Primary Open-angle Glaucoma: Insights on Early Detection, Classification, and Management

By Nicolas Fontaine, OD, MSc, and Pierre Forcier, OD, MSc

Glaucoma is the second most common source of worldwide blindness. The progressive neuropathy associated with glaucoma is often underdiagnosed in the early stages. Earlier detection through routine eye examination, particularly in high-risk individuals, and the implementation of sensitive and specific tests would improve the initiation of management at the outset of glaucoma, which could reasonably be expected to reduce glaucoma progression and improve the functional implications for the glaucomatous patient. This issue of Optometry Rounds presents the current state of knowledge on the different types of glaucoma, particularly on primary open-angle glaucoma (POAG), as well as the latest diagnostic and management techniques.

Glaucoma is a chronic, progressive neuropathy of the optic nerve. It is currently theorized that the optic disc is the site of injury.1 From an anatomical point of view, the injury is characterized by destruction of the retinal ganglion cells and their axons. Physiologically, this results in a partial loss of transfer of the nerve impulses from the retina to the visual cortex. There are multiple clinical sequelae. The most typical impairment involves a loss of sensitivity in the visual field, which is the result of scotomas (relative or absolute) whose magnitudes vary with the stage of disease progression. Some patients also suffer the loss of contrast sensitivity, even decreased visual acuity, depending on the location of the injured ganglion nerve fibres. In advanced stages of glaucoma, pupillary reflex changes are also observed.

The functional consequences of uncontrolled glaucoma can be severe for the patient; eg, loss of driver’s license, restrictions in many sports and leisure activities, reduced mobility, decreased independence for numerous daily activities, and, ultimately, blindness.

The optometrist is optimally positioned to detect glaucoma in its earliest stages, to use the most effective investigative techniques to confirm the diagnosis, and to initiate prompt vision-preserving management. At the time of writing of this article, legislation on the role of optometrists varies across provinces, from independent diagnosis and management of primary open-angle glaucoma (POAG; Ontario and British Columbia) to co-management with ophthalmologists (Alberta, Quebec), treatment of emergency angle closure (New Brunswick), and early detection/diagnosis and referral (remaining provinces and territories). This article is intended to present current and future-looking methods of classification, diagnosis, and management.

Classification

Glaucoma is divided into several subcategories, based primarily on current understanding of anatomy and pathogenesis. This classification is primarily aimed at determining the etiology and the physiological mechanisms that lead to this neuropathy. Its clinical usefulness is to guide the clinician in selecting the most appropriate therapy.

Current understanding of the causative factors at the origin of the apoptosis of the nerve fibres that circulate through the optic nerve head (ONH) lead to 2 main hypotheses.7 One relates to a possible hypoperfusion of the ONH, which in turn could be the result of several systemic conditions, including (but not limited to) blood vessel autoregulation anomalies, vasospasm, cardiovascular disease, nocturnal vascular hypotension, reduced cerebral perfusion, migraines, and Raynaud disease. ONH perfusion is also the result of the balance between blood pressure (BP) to the ONH (short posterior ciliary arteries and circle of Zinn) and intraocular pressure (IOP); there has been renewed interest in the measurement and interpretation of ocular perfusion pressure, which is the difference between BP and IOP. Alternatively, it has been proposed that the fibre apoptosis could be the result of the posterior ectasia of the
lamina cribrosa. The distortion of the pores of the lamina “strangles” the nerve fibres. This ectasia is the result of either a weak lamina cribrosa or elevated IOP. In both instances, axoplasmic flux is interrupted, causing progressive cellular death.

The actual mechanism leading to glaucomatous optic atrophy may be a combination of both theories. Nevertheless, IOP plays an important role in both theories. Elevated IOP is rarely caused by overproduction of aqueous humor, but rather is almost always the result of impaired outflow. It is for this reason that glaucomas are primarily classified according to the underlying cause of IOP elevation.

