## Contents

### Section 1
Learning Objectives for Phase 2 (4th & 5th Year)  
Page 2

### Section 2
On-Line Resources  
Page 5

### Section 3
Tutorial Material

- Ophthalmoscopy  
  Page 6
- Eye Accident & Emergencies  
  Page 8
- Cataract  
  Page 12
- Uveitis & Floaters  
  Page 17
- Eye Banking and Corneal Transplantation in New Zealand  
  Page 21
- Pupil Abnormalities  
  Page 24
- Ocular Surface Inflammation and Allergy  
  Page 28
- Retinal Conditions & Signs of Disease  
  Page 30
- Oculopastics Overview  
  Page 33
- The Acute Red Eye  
  Page 42
- Glaucoma  
  Page 48
- Paediatric Ophthalmology  
  Page 52
- Dry Eye and Sjögren’s Syndrome  
  Page 57

NB. These tutorial notes are to be read in conjunction with the 4th Year Handout and with the 
recommended reading selections, as listed in the 5th year program (University Dept of 
reading from the 5th year program is included in Section 4 of this handout.

### Section 4
Self-directed learning tool:

- Ophthalmology Quiz  
  Page 63
- Quiz Answers  
  Page 69

Recommended Reading  
Page 71

Clinical Scenarios – Links in the MBChB Portal (http://mbchb.auckland.ac.nz)  
Page 73

### Section 5
Contact Details  
Page 75
## Ophthalmology (Phase 2, Years 4 and 5)

### Expected learning outcomes:

<table>
<thead>
<tr>
<th>Domain</th>
<th>Acquisition and Application of Medical Knowledge</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Review and reinforce pre-clinical knowledge applicable to eye disease</td>
</tr>
<tr>
<td>2</td>
<td>Develop an appreciation of key ocular symptoms and signs and the underlying pathological processes</td>
</tr>
</tbody>
</table>
| 3      | Knowledge of symptoms, signs and main differential diagnoses of key ophthalmic presentations:  
|        | a. The acute red eye  
|        | b. Sudden visual loss and visual impairment  
|        | c. The squinting child  
|        | d. Ocular trauma with visual impairment |
| 4      | Appreciation of the ocular manifestations of systemic disease, e.g. diabetes mellitus, hypertension, collagen diseases. Understand the pathology of diabetic retinopathy, its classification and prevention/treatment |
| 5      | Understand the causes, management and health burden of senile cataract |
| 6      | Understand the causes, management and health burden of age related macular degeneration |
| 7      | Understand the causes, management and health burden of chronic open angle glaucoma |
| 8      | Establish relevance of key elements of pathology, microbiology and immunology to eye disease |
| 9      | Basic ocular therapeutics |

### Domain | Professional, Clinical and Research Skills |
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Take an appropriate ophthalmic history</td>
</tr>
<tr>
<td>2</td>
<td>Assessment of visual acuity (near, distance, colour)</td>
</tr>
<tr>
<td>3</td>
<td>Visual field assessment to confrontation / central field by Amsler grid</td>
</tr>
<tr>
<td>4</td>
<td>Examination of the anterior segment – conjunctivae, cornea, iris/pupil</td>
</tr>
<tr>
<td>5</td>
<td>Pupillary examination – near triad, response to light, consensual, swinging light test</td>
</tr>
<tr>
<td>Domain</td>
<td>Professional, Clinical and Research Skills cont.</td>
</tr>
<tr>
<td>--------</td>
<td>-----------------------------------------------</td>
</tr>
<tr>
<td>6</td>
<td>Examination of the red reflex by ophthalmoscope</td>
</tr>
<tr>
<td>7</td>
<td>Examination of the posterior segment using the ophthalmoscope including assessment of optic disc, maculae and vasculature</td>
</tr>
<tr>
<td>8</td>
<td>Assess presence of squint, corneal reflexes, cover test and eye movements</td>
</tr>
<tr>
<td>9</td>
<td>Basic elements of anterior segment slit-lamp examination</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Domain</th>
<th>Hauora Maori</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Develop awareness of the patient’s cultural background and usage of effective communication to facilitate appropriate ocular examination</td>
</tr>
<tr>
<td>2</td>
<td>Appreciation of epidemiology of different ocular conditions as well systemic diseases with ocular manifestations (e.g. diabetes) affecting Maori and Pacific Island population</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Domain</th>
<th>Population Health and Primary Health Care</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Knowledge of important causes of blindness in New Zealand and worldwide</td>
</tr>
<tr>
<td>2</td>
<td>An appreciation of the impact of visual disability on the patient’s lifestyle, family and community</td>
</tr>
<tr>
<td>3</td>
<td>Awareness of the community agencies for the support of visually impaired (e.g. Royal New Zealand Foundation of the Blind, Glaucoma New Zealand)</td>
</tr>
</tbody>
</table>
On-Line Resources

The Ophthalmology Department’s website address is http://ophthalmology.auckland.ac.nz or it can be accessed via the University of Auckland’s homepage, under the School of Medicine.

Materials pertinent to the 5th year teaching program can be found by opening the Teaching page and following the sub-menus listed.

Teaching materials available on-line for use include:

1. **CAL – computer assisted learning.**
   There are 4 subspecialty sections, each containing a series of clinical images, with questions to be answered. The correct responses are supplied on the answer pages.
   The standard of questions presented here corresponds to the level of questions set for the clinical exam paper, to be sat at the end of the Ophthalmology attachment.

2. "**The Eyes Have It**" is an interactive teaching program, courtesy of the University of Michigan Kellogg Eye Center, about disorders of the eyes and visual system. In the Instructional Mode, you will see photographs or captioned streaming videos supplemented with text. In the Quiz Mode, you will have a chance to test your knowledge and be scored.

3. 5th Year Handout 2011 (PDF file) and Tutorial Presentations as given in the Auckland rotation (PDF Files).

4. Clinical Skills Videos. These videos demonstrate the variety of skills to be acquired during the Ophthalmology attachment as listed in the expected learning outcomes.
Ophthalmoscopy
Associate Professor Jennifer Craig, Dr Charlotte Jordan

<table>
<thead>
<tr>
<th>Direct Ophthalmoscopy</th>
<th>Indirect Ophthalmoscopy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monocular</td>
<td>Usually binocular</td>
</tr>
<tr>
<td>Erect image (upright)</td>
<td>Inverted image, laterally reversed</td>
</tr>
<tr>
<td>High magnification (x 15)</td>
<td>Lower magnification (x2 – x3)</td>
</tr>
<tr>
<td>Restricted field of view (7 - 8°)</td>
<td>Increase field of view (approx. 30°)</td>
</tr>
<tr>
<td>Provides view to the arcades</td>
<td>Provides view to ora serrata (with scleral indentation)</td>
</tr>
</tbody>
</table>

Heine Mini 3000 Ophthalmoscope

Examination:

Red Reflex
External eye
The normal fundus

The normal disc

Cup-to-disc ratio: 0.5

http://www.foto-web.com/simuladores/
Fundus examination simulator (click on picture)
(n.b. Download does not work via PC link!)
Eye accident and emergency
Dr James McKelvie

Topics covered in this session:
1. Overview of history taking
2. Examination basics
3. Common conditions presenting to A&E
4. Sight threatening conditions
5. Life threatening conditions

1. The History
Specific considerations for ophthalmic history:
- Trauma (High speed, metal)
- Chemical (acid/alkaline)
- Light (U.V./I.R)
- No precipitating event

2. Examination basics
A systematic approach is essential so as not to miss important signs

Anatomical approach commonly used and easy to remember

Anatomical approach to exam
- VA
- Skin/Lids
- Conjunctiva
- Cornea
- Anterior chamber
- Lens
- Vitreous
- Macula/Retina
- Optic nerve
- Orbit/bone
- Neurological

3. Common A&E problems (Case studies)
GP referral
Grinding metal 2/7 ago
Eye red, irritated and sore
Flourescein staining showing “scratches” on cornea

What important features must you ascertain from the history?
What do you examine first?

High velocity and no safety glasses are risk factors for penetrating injury and
extraocular foreign body – need to exclude with careful examination
Common A&E problems: history of trauma and examination shows...
Self referral
Playing squash yesterday and hit in RE with ball.
“Blurry” vision since
VA R 6/48, 6/30ph

What important features must you ascertain from the history?
What must you check on exam?

Common A&E problems: red eyes, lids and cornea
GP referral with red eyes 7/7
No improvement with chloramphenicol
Gritty, itchy eyes, started in RE then LE 2/7 later.
VA 6/6, 6/6

What important features must you ascertain from the history?
What must you check on exam?
How can you confirm the diagnosis?
What treatment is indicated?

Common A&E problems: red eyes, lids and cornea
GP referral with L corneal ulcer that stains with fluorescein
VA L 6/18

What important features must you ascertain from the history?
What should you check on exam before instilling fluorescein and topical anesthetic?
How can you confirm the diagnosis and what is your differential for corneal ulcers?

Chemical and thermal /UV injuries
Referral from Emergency Department with chemical injury to RE 2/7 ago.
What is the immediate management of chemical injuries that involve the eye?
What is worse acid or alkali?
Why is it important to assess the limbus?

Chemical and thermal /UV injuries
Grinding and welding a trailer at home 2/7 ago.
LE sore and red from 10pm and now sensitive to bright light as well.

What is the diagnosis?

More red eyes, no precipitating event
GP referral with viral conjunctivitis and no improvement with chloramphenicol
Sore red eye for last 3/7.
Happened out of the blue.
Similar episode 12/12 ago but did not seek help
VA L 6/18
What features in the history are you interested in?
What examination findings are consistent with diagnosis?
What investigations would you like to do (if any) and why?

4. Sight threatening problems (painful)
GP referral with rapid onset (4/24) painful++ red eye cloudy cornea.
VA 6/24

What are the essential features of the examination you must check?
Why is the cornea hazy?
Why is the pupil mid dilated and sluggish?
Why is this a sight threatening problem that requires urgent management?

   Sudden onset painless loss of vision
Optometry referral – patient woke with painless loss of vision in one eye
VA 6/60
Differential diagnosis includes:
   • CRVO/BRVO
   • CRAO/BRAO
   • Retinal Detachment
   • Ischaemic optic neuropathy
   • Optic neuritis

   Intermittant loss of vision
GP calls with possible referral. 65 year old man with extensive history of left-sided headaches with general ache around his shoulders, mild weight loss and intermittent episodes of visual loss over past week.

