Department of Ophthalmology

Ophthalmology and Eye Disease
Overview of 4th and 5th year course

School of Medicine
Faculty of Medical and Health Sciences
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## 4th Year Teaching 2016
### Lecture Timetable

**Course Co-ordinators: Dipika Patel & Charles McGhee**

**Venue: 505-011**

2 sessions: 4 hours in total

### 1. Thursday 30th June 8.30 – 10.30am

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<tr>
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<td>8.30 – 8.35am</td>
<td>Introduction</td>
<td>Professor Charles McGhee</td>
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<td>8.35 – 9.00am</td>
<td>Applied Clinical Anatomy &amp; Physiology</td>
<td>Dr Bia Kim</td>
</tr>
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<td>9.00 – 9.25am</td>
<td>Ocular Pharmacology</td>
<td>Dr Ilva Rupenthal</td>
</tr>
<tr>
<td>9.35 – 10.00am</td>
<td>Symptoms of Eye Disease</td>
<td>Dr James McKelvie</td>
</tr>
<tr>
<td>10.00 – 10.25am</td>
<td>Signs of Eye Disease</td>
<td>Assoc. Prof Dipika Patel</td>
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### 2. Thursday 30th June 11.00 – 1.00pm

<table>
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<tr>
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<th>Session</th>
<th>Speaker</th>
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<tbody>
<tr>
<td>11.00 – 11.25am</td>
<td>Acute Red Eye</td>
<td>Assoc. Prof Jennifer Craig</td>
</tr>
<tr>
<td>11.25 – 11.50am</td>
<td>Ocular Trauma</td>
<td>Professor Charles McGhee</td>
</tr>
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<td>12.00 – 12.25pm</td>
<td>Diabetic Eye Disease</td>
<td>Dr Stuti Misra</td>
</tr>
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<td>Vision Loss</td>
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Outline
The ophthalmology course comprises one day of lectures in 4th year and 7 days of clinical exposure / tutorials in 5th year, which should provide the student with an overview of ocular disease and its manifestations in relation to basic science and other clinical subjects, in addition to establishing core skills. Material taught in fourth year will be built upon in fifth year; acquisition of the relevant knowledge will be assumed and not repeated in fifth year.

The fourth year course will be examined at the end of the semester.

The core objectives are

1. Review and reinforce pre-clinical knowledge applicable to eye disease
2. Appreciation of key ocular symptoms and signs and underlying pathogenesis
3. Key clinical problems encountered in as an RMO/General Practitioner.
4. Appreciation of the ocular manifestations of systemic disease e.g. diabetes mellitus
5. Knowledge of the important causes of blindness in New Zealand and worldwide
6. Establish relevance of key elements of pathology, microbiology and immunology
7. An appreciation of the impact of visual disability on the patient's lifestyle, family and community
8. Basic ocular therapeutics
9. To establish core clinical skills in the assessment of patients with disorders of the visual system.

Core clinical skills to be acquired:

A. Taking an appropriate ophthalmic history
B. Assessment of visual acuity (near, distance, colour)
C. Assessment of visual field to confrontation / central field by Amsler grid
D. Examination of the anterior segment - conjunctivae, cornea, iris and pupil
E. Pupillary examination - near, light, consensual, swinging light test
F. Examination of the red reflex by ophthalmoscope
G. Examination of the posterior segment using the ophthalmoscope
H. Assess presence of squint, corneal reflexes, cover test and eye movements
I. Basic elements of slit-lamp examination

Key patient presentations to be considered:

1. The acute red eye
2. Sudden visual loss and visual impairment
3. The squinting child
4. Ocular trauma with visual impairment
Lecture 1: Anatomy of the eye

Fig. 1 Sagittal section of the eye.

Fig. 2 Layers of the retina.

Fig. 3 Diagram of the retina.
Fig. 4 Eyelids and eyeball in sagittal section.

Fig. 5 Eyelid structure anteriorly.

Fig. 6 The nerves and muscles of the orbit.
Lecture 1 (cont’d)

Anatomy and Physiology of the Eye

Overview of the major functions associated with ocular structures.

1) **Tears**: lubrication, oxygen transmission, clarity of vision, immunological protection.

2) **Conjunctiva**: smooth surface between eyelids and cornea, oxygen transmission, tear production, tear film adherent, defence, protection and integrity against micro-organisms and infection.

3) **Eyelids**: protection, tear film production and distribution, tear flow and drainage.

4) **Cornea**: refractive surface of fixed power, clarity, protection against micro-organisms, transparent smooth surface, oxygen transmission, ocular rigidity.

5) **Sclera**: protection, ocular rigidity, opaque, attachment of extra-ocular muscles.

6) **Lens**: refractive surface of variable power, accommodative mechanism, smooth interface with aqueous, transparency.

7) **Iris/Pupil**: restriction of light to retina (variable aperture), reduction of light scatter within the eye.

8) **Ciliary body/ ciliary epithelium**: accommodation, attachment of lens, production of aqueous humour (nutrient supply to lens and cornea and waste metabolite removal from globe).

9) **Choroid**: provides oxygen and nutrition to outer retina.

10) **Vitreous chamber**: vitreous humour maintains ocular shape, protection of ocular structures during trauma.

11) **Retina**: provides visual sensation and conversion of light images into nerve impulses. Supported by the retinal pigment epithelium, which is a layer of cells external to the retina essential for the formation of photopigments, the renewal of photoreceptors, the reduction of damage due to scattered light and the transportation of water and nutrients to the retina.

12) **Optic nerve**: transmits electrical signals to the brain via the optic nerve, optic chiasm, optic radiation, lateral geniculate nucleus and occipital cortex for further processing of the visual image.
Retinal physiology

The electromagnetic spectrum humans are visually responsive to is light between 400-700nm.

The functioning of rods and cones differs under scotopic (low light) versus photopic (daylight) light levels, location within the visual field, retinal location as well as stimulus size and position.

When a photon collides with the retina it is transmitted through the inverted retina to the photoreceptors where photochemical transduction results in the conversion of the light signal to an electrical signal.

Rods and cones

Rods and cones process the visual signal in characteristic ways and are described by performance functions which include the rod and cone spectral sensitivity curves as well as light and dark adaptation curves among other measures. They also differ in the minimum detectable number of photons required for visual processing as well as their ability to process signals across visual space and over time; namely their spatial and temporal summation.

Rods are more suited to processing the signals within visual images seen at low light levels (twilight, scotopic conditions) while cones process visual images seen at higher levels of luminance (bright daylight, photopic conditions).

Cones are responsible for the detection of colour and fine details (visual acuity) and are more concentrated in the central visual field. Rods are responsible for the detection of peripheral movement.

Visual Performance

Perceptual stimulus response functions therefore provide an understanding of visual dysfunction and may be used to indicate and facilitate earlier detection of ocular disease and refractive errors.

Form/Spatial Vision: clinically measured by visual acuity which varies with contrast, brightness, eccentricity and the testing procedure. Reflects the rod/cone distributions.

Colour Vision: hue, saturation, brightness and colour interactions are indicative of cone function and the associated processing of the visual signal. Colour vision testing may therefore provide indications for early detection of disease (i.e. saturation testing; blue/yellow vs. red/green defects).

Binocular Vision: associated with central vision. Requires clear images from both eyes, co-ordinated eye movements and intact cortical fusion mechanisms.

NOTES:
Lecture 2: Ocular Pharmacology
Dr Ilva Rupenthal

Ocular Pharmacology – An Introduction
Dr Ilva Rupenthal
Senior Lecturer and Director of the Buchanan Ocular Therapeutics Unit
Department of Ophthalmology
Faculty of Medical and Health Sciences
The University of Auckland

Pharmacokinetics vs. Pharmacodynamics

- Pharmacokinetics: What the body does to the drug
  - ADME: Absorption, Distribution, Metabolism, Elimination
  - Parameters: Cmax, tmax, t1/2, AUC, k
  - How often? How much?

- Pharmacodynamics: What the drug does to the body
  - Pharmacological effect
  - This will be covered in detail in 5th year

Absorption
How the drug gets into the eye...

- Route of administration
  - topical
  - intracameral
  - intravitreal
  - perocular
  - systemic
- Membrane properties (cornea)
- Drug characteristics (pKa, logP and MW)
- Dosage form

Ocular Administration Routes

- Intracameral
- Intravitreal
- Systemic (oral, parenteral)
- Topical
  - SUBCONJUNCTIVAL
  - PERIORBITAL
  - INTRASCLERAL
  - SUBCONJUNCTIVAL

90% of eye medications
Topical Ocular Drug Delivery

- Ease of application
- Direct application to target site
- Smaller drug dose required
- Rapid onset of action
- Contamination of drops
- Need for preservative → local toxicity
- Limited penetration (A) and fast elimination (E)
- Systemic absorption → side effects

Membrane Properties - Cornea

- Cornea as important mechanical barrier
- Main pathway for ocular penetration
- Sandwich-like structure
- Only drugs with MW <5kDa and logP of 10-100 can pass
- Thickness may affect drug permeation

Dosage Forms - Solutions

- homogeneous mixture composed of only one phase
- solute (drug) is dissolved in solvent (buffer)
- account for >90% of ophthalmic formulations
- β-blockers, PGA, α-agonists, CAI, some AB
- good stability
- easy to prepare
- low cost
- fast drainage → limited residence time
- low drug permeability through cornea
- drug bioavailability generally <10%

Dosage Forms - Suspensions

- heterogeneous mixture composed of two phases
- internal solid phase (drug) is dispersed throughout the external liquid phase (buffer)
- steroids (Pred Forte, Maxidex, Flucon)
- reduced drainage as particles remain in lower lid
- prolonged residence time → higher drug bioavailability
- high cost, sterilisation may cause physical instability
- particle size <10 µm → foreign body sensation → tearing
- particle aggregation/sedimentation
- MUST be shaken before use
Dosage Forms - Ointments

- semisolid preparation intended for external application
- drug in hydrocarbon base (no water!)
- Chloramphenicol and Acyclovir
- high viscosity → reduced drainage
- better absorption → higher bioavailability
- no stinging upon application
- oily base → no dilution by tears
- no preservative required
- suitable for moisture sensitive drugs
- lubricating nature
- blurred vision → application only at night time
- greasy → discomfort/irritation → reflex tearing

Distribution

How the drug moves around the eye...