**Primary or secondary**

The first distinction is made with respect to the primary (idiopathic) or secondary nature of the neuropathy. Glaucoma is deemed to be primary in the absence of an identifiable cause. Otherwise, it is identified as secondary to a clinically identifiable causal agent.

**Open or closed iridocorneal angle**

Glaucoma is also classified based on the configuration of the first anatomical portion of the aqueous humour outflow pathway: the iridocorneal angle. This structure delineates the most peripheral portion from the anterior chamber, where the iris and cornea meet at the anterior part of the ciliary body and trabecular meshwork. The angle is deemed open on gonioscopic examination if there is no restriction of the flow of aqueous humor; otherwise, the glaucoma is called “closed angle,” further to apposition of the trabecular meshwork and iris root. Gonioscopy or optical coherence tomography (OCT) is used to determine the nature of the angle. Open- and closed-angle glaucoma can both be primary or secondary.

The main focus of this article will be primary open-angle glaucoma (POAG), the most common form in Western nations, in which aqueous outflow is reduced despite the lack of angle obstruction.

**Acute or chronic glaucoma**

Glaucoma is considered acute when its onset is sudden. It is characterized by IOP elevation as high as 70 mm Hg in some cases. If not treated quickly, damage to the optic nerve will occur rapidly. Acute glaucoma is usually seen in closed-angle glaucoma or as a consequence of a blunt or a severe ocular trauma, but can present in other forms, including inflammatory glaucoma and glaucomatocyclitic crisis (Posner Schlossman syndrome). Chronic glaucoma is of a more insidious nature, since it will develop very slowly, remaining mostly asymptomatic, and could span many years if not treated. Damage is of a more progressive nature.

**Congenital and developmental glaucoma**

A minority of glaucomas are defined as congenital (occurring before the age of 3–5 years) or developmental (appearing between 5–16 years of age). Discussion of these forms of glaucoma is beyond the scope of this article.

**Ocular hypertension**

Ocular hypertension is a condition where IOP is consistently ≥21 mm Hg; ie, the upper limit of the 95% confidence interval of normal IOPs in a Western population. A diagnosis of glaucoma cannot be made if elevated IOP is not accompanied by clinically objective deterioration of the optic disc and related visual field; such patients are classified as POAG suspects in the Canadian Ophthalmological Society (COS) guidelines. It is interesting to note that, as demonstrated by the Ocular Hypertension Treatment Study (OHTS), fewer than 10% of subjects presenting with ocular hypertension only (ie, without any other ocular or systemic abnormality) developed POAG over a period of 5 years. In contrast, approximately 61% of POAG cases present with a mean pressure <21 mm Hg. By itself, IOP is a poor screening option, because of low sensitivity/specificity which give it a poor predictive value. However, elevated IOP has been found to be an important predictor of POAG development, as shown in the OHTS and the Barbados Eye Studies, as well as a useful marker of disease progression. The Baltimore Eye Survey pointed to strong evidence of a causal relationship between IOP and POAG. These findings underline that glaucoma is a multifactorial entity and that IOP is only one factor in its development; however, IOP is the only risk factor that can currently be modified with medical treatment.

**Epidemiology**

After cataracts, glaucoma is the second most common source of worldwide blindness, affecting 60 million people in 2006. Projections indicate that this number will reach 80 million in 2020. POAG is the cause of 12% of blindness cases in the world. The prevalence of glaucoma varies greatly from one study to another, but it can be estimated that 45%–55% of glaucomas in North America are POAG, about 15% are primary closed-angle glaucoma (PCAG), 30% are secondary glaucomas, and 5% are congenital glaucomas. The National Coalition for Vision Health stated, in 2011, that at least 300 000 Canadians were affected with glaucoma.

In industrialized countries, approximately 50% of the individuals with glaucoma are unaware they have this condition, do not seek medical assessment, and do not receive appropriate care. However, the cost/benefit ratio does not justify widespread screening for the disease as a public health policy. Rather, the performance of opportunistic screening by a primary-care eye specialist is considered to be the most efficacious way to address this issue.

