

trabecular meshwork blockage, failure of glaucoma surgery, cataract and posterior capsule opacification, and proliferative vitreoretinopathy. In their study, **Ochiai and Ochiai** (Japanese Journal of Ophthalmology 2002; 46: 249-253) found, like others, **higher levels of the active form of TGF- β 2 in the aqueous of patients with glaucoma**, diabetes, and both glaucoma and diabetes. Interestingly, they found the levels in the **glaucoma patients to be higher compared to the diabetic patients**. However, where this TGF- β 2 comes from (leakage from the blood aqueous barrier or local production) is uncertain. However, since TGF- β 2 is auto inductive, *i.e.*, it stimulates further cellular production of TGF- β 2, raised levels of TGF- β 2 may then persist. The exact role in primary and secondary pathogenesis of glaucoma and diabetes is still uncertain, but some of the more recent studies using the human antibody to TGF- β 2 in patients following glaucoma surgery and cataract extraction may help us to clarify the role of this interesting and powerful growth factor in a variety of important clinical situations.

Scleral thickness

Comment by Mark Johnson

Glaucoma is a disease characterized by an elevated IOP. The elevated IOP generates biomechanical stresses in the sclera, which may be related to the optic nerve fiber damage seen in glaucoma. If the sclera is viewed as a uniform spherical shell (a reasonable first approximation), then the stresses in the sclera are proportional to the IOP and to the radius of the eye, and are inversely proportional to the thickness of the sclera. In regions where the sclera is thinner, the stresses will be higher. In the region of the optic nerve, the scleral canal creates a stress concentration factor that roughly doubles the scleral stresses in the neighborhood of the opening.

Downs *et al.* were interested in investigating the thickness of the sclera in the immediately neighborhood of the scleral canal. In previous work (Investigative Ophthalmology and Visual Science, December 2001), Downs *et al.* had shown that the thickness of the primate sclera was thicker in the foveal region and near the sclera canal. This is consistent with the idea of reducing the otherwise elevated stresses around the scleral canal to minimize the impact of these stresses on the optic nerve. In their current study (395), Downs *et al.* examined the thickness of the primate sclera in the immediately vicinity of the scleral canal. They found that, surprisingly, immediately adjacent to the scleral canal, the sclera was very thin. However, the thickness of the sclera rapidly increased within a short distance (0.6-1 mm) and reached a maximum thickness in this region. This thickened region may be a



serve the function of a reinforcing ring that shields the optic nerve from scleral stresses. The authors hypothesize that the elevated pressure associated with glaucoma may cause thinning of the sclera in the region of the scleral canal, and that this thinning will elevate the already increased stresses in the sclera. Transmission of these stresses to the lamina cribrosa and/or blood vessels passing through the sclera may have pathological consequences.

Intravitreal cell injections

Comment by Marty Wax



There is a somewhat popular hypothesis that has as its premise, the idea that the main problem in macular degeneration is a specific defect in the retinal pigment epithelium (RPE) in aging eyes. It is unclear whether this problem resides in the inherent function of the RPE cells, or whether age-related macular degeneration (ARMD) results from defective cell adhesion interactions of the RPE with Bruch's membrane. Nevertheless, the hypothesis remains that one potential therapy for this disorder may be achieved by replacing defective or absent RPE cells in susceptible or appropriately diseased ARMD patients. If that is the aim, where will the replacement cells come from? Jordan *et al.* (622) advocate that one such source may be from autologous cell transplantation using iris pigment epithelial (IPE) cells which are placed into the subretinal space of eyes with ARMD. Presumably, healthy IPE cells could provide a source of cytokines for degenerating cells, thereby achieving cell 'rescue', or perhaps serve as a viable substitute for the decaying RPE cells themselves. In this study, the authors isolated IPE cells from Long Evans rats and injected them into the vitreous cavity of Wistar rats, without preculturing. They also injected free melanin granules separately in a similar manner. What they found was that both the free melanin granules, as well as the injected IPE cells, accumulated in the prepapillary region of the optic nerve head and that no proliferative vitreoretinopathy accompanied this process. No IPE cells were found either on or integrated into the peripheral retina. No integration, but rather only deposition, occurred with IPE cells that were found in the area of the disc head. The fact that both IPE cells and free melanin granules accumulated in the same area suggests that a passive mechanism was attributable to this process. Perhaps one explanation for this is an effect similar to the low speed centrifugation that occurs in a swinging bucket rotor centrifuge; in this case, the recipient eye is the swinging bucket. Intravitreal injection of autologous IPE cells directed towards the optic nerve head region may be possible, but unfortunately such experiments do little to convince us that any functional integration of the in-