Drug characteristics (pKa, logP, MW)

\[ \text{pH} = \text{pK}_a - \log \left( \frac{[\text{ionised}]}{[\text{un-ionised}]} \right) \]

\( \text{pK}_a \) (homatropine) = 9.7

@ pH 7: ≥ 99% of drug in unionised form (lipophilic) → can penetrate lipophilic epithelium

@ > pH 7 → more ionised drug → easily diffuses through hydrophilic stroma

Metabolism

How the drug is broken down...

- First-pass
- Enzyme induction/inhibition
- Drug activation

Metabolism – Drug Activation

- Ester prodrugs → more lipophilic → better absorption/penetration into cornea → activation by esterases
Most drugs
- Aspirin
- Phenytoin
- Ethanol

Kinetics
- First order
- Zero order

Elimination
How fast the drug leaves the eye...

Nasolacrimal drainage

Elimination – Nasolacrimal Drainage
- Normal tear volume: 7-10 μl
- Lower lid can hold: < 30 μl
- Eye dropper delivers: 40-70 μl
- Tear fluid turnover doubles after eye drop application ➔ washout
- Reflex tearing due to irritation (pH, foreign body sensation)
- > 80% of drug leaves via nasolacrimal duct
- High surface area and blood supply in nose ➔ systemic absorption ➔ side effects (β-blockers, steroids)

Correct Eye Drop Application
- Shake bottle before use (suspensions!)
- Apply one drop into lower lid pocket
- Close eyes and obstruct duct
  - Normal blinking ➔ <10% of drug remains after 5 min
  - Closed eyes ➔ >50% of drug remains after 5 min
  - Nasolacrimal obstruction ➔ >80% of drug remains after 5 min
  - Higher absorption across cornea
- Leave 5 min between drops (washout effect)

Second Drop Washout Effect
- Second drop washout effect
- If drug A is followed by drug B
  @ 30 sec: 50% of drug A is washed out
  @ 120 sec: 17%
  @ 5 min: 5%
- Leave 5 min between drops
- Less washout ➔ higher absorption
- Combination rather than separate drops
Generic Bioequivalence
Is one eye drop the same as another?

Two drops of the same drug may not be bioequivalent:
- pH of formulation ⇒ drug ionisation ⇒ solubility/permeation
- Particle size of drug in suspension ⇒ drainage/solubility
- Addition of preservatives ⇒ corneal permeation/absorption

Benzalkonium Cl (0.01%) may increase drug absorption by 50%
but also causes corneal toxicity

10 things you need to know

1. Shake bottle before use ⇒ suspensions!
2. Apply only one drop ⇒ drainage↓ side effects↓
3. Close eyes and obstruct duct ⇒ drainage↓ absorption↑
4. Leave 5 min between drops ⇒ washout ↓ absorption↑
5. Two eye drops of the same drug concentration may not be bioequivalent ⇒ absorption & elimination ⇒ efficacy & side effects

6. Topically applied drops can cause systemic side effects ⇒ β-blockers, steroids
7. Preservatives can cause ocular toxicity and allergies
8. Ointments and suspensions exhibit longer drug action than drops ⇒ drainage ↓ absorption↑
9. Use ointments only at night time ⇒ blurred vision
10. Corneal conditions may influence drug absorption ⇒ membrane properties

THANK YOU
Any questions or feedback email rupenthal@auckland.ac.nz
LECTURE 3: Symptoms of Eye Disease –

Dr James McKelvie

(Further notes on Signs & Symptoms can be found in Appendix 1)

History

A careful history will:

- Suggest the disease & its cause
- Direct your clinical examination
- Direct your investigations
- Place the problem in the context of the patient and their circumstances as a whole and allow you to plan their management.

**Standard format:** Presenting complaint and Symptom characteristics

**Vision loss**

- Sudden or gradual
- Constant vs. Intermittent- duration
- Extent of visual loss: blur vs. complete blackout
- Part of the field involved
- Associated symptoms e.g. headache, haloes

**Red eye**

- Unilateral / bilateral
- Associated symptoms
  - Pain
  - Vision affected
  - Discharge

**Pain**

- Character of the pain: sharp vs. dull
- Onset
- Exacerbating or relieving factors
- Associated symptoms e.g haloes or blurred vision
- Past ocular history e.g trauma, shingles
- Systemic enquiry, consider referred pain
Photophobia = light sensitivity

Usually due to

- keratitis
- uveitis

Double vision

- Onset e.g. after head injury
- Associated symptoms e.g. headache or blurred vision
- Past history e.g. of squint
- Systemic enquiry e.g. diabetic, myasthenia
- Two distinct images are seen: horizontal or vertical displacement relative to each other
- May be monocular or binocular
- Where are they relative to each other?

Flashes & Floaters

- Flashes originate from stimulation of the neurosensory retina by traction from the vitreous
- Floaters due to degeneration of the vitreous
- Sudden onset suggests a posterior vitreous detachment.
- Large red floaters or a drop in vision suggest vitreous haemorrhage
- Could suggest retinal detachment when severe e.g. Shower of floaters, many flashers, accompanied by a "black curtain" over part of the visual field in one eye.

Past Ocular History

- Spectacle wear
  - Myope
  - Hypermetropia - presbyopia
- Contact lens wear
- Squint or amblyopia
- Surgery or trauma
Past Medical History

- Vascular history if acute visual loss
- Rheumatological history in patients with uveitis
- Diabetes
- PMR

Medications & Allergies

- Give you an idea of the patient’s general state of health
- Remember systemic medications have ocular effects
- Ocular medications have systemic effects

Family History

Ocular

- Glaucoma
- Squint
- Cataract
- Poor vision
- Age Related Macular Degeneration
- Retinal Detachment

Social History

- Smoking / Diet
- Occupation- implications of visual disease
- Living situation- e.g elderly and lives alone.

NOTES:
(Further notes on Signs & Symptoms can be found in Appendix 1)

Examination of the eye consists of Visual Acuity & Slit Lamp

Visual acuity

- Unaided
- Pinhole
- Glasses if available
- Near vision

Slit Lamp Examination

- Inspect the structures of the eye from anterior to posterior

Fluorescein Drops

- Stains epithelial defects
- Assessing tear film
- Detecting leaks
- Checking intraocular pressure

Intraocular pressure

- IOP Assessment – goldman tonometry

Fundus examination

- **Must** dilate pupils: Tropicamide eye drops

Colour vision

- Ishihara plates

Visual fields

- Test on confrontation, preferably with a red target
Red Reflex

Extraocular muscles

Pupil reactions

- Responses to light
  - Direct
  - Consensual
- RAPD
- Response to accomodation

Case Study: Down Syndrome

- Epicanthal folds
- Up-sloping palbebral fissures
- Iris Brushfield Spots
- Blepharitis
- Epiphoria Congenital naso-lacrimal duct obstruction
- Strabismus
- Keratoconus
- Cataract
- Refractive Problems

NOTES:
The Acute Red Eye

Jennifer P. Craig
Associate Professor
Department of Ophthalmology
jp.craig@auckland.ac.nz

Question 1 – acute red eye

• 34 year old male
• Eye ‘a bit sore’
• Sensitive to light
• Vision slightly blurry

Sight threatening or self limiting?

Question 2 – acute red eye

• 78 year old female
• Sore (uncomfortable) since this morning
• Couldn’t open eyes easily first thing
• Intermittent blurring of vision

Sight threatening or self limiting?