**Risk Factors**

The most important risk factors of POAG include older age, black and Hispanic race, elevated IOP, family history of glaucoma, myopia, and low diastolic perfusion pressure (Table 1). Age >55 years was shown by Tuck et al to be associated with a significant increase in the prevalence of POAG, reaching a maximum rate (4.3%) in individuals >80 years. The Blue Mountain Eye Study found nearly double the rate of POAG (8.2%) in this same >80 year age group. Black individuals are at a 4–5 times higher risk than Caucasians, and the relative risk for Hispanics lies between these 2 populations. The prevalence rates determined by Rudnicka et al were 4.2% for blacks, 2.1% for whites (including Hispanics), and 1.4% for...
Table 1: Risk factors and signs for presence of open-angle glaucoma with level 1 evidence

<table>
<thead>
<tr>
<th>Ocular risk factors and signs</th>
<th>Non-ocular risk factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• IOP</td>
<td>• Increasing age</td>
</tr>
<tr>
<td>- Elevated baseline IOP</td>
<td>• African descent</td>
</tr>
<tr>
<td>• Optic disc</td>
<td>• Hispanic ancestry</td>
</tr>
<tr>
<td>- Deviation from the ISNT rule^</td>
<td>• Family history</td>
</tr>
<tr>
<td>- Increased optic disc diameter</td>
<td>• Genetics</td>
</tr>
<tr>
<td>- Parapapillary atrophy</td>
<td>- Myocillin</td>
</tr>
<tr>
<td>- Disc hemorrhage</td>
<td>- Optineurin</td>
</tr>
<tr>
<td>• PXF</td>
<td>- Apolipoprotein</td>
</tr>
<tr>
<td>• Thinner CCT</td>
<td>• Migraine</td>
</tr>
<tr>
<td>• Pigment dispersion</td>
<td>• Corticosteroids</td>
</tr>
<tr>
<td>• Myopia</td>
<td></td>
</tr>
<tr>
<td>• Decreased ocular perfusion pressure</td>
<td></td>
</tr>
</tbody>
</table>


Asians. They also found that whites had the highest odds ratio per decade increase in POAG (2.1) compared with blacks and Asians (1.6 each). A positive family history is associated with a 3.7-fold higher risk. Other risk factors include vertical enlargement of optic nerve cupping, a thin cornea (correlated with a thin lamina cribrosa), and reduced blood flow to the optic nerve, arterial hypertension, diabetes, migraines, and significant myopia (increased axial length).

Clinical Examination

The diagnosis of glaucoma is based on the atrophic anatomical changes of the optic nerve, more specifically at the exit of the eyeball. The following, in detail, are the investigations and tests available to optometrists in order to help make a proper diagnosis.

Examination of the ONH and the ganglion nerve fibres

Several clinical tools are available to assess the integrity of the optic disc. Slit lamp examination combined with a fundus positive lens (60, 78, 90D) allows for maximum stereoscopic observation of the disc when the iris is dilated. This examination is essential to observe subtle changes in the ONH. These changes can manifest in many ways: localized or diffuse optic cup expansion, peripapillary atrophy or changes in blood vessels.

The cup-to-disc ratio is expressed as the fraction of the size of the optic disc relative to the total diameter of the optic nerve head. This diameter is an important factor when assessing the disc cupping since a large optic nerve will produce a greater physiological cupping compared to a smaller optic nerve. The cup-to-disc ratio is stable over time. A progressive loss of optic nerve axons will produce a localized or generalized enlargement of the cup-to-disc ratio. This enlargement, when present, is almost pathognomonic of glaucoma, as the alternative causes are rare conditions such as temporal arteritis, certain orbital tumours, and carotid artery disorders. Since the cupping is usually bilateral and symmetric, a difference >0.2 between the eyes combined with similar optic nerve diameters is considered suspicious for glaucoma. The expansion of the disc occurs more rapidly at the surface compared to the depth. When the depth of cupping increases, one can observe the "laminar dot sign" which results from the exposure of the cribiform plate fenestrations.