Any additional questions for GP?
What is the differential diagnosis?
What investigations would you like on arrival? What are the expected results in this condition?
What should you include in your exam and why?
How can you confirm the diagnosis? What is the treatment and when should it be started?

5. Life threatening
Orbital vs preseptal cellulitis
Referral from SSH. 5 year old boy presents with swollen red RE. Not able to check vision
?orbital cellulitis.

What is orbital cellulitis and how does it differ from preseptal cellulitis?
How can you clinically differentiate these two conditions?
What investigations or imaging is required?
Why is orbital cellulitis a potentially life threatening problem?

**What is going on here?**
GP referral with binocular diplopia for one month
Patient complains of feeling hot at night but currently afebrile and FBC Normal

What is your differential diagnosis?
What investigations would be helpful to confirm the diagnosis and plan management?

**Ward call**
You are a house officer and have been asked by a nurse on the urology ward to see a man with double vision that started 2 hours ago and eyes look in “funny position”.

What is the abnormality?
Do you need to do anything about it now or can you leave a message for the team in the morning?
What is the next step in management?

**Don’t forget...**

<table>
<thead>
<tr>
<th>Sight threatening</th>
<th>Life threatening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute glaucoma</td>
<td>Third nerve palsy with pupil involvement</td>
</tr>
<tr>
<td>Giant cell arteritis</td>
<td>Orbital cellulitis</td>
</tr>
<tr>
<td>Intraocular foreign body</td>
<td></td>
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</tbody>
</table>
Cataract: past, present and future
Professor Charles McGhee

Blindness from cataract:
Globally, 25 million cataract blind
Requires 36 million procedures to treat <6/12

Basic Lens Anatomy –

- Transparent, biconvex structure
- Lies behind iris, suspended by zonules – loss of zonules leads to subluxated lens
- Approximately 10mm diameter, 4 mm thick

Tends to opacity (cataract) with age

The eye has two principal focusing structures
a) The cornea 2/3rd – approximately 40 dioptres
b) The crystalline lens 1/3rd – approximately 20 dioptres
c) If a lens cataract is removed the focussing power (20D) needs to be replaced
d) Historically replaced by spectacles, in last 30 years mainly by intraocular lenses

Cataract assessment:
acuity, ophthalmoscope and slit lamp

Describing cataract anatomy:
Anterior subcapsular
Cortical
Nuclear
Posterior subcapsular

The global burden of cataract
- 25 million blind (classified as 20/400)
- Additional 2 million new cases per annum
- 110 million severe visual impairment
- 90% of blindness in developing world
- Only 7 million procedures per annum
Cataract - multiple aetiologies
- Congenital
- Inherited
- **Age-related (the majority)**
  - Metabolic – e.g. diabetes
  - Toxic – e.g. corticosteroids
  - Traumatic – e.g. irradiation
  - Secondary – e.g. ant. uveitis

Cataract aetiology in developing world:
repeated dehydrational crises

Cataract in NZ
The Auckland Cataract Study
- Provided a prospective snapshot of 500 patients with cataract, cataract services, and state of the art surgical techniques in a public hospital service in the year 2000
- Also highlighted public cataract waiting list issues and access to appropriate government funded care in a major metropolitan area (now resolved)
- Results published in a series of scientific publications in the British Journal of Ophthalmology and Clinical and Experimental Ophthalmology

The Auckland Cataract Study: Systemic Disease
The majority of those with significant cataract have General Health issues

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>25%</td>
</tr>
<tr>
<td>Cerebral vascular disease</td>
<td>12%</td>
</tr>
<tr>
<td>Diabetes Melitus</td>
<td>11%</td>
</tr>
<tr>
<td>Ischaemic Heart disease</td>
<td>10%</td>
</tr>
</tbody>
</table>

Rx
- Aspirin 42%
- Warfarin 6%

Auckland Cataract Study: commonly associated ocular disease
Ocular co-morbidities in cataract patients -

<table>
<thead>
<tr>
<th>Condition</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open-angle glaucoma</td>
<td>10%</td>
</tr>
<tr>
<td>Diabetic retinopathy</td>
<td>6%</td>
</tr>
<tr>
<td>Vein occlusion</td>
<td>2%</td>
</tr>
<tr>
<td>Advanced AMD</td>
<td>1%</td>
</tr>
</tbody>
</table>
Cataract Surgery
Ancient Techniques
   Couching
Current cataract extraction techniques
   1. Intra-capsular – now mainly in developing world
   2. Extra-capsular – some use in developed world
   3. Phacoemulsification – most popular technique

Phacoemulsification Basics
Use of high frequency ultrasound to emulsify cataract performed through a smaller incision (6mm verses traditional 10mm incision of ECCE procedures)

Phacoemulsification hand piece
   • Piezoelectric crystal vibrates ultrasonically
   • Driven electrically
   • Frequency 25,000 – 60,000 Hz
   • Phaco “needle” is hollow (0.9mm diam) with central aspiration port
   • Needle surrounded by soft irrigation sleeve with two ports which maintains fluid in ant. chamber

Late 1990’s Intra-ocular lenses: small incision phacoemulsification and the evolution of foldable and injectable Intraocular lenses.
Folding or injecting the 6mm diameter IOL enables the incision size to be reduced to 3mm width for no suture phaco with incisions moving from largely scleral to corneal based

Corneal Incision structure
One and two step corneal incisions
   • Quick
   • Single instrument
   • No suture
   • Enables topical anaesthetic approach
   • Popular

What’s new in IOL design?
Considering -
   PCO and square edge
   Correction of spherical aberration
   Multifocal / accommodative IOLs
   Coloured IOLs & macular protection
   Specialised IOLs for reconstruction
The Auckland Cataract Project

**Summary 1**
In a public health system, a predominantly elderly, female population, frequently with significant systemic illness and co-existing ocular diseases, relatively advanced cataracts and poor visual acuity, presented for cataract surgery. The majority of subjects (97.5%) underwent small incision, phacoemulsification, local anaesthetic, day-case surgery. The remaining 2.5% underwent extra-capsular cataract surgery.

**Summary 2**
Despite co-existing eye disease, almost 90% of patients achieved best spectacle corrected vision (BSCVA) of 6/12 [20/40] or better which meets the NZ & UK driver’s licence standard. Although approximately 5% of eyes sustained an adverse intra-operative event only 1.5% of eyes exhibited poorer post-operative BSCVA than anticipated.
Uveitis & Floaters
Dr. M. Pradhan, Dr T. Malik, Dr M. Donaldson

Introduction
• Inflammation of uveal tissue
  Involving iris, ciliary body, choroid
• Classification
  – Anterior
  – Intermediate
  – Posterior
  – Panuveitis

Uveitis Associations:
• Idiopathic,
• HLA B27 +ve
  – Ankylosing spondylitis
  – Juvenile chronic arthritis
  – Psoriatic arthropathy
  – Reiter’s syndrome
• Sarcoidosis
• Behcets
• Inflammatory bowel disease
• Collagen vascular disorders
  SLE
  Polyarteritis nodosa
  Wegener’s
• Infection
  – Toxoplasma, Toxocara, Lyme
  – HSV, HZV, HIV, CMV
  – Tuberculosis, syphilis, leprosy
  – Candidiasis
• Trauma
  Sympathetic ophthalmia

Symptoms of Anterior Uveitis -
• Aching pain
• Redness
• Photophobia
• Blurring of vision
• Pain on accommodation
**Signs of Anterior Uveitis** -
- Decreased acuity
- Unilateral red eye
- Circumciliary conjunctival injection
- Small or irregular pupil
- Inflammatory products in Anterior Chamber (AC)
- Sequelae

**Inflammatory Products:**
Cells – graded as +1 to +4
Protein (flare) - +1 to +4
Fibrin – Grade +4 flare
Keratic precipitates
- Fine, non-granulomatous
- Mutton-fat, granulomatous

Iris Nodules – Koepp, Busacca

**Sequelae**
- Posterior synechiae
- Seclusio pupillae
- Occlusio pupillae
- Iris bombè
- Sectoral iris atrophy - HZO
- Low IOP - hypotony
- Glaucoma
- Cataract
- Cystoid macular oedema
- Neovascularisation

**Glaucoma**
- Inflammation of the trabecular meshwork
- Steroid responsive glaucoma – in 20 %, due to topical treatment
- Mechanical - ‘pupil block’

**Symptoms of Posterior Uveitis**
- Floaters
- Blurred vision
**Signs of Posterior Uveitis**

- Inflammatory products
- Visible focus of chorioretinal inflammation
- Inflammatory consequences
  - Macular oedema
  - Vascular sheathing / occlusions
  - Optic disc swelling

**Management of uveitis**

**Anterior:**

- Topical steroids (intensive and early)
- Cycloplegics (pain relief and prevent posterior synechiae)
- Topical / oral Antivirals (aciclovir) – HSV, HZO
- Local steroid injections (subconjunctival / sub tenons)
- Systemic steroids

**Posterior / panuveitis**

- Orbital floor / posterior subtenons Steroid injection
- Systemic steroids
- Antibiotics, anti-TB therapy
- Antivirals – oral (HZO, CMV, HIV), intravitreal ganciclovir (CMV)
- Immunosuppression

**Floaters**

**Causes**

Posterior vitreous detachment - commonest

Blood
  - Neovascularisation eg diabetes
  - Torn peripheral retina

Inflammatory products – intermediate / posterior uveitis

**History...**

Onset

Flashing lights
Reduced vision

Other symptoms e.g. field loss

Other history - ocular e.g. myopia, surgery, trauma
  - systemic e.g. diabetes
Retinal tear
Floater – haemorrhage / PVD
Photopsiae / flashes – traction at edge of tear

Retinal detachment
Secondary to PVD and retinal tear
Floater / flashes and loss of field
‘Curtain’ effect

Diabetic eye disease – neovascularisation at optic disc

Diabetes – preretinal and vitreous haemorrhage due to neovascularisation

Vitreous haemorrhage – blurred vision and floaters
Eye Banking and Corneal Transplantation in New Zealand
Louise Moffatt, Manager, New Zealand National Eye Bank
Associate Professor Trevor Sherwin

“The acquisition, evaluation & supply of high-quality corneal and other tissue to all New Zealanders needing a transplant to restore their sight”