Question 3 – acute red eye

• 29 year old male
• Concerned about red eye that appeared suddenly
• Not painful

Sight threatening or self limiting?
Acute Red Eye
Differential Diagnosis

- Conjunctivitis
- Keratitis
- Uveitis/Iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

For Acute Red Eye Flow Chart, see Auckland Eye Manual: pages 19-20

Conjunctivitis

<table>
<thead>
<tr>
<th>History</th>
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<tbody>
<tr>
<td>Past ocular disease ✗</td>
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<tr>
<td>Vision ✓</td>
</tr>
<tr>
<td>Pain and severity ✓</td>
</tr>
<tr>
<td>Photophobia ✓</td>
</tr>
<tr>
<td>Ocular discharge ✓</td>
</tr>
<tr>
<td>Systemic symptoms ✓</td>
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</table>

<table>
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<tr>
<th>Signs</th>
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<tbody>
<tr>
<td>Vision ✗</td>
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<tr>
<td>Discharge ✓</td>
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<tr>
<td>Redness and distribution: Bulbar and palpebral</td>
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<tr>
<td>Corneal clarity ✓</td>
</tr>
<tr>
<td>Pupil size and mobility ✓</td>
</tr>
<tr>
<td>Intraocular pressure ✗</td>
</tr>
</tbody>
</table>

Distribution of redness

- Circum-corneal / ciliary
- Bulbar / palpebral

Conjunctivitis: Management

- Largely self-limiting
- Swab and identify responsible organism
  - Bacterial
    - Chloramphenicol
    - Fucithalmic
  - Viral
    - No specific treatment
  - Chlamydial
    - Systemic tetracycline
Acute Red Eye
Differential Diagnosis

- Conjunctivitis
- Keratitis
- Uveitis/Iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

Keratitsis

History
Past ocular disease ✓
Vision ✓
Pain and severity: ++ to +++ ✓
Photophobia ✓
Ocular discharge ✓
Systemic symptoms ✓

Signs
Vision ✓
Discharge ✓
Redness and distribution: Ciliary ✓
Corneal clarity ✓
Pupil size and mobility ✓
Intraocular pressure ✓

Microbial keratitis
- Bacterial
- Viral
- Fungal
- Acanthamoeba (contact lenses)

Viral keratitis:
Herpes Simplex Virus

Keratitis: Management
- Corneal scrape
- Mydriatic
- Start intensive antimicrobials immediately
- Bacterial
  - Monotherapy: ciprofloxacin
  - Dual therapy: fortified Kefzol & Tobrex
- HSV
  - Acyclovir ointment 5x daily
Acute Red Eye
Differential Diagnosis

- Conjunctivitis
- Keratitis
- Uveitis/Iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

For Acute Red Eye Flow Chart, see Auckland Eye Manual, pages 19-20

Anterior Uveitis

<table>
<thead>
<tr>
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<th>Signs</th>
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</thead>
<tbody>
<tr>
<td>Past ocular disease</td>
<td>Vision ✓</td>
</tr>
<tr>
<td>Decreased vision</td>
<td>Discharge X</td>
</tr>
<tr>
<td>Pain and severity</td>
<td>Redness and distribution: Ciliary</td>
</tr>
<tr>
<td>Photophobia: +++</td>
<td>Corneal clarity X</td>
</tr>
<tr>
<td>Ocular discharge</td>
<td>Pupil size and mobility: Small (miotic)</td>
</tr>
<tr>
<td>Systemic symptoms</td>
<td>Intraocular pressure X</td>
</tr>
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</table>

For Ophthalmic History and Ophthalmic Examination Tips, see Auckland Eye Manual, pages 1-18
For Anterior Uveitis, refer to Auckland Eye Manual pages 151-152

Acute Anterior Uveitis / Iritis

Posterior synechiae

Aetiology

- Idiopathic
- Ankylosing spondylitis
- Reiters syndrome
- Juvenile arthritis
- Psoriatic arthropathy
- Sarcoidosis

Anterior Uveitis: Management

- Subdue Inflammation
  - Topical corticosteroids
- Prevent Posterior Synechiae
  - Mydriatic e.g. cyclopentolate
- Reduce IOP if elevated
  - Betablocker e.g. timolol
Acute Red Eye
Differential Diagnosis

- Conjunctivitis
- Keratitis
- Uveitis/Iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

For Acute Red Eye Chart, see Auckland Eye Manual: pages 19-20

Acute Angle Closure Glaucoma

History
Past ocular disease X
Decreased vision: +++
Pain and severity: +++
Photophobia X
Ocular discharge X
Systemic symptoms X

Signs
Vision ↓ +++
Discharge X
Redness and distribution: Ciliary
Corneal clarity ↓
Pupil size and mobility: Fixed mid-dilated
Intraocular pressure ↑↑

ACAG: Anatomical predisposition

Iris bombé

pupillary block

For ACAG: Anatomical predisposition, see Auckland Eye Manual: pages 19-20

Acute Angle Closure Glaucoma

- Red eye
- Hazy cornea
- Pupil mid-dilated
- Pupil non-reactive
- High IOP

For Acute Angle Closure Glaucoma, refer to Auckland Eye Manual: pages 147-148

AACG: Acute Management

- Reduction of IOP (Typically > 50mmHg)
  - Topical Agents
    - Prostaglandin analogues
    - Betablockers
    - Alpha agonists
  - Systemic Agents
    - Acetazolamide
    - Mannitol

For AACG: Acute Management, see Auckland Eye Manual: pages 19-20

Incidence
- 1/1000 (Caucasian) to 1/100 (Asian) > 40 yrs
- Ratio M:F is approximately 1:4

Predisposition
- Short eye
- Narrow angle
- Large lens

Therefore the older female hypermetrope is at risk

For Acute Angle Closure Glaucoma Incidence, see Auckland Eye Manual: pages 19-20
Aim: to re-establish normal aqueous flow & maintain IOP reduction

- YAG laser iridotomy
- Crystalline lens extraction
- Surgical iridectomy
- Trabeculectomy

**AACG: Surgical management**

**Acute Red Eye Differential Diagnosis**

- Conjunctivitis
- Keratitis
- Uveitis/Iritis
- Acute Angle Closure Glaucoma
  - Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

**Anterior Scleritis**

- Relatively rare
- Very severe boring pain
- Focal injection of sclera
- Can lead to blindness if untreated
- Associated with systemic disease

**Anterior Scleritis**

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<td>Redness and distribution: Sectorial, bluish</td>
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<td>Corneal clarity ✓</td>
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<td>Pupil size and mobility ✓</td>
</tr>
<tr>
<td>Systemic symptoms ✓</td>
<td>Intraocular pressure ✓</td>
</tr>
</tbody>
</table>

**Anterior Scleritis: Systemic associations**

- Rheumatoid arthritis
- Herpes Zoster Ophthalmicus
Acute Red Eye
Differential Diagnosis

- Conjunctivitis
- Keratitis
- Uveitis/iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

Episcleritis

- Relatively common
- Mild ocular discomfort
- Mild superficial injection
- Usually requires no treatment
- Seldom associated systemic disease

Episcleritis

<table>
<thead>
<tr>
<th>History</th>
<th>Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Past ocular disease</td>
<td>Vision X</td>
</tr>
<tr>
<td>Vision X</td>
<td>Discharge X</td>
</tr>
<tr>
<td>Pain and severity: +</td>
<td>Redness and distribution:</td>
</tr>
<tr>
<td></td>
<td>Sectorial, pink</td>
</tr>
<tr>
<td>Photophobia X</td>
<td>Corneal clarity X</td>
</tr>
<tr>
<td>Ocular discharge X</td>
<td>Pupil size and mobility X</td>
</tr>
<tr>
<td>Systemic symptoms X</td>
<td>Intraocular pressure X</td>
</tr>
</tbody>
</table>

Subconjunctival Haemorrhage

- Focal bleeding under conjunctiva
- Severe coughing
- Valsalva manoeuvre
- Rarely systemic hypertension
- Requires no treatment
Subconjunctival haemorrhage

**History**
- Past ocular disease ✓
- Vision ✓
- Pain and severity ✓
- Photophobia ✓
- Ocular discharge ✓
- Systemic symptoms ✓

**Signs**
- Vision ✓
- Discharge ✓
- Redness and distribution: Opaque red
- Corneal clarity ✓
- Pupil size and mobility ✓
- Intraocular pressure ✓

For Subconjunctival Haemorrhage, refer to Auckland Eye Manual: pages 19-20; image [post-trauma] page 41

Acute Red Eye

**Differential Diagnosis**
- Conjunctivitis
- Keratitis
- Uveitis/iritis
- Acute Angle Closure Glaucoma
- Anterior Scleritis
- Episcleritis
- Subconjunctival Haemorrhage
- Ocular Trauma

Ocular trauma!!

Beware of picking your nose ....

For Trauma, refer to Auckland Eye Manual: pages 35-49

The Acute Red Eye

- A systematic examination will enable appropriate management / referral decisions to be made.

THANK YOU!

For Acute Red Eye Flow Chart, see Auckland Eye Manual: pages 19-20
Epidemiology of eye trauma

- Eye injury admissions: 15.2 per 100,000
- Penetrating injuries: 3.6 per 100,000
- 40% of monocular blindness is traumatic
- Two thirds are male
- Majority are of working age

Causes and mechanisms

Assessment of eye trauma

- Rule out life threatening injuries
- Treat chemical injuries
- Rule out globe threatening injuries
- Examine both eyes and lids
- Consider radiological imaging
- Plan management

Establish mechanism

Ocular history

- Visual symptoms
  - Visual acuity, floaters, pain
- Past ocular history
- Previous injuries or ocular surgery
- Past medical history
Eyelid injuries

Haemorrhage

Orbital blow out fracture

Orbital blow out fracture

Orbital blow out fracture

Orbital blow out fracture - signs

- Black eye (haematoma)
- Infra-orbital nerve anaesthesia
- Double vision (upgaze & downgaze)
Radiological imaging

Globe injury
- Sub-conjunctival haemorrhage
- Corneal abrasion
- Foreign body
- Blunt ocular trauma
- Penetrating injury

Subconjunctival haemorrhage
- Coughing
- Vomiting
- Trauma
- Raised BP

Corneal abrasion

Corneal foreign body

Corneal foreign body
Corneal foreign body
Blunt trauma to globe
Penetrating eye injury
Penetrating eye injury

Aetiology of chemical injuries
- Industrial
  - Alkali’s & acids
  - High pressure & temperature
- Farming
  - Ammonia in fertilizers
- Home
  - Cleaning agents
- Assault
  - Alkali & acids

Epidemiology of chemical injuries
- USA
  - Assault No 1 cause of alkali injuries
  - <10% industrial injuries chemical
- Croyden Eye Unit UK
  - 221 chemical injuries
  - 89% accidental (63% at work)
  - Only 11% assaults
  - ~50% alkali, males (76%) 16-25 yrs

Remember normal anatomy

Chemical injury
- Alkalis
- Acids
- High pressure
- High temperature
- Particulate matter
Chemical injury: alkalis

- Ammonium ions penetrate instantaneously
- NaOH (Na Hydroxide, caustic soda, lye)
  - Hydroxyl ions take 3-5 minutes
  - Typically appears in drain cleaner
- Ca(OH)₂ (Ca Hydrate, Lime, Quicklime)
  - Plaster, mortar, cement
  - Particulate nature prolongs exposure
- All penetrate rapidly through the cornea & anterior chamber causing saponification and cell death

Chemical injury: acids

- Weak acids
  - Precipitate proteins within the corneal and conjunctival epithelium creating a partial barrier to further ingress
- Strong acids
  - Overcome layer of protein precipitation and enter anterior chamber with results indistinguishable from strong alkali burns

Chemical injury

Safety glasses!

