

# BJO at a glance

Creig Hoyt, Editor

## WHAT SHOULD BE THE DRUG OF CHOICE IN THE TREATMENT OF OPEN ANGLE GLAUCOMA?

A little over two decades ago topical  $\beta$  blockers became the primary treatment for chronic open angle glaucoma.  $\beta$  Blockers replaced cholinergics (primarily pilocarpine) despite the fact that  $\beta$  blockers were seen as considerably more expensive. The advantage of  $\beta$  blockers was their apparent increased effectiveness, and the minimal amount of local side effects. Despite the fact that systemic side effects associated with their use are not inconsiderable,  $\beta$  blockers have remained the drug of choice for initial treatment of chronic open angle glaucoma. A controversy moderated by Ivan Goldberg addresses the question of whether  $\beta$  blockers should now be replaced by some of the newer agents available for treatment of open angle glaucoma. What are these newer agents? They are primarily  $\alpha_2$  agonists, prostaglandin analogues and related drugs, and topical carbonic anhydrase inhibitors. Some of them appear to offer equal or perhaps even better hypotensive effect than  $\beta$  blockers. Moreover, the side effects associated with their use appear to be primarily local, non-systemic, and not life threatening. At least for the moment, the use of  $\beta$  blockers appears to be more cost effective than the use of the newer agents. Nevertheless the question is posed: is it time to replace a group of drugs with significant systemic side effects with drugs that, although slightly more costly, have primarily local side effects? **See p 691**

## IS THIS REALLY A CASE OF TOXOPLASMA RETINOCHOROIDITIS?

One of the appealing features of clinical ophthalmology is the ability to examine directly most structures of the eye. The ability to establish the appropriate diagnosis from physical examination alone

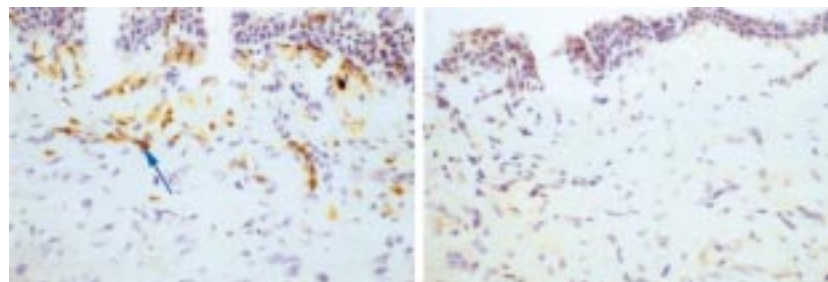
is rewarding. Nevertheless, the limitations of clinical observations in ophthalmology have been brought to our attention. Interobserver variability in the evaluation of the optic nerve head in the patient suspected of having open angle glaucoma is well established. Gilbert and co-workers studied the reliability of uveitis experts' interpretation of retinal photographs in establishing the diagnosis of toxoplasma retinochoroiditis. Five uveitis experts were asked independently to classify retinal photographs into four categories—definite, probable, possible, or not toxoplasma retinochoroiditis—without any additional clinical information. Although there was moderate agreement among the experts in these studies, there was substantial inter-observer variation. This study concluded that in a low prevalence setting uveitis experts are more likely to diagnose toxoplasma retinochoroiditis and perhaps treat patients who do not have the disease than to miss patients who have the disease. **See p 636**

## OUTCOMES OF CORNEAL GRAFTING IN PATIENTS WITH HERPETIC KERATITIS

Regrettably, herpetic keratitis remains a significant cause of visual morbidity. Recurrent herpetic infection may lead to significant corneal scarring that adversely affects visual function. Prior to the availability of systemic antiviral therapy survival rates for corneal grafting in patients with herpetic keratitis were considered poor. Recently it has been suggested that corneal graft survival rates can be increased if patients undergo systemic antiviral therapy prior to surgery. Garweg and co-workers report the outcome of corneal grafting in patients with stromal keratitis of herpetic and non-herpetic origin. In a study group of 384 immunocompetent adults they found that the 5 year graft survival for patients with herpetic keratitis was similar to those obtained in individuals with non-herpetic keratitis. They attribute these relatively good results to the instigation of combined antiviral and local immunosuppressive therapy immediately after transplantation. **See p 646**

## WHAT MEDIATES THE DEVELOPMENT OF CHRONIC CONJUNCTIVAL INFLAMMATION?

Although normal wound healing is usually a surgeon's ally it may in some cases be the primary complication. Certainly in the case of many forms of glaucoma surgery and vitreoretinal surgery chronic inflammation is a recognised risk factor for failure of the procedure. The immune system is known to play an essential role in wound healing following glaucoma surgery. Yet, for appropriate wound healing to occur the immune system must also deactivate itself in order to avoid the consequences of a persistent population of activated inflammatory cells. A failure of the immune system to deactivate itself might be an important factor in the development of chronic inflammation where persistent inflammatory cells would stimulate fibroblasts and excessive scar tissue production. Chang and co-workers studied conjunctival tissue from glaucoma patients at the time of their filtration surgery. Using an immunohistologic technique they identified fibroblasts that produced interferon  $\beta$  and T lymphocytes in the conjunctival specimens. This suggests that the development of this interaction may contribute to the development of chronic inflammation and excessive scar tissue formation in glaucoma patients. **See p 611**



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