Tissues Supplied:
- **Corneas [220 – 250 per annum]**
  - Viable, unaltered, sight-restoring
  - Stored in warm organ culture (34°C), up to 21 days
- **Sclera [140 – 160 per annum]**
  - Structural, reconstruction
  - Stored refrigerated, up to 6 months
- **Amniotic membrane [15 – 20 per annum]**
  - Structural/biological, surface ‘bandage’
  - Stored frozen (-80°C), up to 2 years

Tissue Donation: the process

**Organ donation:** heart, liver, kidneys, lungs, pancreas - 50 per year
**Tissue donation:** corneas, skin, heart valves, bone – many 1000’s per year

Suitable donors identified – by transplant coordinators, medical staff, OR referral from families
Next-of kin contacted - information provided about donation
  - possibility raised, no coercion
  - if consent given, process explained
Retrieval:
  - careful surgical procedure in hospital, mortuary or funeral home
  - donor treated with dignity & respect
  - no delay to funeral arrangements
  - no visible difference, viewing can occur

- **Driver’s licence is indication of wish only**
  Importance of discussion with family/whanau
Donor requirements:
Age: 10 – 85 years
Time: within 24 hours

Medical contraindications:
- Death of unknown cause
- Infectious disease: - Hepatitis B,C, HIV, meningitis
- Systemic viral infection
- CNS diseases, progressive dementia
- Leukaemia, lymphoma
- Previous eye disease or surgery, laser surgery
- Various congenital disorders

Generally suitable:
- Cancers
- Bacterial septicaemia
- Heart disease, respiratory disease
- Diabetes, arthritis
- Vision problems – glasses/contacts usually ok

Eye Bank Operations
- Donor acquisition from Auckland area – within 24 hours
- Links with donor sources: Mortuary, Hospitals, other transplant services
- Donors selected by strict criteria for suitability
- Long-term storage in 34°C culture system – up to 21 days
- Extensive testing for infectious disease & contamination
- Quality control / sterility of highest standard
- Minimum endothelial cell count

Eye bank output:
- Supply 100% of all transplanted corneas in NZ
- Schedule of planned, elective surgery: 6+ grafts per week
- Reduction of surgical waiting lists

Corneal Transplantation:
Oldest form of transplant – early 1900’s

Most common transplant operation performed
USA 45,000 pa
UK 9,000pa
NZ 250 pa

Avascular
- few rejection problems
- no systemic immunosuppression required
- 12-18 months for optimal vision
Indications for Corneal Transplantation in New Zealand:

Keratoconus - 47%
- Acquired abnormality – cornea protrudes, thin & distorted
- Bilateral, progressive, occurs at young age (teens, twenties)
- High prevalence in NZ – 50% of transplants
- Does keratoconus progress more rapidly in NZ?

Keratopathy/oedema - 18%
- Painful epithelial blisters, scarring
- Association with glaucoma & following cataract surgery

Corneal Dystrophies – 10%
- Intrinsic genetic disorders, or aging
- Epithelial abrasion/erosion, endothelial cell loss & dysfunction

Viral/bacterial keratitis – 10%
- Inflammation – opacification – vascularisation - ulcers
- Risk of rejection higher – blood vessels reduces graft tolerance

Trauma – 6%
- Perforation – physical/chemical injury
- Heavy male preponderance
Pupil Abnormalities
Dr Stephen Best, Dr Hussein Patel

Pathway of the papillary light reflex consists of:
Afferent visual pathway
- retinal receptor cells
- bipolar cells
- ganglion cells
- optic nerve and tract
- Pretectal nucleus of the high midbrain
- Edinger-Westphal nucleus

Efferent visual pathway
- Two neurone pathway via the
  - Oculomotor nerve
  - Constrictor muscle of the iris

Anatomy of the papillary reflexes
The size of the pupils depends on the balance of sympathetic and parasympathetic activity supplying the iris (efferent visual pathway):
- sympathetic activity tends to dilate the pupil
- parasympathetic activity tends to constrict the pupil

Normal Light Reflex

Examination of Pupils
- before dilating
- size, symmetry
- shape
- near reflex
- light reflex
- RAPD (direct and indirect response)

Anisocoria
Difference in pupil size between the eyes - may be physiological or pathological
Physiological anisocoria
  - normal variation in pupil size
  - 20% of individuals
  - Usually less than 1mm

Local Factors affecting pupil size
  - Topical medications:
    - mydriatics/miotics/other agents
  - Trauma:
    - traumatic mydriasis / sphincter rupture / surgical trauma / posterior synechiae
  - Disease processes:
    - uveitis / acute angle closure glaucoma
  - Systemic medications:
    - narcotics = miosis

Conditions with Pathological pupil size
Abnormally small pupil:
  - Horner’s Syndrome
  - Argyll Robertson pupil
Abnormally large pupil:
  - Adie’s Tonic Pupil
  - Pupil involved 3rd nerve palsy
  - Bilateral dilated pupils

Horner’s Syndrome
  - Oculosympathetic paresis....interruption of the sympathetic supply along the three neuron pathway
  - Miosis
  - Ptosis
  - Apparent enophthalmous
  - Cutaneous anhydrosis
  - Other features – transient hyperaemia/iris hypopigmentation in congenital cases
  - Diagnosis confirmed by topical cocaine test
  - Abnormal pupil fails to dilate whilst the normal pupil will dilate (loss of noradrenaline at nerve junction)
- Other associated clinical signs and symptoms...
  (headache / apical lung pathology / long tract neurology signs ) will determine appropriate investigations

**Argyll Robertson Pupil**
- Specific sign of neurosyphilis
- Small and irregular pupils
- Usually bilateral but asymmetric
- Do not respond to light but near response normal (light-near dissociation)

**Adie’s Pupil**
Postganglionic parasympathetic denervation:
- Causes: idiopathic, viral, diabetes, trauma
- Glare / accommodative difficulties
  - Mydriasis
  - Light – near dissociation....slow constriction on prolonged near effort and slow re-dilation to distance
  - Usually young females – 90% unilateral initially , but often becomes bilateral
  - Pupil becomes tonic with time....even miotic
  - If decreased tendon reflexes present = Adie’s syndrome
  - Diagnosis confirmed by **denervation hypersensitivity** to weak cholinergic (pilocarpine 0.1%)... abnormal pupil will constrict whilst normal pupil remains un-effected
  - Aberrant re-innervation of pupillary sphincter muscle ... contractions of part of the pupil margin (**vermiform movement**)  

**Causes of CN III Palsy**
- Microvascular infarction
  - Occlusion vaso nevorum
  - Risks: diabetes, hypertension, artherosclerosis, hyperlipidaemia
- Compressive lesion
  - Aneurysm (usually post communicating artery)
  - Tumour
- Trauma

**What to look for if there is anisocoria?**
- Make sure patient has not had any eye drops instilled
- Check for prescription, over the counter ‘herbal’ and illicit drug use.
- Any History of eye surgery (iatrogenic)
- Check for signs such as ptosis, or ocular motility problems

**Relative Afferent Pupillary Defect (RAPD)**
- The presence of RAPD in the absence of gross ocular disease indicates a neurological lesion of the anterior visual pathway (afferent system)
- Detected using the ‘swinging flashlight test’
- Abnormal pupil respond to consensual light but not direct light
Causes of RAPD
- Optic nerve disorders
- Chiasmal compromise
- Optic tract lesions
- Large retinal detachments or macular lesions, dense vitreous haemorrhage
- RAPD not produced by cataract, refractive error, lesions posterior to the lateral geniculate body, non-physiologic visual loss

Ocular Surface Inflammation and Allergy
Dr David Pendergrast

Papillae versus Follicles

<table>
<thead>
<tr>
<th>Papillae</th>
<th>Follicles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic inflammation</td>
<td>Acute inflammation</td>
</tr>
<tr>
<td>Allergy</td>
<td>Viral</td>
</tr>
<tr>
<td>C/L, sutures, prosthesis</td>
<td>Chlamydial</td>
</tr>
<tr>
<td>Cobblestones</td>
<td>Toxic</td>
</tr>
<tr>
<td>Central vascularity</td>
<td>Pale lesions</td>
</tr>
<tr>
<td></td>
<td>Surrounding vessels injected</td>
</tr>
</tbody>
</table>

Allergic Conjunctivitis:

**acute to chronic**
- Acute hay fever conjunctivitis
- Seasonal allergic conjunctivitis
- Perennial allergic conjunctivitis
- Vernal keratoconjunctivitis
- Atopic keratoconjunctivitis

Vernal Keratoconjunctivitis
- Age 9 to 19
- Boys > Girls
- Warm dry climates
- Symptoms: itching, mucus, photophobia
- Signs: superior tarsal or limbal papillae
- Pseudogerontoxon
- Peripheral fibrovascular pannus
- Shield ulcer

Shield Ulcer:
- Persistent Epithelial defect
- Physical trauma from papillae, rubbing
- Chemical trauma from inflammatory mediators
- Mucous plaque formation
Section 3. Tutorial Notes

Ophthalmology, University of Auckland

Atopic keratoconjunctivitis

- Adult onset
- Symptoms: itch, photophobia, watering
- Signs: redness
- Fine papillary reaction
- Periorbital atopic eczema
- Microbial keratitis esp. opportunistic
- Deep corneal vessels and scarring

Pathophysiology: Mast Cells

Mast cell degranulation in response to:

- Allergens and IgE
- Physical trauma (rubbing)
- UV exposure
- Increased ocular surface temperature
- Bacterial lipopolysaccharides

Therapeutic options I: for milder disease

- Avoidance of allergens and rubbing
- Cold compresses
- Topical antihistamines: rapid onset
- Systemic antihistamines: slower onset
- Mast cell stabilisers: preventative use
- Topical NSAIDs: Acular has some effect
- Dual action agents: best current therapy
  - Patanol

Therapeutic options II: for sight threatening disease

- Topical corticosteroids
  - Introduce at high frequency, tail off rapidly
- Topical cyclosporine 2% ointment
- Systemic immunosuppression
- Surgery:
  - Excision of papillae
  - Superficial keratectomy

Review images in power-point presentation!
Signs of Retinal Disease  
Dr Tahira Malik, Dr Monika Pradhan

Blood supply of the retina
• CRA - real artery
• Atherosclerosis
• AV crossings
• Capillary-free zone – foveal avascular zone