Eye Chemical Injuries 101

RULE 1
In chemical injuries irrigate, irrigate, irrigate before taking full history or performing ocular examination!

Emergency Treatment: irrigation

- Immediate management
  - Copious irrigation at scene of incident
  - Ongoing irrigation for 1-2 hours
  - SINGLE MOST USEFUL Rx
  - Use any neutral fluid available
**Eye Chemical Injuries 101**

**RULE 2**

Remove all particulate matter as soon as possible to avoid ongoing chemical injury.

---

**Gentle but copious irrigation until pH is neutral (7.4-7.4), AT LEAST 20 mins**

**Mild chemical burn: mainly conjunctiva**

**Moderately severe cement injury: 1 and 3 weeks post event**

**Severe firework injury with Amniotic membrane transplant (AMT)**

**Medium term medical Rx**

- G. preservative free antibiotic
- G. preservative free prednisone**
- G. atropine 1% 1-2 per day
- G. ascorbate 10% 2 hourly
- G. citrate 10% 2 hourly
- Oral ascorbate 2G daily
- Oral analgesia for mod/severe pain
- Consider systemic doxycycline 100mg BD
Vitamin C (ascorbate)
- Vital to collagen synthesis
- Ascorbate depleted from cornea by severe alkali or acid injuries
- Scorbatic cornea prone to lysis
- Oral and topical ascorbate reduces risk of perforation

Citrate (Citric Acid)
- Powerful chelation agent for calcium
- Depletes calcium from PMN plasma membrane
- Reduction in calcium reduces 2nd wave of PMN extravasation from vessels
- Reduces PMN related inflammation
- Works in concert with Ascorbate

Surgical rehabilitation
- Symblepharolysis
- Cicatricial entropion/ectropion repair
- Debridement of epithelium
- Amniotic membrane transplant
- Limbal stem cell transplant
- Keratoprosthesis

Conclusions
Diabetic Eye Disease

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University of Auckland
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Organs affected
- Large blood vessel disease
  - Heart
  - Nervous system
  - Peripheral vascular disease
- Small blood vessel disease
  - Eyes
  - Kidneys
  - Nervous system

Ocular surface: Tear film and Cornea
- Tear film - Dry eye
  - Reduced tear quantity
  - Reduced tear film stability
- Reduced corneal sensitivity
- Greater risk of viral and fungal infections

Cornea
- Corneal nerve damage
- Keratopathy
  - Superficial punctate keratitis
  - Recurrent corneal erosions
  - Persistent epithelial defects
Iris, Pupil and Crystalline lens

- Miotic Pupil
- Iris neovascularisation
- Lenticular induced myopic shift!
- Cataract (post-subcapsular)

Retina

Diabetic retinopathy

Retina

- Two main retinal diseases in the older patient
  - Diabetic retinopathy
  - Age related macular degeneration
  Can you SEE the difference?????

Retina

- Diabetic retinopathy
  - Accounts for >90% blindness under the age of 60

- Age related macular degeneration
  - Accounts for >90% blindness over the age of 60
Key differential in retinal macular haemorrhages

- Principally, either diabetic maculopathy* or age related macular degeneration
- Less commonly branch retinal vein occlusion*
- Differentiate can be tricky:
  - Age ARMD > 60 years
  - Age DR < 60 years
  - Diabetic maculopathy* by itself is uncommon
    - Look for retinopathy beyond macula*

Clinical features

- Red bits (small and or large)
  - Blood: microaneurysms, haemorrhages
- White/yellow bits
  - Cotton wool spots, drusen & exudate
- Brown/black bits
  - Laser, pigment
Red Bits: Large
And Yellow bits
And brown/black bits

More than one vessel involved

All the retinal signs accounted for!!

• Blocked vessels lead to
  • retinal haemorrhages
  • Cotton wool spots
  • Abnormal retinal vessels
    • Venous “sausaging”, irregularity
    • Intra-retinal microvascular anomalies
  • Disc new vessels
  • Retinal new vessels
All the retinal signs accounted for!!

- Leaking vessels lead to
  - Protein (hard exudate)
  - Haemorrhage
  - Fluid (diffuse and local)
  - Intra-retinal and Sub-retinal

Diabetic retinopathy (DR)

- How do we classify it

- Background: bits and bobs but good vision!

- Pre-proliferative/ Proliferative: Ischaemic signs of varying degree leading to “new vessel growth”

- Maculopathy: involvement of the macula

Classification of retinopathy

**vessel blockage**

- No retinopathy
- Background
- Non-proliferative
- Increasing Ischaemia
- Proliferative

**vessel leakage**

- No leakage
- Leakage
  - Focal
  - Diffuse
  - Ischaemic
Non-proliferative Signs

- Microaneurysms
- Intraretinal haemorrhages
- Hard/Soft exudates

Venous changes

Proliferative retinopathy

- Neovascularisation

Intra-retinal microvascular abnormalities (IRMA)
Diabetic Macular oedema

Classification of maculopathy
vessel leakage/blockage

Maculopathy
• Focal
  • Fluid, lipid and proteins leak from a focal group of microaneurysm often leaving a well defined yellow ring – ring or circinate exudate
• Ischaemic
  • Capillaries underlying the fovea are all occluded
• Diffuse
  • All the capillaries leak.
Norma vasculature
Fluorescein angiogram

Ischaemic maculopathy

Screening Programs in NZ
• About 25% of NZ population has ‘pre-diabetes’

Social Engineering
• Healthy Living
  • Exercise
  • Weight loss
  • Support groups

Expensive, difficult to measure outcomes, politically unpopular
Why should we be interested?

- Increasing numbers worldwide: 552 million by 2030
  - International Diabetes Federation
- Personal cost
  - Morbidity, reduced life expectancy etc
- Economic cost in New Zealand
  - Diabetes NZ estimated $600 million spent in 2008
  - Projected cost for 2020 $1.61 billion
  - Ministry of Health 2009, Report on NZ Cost of Illness

Why are we interested?

- Retinopathy present in 1/3rd of diabetics
- About 4-8% have retinopathy at diagnosis
- Leading cause of blindness in working age-group
- Prompt recognition and treatment of sight-threatening eye disease can prevent sight-loss

Treatment of retinopathy

- Historically no treatment
  - "diabetics died" before chronic complications developed
- Historically poor systemic treatment
  - Laser protocols for blockage e.g. pan retinal photocoagulation
  - Laser protocols for leakage e.g. focal laser
- Bevacizumab (Avastin)
  - Help reducing macular oedema and neovascularisation

General management

- Education and support
- Institute good diabetic control
- Lifestyle changes
  - Weight loss
  - Dietary modification
  - Exercise
  - Cessation of smoking
- Carbohydrate control
  - Diet
  - Oral hypoglycemics
  - Insulin
- Control BP & Lipids
- Monitor renal function
- Management of the complications of diabetes

Programmes “Get Checked”. Budget 2006 $76m for obesity. CM “Lets Beat Diabetes”
Risk of retinopathy

- Age
- Duration of DM
- Glycaemic control
- Hypertension
- Lipid status
- Anaemia
- Pregnancy
- Obesity
- Smoking
- Alcohol use
- Other Systemic disease
- Ethnicity

Summary

- Blood and stuff in both retinae = diabetic retinopathy if pt < 60yrs
- If macula involvement = maculopathy
- If no macula involvement think “blockage”

Questions....
(Further notes on the causes of vision loss can be found in Appendix 2)

Patient 1: 58 year old  
**PC:** Loss of vision in his left eye  
- Better in the morning, worse through the day.  
- Fluctuates  
- Worse when tired.  
- Associated with diplopia- and then diplopia resolves and he has no vision in left eye.

**Differential Diagnosis of Ptosis**  
- Trauma  
- Orbital Cellulitis  
- III Cranial Nerve Palsy  
- Myasthenia

Patient 2: 21 year old  
**PC:** Increasing difficulty seeing out of his right eye.  
- Sticky discharge  
- Used antibiotics without much improvement.  
- Vision improves with a blink.