The integrity of the neuroretinal ring is evaluated by observing the colour, the thickness of the fibre layer, and by the "ISNT" rule. This rule of thumb states that the widest portion of the neuro-retinal ring is in the inferior portion (I), followed by the superior section (S), the nasal side (N) and finally the temporal side (T). Noncompliance with the "ISNT" rule and/or a whist desaturation of the neuroretinal rim colour is characteristic of glaucomatous optic atrophy.

Drance hemorrhage is one of the vascular changes observed in glaucoma. These hemorrhages are discrete, solitary, and are located directly along the border of the optic nerve and follow the regular architecture of the ganglion nerve fibres. They are linear and can be confused with a blood vessel. A bayonet vessel sign is visible when the path of the blood vessel passes under the edge of the cup and is then displaced at its origin at the cup margin. Finally, one can observe the exclusion of a circumlinear vessel. The latter is a small arteriole or venule that is localized at the inner border of the neuroretinal ring before it heads towards the macula. It is found in 50% of normal discs (Figure 1).

Finally, peripapillary atrophy can be observed in 2 zones. The β zone corresponds to the temporal crescent often seen in myopia (sclera and choroidal vessels) and the α zone, peripheral to the β area, describes a combination of hyper-
hypopigmentation. These zones are larger in patients with glaucoma compared to a normal population.\textsuperscript{31,33}

**Gonioscopy**

Gonioscopy is an essential test in the diagnosis and treatment of all types of glaucoma. Its primary function is to assess the iridocorneal angle (open or closed).\textsuperscript{8,34,35} Gonioscopy is also used to rule out neovascular glaucoma, the presence of pseudoexfoliation, and all other anatomical changes in the iridocorneal angle that could lead to glaucoma.\textsuperscript{7} Gonioscopy should also be performed on follow-up evaluations of patients with established glaucoma. Dr. Wallace Alward has developed an excellent instructional website for gonioscopy (http://www.gonioscopy.org).

**Tonometry**

As IOP reduction remains the only therapeutic option to minimize glaucoma-related optic nerve damage, tonometry is mandatory for patient follow-up and in the establishment of the target pressure for therapy. A wide variety of tonometers are available; however, Goldmann applanation tonometry, developed in 1957, remains the standard for measurement of IOP. Because IOP undergoes significant diurnal variation, it is essential to take several measurements at different times to confirm elevated IOP.

**Pachymetry**

Central corneal thickness (CCT) has been shown to be an important factor in the diagnosis of glaucoma.\textsuperscript{31,36} and can also have a profound influence on accuracy of IOP measurement. In general, the thinner the cornea, the greater the underestimation of IOP. It is recommended to measure CCT in individuals with suspected or confirmed glaucoma, with or without ocular hypertension.\textsuperscript{8,34,35} CCT is measured by pachymetry; ultrasonic instrumentation is recommended over optical, and is accurate, widely available, low cost, transportable, and can be used with opaque cornneas.\textsuperscript{8} There is no precise formula accepted by the scientific community that correlates IOP and CCT; however, industry-developed conversion tables indicate that a 1-mm Hg change can be roughly estimated per 15-µm change in thickness. Be aware of factors that may alter CCT, including measurement within 2 hours of the patient awakening, dry eyes, Fuchs endothelial dystrophy, extended contact lens wear, and use of prostaglandin analogues.\textsuperscript{36}

**Corneal rigidity**

Relative corneal rigidity may be assessed with an ocular response analyzer. This machine "corrects" measurement of IOP relative to the strength/rigidity and thickness of the corneal tissue. Some studies have shown that a weaker cornea is linked with a weaker lamina cribrosa, putting the patient at even greater risk when IOP becomes elevated.\textsuperscript{39}