Signs of Retinal Vascular Disease
• Vascular changes – arteriolosclerosis, AV crossing changes, venous tortuosity
• Microaneurysms
• Haemorrhages
• Hard exudates
• Cotton wool spots
• Neovascularisation

Vessel wall changes  Vascular changes
Silver wiring of arterioles  Venous dilatation and tortuosity
Copper wiring of arterioles  AV crossing changes
Arteriolar attenuation
AV crossing changes

Microaneurysms
• Tiny round red dots
• In inner nuclear layer
• Saccular outpouchings from vessels due to lack of pericytes

Retinal haemorrhages
Appearance ➔ Localisation ➔ Aetiology
• Dot & blot haemorrhages – middle compact retinal layers – diabetic and hypertensive retinopathy
• Flame-shaped haemorrhages – superficial nerve fibre layer – vein occlusion
• Pre-retinal haemorrhages – between posterior hyaloid face and retina – trauma, Valsalva manœuvre, neovascularisation
Retinal haemorrhages – Roth spots
Caused by

- Endocarditis
- Anaemia
- Haematological diseases

Hard exudates

- Lipid deposition
- Outer plexiform layer
- Abnormal vascular permeability
- Circinate pattern
- Macular star

Cotton wool spots

- Ischaemic infarction
- Nerve fiber layer
- Axoplasmic transport defect
- Diabetes
- Hypertension
- SLE
- Leukemias

Neovascularisation

hypoxia $\Rightarrow$ vasoformative factors $\Rightarrow$ neovascularisation

- in the retina NVE
- on the optic disc NVD
- on the iris NVI / rubeosis iridis
- in the angle NVA

Causes:

- Diabetes
- Retinal vein occlusion
- Retrolental fibroplasia
- Sickle-cell disease
- Inflammatory diseases
Oclusive Vascular Diseases Signs

**Venous occlusion:**
- Dilated venules
- Haemorrhages
- Oedema

**Retinal arteries occlusion:**
- constricted arterioles
- column interruption
- retinal oedema
- cherry red spot
- visible emboli

Degeneration/dystrophy signs
- Drusen
- Atrophy
- Pigmentation
- Scarring
  - result of disease
  - iatrogenic – laser scars

Elevations of the Retina
- by fluid – Retinal Detachment
- by solid mass - tumours
- Choroidal neovascularisation

Signs of Inflammation-
**Posterior uveitis**
- Vitreous opacities, cells, snowballs
- Choroiditis – deep yellow or grey lesions
- Retinitis – white cloudy lesion with obscuration of retinal vessels
- Vasculitis – periphlebitis (veins) or periarteritis – fluffy haziness around blood column
- Granlomas – choroidal or preretinal
Oculoplastics Overview  
Dr Neena Peters

Functions of the eyelids
1. Light protection and regulation  
2. Keep cornea moist  
3. Mechanical protection of globe

Lacrimal pump

ELYELIDS (surface anatomy)
- Upper lid [UL] covers superior 2mm of cornea (10 – 2’o clock)  
- Lower Lid [LL] margin JUST touches inferior limbus  
- Distance between UL margin & central pupillary reflex is margin reflex distance (MRD1) and normally, is 3.5 mm  
- Space between UL & LL is palpebral fissure height (PFH) and normally, it is 9-12 mm.  
- A puncta (one in each lid) lies in close apposition to globe

To summarize-
Normal parameters for lid position evaluation:
- A = Skin crease 10 mm in females 8-10 mm in males
- B = MRD1 – 3.5 – 4.0 mm
- C = PFH – 10 -11mm
- Inferior scleral show – Nil
- Lagophthalmos (inability to close eyes) – Nil
- Bell’s phenomenon (globe movement on eye closure) – Up and out

Eye Lids (Internal Anatomy)
Two halves – Lamellae
- Septum
- Landmark – Grey Line
  - Anatomical
  - Surgical

Orbital Septum
- thin sheet of fibrous tissue
- natural barrier between eyelid and orbit

Eyelid: muscular and nervous control

Eyelid Retractors:
- Levator Aponeurosis (3rd CN. Parasymp)
- Muller’s muscle (Sym)

Eyelid Protractor:
- Orbicularis Oculi (VII N)

Abnormal Upper Eyelid Position

PTOSIS (droopy eyelid)

Levator Palpebrae superioris (LPS)
- LPS action – from extreme down gaze to extreme up gaze, by blocking the Frontalis
- Important while planning surgical management of ptosis
- LPA - <4mm - poor
  5 – 7mm – fair
  >8mm - good

Causes of Ptosis?

Congenital
Acquired

- Involutional
- Neurogenic
- Myogenic
- Mechanical

Involutional (Aponeurotic dehiscence))

- the most common type of ptosis
- Long term contact lens wear
- Features:
  - Good LPS action
  - High lid crease
  - No lid lag on down gaze
- Frontalis overaction (s/o visual axis obstruction by ptotic lid)

Neurogenic:

- Third Cranial nerve palsy

Note:

- Total ptosis
- Exotropia
- Hypotropia
- Mydriasis

Neurogenic:

- Horner’s syndrome

Note:

- subtle ptosis (sympathetic innervation to Muller’s & smooth muscle of lower lid affected)
- elevation of LL (apparent enophthalmos)
- miosis (sympathetic innervation to Pupil (dilators) affected)
**Myogenic:**
- Chronic Progressive External Ophthalmoplegia (CPEO)
- Myasthenia gravis [diurnal variation]
- Ptosis is one component of these syndromes

**Mechanical:**
- UL tumors
  - BCC, SCC, Sebaceous Cell Ca
  - Hemangioma
  - Lacrimal gland masses
- Lid edema as post-trauma/ surgery

**Congenital Ptosis**

Features:
- Fair to Poor LPS action
- Faint/ Absent lid crease
- Lid lag on down gaze
- +/- poor upgaze

NB. Risk of amblyopia

Ptosis
Must assess
- Pupils
- EOM, CN II
- Levator function
- Orbicularis power
- Bell’s reflex
- Evert UL

**Abnormal Upper Eyelid Position**

**Lid Retraction**

Causes:
- Congenital
- Acquired
  - Thyroid Eye Disease
  - Midbrain lesions
  - Parinaud’s syndrome
Abnormal Lower Eyelid Position

**Ectropion**
(Eyelid margin is away from the globe)

**Causes:**
- Involutional
- Cicatricial
- Paralytic (VII CN palsy)
- Mechanical

Prolonged ectropion leads to metaplastic changes in exposed conjunctiva

**Involutional**
Due to laxity of canthal tendons (structural support of lids at medial & lateral ends) with age

**Cicatricial**
e.g. traumatic scar at cheek pulling the lower lid down

**Paralytic (VII CN palsy)**
Facial nerve supplying Orbicularis Oculi is affected, as in Bell’s palsy, Parotid tumor excision, Acoustic Neuroma

**Mechanical**
Lower eyelid edema causing mechanical ectropion

Abnormal Lower Eyelid Position

**Entropion**
(Eyelid margin rolled in)

**Causes:**
- Involutional
- Trauma
- Cicatricial
Entropion leads to trichiasis (misdirection of lashes), which may cause punctate epitheliopathy of cornea to frank corneal ulcers.

Cicatricial
- Ocular Cicatricial Pemphigoid
- Stevens Johnson syndrome
- Trachoma (developing countries)
- Chemical injury

Contraction and thus, shortening of posterior lamella of the eyelid: which pulls and rolls in the eyelid margin along with eyelashes.

Common Eyelid Lesions

Benign Eyelid Lesions
- Chalazion
  - chronic, granulomatous inflammation of meibomian glands involving posterior eyelid lamellae
  - Treatment:
    - warm compresses/ antibiotic ointment, if recent onset
    - Incision & Curettage in resistant cases

- Stye
  - tender, acute inflammation of sebaceous glands of Zeiss or sweat glands of Moll at the base of eyelashes in anterior eyelid lamellae
  - Treatment:
    - warm compresses, antibiotic ointment
    - Expression of pus ± lash removal
    - Keep a close watch, as in severe cases, stye may worsen to orbital cellulitis

- Xanthelasmas
  - yellowish, sessile plaques
  - S/o hypercholesterolaemia
  - Treatment:
    - removal for cosmesis

Malignant Eyelid Lesions
- Basal cell cancer
  - Commonest lid malignancy (90%)
  - Risk factors:
    - Fair skin & UV exposure
  - Features:
    - Nodular/ulcerative with rolled edges
    - Loss of lashes
    - Telangiectatic blood vessels on surface
  - Treatment: Excision with wide margins ± Cryotherapy
• Squamous cell cancer
  - Fast growing skin cancer
  
  **Features:**
  - Nodular
  - Hyperkeratotic surface
  
  **Treatment:** Excision Biopsy with wide margins ± radiotherapy depending upon the depth of involvement

**Orbit**

**Orbit Examination**

- Exophthalmometer – axial/non-axial, look from above/below

- Optic nerve function
- Pupils

- Extra-ocular movements, CN V
- Lids – ptosis, retraction, masses, scleral show

- Palpate – masses, pulsatile
- Lymph nodes

- Auscultate ?Buits

**Common Orbital disorders**

**Pre-septal cellulitis**

Aetiology: Trauma, Insect bite, Stye

**Features:**
- Inflamed, oedematous lids
- Nil/ mild pain on eye movements
- Visual acuity good
- Intact Optic Nerve function

**Orbital Cellulitis**

Aetiology:
  - Sinusitis (commoner in children)
  - Trauma/ Tumor in adjoining sinuses

**Features:**
- Proptosis
- Inflamed pre-septal tissues
- Painful/ limited eye movements
- Drop in Visual acuity
- Compromised Optic Nerve function
Thyroid Eye Disease

- Proptosis
- Restrictive Myopathy
- Optic Neuropathy

- Lid signs:
  - lid lag, lid retraction
  - Lid swelling, lagophthalmos
- Ocular surface Inflammation
  - esp. over horizontal recti muscles
  - Dry eyes
  - Exposure keratopathy
  - Glaucoma

CT scan features in Thyroid eye disease

- Thick extra-ocular (recti) muscles
- Maximum diameter of globe beyond lateral orbital rim
- Tenting of optic nerve on right side (d)

Keep a close watch on:

- Corneal exposure – treat with lubricants/ taping eyelids shut
- Optic nerve functions due to its possible compression by enlarged extra-ocular muscles – might need urgent surgical intervention

Orbital Fracture

Classical Signs:

- Hypotropia
- Enophthalmos
- Restricted motility, esp. vertical gaze
- Infra-orbital anesthesia
Lacrima Drainage System & Common Disorders

Eyelid movement ➔ Lacrima Pump

Acute dacryocystitis:
- Complete block
- Stagnant tears in sac
- Recurrent infections

Where is the block? Syringe & Probe

Dacryocystorhinostomy (DCR)

Congenital Naso-lacrimal Duct Obstruction
- Symptoms start soon after birth
  - Watering ± discharge
  - Fluorescein dye disappearance delayed

Treatment Options in order of preference:
- Conservative [Lacrimal Massage ± Antibiotics]
- Probing & Syringing
- Intubation/ Balloon Dacryoplasty
- Dacryocystorhinostomy

Nearly 95% resolve conservatively by the end of one year of age
The Acute Red Eye
Dr Shenton Chew, Assoc. Professor Jennifer Craig, Professor Charles McGhee

What is a red eye?
• Dilation of superficial ocular vessels
  – Conjunctiva
  – Episclera
  – Sclera

Systematic Approach to Diagnosis
1. History
2. Vision
3. Discharge
4. Appearance
5. Pupils

1. History
• Past ocular disease / symptoms
• Decreased vision
• Pain & severity
• Photophobia
• Ocular discharge
• Associated systemic associations

2. Vision
• Snellen visual acuity
• Pinhole

3. Discharge
• Serous / watery
• Mucoid
• Purulent
• Mucopurulent

4. Appearance – view presentation

5. Pupils
• Miosis
• Mid-dilated
• Sluggish / no reaction to light
Causes:
- Conjunctivitis
- Subconjunctival haemorrhage
- Keratitis
- Episcleritis
- Scleritis
- Acute anterior uveitis
- Acute angle closure crisis
- Ocular trauma

Conjunctivitis
- Infective
  - Viral
  - Bacterial
  - Chlamydial
- Allergic
  - Seasonal (hayfever) / Perennial (dust mites)
  - Vernal
  - Atopic

History: Associated features
- Viral:
  - URTIs
  - Pre-auricular lymphadenopathy
- Allergic:
  - Atopy/hayfever
  - CL wear
- Chlamydial:
  - Urethritis (Reiters)

Vision: Generally normal unless there is
- Excessive lid swelling
- Excessive discharge
- Corneal involvement

Discharge
- Viral: Watery
- Allergic: Mucoid/watery
- Bacterial: Purulent
  - Chlamydial: Mucopurulent

Pupils: Normal
Conjunctivitis: Management

- Swab and isolate responsible organism
- Bacterial = Chloramphenicol
- Viral = Supportive Rx (compresses, lubricants)
- Chlamydia = Azithromycin/doxycycline

Keratitis

History:
- Painful (foreign body sensation)
- Photophobic
- Tearing
- Hx of CL wear/trauma

Vision: Decreased
- Especially if involves visual axis

Discharge: Watery, purulent
- (depends on cause)

Appearance:
- Circumcorneal injection
- Corneal infiltrate/hazy cornea
- Overlying epithelial defect

Pupils: Normal

Keratitis - Management
- Isolate responsible organism
  - Corneal scrape
  - Intensive treatment and close follow-up

Acute angle closure crisis
- in IOP due to obstruction of aqueous outflow by complete or partial closure of the angle by peripheral iris
- Incidence
  - 1/1000 in > 40 y.o.
- Female: Male
  - 4:1

Anatomical predisposition
- Short eye
- Narrow angle
- Large lens
- OLDER HYPERMETROPE
History:
- Intense ocular pain & headache
- Nausea & vomiting
- Photophobia
- Premonitory symptoms
- Hypermetrope

Vision: Very blurred
- Secondary to corneal oedema

Discharge: None

Appearance:
- Circumcorneal injection
- Cloudy cornea
- Optic nerve head swelling
  - If prolonged attack

Pupils: Mid-fixed, dilated
- Circumcorneal injection
- Cloudy cornea
- Optic nerve head swelling
  - If prolonged attack

Management:
- Reduce IOP (often starts > 50 mmHg)
  - Medical
    - Topical:
      - Alpha-agonist, Beta-blockers, Mitotics (Pilocarpine)
    - Systemic:
      - Carbonic anhydrase inhibitors (Diamox), Osmotics (Mannitol)
  - Surgical
    - Peripheral iridotomy
    - Clear lens extraction/trabeculectomy

Acute Anterior uveitis/iritis

Aetiology:
- Idiopathic
- Ankylosing spondylitis
- Reiter’s syndrome
- Juvenile arthritis
- Psoriatic arthropathy
- Sarcoidosis
History:
  - Moderate aching pain
  - Photophobia
  - Past history (esp if HLAB27)
  - Systemic symptoms

Vision: Blurred

Discharge: None

Appearance:
  - Circumcorneal injection
  - Clear cornea

Pupils: Miotic / sluggish response to light

Management:
  • Subdue inflammation
    - Topical corticosteroids (g. predforte)
  • Prevent posterior synechiae
    - Mydriatics (g. cyclopentolate)
    - Watch for elevated IOP
    - Topical ocular hypotensives (g. timolol)

Scleritis/Episceritis

<table>
<thead>
<tr>
<th>Scleritis</th>
<th>Episceritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Relatively uncommon</td>
<td>• Relatively common</td>
</tr>
<tr>
<td>• Severe boring pain</td>
<td>• Mild ocular discomfort</td>
</tr>
<tr>
<td>• Focal injection of deep scleral vessels</td>
<td>• Focal injection of episcleral vessels</td>
</tr>
<tr>
<td>• Associations</td>
<td>• Generally no associations</td>
</tr>
<tr>
<td>- Herpes Zoster Ophthalmicus</td>
<td>- Usually requires no treatment</td>
</tr>
<tr>
<td>- Severe Rheumatoid Arthritis</td>
<td>- g. lubricants/g. voltaren</td>
</tr>
<tr>
<td>• Can lead to blindness if untreated</td>
<td></td>
</tr>
<tr>
<td>- po. prednisone</td>
<td></td>
</tr>
</tbody>
</table>
Subconjunctival haemorrhage

- Unilateral localised sharply extravasated blood
  - (bleeding under conj)
- Associations
  - Severe coughing
  - Valsalva manoeuvre
  - Anticoagulants
  - Systemic hypertension

Ocular trauma

<table>
<thead>
<tr>
<th>Blunt ocular trauma</th>
<th>Sharp ocular trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Orbital fractures</td>
<td>Lid laceration</td>
</tr>
<tr>
<td>Contusion</td>
<td>Corneal abrasion/FB</td>
</tr>
<tr>
<td>Hyphaema</td>
<td>Penetrating injury/intraocular FB</td>
</tr>
<tr>
<td>- Rebleed 20%;</td>
<td></td>
</tr>
<tr>
<td>- Glaucoma 7%</td>
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</tr>
</tbody>
</table>

Conclusions:

- Systematic Approach
  - History
  - Vision
  - Discharge
  - Appearance
  - Pupils
- BE AWARE OF SIGHT THREATENING CONDITIONS!
Glucoma
Dr Justin Mora

Glucoma is an optic neuropathy with a specific pattern of axonal loss which may be associated with elevated intraocular pressure and a typical pattern of visual field loss

- A disease of the optic nerves
- Two principal types
  - **Open angle glucoma**
    - Primary – presumed angle predisposition
    - Secondary – cells, inflammation
  - **Closed angle glucoma**
    - Primary – narrow anterior chamber angle
    - Secondary – tumours, synechiae

**Open Angle Glaucoma:**
It affects 2-3% of people over 60
2nd leading cause of blindness in N.Z.
In N.Z. 95% of glucoma of this type
There are significant racial variations
Risk factors: FHx, myopia, HT

**Four Key Components to Glaucoma Assessment**
1. Intraocular Pressure
2. Angle Assessment
3. Optic Nerve
4. Visual Fields

**IOP Assessment**
- Goldman tonometry is the “gold standard”
- Applanates over 3.02 mm so tear meniscus pressure and corneal rigidity are balanced
- The inward pressure of the tonometer equals the IOP
- Will vary with corneal abnormalities
- “Normal IOP” is 6 - 22 mmHg
- 95% of normals fall within this range
- Ocular hypertension > glucoma
- 25-30% of glucoma in N.Z. is normal pressure glucoma
- Proportion varies markedly with race

**Students should understand the principles behind:**
Normal aqueous flow
Gonioscopy
Closed Angle Glaucoma
- Represents 5% of glaucoma in N.Z.
- Rapid onset with pain, redness, blurring and a mid-dilated pupil
- Caused by a rapid elevation of pressure inside the eye
- Treated with laser iridotomy

Optic nerve cupping
- A normal optic nerve has 1,000,000 axons
- Half can be lost before any visual loss occurs
- Visual loss starts in the periphery and affects the central vision last

Histological changes
Cup:disc Ratio
Optic nerve imaging
Visual Fields

Other causes for Visual Field loss -
Infantile Glaucoma
- Incidence: 1:10,000 (1:2500 - 1:20,000)
- Usually bilateral, males > females
- Usually sporadic: 10% inherited as AR with variable penetrance. 5% sib/child risk
- Onset: 40% in utero, 50% <1 yr, 10% > 1 yr
- Hazy corneas
- Tearing
- Photophobia
- Buphthalmos

Glaucoma Treatment
- None
- Medications – local, systemic
- Laser - laser trabeculoplasty, iridotomy
- Surgery:
  - Pediatric surgery
  - Drainage surgery – trabeculectomy, tubes
  - Cyclodestruction
**Glaucoma Medications**

*Increase Aqueous Outflow Through Trabecular Meshwork*
- Miotics: Pilocarpine

*Increase Uveoscleral Aqueous Outflow*
- Prostaglandin Analogue: Xalatan, Travatan, Lumigan

*Reduce Aqueous Production*
- a) B-Blockers: Timoptic, Betagan, Betoptic
- b) CAI Inhibitors: Diamox, Trusopt, Azopt
- c) Alpha Adrenergic Agonists: Iopidine, Alphagan

*Reduce Vitreous Volume by Osmosis*
- Osmotic Agents: Mannitol, Glycerol

**Systemic side effects of Beta Blockers** -
- Bradycardia, heart block, asystole
- Heart failure (may interact with others)
- Shortness of breath and bronchospasm
- Apnoea in infants
- CNS - confusion, delerium, depression
- Reduced exercise tolerance
- Impotence and loss of libido
- Masks symptoms of hypoglycaemia

**Ophthalmic Medications – Questionnaire**
- How many drops should be used per dose?
- Name 5 ways to reduce the systemic effect of drops
- Minimum time between instillation of two different drops to prevent major washout?
- What is the most likely reason a glaucoma drop doesn’t produce a good response?
- Name an eyedrop that is safe in pregnancy and lactation

**Marijuana and Glaucoma**
- Advocated initially in the 70's
- Limited options for glaucoma treatment: miotics, epinephrine, acetozolamide
- Various studies have produced data from a total of 300 volunteer subjects
- Largest single study group was 40 people
- Inhaled marijuana lowers IOP in 60-65 %
- One smoke reduces IOP by 25 %
- Impressive results but......