Patient 3: 18 year old girl  
**PC**  
- Sore left eye after a party.  
- Red  
- Poor vision  
- Light sensitive

Patient 4: 25 year old  
**PC**  
- Red painful left eye  
- Loss of vision  
- Photophobic  
- Recently under stress and quite tired

Patient 5: 27 year old  
**PC:** Sudden loss of vision in left eye  
- Woke one morning - put her contact lenses in and noticed she couldn’t see clearly  
- Developed associated pain  
- No problems in the right eye

**On examination:**  
<table>
<thead>
<tr>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA 6/6</td>
<td>6/36</td>
</tr>
<tr>
<td>Pupils</td>
<td>Left RAPD</td>
</tr>
<tr>
<td>Colour Vision 14/14</td>
<td>3/14</td>
</tr>
<tr>
<td>VF Normal</td>
<td>Central defect</td>
</tr>
</tbody>
</table>

**Fundus examination - Diagnosis?**
**Optic Neuritis**

- **Clinical features:**
  - Decreased VA
  - Decreased Colour Perception
  - Relative afferent pupillary defect
  - Normal or swollen optic nerve
  - Often associated with MS

**Patient 6: 62 year old man**  
(Refer Auckland Eye Manual - section 9-3 p 185)

**PC:** Loss of vision
- Flashes for 1 week.
- Spiders in front of his eyes.
- Shadow in front of eye

**Past Ocular History:** ??

**Patient 7: 57 year old man**  
(Refer Auckland Eye Manual - sections 9-12,13,14 p 199-202)

**PC:** Loss of vision
- Previous days he had loss of vision that lasted approximately 5 min in same eye.
- Sudden blackening of vision

**PMH**
- Smokes 40/day
- Hypertension
- Hyperlipidaemia

**On examination:**

<table>
<thead>
<tr>
<th></th>
<th>Right</th>
<th>Left</th>
</tr>
</thead>
<tbody>
<tr>
<td>VA</td>
<td>CF only</td>
<td>6/6</td>
</tr>
<tr>
<td>Pupils</td>
<td>Right RAPD</td>
<td></td>
</tr>
<tr>
<td>Colour Vision</td>
<td>Unable</td>
<td>14/14</td>
</tr>
</tbody>
</table>

**Fundus examination** - Retinal vein occlusion

**Patient 8: 72 year old man**  
(Refer Auckland Eye Manual - section 9-11 p 197)

**PC:** Big sneeze- and when opened his eyes noticed flies all over his wife.
- Questions?
  - Only right eye.
  - Flies moves with his eyes.

**PMH**
- Diabetic- IDDM 20 years
  
**Patient 9: 68 year old woman**  
(Refer Auckland Eye Manual - section 9-10 p 195)

**PC:** Noticed that while she was knitting her needles were bent rather than straight.

- Gradually became worse.

**Metamorphopsia:** Age related macular degeneration

**Gradual loss of vision** -
- Age related macular degeneration
- Diabetic retinopathy
- Chronic open angle glaucoma
- Extreme myopia
- Cataract
Patient 10: 72 yr presenting with LOV left eye- down to NPL

One day later: now NPL in Both eyes

Clinical Challenge?
Constitutional Symptoms?

GCA-Visual loss

* Majority have profound visual loss.
  * 20% experience antecedent transient visual loss.
  * Bilateral involvement is common.
  * 1/3 lose vision in second eye within 2 days
  * 1/3 within one week
  * 1/3 within one month

Start Treatment with Steroids!

Biopsy is the Gold Standard
Temporal Artery Biopsy
The Auckland Eye Manual

Available free from Google Play Store or Apple Apps.
OPHTHALMOSCOPY

Students must be familiar with the following key elements

1.) How to switch a standard ophthalmoscope on and off. The importance of using the lowest illumination necessary. Advantage of low room illumination where possible.

2.) The focusing dial: clockwise (yellow/white) for plus lenses (+1 to +20), anticlockwise (red) for minus lenses (-1 to -35). Importance of focusing - dialling in lenses compensates for both the observer's and the patient's refractive errors eg. if the observer has no refractive error and the patient is myopic, minus lenses will be required to focus on the retina.

3.) Observer should remove glasses unless astigmatic (and patient should remove glasses unless very, very myopic) to enable the ophthalmoscope to be held as close to the observers eye as possible.

4.) Right eye, right hand, and right forefinger on dial of ophthalmoscope should be used to examine patients R eye, and vice versa for left. Keep top of ophthalmoscope close to observer's brow such that the ophthalmoscope aperture is close to the eye.

5) Patients must be given clear instructions. Ask patient to look directly ahead or slightly upward, asking patient to look at a target may be confusing since the observer may well block the target causing patient confusion and eye movement.

6.) Put approximately +5 into dial and observe red reflex from 20-30 cm. The red reflex will usually be brought into sharp focus by dialling the focus dial anti-clockwise.

7.) Stand to the side of the patient, place hand on patients forehead (or shoulder) and whilst keeping pupil aligned move closer to eye from a slightly temporal approach. Dial lenses to bring retina into focus.

8.) The patient's pupil is like a keyhole, the nearer you are the more you see, the observer should bring the ophthalmoscope very close to the patients eye (3-5cm) whilst keeping it close to their own. The importance of pupil dilatation (g. tropicamide) will be noted from lecture and practicals (fifth year).

9.) Magnification is approximately x15 and the first object encountered is usually the optic disc which will fill much of the undilated view. If the optic disc is not visualised the vessels should be followed to the disk, remembering that the “V” created by the branching of vessels always points to the disc.

10.) Students should follow a methodical pattern of examination. A six point fundal examination routine should be followed: a) disc b) superior temporal arcade vessels and retina c) inferior temporal arcade vessels and retina d) inferior nasal arcade vessels and retina e) superior nasal arcade vessels and nasal retina f) macula. Always ask the subject to look in the direction of the portion of retina you are examining ie look up for superior retina, look left for nasal aspect of right retina etc.
Aim - Learn to test and understand the fields of vision and pupil reflexes. Both can be taught in minutes, done in seconds, and should from now on be part of the routine comprehensive student examination of any patient e.g. on admission to a medical ward, accident and emergency etc.

ASSESSING PUPIL REFLEXES

LIGHT REFLEX: Review and understand, using available diagrams, that the pupillary light reflex consists of a four neurones reflex arc:

1st neurone: Connects the photoreceptors in retina with the pre-tectal nucleus in the midbrain at the level of the superior colliculus. Decussate in chiasm.

2nd neurone: Connects pretectal nucleus to both the ipsilateral and contralateral Edinger Westphal nuclei, this is why a unilateral light response evokes bilateral and symmetrical pupillary constriction.

3rd neurone: Connects the Edinger Westphal nuclei to the ciliary ganglion. In the orbit, the parasympathetic fibres pass in the inferior division of the third nerve to reach the ciliary ganglion. (only parasympathetic fibres synapse here)

4th neurone: Leaves the ciliary ganglion and passes with the short ciliary nerves to innervate the sphincter pupillae.

NEAR REFLEX: Remember the near triad 1) increased accommodation 2) convergence of the visual axes 3) constriction of the pupils. The term light-near dissociation identifies a condition where the light reflex is absent or abnormal but the near reflex is intact. Since vision is not a prerequisite for the NEAR REFLEX there are no disease entities in which the light reflex is present but the near absent.

Introduction to pupillary assessment

Normal pupils are near equal in size. Remember the normal pupil reflexes.

1. Response to a light shone into one pupil.
   a. Direct response = pupil constriction in that eye.
   b. Consensual response = Simultaneous constriction of the other pupil.
   c. Swinging light test = Rapid changes of illumination from one eye to the other. Both pupils should stay equally constricted.

2. Response to Near

   Accommodation, convergence and miosis.
Practical Skills 2: Pupillary responses

PUPILLARY PATHWAYS AND REFLEXES

TO PUPIL efferent
OPTIC N. afferent
CILIARY GANGLION
THIRD NERVE
Edinger-
Westphal

Ambient light

Flashlight right eye

Flashlight left eye

NORMAL REACTIONS in both eyes

EFFERENT DEFECT Left eye

AFFERENT DEFECT Left eye

eg, atropine in eye third nerve palsy
eg, optic neuritis
PRACTICAL SKILLS 2: (cont’d)

Students should consider a series of sites for lesions and predict what effect such a lesion would have on the pupil reflexes of both eyes.

a) Complete optic nerve / retinal Total afferent pupillary defect (TAPD)

b) Incomplete optic nerve or mod severe retinal Relative afferent pupillary defect (RAPD)

c) Central (Argyll Robertson) bilateral, small irregular pupils, light near dissociation, difficult to dilate (syphilis)

d) Third nerve palsy Pupil dilated, no response to light - direct or consensual. Emergency ? aneurysm.

e) Postganglionic (Holmes Adie) Denervation of the postganglionic supply to the sphincter pupillae, possibly post-viral. Pupil large and regular (initially) Light near dissociation - light absent or very slow, near very slow / tonic, dilatation slow.

Finally consider the effect of sleep, alcohol and opiates on pupils - small as a result of decreased inhibition of the 3rd nerve nucleus. Alarm large, death and AACG fixed. NB: Students are expected to review the anatomy of Oculosympathetic palsy (Horner's Syndrome), the three neurone arc (posterior hypothalamus - ciliospinal centre of Budge [C8-T2] - superior cervical ganglion to the dilator pupillae [via ophthalmic division of the trigeminal nve, then nasociliary and long ciliary nerves]) and causes of Horner's syndrome (Congenital, carotid aneurysm, lesions of the neck e.g. trauma or surgery, brain stem vascular disease or MS, Pancoasts tumour of the lung etc.) in their private study period. Therefore this will not be covered in this practical session.

INTRODUCTION TO VISUAL FIELD ASSESSMENT

BACKGROUND:
Revise relevant anatomy: optic nerves, decussation at chiasm, optic tracts, optic radiation and occipital cortex. Understand relative preservation of spatial relationship of fibres, both horizontally, apart from decussation and vertically, e.g. lower half retinal fibres remain in lower half of pathways. Preponderance of macula fibres in centre of optic nerve. Homonymous and bitemporal defects.