**Visual field testing**

Several automated instruments measuring the visual field are currently available. Those that measure the central 30° limits (white-on-white) are considered standard practice in patients with confirmed or suspected glaucoma. The automated measurement of the visual field makes it possible to detect functional loss and to establish a point of reference to assess the evolution of the disease over time. In order to obtain reliable values and to take into account the learning curve, it is recommended to repeat the first measurements. However, up to 50% of the ONH axons may be damaged before standard automated perimetry detects visual loss.\textsuperscript{38}

Most systems are programmed with integrated statistical analysis software to highlight the evolution of the changes in the visual field over time.

Another means of detection of field loss resulting from glaucoma is short-wavelength automated perimetry (SWAP), which makes use of a blue target on a yellow-lit background.\textsuperscript{19} This technology is more sensitive than regular perimetry, but is sometimes difficult to perform in older people presenting with a change in the lens index of refraction.

A second type of visual field that is often used clinically, which is fast and inexpensive, is frequency-doubling technology (FDT) perimetry. Although highly sensitive and very specific for the detection of glaucoma compared to standard automated perimetry, there are shortcomings in this system for follow-up of visual-field narrowing with time.\textsuperscript{40-46}

**Medical imaging: analyzers of ONH and retinal nerve fibre layer**

**OCT**

OCT is the most common technique used clinically for the analysis of optic neuropathies that are glaucomatous in nature. The use of an optical coherence interferometer allows one to achieve in vivo retinal sections of near-histological resolution (<10 µm) despite a nondilated pupil and slightly opaque ocular media.\textsuperscript{17} A normative database taking into account age and ethnicity allows for the comparison of the patient’s optic nerve parameters with those of a defined population. Integrated statistical software can show, using graphs, the evolution of the condition over time.

Anterior segment OCTs can also help the practitioner determine the structure of the iridocorneal angle. Not only does this tool provide a sharp view of the angle but it helps to quantify the angle and its evolution over time. Scheimpflug technology is also a useful tool that provides an automatic calculation of the anterior chamber angle, volume, and depth. Anterior segment OCTs and Scheimpflug technology also allow for the evaluation of pachymetry but over the entire corneal surface. Compared to ultrasonic pachymetry this mapping could be beneficial in cases of highly irregular corneas.

**Scanning laser polarimetry (GdxVCC)**

Scanning laser polarimetry is another imaging technique that objectively measures the thickness of the peripapillary ganglion nerve fibres and compares them with a population of healthy subjects. This technique uses the phenomenon of birefringence of the ganglion fibre axons to measure the phase delay of the polarized light. The delay is directly proportional to the thickness of the ganglion fibre. Scanning laser polarimetry is reliable and reproducible. It analyses 17 parameters and compares them with a database. A probability score (nerve fibre indicator) is then calculated to facilitate the analysis of the parameters.
Suspected glaucoma
Early glaucoma
Moderate glaucoma
Advanced glaucoma

Several sequential parallel images are captured through a nondilated pupil at different depths, beginning above the surface of the retina. Once the images are aligned, the software creates a 3-dimensional image of the optic nerve. The diagnosis and the progression of glaucoma are facilitated with user-friendly software. The print-out results are easy to read and can be compared statistically from visit to visit in order to detect subtle changes that could develop in the optic nerve.

Optic disc photography

This inexpensive technique (stereoscopic or not) remains a simple method of documenting the optic disc in order to identify a potential evolution of the disease over time. The enlargement of the disc excavation, which is a manifestation of progressive ganglion fibre atrophy, will indeed be more easily detected by comparing images of the papilla over time. This technique provides good visualization of the ONH and facilitates diagnosis of Drance hemorrhage. Moreover, image acquisition software now allows for easy comparison of photographs. However, the majority of studies to date have found only poor to moderate expert agreement in assessing progressive changes with optic disc photography and only a few showing good to excellent agreement.