- Duration of effect only 3-4 hours
- For a consistent response one would have to smoke:
  - 8-9 / day
  - 3000 / year

(JAMA 1980; 244(22): 2498)

No green light for grass in glaucoma!

Diagnose with Care
- Patients fear glaucoma
- Treatment is for a lifetime
- Treatment can carry significant morbidity, even mortality
- If the signs are soft and the optic nerve still quite healthy, then watch for progression before starting treatment

Treat Aggressively -
- The more nerve damage the lower the target pressure
- Trial a drop and alter if poorly effective
- If some effect but not enough add another drop
- If control with drops is poor then surgery

Glaucoma Surgery to Improve Aqueous Drainage
- Laser Surgery
  - Laser Trabeculoplasty, Laser Iridotomy
- Paediatric Surgery
  - Goniotomy, Trabeculotomy
- Filtration Surgery
  - Trabeculectomy
- Aqueous Shunt Surgery
  - Molteno Implants

Glaucoma Surgery to Reduce Aqueous Production
- Cyclodestruction
  - Cyclocryotherapy
  - External Laser Cyclophotocoagulation
  - Endoscopic Laser Cyclophotocoagulation
Paediatric Ophthalmology and Strabismus
Dr Justin Mora

Key issues in Paediatric Ophthalmology
• Assessing vision in children
• Assessing strabismus
• Types of strabismus
• Management of strabismus

Nasolacrimal Duct Obstruction
• Common, congenital, failure to canalize
• Recurrent tearing and infections
• 95 % resolve by 12/12. If not, unlikely to
• Surgery to probe duct and open

Leukocoria (White Pupil)
• Any opacity in the visual axis
• Corneal e.g.: glaucoma, metabolic, trauma
• Aqueous and vitreous e.g.: uveitis
• Lens e.g.: cataract
• Retinal e.g.: retinoblastoma, retinopathy of prematurity, retinal inflammatory disease

Retinoblastoma
• Malignant. 1 in 20,000
• Mutation of tumour suppressor gene at 13q14.1
• 65 % sporadic, 25 % heritable, 10 % inherited with FHx
• 1/3 bilateral
• Rx gives high survival
• Risk of other malignancies with heritable forms

Congenital Cataract
• Occurs in about 1 in 2000
• 65% sporadic
  20% inherited
  15% systemic or ocular problems e.g.: Down’s, Peter’s
• Detected by absent red reflex
• Surgery ideally performed by age 4-6 weeks
• Vision corrected with contact lenses
• Intraocular lens implants possible after age 6 months
**Congenital Glaucoma**
- 1 in 10,000. Congenitally abnormal drainage angle
- May be associated with systemic conditions
- Photophobia, tearing, hazy corneas and buphthalmos (enlargement of the eye)
- The management is generally surgical

**Strabismus = squint = misaligned eyes**
- Esotropia = ET = convergent squint
- Exotropia = XT = divergent squint
- Hypertropia = Eye is deviated up
- Hypotropia = Eye is deviated down

**Normal Visual Development**
- At birth: VA = 3/60, no fixation, variable XT
- VA = 6/12 by 6-12 months
- Infants usually hyperopic
- Eyes should be straight by 2 months with good fixation
- **Any strabismus at 3 months needs assessment**

**Measuring Visual Acuity**
- Infant: fix and follow, preferential looking tests, asymmetrical objection to occlusion, fixation preference, optokinetic nystagmus
- 2 yrs: Kay’s Pictures
- 2 ½ yrs: Tumbling E’s
- 3 yrs: Sheridan-Gardner
- 4-5 yrs: Smelled Acuity

**Amblyopia (Dull Eye)**
- Poorer development of the visual cortex due to a blurred visual input. Brain problem (not an eye problem)
- The younger the child the greater the risk but also a greater the likelihood of successful treatment
- System fixed and no treatment possible by 7-8 years
Causes of Amblyopia

- Refractive
  - anisometropia > astigmatism > hyperopia > myopia
- Strabismus - treating amblyopia prior to surgery improves stability of outcome
- Stimulus deprivation e.g.: cataract, overpatching

Amblyopia Treatment

- Patching: Good eye is occluded (patched)
  - part-time vs full-time occlusion
  - full time max 1 week per year of age
  - recent studies suggest 2 hrs = 6 hrs per day
  - compliance is the key
- Penalization: good eye is blurred with Atropine. Beware of cycloplegic toxicity: facial flushing, rapid heart rate, confusion, irritability, seizures

Management Issues

- Cycloplegic refraction is vital
  - allow 40 mins for cycloplegia
- Strabismus is assessed with prism cover tests in 9 cardinal gaze positions depending on concerns
- Motility is assessed, versions and ductions
- The media and fundi are examined

Assessing Strabismus

- Corneal Light Reflex Test
  - Reflexes should be symmetrical just nasal to visual axis
  - Reflex displaced temporally = Esotropia
  - Reflex displaced nasally = Exotropia

- Cover Test
  - cover straight eye
  - if other eye moves it was deviated
  - if it moves in = exotropia / divergence
  - if it moves out = esotropia / convergence
Prism Cover Testing
- Allows angle of deviation to be measured
- Cover test performed with prism over deviating eye
- Prism adjusted until any movement is negated
- Performed at near and distance and in different gaze positions
- Tables and experience used to calculate amount of surgery for deviation measured

Pseudoesotropia
- Broad epicanthic folds
- Medial sclera is buried with lateral gaze so the eyes look esotropic / convergent
- Corneal light reflex and cover test confirms straight
- The only “Strabismus” a child will “grow out of”

Infantile Esotropia
- Onset from birth to 2 months of age
- Due to poor fusion
- Usually large angle, other motility issues: IOOA, DVD, latent nystagmus
- Need to treat amblyopia before surgery
- Surgery for fusion (stability) and 3D
- Ideal time to operate is 6 - 12 months
- Results poor if operate > 2 years
- 50 % require further surgery

Refractive Esotropia
- Onset 18 mths to 5 years
- Due to hyperopia and accommodative response stimulating convergence
- Many straighten with glasses alone, if given full hyperopic correction
- Some with residual ET also require surgery

Intermittent Exotropia
- Onset 2 - 5 years
- Usually worse at distance
- May close eye in bright light
- 60% progress to constant XT, 35% stable, 15% improve
- Surgery to preserve depth perception or for cosmesis
- Control & proportion of time XT important
Superior Oblique Palsy
- Often congenital, may break down later in life. May be acquired. e.g.: trauma
- SO underaction, IO overaction, ipsilateral hypertropia worse on contralateral gaze and ipsilateral tilt
- Surgery often IO weakening or SO tuck

Principles of Strabismus Surgery
- Muscles can be
  - weakened (recession, myotomy, myectomy)
  - strengthened (resection, tuck)
  - repositioned (transposition, Faden)
- Surgery on paralyzed muscles is poorly effective
- Amount of surgery depends on size of squint
Dry Eye and Sjögren’s Syndrome
Associate Professor Jennifer P. Craig
PhD MCOptom FAAO FBCLA

The Tear Film
The tear film, which bathes the ocular surface, has a complex structure and composition and has a number of important functions:
- Optical
- Mechanical
- Nutritional
- Defensive

Lacrimal functional unit
The lacrimal functional unit comprises the cornea, conjunctiva, eyelids, lacrimal gland, accessory glands and the connecting neural reflexes. Dysfunction of any component of the unit can alter the tear film quantity or quality and result in signs and symptoms of dry eye.

Dry Eye Definition
“Dry eye is a multifactorial disease of the tears and ocular surface that results in symptoms of discomfort, visual disturbance, and tear film instability with potential damage to the ocular surface. It is accompanied by increased osmolarity of the tear film and inflammation of the ocular surface”
[2007 International Dry Eye Workshop (DEWS)]

Dry Eye
- Symptoms: dryness, grittiness, irritation, burning, ‘feeling of sand in the eye’
- Prevalence: between 8 and 13% of population affected. More common with increasing age and most common in post-menopausal women
- Environmental conditions and contact lens wear common exacerbating factors

Sjögren’s Syndrome
- Chronic, systemic, inflammatory, auto-immune disorder characterised by lymphocytic infiltration of the exocrine organs
- Affects 0.1 - 4% of the population, no race predilection
- More common in females; ratio of 9:1
- Onset typically in 30’s or 40’s
- Characteristically presents with sicca symptoms:
  - Xerophthalmia (dry eye)
  - Xerostomia (dry mouth)
  - Parotid gland enlargement
  - Extraglandular features e.g. arthralgia, RA, Raynaud’s phenomenon, lymphoma
- Primary SS occurs in the absence of an underlying rheumatic disorder
  1° SS = Dry Eye + Dry Mouth
- Secondary SS is associated with an underlying rheumatic disease, such as SLE, RA, or scleroderma.
  2° SS = Dry Eye + Dry Mouth + Connective Tissue Disease
- Treatment typically centres around symptom management, but patients require monitoring for potential lymphoma development
- Morbidity is mainly associated with decreased exocrine organ function, and mortality with associated conditions such as SLE /RA.
- Patients with primary SS who do not develop a lymphoproliferative disorder have normal life expectancy

Pathophysiology of Sjögren’s Syndrome
- Not fully understood
- Believed that environmental or endogenous antigens can trigger a self-perpetuating inflammatory response in susceptible individuals
- Genetic associations
- Sex hormones believed to play a role
- Viruses may be responsible for triggering onset (e.g. HIV)

‘Vicious circle’
- Structural and functional changes in the lacrimal (and salivary) glands, result in reduced aqueous production and increased tear osmolarity. Tear hyperosmolarity, as a proinflammatory stimulus, induces an inflammatory cascade on the ocular surface causing an immune response in the ocular surface epithelium, and local cytokine and metalloproteinase production. The end result is damage to an epithelium, already vulnerable from poor tear film protection, and evidenced as epithelial erosions, surface irregularity and impaired vision.