VISUAL FIELD ASSESSMENT BY CONFRONTATION:

1. The examiner and subject should be seated approximately 70-100cm apart such that the hand / fingers/ target/ occupies a plane midway between them.

2. Use simultaneous presentation of hands as screen for hemianopia with both of the patients eyes open, then proceed to formal confrontation test.

3. Confrontation test using moving finger as target or counting fingers in peripheral field. The examiner should ask the subject to close one eye at a time in order to examine a single field. The subject should look at the examiners eye (left eye for field of subjects right eye and vice-versa) and the examiner should compare his/her field vision with the patient, movement of the finger or target should be made from the periphery to centre of the visual field in the shape of a diagonal cross. Emphasise the extent of the temporal field which extends to 90-100 degrees.

4. Identifying subtle central scotoma or the blind spot. This can only be done with small targets, eg. hat pin. If time allows this may be demonstrated, but this subject will be covered in "advanced visual fields" in the fourth year attachment.
VISUAL PATHWAYS WITH ASSOCIATED FIELD DEFECTS

Visual field, fixation central
Right half = dotted lines
Left half = solid lines

In all the following diagrams, the fields shown reflect the effects of total interruption of the indicated structures.
In REAL life, partial impairment is more the rule than the exception, of course.

1 OPTIC NERVE (monocular loss of vision)
   a. optic neuritis
   b. optic atrophy

2 OPTIC NERVE MERGING WITH CHIASM
   (monocular loss of vision associated with contralateral impairment of temporal field)

3 OPTIC CHIASM (bitemporal hemianopsia)

4 OPTIC TRACT (noncongruous homonymous hemianopsia)

5 TEMPORAL LOBE (upper, homonymous hemianopsia)

6 GENICULATE BODY (rare, total homonymous hemianopsia)

7 PARIETAL LOBE (lower, homonymous hemianopsia)

8 OCCIPITAL LOBE (Depending upon portions involved, can produce variety of homonymous hemianopsias. They range from total, to small homonymous scotomas. They have in common a high degree of congruity since fibers serving corresponding retinal points are sorted closer together as they approach cortex.)
PRACTICAL SKILLS 3: EXTRA-OCULAR MUSCLE MOVEMENTS AND COVER TEST

Introduction

Revise the essentials. Ocular movements are performed by the 6 extraocular muscles. Each muscle moves the eye particularly in one direction (primary action) but also has other less pronounced effects (secondary actions). Movements of one eye are referred to as ductions (e.g. abduction, adduction), whilst movements of both eyes together are termed versions (e.g. laevoversion and dextroversion).

In adults and older children failure of the two eyes to move together in synchrony and maintain the visual axes parallel, results in diplopia. In young children with a squint, in whom the visual axes are not parallel, the brain will suppress the image from the deviating eye and hence the child will not complain of diplopia. Prolonged periods of such suppression will result in a form of amblyopia ("lazy eye").

Any patient with a suspected squint should be examined using the cover test and testing of the extraocular movements. A squint may be termed concomitant or incomitant. In concomitant squints, the deviation does not vary with different positions of gaze, whereas in incomitant squints the deviation does increase or decrease depending on which direction the eyes are looking. Incomitant squints usually result in diplopia and are due to disease of the muscles or nerve supply to the muscles.

The six extraocular muscles and their actions (noting the nerve supply) :

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Action(s)</th>
<th>Nerve Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medial rectus</td>
<td>Adduction</td>
<td>III</td>
</tr>
<tr>
<td>Superior rectus</td>
<td>Elevation</td>
<td>III</td>
</tr>
<tr>
<td>Inferior rectus</td>
<td>Depression</td>
<td>III</td>
</tr>
<tr>
<td>Inferior oblique</td>
<td>Extorsion and elevation</td>
<td>III</td>
</tr>
<tr>
<td>Superior oblique</td>
<td>Intorsion and depression</td>
<td>IV</td>
</tr>
<tr>
<td>Lateral rectus</td>
<td>Abduction</td>
<td>VI</td>
</tr>
</tbody>
</table>

NB> The third cranial nerve also supplies the levator palpebrae in the upper eyelid and the parasympathetic constrictor fibres to the pupil.

EXTRA-OCULAR MUSCLE MOVEMENTS AND COVER TEST

When testing eye movements the eyes should be moved into the nine positions of gaze in order to test each of the extraocular muscles. The patient should be told to report any double vision.

The vertical recti muscles are tested when the eye is ABducted (superior rectus - elevation, inferior rectus - depression), and the oblique muscles with the eye in ADduction. Because of their insertions, when the eye is adducted the superior oblique acts purely to depress the globe and the inferior oblique to elevate it.

For those who prefer simple memory aids it might be remembered that to test the vertical actions of the inferior/superior rEcti the eye should be deviated towards the Ear, and to test the vertical actions of the inferior/superior Obliques, the eye being tested should be deviated towards the nOse.

If the patient complains of diplopia he should be asked whether the images are separated horizontally (horizontal recti paresis), vertically (vertical recti or obliques) or are just tilted (obliques). He should also be asked in which direction the separation is greatest and which image disappears on covering either eye. The image further away is derived from the eye with the paretic muscle.
The COVER-UNCOVER TEST to demonstrate strabismus (esotropia / exotropia)

This is the basic examination for the patient with a squint. Only by carrying out this test can one determine whether a patient, particularly a young child, has a squint or not. This is a monocular test designed to reveal a squint. Ask the patient to fix on an interesting target (e.g. picture, letter), cover one eye (non-squinting) with a hand held occluder:

1) In a non squinting person, when either eye is covered the other eye does not move
2) In a person with a convergent squint then the squinting eye will move from its initial (in-turning) position, in an outwards direction to take up fixation when the other eye is covered.
3) In a divergent squint, the diverging eye moves inwards to take up fixation when the non-squinting eye is covered.

Ideally this cover test should be performed for a target both in the distance (i.e. 6 metres or more) and for near. The near target should also have some degree of detail or pattern (i.e. not a light/ pen torch) in order to stimulate accommodation. This near examination is essential because some squints are only apparent, or are increased by accommodation, or may only occur for near (e.g. convergence excess) or for distance (e.g. intermittent exotropia).

Uncover component: when the non-squinting eye is uncovered, the strabismic eye which has temporarily taken up fixation will return to its original position ie; nasally in a convergent squint, temporally in a divergent squint.

The ALTERNATE COVER TEST to demonstrate phorias (esophoria/exophoria)

If the cover-uncover test is normal an alternate cover test may be performed to demonstrate an underlying phoria. In this test, both eyes are straight at the beginning and end of the procedure, one eye is covered for about 2 seconds and then quickly moved to cover the other eye. As the occluder is moved from the first eye a small compensatory movement may be identified ie. nasally in exophoria or laterally in esophoria.

The cover / uncover test should usually be used in conjunction with the Corneal light reflection test when attempting to identify squints.

CORNEAL LIGHT REFLECTION TEST

1. This is a good preliminary screening test for squint
2. Hold a pen torch in front of, and pointing towards, the child’s eye from a distance of 33cm. Observe the position of the corneal light reflexes in relation to the pupils.
3. The reflexes should be in the same position in each eye i.e. symmetrical.
4. A reflex which is displaced outwards in one eye (i.e. temporally) means that the eye is turned inwards – a convergent squint.
5. A reflex which is displaced inwards (i.e. medially) in one eye means that the eye is turned outwards – a divergent squint.
Clinical history:

It is essential to establish the nature and characteristics of the patient's complaint. The duration of and the circumstances surrounding the onset of symptoms need to be ascertained, whether the onset was gradual or sudden, unilateral or bilateral, whether the problem is intermittent, persistent or progressive, and whether there are any relieving or exacerbating factors. Any associated symptoms – ocular or systemic, should be noted. The format is as with any general medical history but with an ocular emphasis and a relevantly directed systemic review.

Presenting complaint

History of presenting complaint

Past ocular history: Squint, amblyopia, spectacles, contact lenses, trauma or surgery.

Past medical history: Direct as indicated by ocular history, e.g. a detailed vascular history in a patient with sudden painless loss of vision

Systemic enquiry: E.g. squints, glaucoma, cataract and diabetes.

Medications and allergies: e.g. squints, glaucoma, cataract and diabetes.

Family history of eye or systemic disease: e.g. squints, glaucoma, cataract and diabetes.


Key Symptoms:

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Comments and examples</th>
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<tbody>
<tr>
<td>Visual loss</td>
<td>Is it unilateral or bilateral? For example, unilateral visual loss is likely to be ocular whereas bilateral is more likely to be a result of visual pathways or cortical involvement. Is it sudden or gradual? Does it only involve one part of the field or the complete field? Is it transient, constant or progressive? Gradual loss in one eye may be cataract whereas sudden loss suggests a vascular aetiology; transient loss may be amaurosis fugax, a premonitory sign of a CVA. Loss of the superior field in one eye may be due to occlusion of a branch of a retinal vessel or a retinal detachment. Central visual loss or distortion, with preservation of the peripheral field, may be due to age-related macular degeneration. Eliciting other symptoms may contribute to the diagnosis. For example, a history flashing lights in one eye and a shower of floaters (mobile spots in front of the eyes) a week prior to a history of visual loss may suggest retinal detachment, whereas visual loss with sudden onset of dark or red blobs suggests a vitreous haemorrhage. Accompanying headaches, temporal artery tenderness, or jaw claudication may direct the examiner to consider giant cell arteritis.</td>
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<tr>
<td>Pain</td>
<td>What is the character of the pain? Is it sharp or dull, intermittent or constant? A sharp pain or irritation may suggest a sub-tarsal or corneal foreign body or corneal abrasion. A deep or boring pain is more likely to may suggest serious disease, as does the presence of a red eye. Is the pain exacerbated by reading, or on accommodation, as in iritis?</td>
</tr>
<tr>
<td>Redness</td>
<td>Unilateral or bilateral? Conjunctivitis is more likely to be bilateral and accompanied by purulent discharge, whereas iritis or acute glaucoma usually present unilaterally, but with pronounced pain. What is the distribution of the redness? Is it diffuse, mainly affecting the tarsal conjunctiva as in conjunctivitis or sectorial on the bulbar conjunctiva as in</td>
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</table>
episcleritis or scleritis? Localisation around the cornea (circumcorneal) suggests a more serious inflammation such as keratitis (corneal inflammation) or anterior uveitis.