Management

Treatment strategy

The first step in treatment is identification of the stage of the disease. The Canadian Glaucoma Strategy Forum defined 4 clinical stages of POAG (Table 2). These stages indicate an increasing risk level of functional vision loss. Consequently, more aggressive treatment is required for more advanced stages of the disease. Target goals must also take into account the life expectancy of the patient, risk factors, diurnal variations in IOP, and the attitudes of patient with respect to treatment (compliance).

Pharmacological treatment

As stated previously, the only risk factor of POAG in which medical intervention can have an effect is IOP. In the OHTS, pharmacological reduction of IOP cut the risk of progression from simple ocular hypertension to POAG by more than half (4.4% versus 9.5% in the observation group; P<0.0001). The COS guidelines recommend the establishment of an upper limit of initial target IOP for each eye, and re-evaluation on follow-up visits based on structural/function changes in the optic nerve. Several classes of drugs can control IOP.

Prostaglandin analogues

Prostaglandin analogue drops are currently prescribed as the first choice for treatment of glaucoma. They have been shown to be more effective in lowering IOP than beta-blockers and with fewer systemic adverse effects. Available prostaglandin analogues include latanoprost (0.005%), travoprost (0.004%), and bimatoprost (0.03%); dosage is 1 drop qhs. The mechanism of action is through increased uveoscleral drainage. The average IOP reduction is 28%–33%. Ocular side effects are allergy, change in iris pigmentation, hypertrichosis, and periorbitopathy (peri orbital fat atrophy is rarer).

Beta-blockers

Beta-blockers reduce IOP through a reduction of aqueous humour production. Selective (betaxolol) or nonselective agents (timolol, levobunolol) can be used, all of which are available in concentrations of 0.25% or 0.5%. The mean IOP reduction is 20%–30%. Beta-blockers are contraindicated in patients suffering from bronchorespiratory problems, but also in some patients with cardiovascular disease and in hypoglycemic patients.

Cholinergic agonists

The principal cholinergic agonist is pilocarpine, although carbachol is another option. The mechanism of action is an increase in the aqueous humour discharge through the trabecular pathway. Pilocarpine is usually prescribed in a concentration of 2% or 4% with qid use; carbachol 1.5% or 3% is administered tid. The efficiency of pilocarpine is high with an average 15%–25% reduction in IOP. There are many disadvantages, including the high frequency of use, frontal

### Table 2: The 4 clinical stages of primary open-angle glaucoma

<table>
<thead>
<tr>
<th>Stage</th>
<th>Symptoms and signs</th>
<th>Treatment objectives with decision to treat</th>
</tr>
</thead>
</table>
| 1 – Suspected glaucoma | IOP ≥ 22 mm Hg  
Asymmetry of vertical C/D ratio > 0.2 between the 2 eyes  
Suspect appearance of the optic disc  
Suspected central-field defect | ≥20% reduction of IOP  
Final IOP <25 mm Hg |
| 2 – Early glaucoma    | Slight glaucomatous changes in the cup; C/D ratio ≥0.65 for an optic nerve of average diameter  
Slight visual-field defect outside the central 10° | ≥20% reduction of IOP  
Final IOP <21 mm Hg |
| 3 – Moderate glaucoma | Moderate glaucomatous changes in the cup; C/D ratio 0.7–0.85  
Moderate visual-field defect outside the central 10° | ≥30% reduction of IOP  
Final IOP <18 mm Hg |
| 4 – Advanced glaucoma | Significant glaucomatous changes in the cup; C/D ratio ≥0.9  
Visual-field defect within the central 10° | ≥ 30% reduction of IOP  
Final IOP <15 mm Hg |

IOP = intraocular pressure; C/D = cup/disc
headache, myosis that affects visual acuity, and accommodative spasm which can reach 2.00 D.

