

NELSON'S EYE

Bulletin 4 February 2004



Introduction

The Nelson Hospital Eye Clinic service is being realigned to match staffing change. Our Specialist Ophthalmic Nurse has left after giving 13 years of enthusiastic, resilient and compassionate assistance to a growing population of patients. Patients, General Practitioners, Optometrists and Hospital Staff will notice the loss of many favourable aspects of Eye Clinic service.

Questionable HR department actions mean that a replacement nurse has yet to be appointed and, anyhow, a new staff member will take considerable time to learn the ropes. Surgery & Outpatient numbers are being scaled back and we request a modicum of understanding from those we attempt to support.

Over the years we have introduced schemes to improve and streamline aspects of our service: diabetic retinopathy photo-screening, triage of paediatric referrals (Orthoptic examination & photo-screening) and day-stay, small incision cataract surgery. Other schemes – such as triage of glaucoma referrals – were abandoned when they proved to be inefficient.

As Glaucoma patient follow-up occupies a large part of our clinic workload we are seeking ways of sharing care of patients whose condition is controlled and needing less frequent Specialist review. Most Glaucoma patients visit their GP regularly and their optometrist every few years so we hope to involve these colleagues in shared follow-up and management of medications.

Thus, this newsletter reviews Glaucoma management.

Glaucoma

Glaucoma is a potentially blinding disease resulting from optic nerve damage. It is typically asymptomatic and may be associated with increasing age, family history, ethnicity, other eye disorders and eye trauma. Detection and monitoring of therapy depends on the triad of optic nerve head assessment, intra-ocular pressure measurement and visual field plotting. Optic disc enlargement (cupping) is the most sensitive sign. Intraocular pressure is usually raised but up to a third of patients with glaucomatous disc and field damage have eye pressures in the “normal range” (Low/Normal Pressure Glaucoma). Therapy aims to lower the intraocular pressure sufficiently to halt progression of glaucomatous damage (target pressure). There is no cure but most patients will achieve control of glaucoma and preserve their vision.

Medical management comprises topical or systemic medication, laser treatment and surgery (drainage operations). Drugs and laser therapy have been shown to be equally effective but most glaucoma is controlled with topical medication. If a particular drug proves inadequate alternative agents or combinations of agents are used. Laser therapy and surgery are considered if maximum tolerated medical therapy cannot control glaucoma

Optic nerve head blood supply is critical to the survival of retinal nerve fibres so vascular hypotension, carotid stenosis and blood hyperviscosity must be kept in mind and, in some patients, are as important as intraocular pressure.

Glaucoma Medications

- **β blockers**

non-selective: Timolol (*Timoptol, ApoTimolol*), Levobunolol (*Betagan*)

β_1 -selective: Betaxolol (*Betoptic & Betoptic-S*)

Action: reduces aqueous production by ciliary body

Side-effects: as for systemic beta-blockade re heart, lungs, CNS, impotence, diabetes & lipids; reduced corneal sensation, dry eye, lid dermatitis, blurring, reduced accommodation

- **α_2 agonists**

Brimonidine (*Alphagan*), Apraclonidine (*Iopidine*)

Action: reduces aqueous production, ? increased uveo-scleral outflow

Side-effects: high allergy rate, lid retraction, conjunctival blanching; dry mouth, fatigue & drowsiness & hypotension

- **carbonic anhydrase inhibitors** (sulfa derivative)

Dorzolamide (*Trusopt*), Acetazolamide (*Diamox* tablet)

Action: decreases aqueous production

Side-effects: metallic taste, GI upset, paraesthesia (fingers & toes), respiratory distress & potassium loss (metabolic acidosis), kidney stones, aplastic anaemia, teratogenic

- **miotics** (cholinergic agents)

Pilocarpine

Action: increases aqueous outflow through trabecular meshwork

Side-effects: red eye, small pupil, accommodative spasm giving eye pain & blurring, uveitis, retinal detachment; salivation, tearing, urinary frequency, diarrhoea, sweating

- **combinations**

Timolol & Dorzolamide (*Cosopt*), Timolol & Pilocarpine (*TimPilo*).

- **prostaglandin analogues**

Latanoprost (*Xalatan*) (special subsidy), Travaprost (*Travatan*) (by sample supply)

Action: increase aqueous outflow via uveo-scleral pathway

Side-effects: red eye, eyelash growth, darkening of iris colour, ocular inflammation, macular oedema, herpes keratitis

Patients should enhance ocular absorption & reduce systemic absorption by applying punctal occlusion through closed lids for two minutes after administering the eye-drop.

All bulletins are available at www.eyenz.com - the eye department website. This also has other eye information and patient education files. Please e-mail us at eyeclinic@nmhs.govt.nz with any comments or requests.