### Photopsia
Flashes of light in one eye with onset of floaters suggest vitreous degeneration with acute detachment of the vitreous from the retina. The flashes are from traction from the vitreous and stimulation of the neurosensory retina. This can result in a hole or tear in the retina and eventually lead to a retinal detachment.

### Diplopia / double vision
Is diplopia monocular (cataract, media opacities) or binocular (misalignment of the ocular axes)? Are the two images next to each other or above and below each other? Are they tilted?

### Photophobia
The patient may complain of light sensitivity. This may be due to glare from cataract or because there is inflammation in the eye due to uveitis or keratitis.

### Discharge
Purulent discharge suggests an infective conjunctivitis, mucous or watery discharge may suggest allergic conjunctivitis.

### Epiphora and lacrimation
Increased tearing can result from any form of irritation or inflammation of the cornea. Overflow can result from increased tearing, or insufficient drainage through the naso-lacrimal system.

### Itchiness
A history of hay fever or allergic eye disease may cause itchy, gritty eyes, as may blepharitis.

### Grittiness and irritation
Grittiness, irritation or foreign-body sensation is usually due to an ocular surface abnormality such as a dry eye, small recurrent erosion or a foreign body.

### Examination:
Having taken an appropriate history, a simple but systematic examination as outlined below will often lead you to a short-list of diagnostic possibilities. Please see Practical Skills and pages 12 – 15 in the recommended text (Ophthalmology. An Illustrated Text. Batterbury and Bowling, Churchill Livingston) for further explanation. As many ocular disorders have systemic associations, a systemic examination may also be required.

<table>
<thead>
<tr>
<th><strong>Comment</strong></th>
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<tbody>
<tr>
<td><strong>General inspection</strong></td>
</tr>
<tr>
<td><strong>Vision</strong></td>
</tr>
<tr>
<td>A Snellen chart, a near reading chart and a pinhole is required.</td>
</tr>
<tr>
<td>Check near and distance vision with the patient’s own spectacles. One eye at a time is tested; improvement using a pinhole suggests a correctable refractive error.</td>
</tr>
<tr>
<td><strong>Colour vision</strong></td>
</tr>
<tr>
<td>Use a red target or Ishihara plates.</td>
</tr>
<tr>
<td>Use a red target to compare colour vision in each eye. Optic nerve disease such as optic neuritis causes the red colour to appear desaturated or washed out, compared to the normal eye. Ishihara plates are used to identify congenital colour abnormalities but may also be used in acquired colour defects.</td>
</tr>
<tr>
<td><strong>Visual fields to confrontation</strong></td>
</tr>
<tr>
<td>A large white (and possibly red) pin is required</td>
</tr>
<tr>
<td>Sit at the same level as the patient, approximately one metre apart. Asking the patient to look at your face and see if any parts are missing can often pick up hemianopias and central scotomas. The patient’s field in each eye individually is then compared with your own, using targets equidistant between you</td>
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</table>
and the patient. Use ‘finger counting’ centrally and then test the peripheral field using a white target coming in from the periphery. The blind spot may be plotted using a red pin if required.

<table>
<thead>
<tr>
<th>Eye movements</th>
<th>Use a pen torch and an occluder are used.</th>
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<tbody>
<tr>
<td>The corneal reflexes should be symmetrical if the eyes are aligned. Performing a cover-uncover test on each eye will elicit a manifest squint, follow this by asking the patient to follow a target or light into the 9 (union jack) positions of gaze to see if this elicits diplopia.</td>
<td></td>
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<tr>
<th>Pupils</th>
<th>Are the pupils mobile, round and equal in size? Is there a ‘direct’ and ‘consensual’ response? Is there a relative afferent defect on the swinging flashlight test? A lack of constriction or a relative dilatation in one pupil indicates optic nerve or extensive retinal disease. Finally check that the pupils constrict on accommodation by asking the patient to look at a near target.</th>
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<tr>
<th>Examination of the external eye –</th>
<th>Examine the eyelids, conjunctiva, cornea, anterior chamber and iris. Are the appearances normal? Is there redness, swelling, or discharge? What is the distribution? Is the cornea bright, shiny and clear? Are there opacities, abrasions, abnormal blood vessels or foreign bodies? Does the cornea stain with fluorescein? Is the anterior chamber deep? Does it have blood or pus cells within it? Does light shining from one side of the eye light up the whole iris? Can you see the iris details clearly- does it have dilated vessels or appear abnormal in any other way?</th>
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<tr>
<th>Pupil dilation</th>
<th>Dilate pupils with a drop of tropicamide 0.5% or 1.0% (onset within 20 minutes, lasts 2 to 4 hours) to examine the red reflex and posterior segment of the eye.</th>
</tr>
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<tr>
<th>Red reflex</th>
<th>Use an ophthalmoscope to assess the eye from approximately a 1/3rd of a metre away, dial in ‘plus’ lenses until the iris and then the red reflex is seen clearly. As some of the light entering the eye is reflected back by the retina, an even red glow is usually obtained. Any opacity within the normally transparent structures of the eye will cause dark obscuration of the red reflex. Cataract is the most common cause of obscuration of part or the entire red reflex.</th>
</tr>
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<tr>
<th>Examination of the posterior segment</th>
<th>Ask the patient to fix on a distant target then systematically examine the ocular fundus. Start with the optic nerve head; follow the vessels along the arcades into each quadrant before examining the retina between the vessels. Then finally examine the macula, which is temporal to the optic disc.</th>
</tr>
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</table>
AIMS:

1. To understand and prioritise the common causes of acute visual loss
2. To understand and manage appropriately less acute forms of visual loss
3. To be acquainted with visual prognosis in order to inform patient
4. To be aware of legal definitions and restrictions related to visual loss
5. Use of support services eg. partial sight/blind registration; blind societies; low visual aid provision; guide dogs for the blind
6. To understand the impact of loss of vision in the family and community
7. Blindness prevention

Introduction to vision loss

Loss of vision involves not only the eye by also the central elements of the visual apparatus. Loss of vision can therefore be subdivided **anatomically** into three subgroups:

- Anterior to chiasm
- Chiasmal
- Posterior to chiasm

Lesions anterior to the optic chiasm, affecting the eye or optic nerve, are likely to produce visual loss in only one eye (or one eye at a time), whereas chiasmal lesions usually lead to bitemporal field involvement and retrochiasmal lesions (posterior to the chiasm) tend to produce hemianopic field defects.

Loss of vision can also be divided **temporally** into acute visual loss and gradual visual loss. Conditions which cause acute visual loss, ie. within a few minutes to few hours, should be treated as ophthalmic emergencies.

Important aspects of history (also see lecture 2)

1. Speed of onset - acute or gradual
2. Extent of visual loss total / central / partial / hemifield
3. One eye or both eyes affected
4. Other ocular symptoms photopsia / flashing lights / "floaters" metamorphopsia (central distortion) ocular pain - consider differential of acute red eye
5. Systemic symptoms headache Jaw claudication etc. (GCA)
6. Systemic disease Hypertension Diabetes Cardiovascular disease
ACUTE LOSS OF VISION

Common causes of persisting* painless acute total/subtotal loss of vision in one eye

1. Ischaemic optic neuropathy - may be arteritic (giant cell arteritis) or non-arteritic
2. Central retinal artery occlusion or large branch retinal artery occlusion
3. Central retinal vein occlusion or large branch retinal vein occlusion

Less common but equally important acute or subacute causes of visual loss in one eye

4. Vitreous haemorrhage (trauma / retinal detachment / diabetic retinopathy)
5. Retinal detachment
6. Retrobulbar neuritis (may be associated with mild degree of pain)

Acute loss of vision affecting both eyes

1. Cerebral infarction involving optic tract, radiation or occipital cortex

*NB. Amaurosis fugax: by definition is a transient loss of vision in one eye lasting usually only a few minutes and at most a few hours - essentially a TIA involving the eye and usually secondary to embolic event from carotid or heart.

Immediate management of sudden loss of vision

These conditions should all be treated as ophthalmic emergencies and the importance of excluding giant cell arteritis cannot be overstated in relation to sudden loss of vision in the contralateral eye.

Students must be familiar with the symptoms, fundal appearances, and management of all these conditions.

COMMON CAUSES OF PAINLESS GRADUAL LOSS OF VISION

1. Age related macular degeneration (ARMD)
2. Chronic open angle glaucoma (COAG)
3. Senile cataract
4. Diabetic retinopathy
5. Extreme myopia

These are very common conditions that the student/graduate will encounter in general medicine, accident and emergency, general practice etc.