**Alpha adrenergic agonists**

The mechanism of action of alpha adrenergic agonists (apraclonidine 0.5% and 1.0%, and brimonidine 0.15% and 0.2%) is 2-fold: an increase in aqueous humour discharge through the uveoscleral pathway and a decrease in the production of aqueous humour. These agents are prescribed tid as monotherapy, or bid as adjunctive therapy. The average IOP reduction is 20%–30%. Allergies are common, but the systemic adverse events are minor (except in patients who are allergic to sulphonamides).

**Carbonic anhydrase inhibitors**

The topical carbonic anhydrase inhibitors available in Canada are dorzolamide 2% and brinzolamide 1%. Both are prescribed as a bid dose. The mechanism of action is a decrease in the production of aqueous humour. The average IOP reduction is 15%–22%. Patients report a bitter taste when the drops flow from the nose to the throat. They are not recommended for patients who are allergic to sulphonamides.

Pharmacological treatment is usually initiated as monotherapy. The current COS guidelines recommend a unicocular trial, with the other eye serving as a control, and to see the patient again for other pressure measurements at the same time of day to eliminate the effect of diurnal changes in IOP. The patient is seen again 3 weeks after initiation of therapy when prostaglandin analogues are prescribed, or 2 weeks later for other classes of drugs. If the goal of reducing IOP is not achieved, one can consider a dual or combination therapy (2 medicines in the same bottle) for practicality. If the above is not enough to stabilize ganglion fibre loss, triple therapy or surgical intervention can be considered.

**Surgical treatment**

Surgical intervention is usually reserved for glaucoma that progresses despite pharmacological treatment or for patients who do not fully adhere to treatment. Given that these procedures are beyond the scope of optometric practice, the following is a brief overview.

Trabeculoplasty consists of applying a series of approximately 50 laser burns on the trabeculum on a 180° surface. The other 180° can be treated thereafter if the first treatment is insufficient. It is performed with an argon laser or a 532 nm Q-switched neodymium-doped yttrium-aluminum garnet (Nd:YAG) laser (selective trabeculoplasty).

Filtration surgery (trabeculectomy) is performed by creating a fistula between the anterior chamber and subconjunctival space so that part of the aqueous humour can be evacuated, after having been filtered through a subconjunctival bubble (bleb) located in the superior portion of the limbus. This surgery is most effective for recalcitrant glaucoma. However, it poses risks of serious complications such as ocular hypotonia, choroidal detachments, or endophthalmitis.

Alternatively, a number of implants are available for insertion at different locations in the anterior chamber or posterior chamber to create a new route for the discharge of aqueous humour.

Canaloplasty is a recent procedure that allows for the catheter-guided insertion of a polypropylene suture along the Schlemm canal. This wire is then stretched to dilate the canal, improving flow of aqueous humour.

Endoscopic cyclophotocoagulation consists of selective ablation of ciliary processes, depending on the target reduction of IOP. It is usually performed using an 810-nm pulsed laser diode and fibre-optic camera to visualize the structures to ablate.

In trabeculodialysis, a corner of the trabecular meshwork on a 60°–120° surface is raised by means of a needle or a trabectome to allow for a better flow of aqueous humour through the usual physiological pathway.

Finally, iridotom y is performed by making a hole in the periphery of the iris with a YAG laser. This allows for the rebalancing of aqueous humour pressures between the anterior and posterior chambers. As a result, the iris becomes flatter, which releases the iridocorneal angle in cases where the angles are narrow, or which reduces posterior surface friction on the zonules of Zinn in cases of pigment dispersion syndrome.

**Patient Follow-up**

Follow-up of glaucoma patients should be scheduled at intervals ranging from 2–12 months, depending on the following factors:

- Baseline severity of the injury
- Risk factors
- Rate of progression
- Patient compliance

The COS guidelines recommend several initial visual field tests at regular intervals in the first 2 years to establish an accurate baseline. Chauhan et al estimated that detection of a mean deviation of -1.0 dB would require 7 visits over 2 years, and 5 visits in 2 years to detect a -2.0 dB change.

