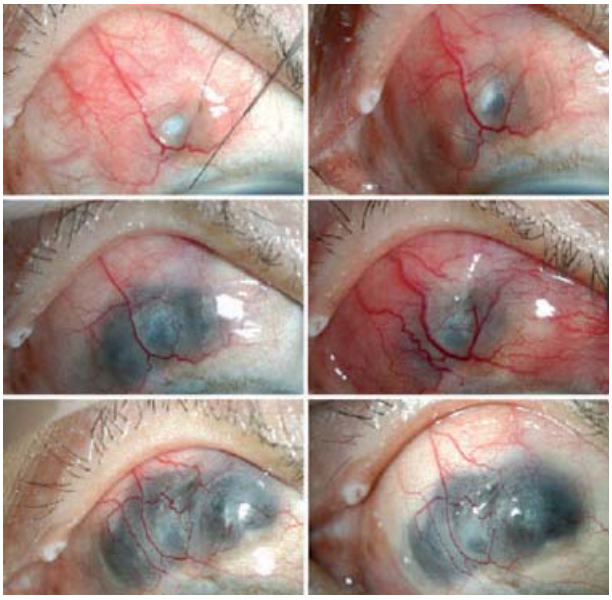

Fig. 1. Onset of scleritis during etanercept therapy. (Top left) 3 month after restarting etanercept retreatment, her scleritis was exacerbated. (Top right) The scleritis was completely limited 3 months after increasing the prednisolone dosage up to 30 mg/day without etanercept (middle left). The scleritis recurred 6 weeks after the second restart of etanercept treatment with 10 mg of prednisolone (middle right). The scleritis was completely reduced 3 months after increasing the prednisolone dosage up to 30 mg/day without etanercept (Bottom left). Two months after

methotrexate treatment, we succeeded in reducing prednisolone to 5 mg (original maintenance dosage) without any recurrences (Fig. 1). To date, there has been no flare-up of scleritis for 12 months.

According to previous reports, TNF-α may play a role in uveitis and scleritis in patients with RA (Di Girolamo et al. 1997; Braun et al. 2005; Smith et al. 2005), but anecdotal reports paradoxically implicated etanercept as a cause of uveitis and scleritis (Lim et al. 2007; Le Garrec et al. 2009). On the other hand, another anti-TNF medication, infliximab, is effective in controlling eye involvements (Braun, et al. 2005). Our case suggests that etanercept may induce intractable scleritis or is ineffective in treating scleritis. Switching anti-TNF treatment from etanercept to infliximab was effective in controlling both RA and scleritis. The discrepancy of efficacy in the treatment of eye involvements might come from the mechanisms of these anti-TNF medications. Infliximab can bind to transmembrane forms of TNF-α, resulting in a breaking down of TNF-α-producing cells, while etanercept cannot affect TNF-α-producing cells because of its lack of binding to transmembrane forms of TNF. Our report does not suggest that infliximab should be preferred over etanercept in treatment of patients with inflammatory disease that may or may not be associated with eye involvements. However, if a patient has scleritis during etanercept therapy, then a change to infliximab therapy may be valuable.

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Beta blocker use and age-related macular degeneration

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doi: 10.1111/j.1755-3768.2011.02117.x

Editor,

Through serendipity, Léauté-Labrèze et al. discovered that propanolol induces regression of infantile hemangiomas (Léauté-Labrèze et al. 2008; Siegfried et al. 2008). A non-selective beta blocker, propranolol, may affect involution of infantile hemangioma by decreasing the expression of vascular endothelial growth factor (VEGF).

Since propranolol may cause decreased expression of VEGF, oral or topical beta blockers may have a beneficial effect on patients with age-related macular degeneration (AMD). We retrospectively compared the use of oral and topical beta blocker medication in patients with wet and dry AMD to identify a possible protective effect of beta blockers against choroidal neovascularization.

The medical records of all new patients who were referred to our group retina practice between January 1, 2007 and December 31, 2008 were screened for inclusion in this study. A diagnosis of AMD was confirmed by reviewing the history, examination, and diagnostic testing. Each eye was categorized as dry or wet AMD based on the age-related eye disease study classification (AREDS Study Group 2001).

