macular pucker (depending on membrane thickness, presence of hyalocytes and vitreopapillary adhesion). In this updated version, the diagram is completed by adding that not only full, but also partial thickness PVD can lead to peripheral and posterior traction, but most importantly by adding the ‘missing link’: anomalous PVD with vitreoschisis can lead to VCR over the (mid)peripheral retina (peripheral membrane), which can act like a scaffold for fibrocellular proliferation, in which RPE cells from retinal tears and hyalocytes in VCR, together with previously identified PVR risk factors, conspire to form PVR membranes.

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Choroidal naevus regression associated with PD-1 inhibitor monotherapy for metastatic cutaneous malignant melanoma

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