

On the lookout: how to save the sight of Australians who have glaucoma

Mark J Walland

Excessive reliance on intraocular pressure to detect glaucoma leaves many affected patients undiagnosed and untreated; visualisation of the optic disc is a dying art that needs to be revived

Intraocular pressure (IOP) is measured by tonometry. In our community, mean IOP is around 15 mmHg, with a normal range (mean \pm 2 SD) regarded as 10–21 mmHg. This information is irrelevant to the diagnosis of glaucoma.

Misconceptions about the importance of IOP in diagnosing and screening for glaucoma continue to result in unnecessary blindness in Australia from glaucomatous optic neuropathy. An aggressive realignment in our thinking is required.

The classic diagnostic triad for glaucoma of raised IOP, arcuate visual field loss and optic nerve head cupping has been inverted. Glaucoma may now be defined as a characteristic, progressive, optic neuropathy showing optic disc cupping as a consequence of neural rim loss. It tends to produce arcuate visual field loss. It is frequently associated with an elevated IOP, but may be diagnosed at any IOP if there is characteristic optic disc and/or visual field damage. This damage indicates susceptibility of that optic nerve to a given IOP.

Early work established a population mean IOP, with an about normal distribution.¹ It is regrettable that this result came to be interpreted as indicating a “normal” (ie, safe) IOP for an individual patient, particularly as that report recognised that glaucoma could occur despite an IOP of less than 21 mmHg. In the Baltimore Eye Survey, more than 50% of patients with newly diagnosed glaucoma had a “normal” IOP on a single tonometry measurement.² The term “normal pressure glaucoma” (NPG) has been coined for such eyes, and they may comprise a third to a half of all open-angle glaucoma cases.^{2,3} Conversely, there is a subset of the population that has IOP elevated above normal, yet does not have glaucoma. This status is labelled ocular hypertension.

Detection of elevated IOP remains important at any time. The prevalence of glaucoma at successive levels of IOP greater than 21 mmHg rises exponentially.⁴ IOP is the single greatest risk factor for glaucoma, and its reduction is the cornerstone of all current treatment. Screening on the basis of IOP alone, however, clearly produces a high rate of potentially disastrous false negatives. Patients continue to undergo “eye-checks” and be reassured that they do not have glaucoma solely on the basis of an IOP measurement. This represents an inadequate standard of care.

Visual field loss is no longer a sine qua non of diagnosis. In recognition that up to 40% of axons may be lost from the optic nerve before visual field loss is evident on standard automated perimetry,⁵ the term “preperimetric glaucoma” connotes this early disease. Rather than being a distinct entity, preperimetric glaucoma really indicates the inadequate sensitivity of this standard psychophysical test in detecting early glaucoma (as compared with its invaluable role in confirming and monitoring established disease). Early detection of disease can thus depend entirely on identifying optic disc cupping, notching or rim loss, and associated retinal nerve fibre layer defects, perhaps in the absence of raised IOP or

visual field loss. Newer and more sensitive tests of specific visual processing such as frequency doubling perimetry or short wavelength automated perimetry may detect glaucoma earlier, and may thus shift the boundary in defining preperimetric glaucoma.⁶

We must also disabuse ourselves of any convenient dichotomy between chronic open-angle glaucoma and acute angle-closure “glaucoma”. (Acute primary angle closure [APAC] causes a painful, unilateral red eye with markedly elevated IOP: patients with APAC may not have optic disc cupping or visual field loss.) It is increasingly evident that a massive and untreated burden of *chronic* angle-closure glaucoma exists, particularly in Chinese and South-East Asian people,⁷ and therefore in our immigrant populations and their families.

