

adequate IOP control; whereas eyes with advanced glaucomatous optic neuropathy are more likely to have poor residual trabecular meshwork function as a result of PAS or non-synechial damage.¹⁶ In such cases phacotrabeculectomy may be necessary to achieve the degree of IOP control required to prevent progression of glaucomatous optic neuropathy. This is a similar theory to that used to explain why laser iridotomy appears to be less effective in controlling IOP in advanced PACG.¹⁷⁻¹⁹ It is probably oversimplifying things to extrapolate data from laser studies to the surgical management of PACG and other issues need to be considered. These include the frequency and consequences of IOP spikes following cataract surgery in angle closure patients and whether target pressures aimed for following surgery in POAG patients should be applied to patients with PACG. Studies investigating the effectiveness of surgical interventions for angle closure should be designed with these factors in mind.

A randomised controlled trial is under way in Hong Kong comparing phacemulsification with phacotrabeculectomy for PACG (CC Tham, personal communication). The results of this and other ongoing trials in Asia investigating the effectiveness of early detection and treatment for primary angle closure are needed to help guide clinicians when making decisions on which interventions are likely to be beneficial to the

patient. From a public health perspective PACG has been projected to be one of the commonest causes of irreversible blindness in the populous countries of Asia.²⁰ If we are to attempt to implement prevention of blindness programmes targeted at PACG we need evidence that our interventions are effective in preventing disease progression and visual loss.

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Diabetic blindness

Prevention of diabetic blindness

E Stefánsson

New technologies or "old fashioned" public health?

We ophthalmologists know how to prevent diabetic blindness, but we are not doing it. The scientific principles of treatment of diabetic retinopathy and prevention of blindness have been known for over 20 years. In spite of this, diabetic eye disease remains a major public health problem with large numbers of people with diabetes going blind worldwide from what is largely a preventable cause of blindness.¹⁻⁶ The problem will expand rapidly in the decades to come with the ongoing worldwide epidemic of type 2 diabetes mellitus.⁷ Is it possible that our

efforts in this field are directed too much towards new inventions in diagnostic technologies and treatment and not enough towards old fashioned public health efforts and health care, using the equipment and knowledge we already have?

Specific treatment for diabetic retinopathy was initially limited to pituitary gland destruction. In the 1970s this was replaced with photocoagulation, and the Diabetic Retinopathy Study⁸ confirmed the benefit of xenon arc or argon laser photocoagulation to reduce the risk of visual loss in people with diabetes with

proliferative retinopathy. A few years later the Early Treatment Diabetic Retinopathy Study⁹ confirmed the utility of macular laser photocoagulation to reduce the risk of visual loss in patients with diabetic macular oedema. In both proliferative diabetic retinopathy and diabetic macular oedema the benefit of laser treatment is critically related to the timing of the treatment. The treatment is highly effective when applied in the early stages of proliferative retinopathy or diabetic macular oedema but less effective and more difficult if the disease is more advanced. The use of laser treatment in diabetic eye disease has revolutionised the treatment of diabetic eye disease and probably millions of diabetic patients have been saved from severe vision loss with this treatment.

While this has been extremely beneficial in many individual cases, a different picture emerges if the situation is examined from a public health viewpoint. Diabetic eye disease remains a major cause of blindness in the world, also in some of the richest societies.^{4, 5, 10}

The public health failure is not universal. Systematic screening programmes for diabetic eye disease and preventive treatment have been organised in some regions and the outcome has been documented.¹¹ The longest experience is in Iceland where systematic screening for diabetic eye disease has been in place for 25 years. In 1980 2.4% of Icelandic people with diabetes were legally blind (visual acuity <0.1) and in 2005 this number is 0.5%. This has been achieved with a "low tech" public health approach.¹²⁻¹³ Similar benefit from a public health approach to diabetic eye disease has been seen in a few other places in northern Europe. In each instance the prevalence of diabetic blindness has gone down and incidence studies have shown that the annual incidence of diabetic blindness can be brought down to 1% or less.¹⁴⁻¹⁵ This is in sharp contrast with surveys from areas where a public health approach with systematic screening and preventive treatment has not been in place. For example, in Wisconsin, Klein *et al*¹⁶ have reported 3.6% prevalence of legal blindness among people with diabetes and 4.6% with partial sight, and Jerneld and Algvare¹⁷ reported 7.7% legal blindness and 9.3% partial sight in a Swedish population that was not being screened for diabetic retinopathy in the 1980s.

The pressing need is for a public health approach using present technology rather than the development of new technologies

The standard of treatment and prevention is universally accepted. The World Health Organization and many professional organisations recommend yearly fundus examination of diabetic patients and preventive treatment as indicated by the DRS (Diabetic Retinopathy Study) and ETDRS (Early Treatment Diabetic Retinopathy Study) studies.¹⁸ The fact remains that these standards of treatment are not generally followed. Campaigns organised to improve this situation such as the Diabetes 2000 program of the American Academy of Ophthalmology and the St Vincent Declaration in Europe have helped, but have not been able to solve the public health problem.

If diabetic blindness can be prevented in Nordic communities of a few hundred thousand inhabitants, there should be no reason why it cannot be replicated in larger communities and around the world. Any community willing to invest in diabetic eye screening can expect the number of legally blind diabetic patients to decrease by twofold to threefold within 10 years and decrease the disability expenditures by an amount

that is many times the initial investment.¹⁹⁻²⁰

Ideally, prevention of diabetic blindness would be supported by efforts to prevent diabetic retinopathy through optimal treatment of blood sugar and blood pressure levels and, ultimately, by the prevention of type 2 diabetes with public and education correction of the lifestyle that leads to obesity and diabetes.²¹ This, however, may be outside the scope of ophthalmology. On the other hand, the prevention of diabetic blindness is very much the duty of ophthalmologists and the public health failure in dealing with it puts the world's ophthalmological community to shame.

IS THERE ANY VALUE IN EARLY DETECTION OF DIABETIC RETINOPATHY?

A considerable research effort is being made in diabetic eye disease. New and older drugs are being studied that may help treat diabetic eye disease⁴ and a number of scientists are studying new technologies to detect and diagnose diabetic retinopathy. These techniques include fluorophotometry and fluorescein angiography and electroretinography, and this issue of the *BJO* (p 17) contains an elegant study by El-Bradey *et al* on scanning laser entoptic perimetry for the early detection of visual defects associated with diabetic retinopathy. It is clear that these techniques are able to detect early diabetic retinopathy and even detect changes in the retina before diabetic retinopathy changes are visible by fundus examination. But what is the value of detecting diabetic change in the retina at this early stage?

In the present clinical situation there is no clinical value in the detection of diabetic retinal disease before the occurrence of microaneurysms. The detection of mild non-proliferative retinopathy also has very little clinical value, in that no treatment would be instituted and the patient would receive the same general advice regarding blood glucose and blood pressure control.²¹ It is only the detection of early macular oedema or neovascularisation that would call for specific treatment. This usually takes place rather late in the development of diabetic retinopathy and is easily detectable by biomicroscopy at the slit lamp.

Patients with diabetes are not going blind for lack of technology or treatment options. They are going blind because they are not receiving treatment that has been well established for more than a quarter of a century. The pressing need is for a public health approach using present technology rather than the development of new technologies.

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