Other important (but less common) causes of gradual loss of vision

1. Tobacco/alcohol (nutritional) amblyopia (usually one eye more severely affected)
2. Toxic optic neuropathies - drugs, heavy metals
3. Retinitis pigmentosa
COMMON CAUSES OF PAINLESS GRADUAL LOSS OF VISION (cont.)

Age related macular degeneration (ARMD)

This may be of very gradual onset, or if associated with sub-retinal neovascularisation and haemorrhage, may present rapidly over a few days to weeks with profound loss of central vision. Students should be aware of the difference between "dry" and "wet" macular degeneration and the progression of the disease and the use of the AMSLER grid.

ARMD is one of the most common reasons for blind registration in adulthood. The prevalence (for all degrees from minor to severe) of ARMD rises from approximately 2% in the 54-64 year old age group to almost 30% in the 75-85 year old age group.

Students should be familiar with the fundal appearances, symptoms and prognosis.

Chronic open angle glaucoma (COAG)

The association of 1. an open anterior chamber angle, 2. chronically raised intra-ocular pressure (normal 11-21mHg), 3. "cupping" of the optic disc (students should recall CDR ratios) and 4. progressive loss of visual field, characterise chronic open angle glaucoma. This is a very common disease that causes gradual progressive field loss if left untreated, it is often picked up by optometrists when patients are being examined for spectacles. It affects approximately 1% of persons between 60-70 years of age and 3% of the population over 75 years. Affected subjects may provide a family history and are usually over 40 years of age.

Students should be familiar with optic disc appearance, prognosis, medical management, and surgical management. The difference between acute closed angle glaucoma and chronic open angle glaucoma must be clearly understood.

Senile and other forms of cataract (also see lecture 9)

This is the most common cause of gradual visual impairment, short of blindness, in the elderly. Indeed, lens opacities which reduce visual acuity to 6/9 or less have a prevalence of almost 50% in subjects over 75 years of age!

Students must be aware of symptoms, signs and associations of cataract. They should also have a clear understanding of the surgical management of this condition, which they can impart to patients. Cataract extraction is the most common elective surgical procedure carried out with an annual extraction rate of greater than 1% in the over seventies.

Diabetic Retinopathy

Diabetes affects 2 to 2.5% of populations of Northern European extraction but may affect 4 to 8% of Maori and Pacific Islanders. There is an approximate 8:1 ratio between NIDDM and insulin dependent cases. Poor control and duration of diabetes are associated with greater risk of diabetic retinopathy. Diabetic retinopathy remains one of the most common causes of blind registration in the NZ.

Students should be familiar with the classification of diabetic retinopathy

Extreme Myopia

Myopia of greater than -8.0 to -10.0 Dioptries is frequently associated with a number of degenerative ocular conditions which may lead to severe visual impairment including: myopic macular degeneration; chronic open angle glaucoma; cataract; and retinal detachment.
DEFINING VISUAL IMPAIRMENT

Students must familiarise themselves with the following during the fifth year course:

a) Range of “normal” visual acuity and refractive error  
b) Blindness registration/partial sight registration  
c) Legal requirements to drive  
d) Local services for the visually impaired  
e) The role of low visual aids

RELEVANCE TO OTHER CURRICULUM THEMES

Visual loss is directly related to many other components of the medical curriculum. Students should review the anatomy of the visual pathways and cerebral/ocular blood supply. Visual loss may be associated with common medical disorders including diabetes, hypertension and cardiovascular disease. Since visual loss of gradual onset primarily affects the elderly there are obvious ramifications in relation to medicine in the community and maintaining independence in the ageing population. Disease prevention by early identification and appropriate management of early diabetic retinopathy or early glaucoma is important. Since visual loss involves not only the eye but also the central visual apparatus there is a significant degree of overlap with neurology themes.
APPENDIX 3: CARE OF THE VISUALLY DISABLED

In every doctor’s practice, they will from time to time come across visually disabled persons. During your clinical attachment, you will visit the Auckland Eye Department Vision assessment Clinic (VAC). This is for people with low vision and it also supplies an electrodiagnostic, rehabilitation, optometric and genetic service. The following are some notes about visual impairment.

**ASSESSMENT OF:**
- VISUAL IMPAIRMENT
- VISUAL DISABILITY
- VISUAL HANDICAP

**VISION ASSESSMENT CLINIC (Auckland Hospital)**

Multidisciplinary, tertiary care clinic, designed to assist patients who have already been assessed by an Ophthalmologist. Referrals are accepted from GPs, Optometrists and Social Workers of RNZFB.

**Ophthalmologist:**
Specialising in medical retinal disorders, electrodiagnostic studies

**Optometrist:**
Experienced in assessing and dispensing optical and electronic Low Vision Aids for the visually impaired

**Visual rehabilitation Coordinator:**
Experienced in rehabilitation of the visually impaired.

**Technical Assistant / Orthoptist:**
Technician for visual field and colour vision testing. Electrodiagnostic technician

**Rehabilitation Instructor from the RNZFB:**
Rehabilitation of the visually impaired, including home visits.

**Clinical Geneticist:**
Genetic counselling for families with inherited opthalmic disorders.

**Measurement of VISUAL IMPAIRMENT**

<table>
<thead>
<tr>
<th></th>
<th>Moderate</th>
<th>Severe</th>
<th>Profound</th>
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Other factors modifying the degree of visual impairment:

1. **Visual Field**
   - Central stroma
   - Very restricted field
   - Patchy visual field
   - Hemianopia – homonymous / bitemporal

2. **Contrast sensitivity**
   - Affected by diabetic retinopathy, cataracts and other conditions

3. **Colour Vision**

4. **Night Vision**
   - e.g. Retinitis Pigmentosa

5. **Fluctuating Vision**
   - Temporary reductions in vision secondary to: vitreous hge / macular oedema / diabetics with fluctuations in blood glucose / temporary e.g. awaiting cataract surgery.
Optical Aids For VISUAL DISABILITY

**Moderate:** Optical appliances for detailed visual tasks
   ▶ NEAR NORMAL vision with aids e.g. low magnification magnifiers

**Severe:** Optical appliances for detailed visual tasks
   ▶ LESS THAN NORMAL vision with aids e.g. higher magnification magnifiers (but higher magnification gives a smaller field)
   
   A realistic goal is reading for information not reading for pleasure.

**Profound:** Optical appliances and OTHER SKILLS for visual tasks e.g. closed circuit TV or other high tech aids.

Aids for Daily Living (ADL) for Visual Handicap

**Moderate:** Meet MOST social expectations visually

**Severe:** Meet MANY social expectations visually

**Profound:** Difficulty with many social expectations

Aids for Daily Living (ADL) for Moderate / Severe:
- Extra lighting, large print, felt-tip pens, Typoscope (reduces glare), heavy lined paper.
- Use of contrast e.g. white cup with coffee / black with milk
- Large print cards, black watch face with white print
- Maxiphone.

Aids for Daily Living (ADL) for Profound: TACTILE
- Elastic lined writing frame, tape measure with tactile markings, self-threading needles, cheque templates, Braille playing cards, talking clock.

Factors modifying the degree of visual handicap:
- Current age, age of onset, social expectations
- Social support (family / community)
- Psychological adjustment to visual impairment
- Self-image (i.e. solitary / extrovert/ organised/ preconceived idea of "blind person")
- Physical health
- Occupation

Royal New Zealand Foundation for the Blind (RNZFB)

Registration Criteria:

**Full:** Visual acuity (VA) not exceeding 6/60 or visual field not wider than 20 degrees.

**Restricted:** VA 6/24 to 6/60

**Temporary:** e.g. awaiting eye surgery
RNZFB Services

<table>
<thead>
<tr>
<th>Visual Impairment</th>
<th>Moderate</th>
<th>Severe</th>
<th>Profound</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Social support groups</td>
<td>+</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>Sport / craft/ cultural (e.g. Ngati Kapo) / youth groups</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Vocational assistance</td>
<td>modify</td>
<td></td>
<td>new technology</td>
</tr>
<tr>
<td>3. Daily living skills</td>
<td>advice</td>
<td>instruction</td>
<td>home visit / intense rehab</td>
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<tr>
<td>O &amp; M</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>4. Talking books</td>
<td>-</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td>5. Guide Dog</td>
<td>-</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>6. Counselling, Field Workers, Child and Family counsellors, Whanau workers</td>
<td>+</td>
<td>+</td>
<td>++</td>
</tr>
</tbody>
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Community Services

Financial Assistance:
1. Disability allowance
Provides for extra expenses incurred because of disability (Means tested) - $40/week
2. Blind Invalid Benefit
Not means tested (<2/60 and <3 degree visual field)
3. Equipment Funding
Assistance through income support service for education, training and employment. Not means tested. Equipment recommended by DSW accredited assessor.
4. Diabetic Educators
Educational sessions. Organise motivational support groups. Advice on modified equipment e.g. blood monitors and click syringes.

Other Community Services
1. “60’s plus”
The over 60’s are entitled to help from “60’s plus”. This includes housework, shopping and transport. Obtained through extramural.
2. Domiciliary Occupational Therapist.
Provide safety aids e.g. rails, non-slip surfaces. The combination of visual impairment and physical problems can compound to make a person less secure in their home environment.
Run by the community e.g. Care and Craft
- a place to go and meet people
- gives relief to care givers
- helps establish social contact and reduces the sense of being cut off through being homebound and not seeing peoples’ faces.
The Department of Ophthalmology
4th floor, Building 504
Faculty of Medical and Health Sciences
The University of Auckland

www.fmhs.auckland.ac.nz/som/ophthalmology