Glaucoma, whether open-angle or closed-angle, is an insidious disease — “the sneak thief of sight”. Its prevalence increases exponentially with age; progression is slow, painless and irreversible; visual acuity is affected only late; and symptoms of peripheral vision loss with mobility difficulties do not occur until gross levels of field constriction have developed. For all these reasons, affected patients tend not to present but instead need to be found. Prevention — or at least slowing — of further vision loss is possible with an effective range of medical, laser or surgical therapies directed at lowering IOP to an individualised target level (which may be considerably lower than 21 mmHg).

The limitation to our preventing blindness in Australia is not so much a lack of therapies, but inadequate case detection. Awareness of the newer concepts in glaucoma has been variable among general practitioners, optometrists and even ophthalmologists. Glaucoma has a prevalence of around 3% in the population aged over 50 years, yet 50% of cases of glaucoma in Australia today remain undiagnosed.⁸ The Melbourne Visual Impairment Project provides a further indictment: of the patients whose glaucoma was detected during the survey and who had seen an eye-care practitioner in the preceding 12 months, nearly half (many of whom had established visual field defects) had not had their glaucoma diagnosed.⁹ Highly sensitive screening programs of the general population for glaucoma are, however, not cost-effective, producing low case yields and a high number of false positives.¹⁰ Despite a panoply of technological wizardry, there remains no device that in a single or screening test can identify early glaucoma with appropriate sensitivity and specificity. Local research is progressing with international collaborators to identify genetic loci responsible for glaucoma,¹¹ but a blood test to identify those genetically at risk is not generally available.

What is to be done? Ophthalmologists diagnose and manage glaucoma, but are referral-dependent and thus see a selected sample. The aim that every Australian aged over 40 years have a general eye examination by an eye-care practitioner remains an elusive goal, but such a strategy could detect glaucoma as well as

other ocular abnormalities, particularly if glaucoma sufferers could be prompted to reveal their diagnosis to first-degree relatives. "More funding" is a mantra so tired as to engender indifference, but would certainly enable more targeted, resource-intensive testing.¹² While the benefit of disease detection seems self-evident, legitimate public health arguments could be mounted that, as progression of glaucoma is slow, diagnosis and treatment of early disease (particularly in older patients with preperimetric glaucoma) is not justifiable. We must also be mindful of the burden of the diagnosis and treatment for the patient.¹³ Screening protocols would therefore be directed not to detecting early glaucoma per se, but rather to finding those with a greater lifetime risk of loss of functional vision (ie, advanced disease, younger age or higher IOP).

In the absence of such a structured program, we are dependent on "opportunistic screening". In this issue of the Journal, Zegers and colleagues present two clinical vignettes illustrating the importance of a family history of glaucoma (page 312),¹⁴ which is an excellent starting point in case detection. Glaucoma suspects can also be identified by general practitioners and optometrists prepared to examine the optic disc. For the former, ophthalmoscopy is a dying art that needs to be rejuvenated in training programs so that visualisation of the optic disc can resume its place in the routine medical check-up. Optometrists, on the other hand, are technologically equipped and trained to detect and diagnose eye disease, are frequently primary eye-care practitioners, and are numerous. The recent move by a vanguard of optometrists into therapeutics and comanagement remains politically vexed. Nonetheless, medical practitioners must accept and encourage the major contribution to glaucoma detection that many optometrists make.

Vision loss from glaucoma is most often preventable. Current therapies are effective if the diagnosis is made early enough. Many treatable patients continue to lose visual field because case-detection strategies are haphazard, incomplete, or suffer from a lingering and inappropriate fixation on elevated IOP as the sole diagnostic criterion. The two simple tasks of asking for a family history of glaucoma and assessing the optic disc (with referral onward of patients with positive findings) represent opportunities to diminish the impact of the commonest preventable cause of irreversible blindness in Australia today.

To heighten awareness of glaucoma, the first-ever World Glaucoma Day has been designated for 6 March 2008 (<<http://www.wgday.net/>>). Glaucoma Australia provides patient and com-

munity education and support, and can be contacted at <<http://www.glaucoma.org.au/>>.

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