Table 1. Patient demographics and beta blocker use.

	All	Wet AMD	Dry AMD	Beta blocker	No beta blocker
Number (*N*)	919	363 (39%)	556 (61%)	288 (31%)	631 (69%)
Age (years)	84	81	86	81	85
Smokers (*N*)	49 (5%)	23 (6%)	26 (5%)	15 (5%)	34 (5%)
Oral beta blocker (*N*)	290 (32%)	115 (32%)	175 (31%)		
Oral non Selective beta blocker (*N*)	58 (6%)	22 (6%)	36 (6%)		
Topical beta blocker	57 (6%)	21 (6%)	36 (6%)		

AMD = age-related macular degeneration.

Patients with wet AMD in either eye were categorized as a wet AMD for the purpose of this study. One thousand and five charts were reviewed. Nine hundred nineteen patients were included in the study.

The study complied with the Declaration of Helsinki. The research protocol was approved by the institutional review board of the University of South Florida.

Of the 919 patients included in the study, 363 had wet AMD in at least one eye. Sixteen (6%) of 363 patients with wet AMD were current smokers and 26 (5%) of 556 patients with dry AMD were current smokers. The average age of patients included in the study was 84 (Table 1).

There was no significant difference in use of oral selective beta blocker, oral non-selective beta blocker or topical beta blocker medications between patients with wet AMD and patients with dry AMD (Table 1).

The variable course of patients treated for wet AMD suggests that there are yet to be determined factors that may affect the clinical course patients with AMD. We chose to look for a link between beta blocker usage and wet AMD because beta blockers are useful in treating infantile hemangioma and they have a long history of safe use for cardiovascular disease.

Our study showed no difference in beta blocker usage between patients with dry AMD and wet AMD (Table 1). The negative findings of this study – that the percentage of patients using topical or oral beta blockers was the same in the wet AMD group and the dry AMD group – suggest that beta blockers do not protect patients from developing wet AMD.

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Dispase-assisted vitrectomy for epiretinal prostheses implantation

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doi: 10.1111/j.1755-3768.2010.02084.x

Editor,

We previously reported about a new surgical method for the implantation of epiretinal prostheses in porcine eyes (Ivastinovic et al. 2010a). While performing vitrectomy as part of the implantation procedure, we noted a strong vitreoretinal adherence hampering a complete removal of the posterior vitreous cortex. However, close proximity between the electrodes and ganglion cells is crucial for successful induction of visual perception with epiretinal implants (de Balthasar et al. 2008). Although in elderly humans posterior vitreous detachment (PVD) is commonly observed during vitrectomy, vitreoschisis might occur at different levels owing to the multilamellar structure of the posterior hyaloid (Sebag 2008). Hence, considerable amounts of vitreous remnants might still remain attached to the inner limiting lamina (ILL). Intravitreal pharmacological adjuncts, such as plasmin and dispase, have been shown to facilitate PVD. While plasmin hydrolyses laminin and fibronectin, which play key roles in the vitreoretinal attachment, dispase additionally degrades type IV collagen in the ILL, which supports the attachment of the vitreous cortex to the ILL (Wang et al. 2004). The incidence of dispase-related complications including preretinal haemorrhage, vitreous inflammation and morphological retinal damage proved to be dose dependent and more likely to occur after prolonged exposure (Wang et al. 2004). To obviate these adverse effects, the injected dose of dispase should not exceed 50 μg, and the incubation should be limited at 15 min (Oliviera et al. 2001). The aim of our study was to evaluate the efficacy and safety of 50 μg dispase at the proposed exposure in porcine eyes with respect to epiretinal prostheses implantation.

We performed an experimental study in eight domestic pigs weighting 20–25 kg. This animal trial was approved by the Austrian legal authorities and the Medical University of Graz. The animals were treated according to guidelines of the Association for Research in Vision and Ophthalmology. Surgeries were performed under sterile conditions in general anaesthesia using standard parameters. One day before surgery, dispase (isolated from Bacillus polymyxa; Roche Diagnostics, Grenzach-Wyhlen, Germany) was diluted with phosphate-buffered saline to 50 μg/0.05 ml