migration of RPE. It appears, however, that, like GA, the atrophy surrounding the scars does not progress indefinitely, and the factors in the more peripheral retina that make it more resistant to the atrophic process in the cases discussed here, and in GA in general, remain to be clarified.

Attention is just now beginning to focus on possible treatments to slow the progression of GA over time. The AREDS study had an insufficient number of patients with GA to show a statistically significant outcome related to the study medication.²⁰ Since GA is a degenerative disorder, a progression which continues chronically over time, it is likely that medication will have to be supplied on a regular basis, whether systemically, pericocularly, or intraocularly. It is possible that the ultimate treatment for CNV will include both antiangiogenic agents to address the neovascular process and trophic factors to promote RPE survival and limit progressive atrophy.

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Retinal tumours

Vasoproliferative retinal tumour

B Damato

Brachytherapy requires further evaluation

Vasoproliferative retinal tumour is an enigmatic disease, characterised by one or more retinal nodules, which are usually located pre-equatorially and inferotemporally, and which cause retinal exudates, macular oedema, and epiretinal membranes. Numerous single case reports and several case series have been published, with the disease described using a variety of terms.^{1–4}

The tumour in question consists mostly of glial cells interlaced with a fine capillary network and dilated, hyalinised blood vessels, some of which are occluded.^{5–7} Exudates, macrophages, and foreign body giant cells are also present. The histology does not indicate a "vasoproliferative" tumour. The term "reactionary retinal gliosis" has been proposed.⁵

Approximately 75% of cases are idiopathic and 25% are secondary to other ocular diseases, such as retinitis pigmentosa, uveitis, retinal detachment, congenital toxoplasmosis, and Coats' disease.^{3, 8} Multiple lesions occur in about 6% of patients without predisposing disease and in 41% of patients with pre-existing ocular disease.³ Multiple lesions can be bilateral, even in the absence of any apparent underlying ocular disease, especially in females.³ Bilateral vasoproliferative tumours have been reported in a pair of monozygotic twins.⁹ There is no sex preponderance. The condition can present at any age but is usually detected between the ages of 40 years and 60 years. A rare, diffuse variety of vasoproliferative tumour exists, which is relatively aggressive and which tends to occur in young females (mean age 19 years).³

Patients tend to present with visual loss, floaters, and/or photopsia. On ophthalmoscopy, the tumour has the appearance of a yellow or pink, intra-retinal mass associated with adjacent hard exudates and occasionally retinal and vitreous haemorrhages. The hard exudates tend to extend posteriorly, eventually involving the fovea. There may also be macular oedema and exudative retinal detachment, which can become total. In advanced stages, there can be neovascular glaucoma. Epiretinal membranes may also develop, which can cause retinal distortion. The natural course of this disease varies from patient to patient, progressing slowly or not at all in some but causing severe ocular complications in others.

Fluorescein angiography shows a rich capillary network and/or telangiectatic blood vessels within the tumour. These leak profusely so that the entire lesion is hyperfluorescent in the late stages of the angiogram. On ultrasonography, vasoproliferative tumours vary in size from 1.0 mm to more than 5 mm, averaging about 3 mm. The internal acoustic reflectivity varies from one tumour to another and can be low, medium, or high. Tumour biopsy may be needed in some patients.

The differential diagnosis includes eccentric disciform lesion, retinal haemangioblastoma, amelanotic melanoma, choroidal haemangioma, and Coats' disease.

Observation is recommended if a small, peripheral lesion is not causing much exudation and if there seems to be no threat to vision.¹⁰ Cryotherapy using the triple freeze-thaw technique can conveniently be applied transconjunctivally in most cases, but repeated treatment may be required, especially if the tumour thickness exceeds 2 mm.³ As early as 1932, Traquair successfully used brachytherapy to obliterate exudative retinal haemangioblastoma.¹¹ Anastassiou *et al* have used essentially the same approach for the treatment of vasoproliferative tumours, and in this issue of the *BJO* (p 447) they report their results with 35 consecutive patients treated with ruthenium plaque radiotherapy. Tumour regression and resolution of the exudation were achieved in 31 patients (89%). The four failures occurred in advanced cases, with total retinal detachment and neovascular glaucoma. After a median follow up of 24 months, the visual acuity was the same as at presentation or better in 20 eyes and only five of the remaining patients had severe visual loss. The main cause of visual loss was cellophane maculopathy.

Vitreoretinal surgery may be required to treat vitreous haemorrhage or an epiretinal membrane. Hard exudates threatening the fovea have also been removed successfully (Groenewald, Damato, unpublished data); however, this treatment requires further evaluation. Advanced disease with exudative retinal detachment and neovascular glaucoma may be treatable by surgical removal of the "toxic tumour." This approach has proved successful in treating severe exudative and neovascular complications developing after radiotherapy of uveal melanoma.¹²

Photodynamic therapy has recently been shown to be effective.¹³ There is scope for investigating agents such as intravitreal triamcinolone and anti-angiogenic factors like ranibizumab (Lucentis), bevacizumab (Avastin), and anacortave acetate (Retaane). We can expect several case reports on such novel therapies in the near future and it will be interesting to see whether any will supersede the brachytherapy described in this issue of *BJO*.

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ROP

The Boer War and fuzzy logic in screening

M P Clarke

Population screening programmes demand considerable commitment and resources, not only from healthcare providers but also from the population to be screened

The origins of population screening have been attributed to events at the beginning of the 20th century when, in the aftermath of the Boer War, steps were taken to improve the health of British children and, therefore, in due course, of recruits to the British army. The principles underlying screening, and the criteria outlined by Wilson and Junger to be applied to proposed programmes,¹ have stood the test of time and will be well known to most readers. In truth these criteria are not fully met by most, if not all, medical screening

programmes. Even if proponents are convinced that they can reliably identify an asymptomatic early stage and apply a treatment that is more effective when received early, the process of screening often brings to light ambiguous states of health which have uncertain natural histories.

Despite these difficulties, screening remains a popular concept. Nobody wants to get ill, or for their children to be ill, and if a simple test and acceptable preventative treatment can stop this happening, so much the better. No one

could argue that detection of early, localised, asymptomatic breast carcinoma, which would have metastasised by the time symptoms had occurred, is not a good, and politically popular, thing.

Things are, however, rarely so clear cut. Population screening programmes demand considerable commitment and resources, not only from healthcare providers but also from the population to be screened. The case where metastasis might have been prevented, despite the value this has for the individual, has to be weighed against the time lost from work for the rest of the population to be screened, the anxiety generated in the inevitable false positives, the definitely unnecessary and possibly inappropriate treatment for other individuals, the appointment slots that could otherwise have been given to the symptomatic, and the potential for harm and litigation among those falsely reassured. And population screening programmes demand a cohesive public health service.

In the United Kingdom, adult population screening programmes exist (for those at risk) for breast and cervical cancer. For children, there are seven age stratified population screening