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 Hard exudates threatening the fovea have also been removed successfully (Groenewald, Damato, unpublished data); however, this treatment requires further evaluation.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

programmes: antenatal care; newborn physical examination, hearing test, and blood tests for phenylketonuria and hypothyroidism; an examination at 6 weeks that focuses on heart, hips and eyes; visual screening between 4–5 years; and height and weight measurement at school entry.² It is notable that three of the seven include a vision component. Preschool vision screening at age 3 has been largely replaced by orthoptic led, school entry screening, which has the advantage of better population coverage and more reliable test procedures.

Screening of whole populations is a relatively inefficient way of detecting disease. Targeted screening, or clinical surveillance, of conditions with multi-system involvement is more rewarding. Examples of such conditions in ophthalmology include diabetes, juvenile idiopathic arthritis, Marfan syndrome, neurofibromatosis, and, of course, retinopathy of prematurity (ROP). Screening for, and treatment of, ROP began on a large scale in the United Kingdom, and many other countries, in the early 1990s, following the first report of the Cryo-ROP study,³ and helped to create paediatric ophthalmology as a specialty in the United Kingdom.

The consequences of premature birth last a lifetime and include visual sequelae such as cerebral visual impairment, refractive errors, strabismus, and amblyopia. In this issue of the *BJO* (pp 000 and 000), O'Connor *et al* argue for targeted screening of very low birthweight children at 2–2.5 years. They demonstrate in their article that a plethora of diverse policies exist in orthoptic departments and children's eye clinics in the United Kingdom with regard to screening of very low birthweight children—a situation similar to that which used to exist in relation to preschool visual screening.⁴ Many, if not all, of these children will also be under the scrutiny of a paediatrician, although it is likely that a diversity of practice exists here too.

It cannot be useful for such diverse policies to continue—either some units are wasting their own, and their patients', time, or other units have patients who are missing out on the chance to reduce the burden of preventable disease. It may be that some local

health services would decide this was a low priority area; however, in an increasingly fragmented, but still national, health service, there should be a national recommendation and rationing decisions should be explicit.

While acknowledging that many existing programmes do not meet the Wilson and Junger criteria, they remain a benchmark against which proposed new programmes should be judged. Central to them is that the condition should have a recognisable, but asymptomatic, early stage at which treatment is more effective than treatment applied later.

Perhaps the most useful visual intervention that could be performed for very low birthweight children would be to refract them between 6 months and 1 year

In targeted screening of very low birthweight children, the primary aim must surely be to prevent visual impairment, especially bilateral visual impairment. Testing of acuity at this age is difficult, particularly if the child is neurologically impaired, and normal ranges are broad. Visual impairment in these children will most commonly be the result of ROP, cerebral visual impairment, refractive errors, and amblyopia. Neither ROP nor cerebral visual impairment are preventable by screening at 2–2.5 years, although it may be argued that early recognition of visual impairment from these causes is valuable in informing developmental and educational strategies. Significant degrees of visual impairment from either cause are, however, likely to be obvious to parents, general practitioners, and paediatricians and will probably prompt referral earlier than at 2 years. Lesser degrees of visual impairment, including monocular impairment, may remain undetected and it may be that in some cases, early detection may allow for more effective treatment.

Strabismus that is not of cosmetic concern to the parents of a very low birthweight child is arguably of no importance except as a marker for neurological sequelae of prematurity and visual impairment. Visual impairment in a strabismic eye of a very low birthweight child may be the result of

cerebral visual impairment and is unresponsive to amblyopia treatment. I would argue that while there may be a case for screening for visual impairment, there is not a case for screening for strabismus itself.

Refractive error is similarly a proxy marker of visual impairment. The situation here is complicated by the fact that many hypermetropic and astigmatic refractive errors, and degrees of anisometropia, are compatible with normal visual acuity in children, and that accurate refraction of small children is difficult. Judgments have to be made about which refractive errors should be corrected, either because they are thought to be affecting acuity, or are likely to do so by causing amblyopia or strabismus. These judgments will be affected by the presence of neurodevelopmental impairment: children with such impairment may benefit from correction of low hypermetropic refractive errors or from an additional reading correction if their accommodation is deficient. What is certainly true is that very low birthweight children have a much higher than normal incidence of significant myopia, and that early recognition and correction of this will be of benefit to all except the most cerebrally visually impaired child.

Perhaps the most useful visual intervention that could be performed for very low birthweight children would be to refract them between 6 months and 1 year, to identify high refractive errors; that, and advice to parents and paediatricians to be alert for, and to refer promptly, very low birthweight children with visual symptoms.

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