The specific tests administered at follow-up visits are generally as follows:

- IOP measurement
- Visual field studies
- ONH imaging and 3-dimensional observation (ideally with photodocumentation)
- Gonioscopy
- CCT measurement (following any event that may have altered it)

**Conclusion**

Glauc oma is an insidious disease: it presents with very few signs and symptoms in its early stages. It develops predominantly in older patients. The visual deficits associated with glaucoma can result in a significant loss of autonomy for affected individuals, including loss of driver’s license, reduction in mobility, and restriction in leisure activities. The optometrist plays a vital role in the early detection, evaluation, management (where provincial legislation permits), and follow-up assessment. Glaucoma is usually detected during a routine eye examination. Since the risk of developing glaucoma increases with age, all individuals >50 years of age should have their ocular health checked on a regular basis.
The diagnosis and follow-up of POAG are stimulating challenges for the practitioner. In addition, by playing their role to the fullest, the optometrist improves the quality of life of his/her patients and helps reduce the cost of health services related to the care of individuals whose vision would otherwise be at serious risk.

MC, a 63-year-old man, presents for a routine eye examination. During the consultation, a positive history of glaucoma in the family is noted (father and sister) as well as a subjective decrease in vision in his left eye. The rest of the case history is unremarkable. Visual acuity is OD 6/6 and OS 6/6. Pupillary reflexes show a slightly afferent pupillary defect in the left eye. The intraocular pressure measured with the Goldmann tonometer and compensated for pachymetry is OD 20 mm Hg and OS 21 mm Hg (pachymetry of 505 and 498 µm, respectively). Gonioscopy reveals a wide-open iridocorneal angle without obstruction of trabecular drainage. An enlarged vertical excavation of the optic disc is found in the left eye (0.6 x 0.4), thinning of the superior neuroretinal ring and the presence of sectorial optic atrophy. The optic nerve of the right eye reveals excavation of the optic disc of 0.4 x 0.4 with a healthy neuroretinal ring (Figure 2).

A 24-2 automated visual field is performed on 2 occasions over a period of 1 week. A significant arcuate defect is visible on the printout of the left eye, while the right eye reveals a visual field with a potential early inferior arcuate scotoma (Figure 3).

The patient is referred for high-resolution optical coherence tomography (OCT) to confirm the presence of glaucoma. OCT clearly shows a significant decrease in ganglion fibres of the left eye in the superior region (Figure 4).

Following collection of these results, a diagnosis of POAG is confirmed and the patient is referred to an ophthalmologist for treatment. He is started on travoprost 0.004% and follow-up appointments, alternating between the ophthalmologist and our clinic, are made for every 6 months.

Dr. Fontaine is an Assistant Professor and Dr. Forcier is an Associate Professor at the School of Optometry, University of Montreal.

*Quebec optometrists are not authorized to initiate glaucoma treatment but can modify the management with initial prescriber’s authorization.
References:


The authors stated that they have no disclosures to report in association with the contents of this issue.

A Partnership for Excellence in Continuing Optometry Education

Optometry Rounds is made possible through independent sponsorships from Alcon Canada and Novartis Pharmaceuticals Canada Inc.

© 2013 School of Optometry & Vision Science, University of Waterloo, and School of Optometry, University of Montreal, which are solely responsible for the contents. The opinions expressed in this publication do not necessarily reflect those of the publisher or sponsor, but rather are those of the authoring institution based on the available scientific literature. Publisher: SNELL Medical Communication Inc. in cooperation with the School of Optometry & Vision Science, University of Waterloo, and School of Optometry, University of Montreal. All rights reserved. The administration of any therapies discussed or referred to in Optometry Rounds should always be consistent with the approved prescribing information Canada.