Clinical findings
- History of ocular sicca symptoms (n.b. not exclusive to SS)
- History of oral sicca symptoms (eating / speaking difficulties)
- Dryness of other mucosal surfaces
- Parotitis / visibly enlarged parotid glands
- Extraglandular involvement

Symptoms related to extraglandular involvement
- Cutaneous symptoms (including dryness, pruritus, Raynaud’s)
- Pulmonary symptoms (e.g. xerotrachea leading to dry cough or recurrent bronchitis)
- GI symptoms due to pharyngeal/oesophageal dryness (causing difficulty swallowing, reflux)
- Cardiac symptoms (e.g. pericarditis)
- Neurological symptoms (e.g. peripheral neuropathy, distal paresthesia, trigeminal / facial nerve palsies)
- Renal symptoms (secondary to interstitial nephritis)
- Debilitating fatigue
Ocular examination (requires Ophthalmology referral)
- Ocular redness (conjunctival hyperaemia, limbal redness)
- Dullness of the corneal reflex (due to surface irregularity)
- Blepharitis or meibomian gland dysfunction (eyelid disease) can also exist, affecting the tear lipid layer. This requires treatment with lid hygiene and warm compresses.
- Poor tear film stability (tear break up in <10 sec abnormal)
- Mucous strands (filamentary keratitis)
- Reduced tear production (reflex tearing) indicating lacrimal gland insufficiency (Schirmer test)
- Ocular surface staining
  - Rose bengal or lissamine green will stain primarily dead/devitalised cells (and cells without mucous cover)
  - Fluorescein will highlight areas of epithelial cell loss

Oral examination (ensure regular dental care)
- Visibly reduced sublingual salivary pool
- Tongue may stick to tongue depressor
- Frequent dental caries.
- Periodontal disease / tooth loss
- Tendency to develop oral candidiasis.
- ENT exam for bilateral parotid gland enlargement
- Sialometry / sialochemistry

Joint examination
- Arthritis may be a component of SS
- Symmetric, polyarticular, inflammatory arthritis suggests underlying RA or a connective-tissue disease such as SLE or scleroderma.
- One third of patients with RA have Sjögren’s syndrome.

Differential diagnoses
- Ig-related Amyloidosis
- Bulimia
- GVHD
- Pancreatitis
- Polymyositis
- RA
- Salivary Gland Tumors
- Sarcoidosis
- Scleroderma
Work-up

Immunological work-up includes:
- RF — positive in most patients with SS
- ANA — typically present in patients with SS
- Anti-SSA/Ro — found in ≈ 50% of patients (75% with 1° SS and 15% with 2° SS
- Anti-SSB/La — present in 40-50% of patients with 1° SS

n.b. titers of these antibodies do not reflect disease activity

Minor salivary gland biopsy
- Biopsy of the minor salivary glands of the lower lip is the single most useful test to confirm the diagnosis of SS.
- A 1.5 to 2cm incision of normal-appearing mucosa allows for the harvesting of 5 or more salivary gland lobules
- Histopathological findings include focal lymphoid infiltration of minor salivary glands.

Classification Criteria
American-European Consensus criteria for diagnosis of 1° SS: 4 of 6 positive responses to criteria below (must include no. 5 or 6)
1. Ocular symptoms (dry eye)
2. Oral symptoms (dry mouth)
3. Ocular signs (abnormal Schirmer without anaesthesia; < 5mm in 5 min / ocular surface staining)
4. Oral signs (abnormal sialometry; unstimulated salivary flow <1.5 mL in 15 min)
5. Positive minor salivary gland biopsy
6. Positive anti–SSA or anti–SSB antibody results

- 2° SS is diagnosed in presence of:
  - a connective tissue disease
  - symptoms of oral or ocular dryness, plus
  - criterion 3, 4 or 5 (ocular/oral signs or positive biopsy)

- For 1° SS, these criteria yield a sensitivity of 97.2% and a specificity of 48.6%
- For 2° SS, the criteria yield a sensitivity of 64.7% and specificity of 97.2%

Treatment of dry eye: Level 1
- Level 1 (Mild symptoms, no corneal signs)
  - Artificial tear supplements
  - tailor viscosity to symptom severity
    - if instilled > 4 times per day, use non-preserved drops
  - modify environmental conditions
  - increase relative humidity
  - avoid windy conditions
  - Avoid anticholinergic / antihistamine medications
Treatment of dry eye: Level 2
- Level 2 (Moderate-severe symptoms with tear film / visual signs, or mild ocular surface staining)
  - Use Level 1 treatments plus:
    - Unpreserved tears or gels ± ointments at night-time
    - Topical steroids (restricted course due to side effects risk)
    - Cyclosporine A (Restasis (US) / local pharmacy preparation)
    - Nutritional supplements (EFAs)

Treatment of dry eye: Level 3
- Level 3 (Severe symptoms with marked corneal changes or filamentary keratitis)
  - Use Level 2 treatments plus:
    - Tetracyclines (low dose, long course)
    - Autologous / allogeneic serum tears
    - Punctal occlusion (to retain tears)
    - Acetylcysteine to break down mucous strands

Treatment of dry eye: Level 4
- Level 4 (Extremely severe symptoms with altered lifestyle, or severe corneal staining, erosions, or conjunctival scarring)
  - Use Level 3 treatments plus:
    - Systemic anti-inflammatory therapy including acetylcysteine
    - Topical Vitamin A
    - Cautery for permanent punctal occlusion
    - Moisture chamber glasses

Treatment of dry mouth
- Sip water frequently
- Sugar-free lemon drops to stimulate saliva
- Artificial saliva (variable patient tolerance)
- Humidifiers for symptomatic relief
- Regular dental visits
  - fluoride treatment / special toothpaste
- Topical anti-fungals as required for candidiasis

Treatment of joint pain
- Arthralgias
  - NSAIDs
- Hydroxycholoquine
- Rheumatoid arthritis
  - Likely to require other disease-modifying medications (under direction of rheumatologist)

n.b. Cyclophosphamide used to treat serious systemic manifestations of SS has been associated with increased risk of lymphoma development
Multidisciplinary care of SS
- Physician (provide overall perspective)
- Rheumatologist (management of connective tissue disease)
- Ophthalmologist (management of ocular surface manifestations)
- ENT specialist (minor salivary gland / parotid biopsies)
- Dentist (for oral prophylaxis and treatment)
± renal physician, chest physician, haematologist, oncologist, as necessary

Common SS medications
- Cholinergic parasympathomimetics (muscarinic agonists)
  (e.g. oral pilocarpine, cevimeline (US))
- Artificial tears
- Artificial saliva agents
- Disease modifying agents (e.g. hydroxycholoquine)
- Immunosuppressants (e.g. cyclophosphamide)
- Immunomodulators (e.g. cyclosporin A)

Complications of SS
- Typically SS patients undergo outpatient reviews 3 – 6 monthly to check for complications
  - Emergence of related disorders (e.g. SLE)
  - Parotid gland infections
  - Parotid tumours
  - Pseudolymphomas / lymphomas
    (18x increased risk of NHL in SS)

Take home message
- SS has a generally good prognosis
- Prognosis more closely aligned with any associated disorders (e.g. SLE)
- Patient education and support is essential
  - Do not underestimate the impact SS, as a chronic condition, can have on daily life
  - Teach patient avoidance tactics and self-care strategies to help manage the symptoms associated with their dry mucosal surfaces
  - Support groups: Sjogren’s Syndrome Society of NZ
    www.sjogrensnewzealand.co.nz
Ophthalmology Quiz

Please choose one answer for each question. There is only one correct answer to each question. To mark yourself add 1 point for every right answer, subtract one point for every wrong answer and give no points if you did not answer the question.

17-20 points - excellent
11-16 points - good
Less than 10 points – further study required!

Question 1
In regard to neoplasms of the eyelid (basal cell carcinoma, squamous cell carcinoma, melanoma). The most likely aetiological factor is:

A) Use of mascara  
B) A hereditary condition  
C) Recurrent herpes infection  
D) Sunlight exposure  
E) Recurrent local trauma

Question 2
A 60 year old male has noted painless decreasing vision in the right eye for the past year. Funduscopic examination reveals a darkly pigmented mass. These findings most strongly suggest a diagnosis of:

A) Diabetic retinopathy  
B) Retinoblastoma  
C) Melanoma  
D) Tay-Sachs disease  
E) Cytomegalovirus retinopathy

Question 3
A 56 year old female has noted increasingly frequent headaches for the past year. She recently had her vision checked, and her intraocular pressure was found to be 34mmHg. The most significant result of this condition, if not treated, is:

A) Cataract formation  
B) Conjunctivitis  
C) Hypertensive retinopathy  
D) Optic neuropathy  
E) Strabismus
Question 4
A 42 year old male notes a progressively enlarging nodule on his right upper eyelid over the past month. This nodule is firm and painless. There is no ulceration. The conjunctiva and the cornea appear normal. Such a lesion can be characterized by all of the following findings EXCEPT one:

A) Resembles a sebaceous carcinoma on gross examination
B) Prone to recurrence
C) Infection of eccrine sweat glands
D) Exhibits granulomatous inflammation
E) Related to sun exposure

Question 5
A 3-year-old child, who had been born prematurely at 30 weeks gestation and then developed hyaline membrane disease at birth, is found to be visually impaired. Funduscopic examination reveals retinal detachment on the left. The most likely pathogenesis of this condition is:

A) Retinal damage by oxygen toxicity
B) Hereditary hexosaminidase A deficiency
C) Germinal matrix hemorrhage at birth
D) Neonatal chlamydial infection
E) Absence of tumor suppressor gene on chromosome 13

Question 6
A 70 year old female has noted the appearance of a "spot" discoloring the sclera of her right eye. Her physician notes a 0.3 cm raised yellowish-white lesion near the limbus. This lesion is LEAST likely to:

A) Appear more often in older persons
B) Decrease visual acuity
C) Increase in size very slowly over time
D) Show elastosis of collagen histologically
E) Have an association with sunlight exposure

Question 7
A 34-year-old female with a recent onset of right eye pain and irritation is noted to have an irregular dendritic corneal ulcer on slit lamp examination. This lesion is probably due to infection with:

A) Candida albicans
B) Pseudomonas aeruginosa
C) Hemophilus aegypticus
D) Acanthamoeba
E) Herpes simplex virus
Question 8
A 17 year old female is 188 cm tall. She has long, thin fingers. Examination of her eyes reveals dislocated lenses. These findings are features of:

A) Marfan's syndrome  
B) Osteogenesis imperfecta  
C) Narrow angle glaucoma  
D) Aging  
E) Infection

Question 9
A 3 year old boy has lost vision in the right eye. Other children in his family have had a similar problem, with many losing vision in both eyes. A presumptive diagnosis of an intraocular tumor is made. Which of the following statements is LEAST appropriate for this condition:

A) Leukocoria is often noted on physical examination  
B) The patient may have inherited a faulty tumor suppressor gene  
C) The tumor is composed of small cells that form rosettes  
D) Dystrophic calcification is often seen histologically  
E) The tumor often spreads via scleral vascular channels

Question 10
A 41 year old female with a history of intravenous drug abuse presents with a 1 day history of severe headache and high fever. She has marked nuchal rigidity. You suspect that her intracranial cerebrospinal fluid pressure is increased. Which of the following funduscopic examination findings is most likely to be present:

A) Deepening of the optic cup with excavation  
B) Arteriolar narrowing  
C) Neovascularization of the optic nerve  
D) Elevation and swelling of the optic nerve head  
E) Flame-shaped haemorrhages

Question 11
An obese 50 year old male has a serum glucose of 9.9mmol/L and a hemoglobin A1C that is increased. He has decreased visual acuity. A funduscopic examination reveals a proliferative retinopathy. Which of the following findings is most characteristic of this process:

A) Capillary microaneurysms  
B) Dot and blot haemorrhages  
C) Neovascularization  
D) Flame-shaped hemorrhages  
E) Hard exudates
**Question 12**
What is the most common cause of amblyopia?

A) Squint  
B) Trauma  
C) Uveitis  
D) Conjunctivitis  
E) Nuclear cataract

**Question 13**
A firm 0.4 cm nodule is noted on examination of the upper eyelid of a 41 year old male. Biopsy of this lesion demonstrates inflammation consisting of lymphocytes admixed with epithelioid cells and giant cells. The diagnosis is most likely to be a(an):

A) Pinguecula  
B) Hordeolum (stye)  
C) Pterygium  
D) Chalazion  
E) Actinic keratosis

**Question 14**
A 62 year old female has decreasing vision associated with deepening of the optic cup with excavation, as seen on funduscopic examination (atrophy of the optic nerve fibers). The disease that led to these findings could have been detected by screening for:

A) Elevated serum glucose  
B) Hypercholesterolemia  
C) Homocystinuria  
D) Increased intraocular pressure  
E) Elevated blood pressure

**Question 15**
An uncommon, but sight-threatening and difficult to treat, complication associated with poor contact lens hygiene affects the cornea of a 30 year old male. His eye has been painful and he has been photophobic for more than a month. The most likely diagnosis is:

A) Band keratopathy  
B) Acanthamoeba infection  
C) Pterygium  
D) Staphylococcus aureus infection  
E) Herpes simplex infection
Question 16
A series of tissue samples for frozen section are sent to surgical pathology to make sure a lid lesion is completely excised. These samples are taken from the margin of a lesion of the medial lower eyelid of a 72 year old male. The lesion has been slowly enlarging for several years, and is now 0.4 cm in diameter, with slightly raised, pearly edges and an ulcerated center.

What is the most likely diagnosis?

A) Basal cell carcinoma
B) Glioma
C) Chalazion
D) Squamous cell carcinoma
E) Malignant melanoma

Question 17
Funduscopic examination of the right eye in a 50 year old male with 6/6 vision reveals a pigmented uveal mass. What is the most likely diagnosis?

A) Basal cell carcinoma
B) Glioma
C) Retinoblastoma
D) Squamous cell carcinoma
E) Malignant melanoma

Question 18
A 62 year old obese male is reviewed by a physician who notes decreased visual acuity and a non-healing ulcer on his left foot. Fasting blood glucose is 9.5mmol/L on two occasions. He is referred to an ophthalmologist, who on funduscopic examination observes a background retinopathy characterized by:

A) Neovascularization of the disc
B) Vitreous hemorrhage
C) Fibrous proliferation
D) Dot and blot hemorrhage
E) Papilloedema
Question 19
A 78 year old male is bothered by chronic grittiness of the left eye. He has recurrent local redness in the eye. You find a slightly raised, pink, vascular, relatively smooth 0.3 x 0.6 cm lesion that encroaches from the conjunctiva onto the corneal surface of his left eye. The fundus examination is unremarkable. Which of the following conditions is most likely to explain these findings:

A) Hordeolum
B) Primary acquired melanosis
C) Pterygium
D) Herpes simplex keratitis
E) Pinguecula

Question 20
Which of the following eye drops has NO cycloplegic effect?

A) Atropine
B) Cyclopentolate
C) Midriacyl
D) Homatropine
E) Phenylephrine
**Ophthalmology Quiz**

*(Answers)*

1. (D) Increasing sun exposure is strongly associated with development of malignant skin tumors.

2. (C) Melanomas produce melanin, which is pigmented (black), and are also a mass lesion.

3. (D) The increased intraocular pressure leads to optic nerve head atrophy.

4. (E) A chalazion involves the meibomian glands of the eyelid, can resemble sebaceous carcinoma and is a granulomatous inflammation that can recur. However, it is not associated with sunlight exposure.

5. (A) The child has retinopathy of prematurity, a complication of high dose oxygen therapy following birth, typically administered because of hyaline membrane disease.

6. (B) Pingueculae are located on the conjunctiva away from the cornea.

7. (E) HSV classically produces a dendritiform corneal lesion and would be the most likely causative factor.

8. (A) Marfan's syndrome is a connective tissue disorder associated with arachnodactyly and subluxation/dislocation of the crystalline lens.

9. (E) Retinoblastoma, is the most common childhood intraocular tumor. A retinoblastoma will invade into the optic nerve, whereas melanomas in adults tend to spread via scleral vascular channels. Histologically “rosettes” of cells are a pathognomonic feature.

10. (D) This patient has findings suggestive of acute bacterial meningitis that can lead to cerebral edema and increased intracranial pressure manifested as papilledema (disc swelling) on funduscopic examination.

11. (C) This is diabetic retinopathy. In proliferative retinopathy, fine new vessels grow upon the retina and into the vitreous and bleed easily, such that vitreal hemorrhages can
obscure vision and contraction of fibrovascular and glial tissue can lead to traction retinal detachment. The other features mentioned may also be seen but new vessels are the defining element of proliferative retinopathy.

12. (A) A squinting eye (Strabismus) is the most common cause of amblyopia in children. Treatment includes a full refractive assessment, provision of appropriate spectacles correction, patching of the better (non-amblyopic) eye, and surgery where necessary.

13. (D) This lesion is due to chronic inflammation of the meibomian glands and the histology suggests the key features of a chalazion.

14. (D) She has optic nerve atrophy from glaucoma, which is most often idiopathic primary open angle type.

15. (B) This protozoal infection is uncommon but responds poorly to antimicrobial therapy and is classically associated with poor contact lens care and swimming in contact lenses or exposing them to tap water.

16. (A) Basal cell carcinomas result from excessive sunlight exposure and are the most common tumour around the eyelids.

17. (E) The melanoma is typically pigmented (with melanin) and is the most common primary intraocular tumour of adulthood.

18. (D) This subject has diabetes mellitus with a diabetic background retinopathy that can be characterized by dot and blot hemorrhages, capillary microaneurysms, flame-shaped hemorrhages and hard exudates but not by cotton-wool spots or new vessel formation which are features of more advanced disease.

19. (C) A pterygium is similar to a pinguecula, but it encroaches onto the corneal surface. It is also related to aging and sun exposure. It is not premalignant.

20. (E) Phenylephrine is α mimetic. There is no sympathetic innervation of the ciliary muscle so it produces mild pupillary dilatation without cycloplegia – i.e. it does not cause loss of accommodation.
Recommended Reading

4th year Handout – with particular reference to material included in the Appendix

Section
Vision Loss
Clinical methods
Visual Assessment Clinic (VAC) notes

Recommended reading as set in the 5th Year teaching program (Auckland), from the recommended textbook, “Ophthalmology. An illustrated colour text” by Batterbury and Bowling

ARMD (p58-9)

Conjunctiva (p34-7)
Corneal inflammation (Keratitis) (p38-9)
Corneal Dystrophies (p40-1)
Correcting Refractive Errors (p84-5)

Epidemiology and functional implications of visual handicap (p90-1)
Eyelids (p26-9)

Floaters (p110-1)

Glaucoma (p42-5)

Inherited Disease (p62-3)
Iritis / Uveitis (p46-9)
Lasers in Ophthalmology (p94-5)

Orbital Disease (p32-3)
Ocular Fundus – Retinal Vascular Disease (p52-57)
Ocular pharmacology (p22-3),
Ophthalmic Examination (p12),
Ophthalmic surgery (p86),
Optic disc (p62),

Pupils (p70),

Retinal dystrophies (p58),

Special Investigations (p14),
Strabismus childhood (p66, 100), Strabismus adult (p68),

Tear secretion and drainage (p28),
The Lens (p48),
Trauma (p72, 74, 98)
Clinical Scenarios – Links in the MBChB Portal (http://mbchb.auckland.ac.nz)

Cataract Surgery Past Present Future
Infant with an altered light reflex (Oph12)
Gradual deterioration in visual acuity over time (Oph07)
Type 2 diabetes mellitus (Endo11)
6 week check (Paed26)
Family with a genetic disorder (MG02)

Dry Eye & Sjogren's Syndrome
Acute or chronic red eye (Oph01)
Swollen and tender joints (Rh02)
SLE / fatigue / pain and swelling in the hands (Rh03)

Eye Accident & Emergency
Acute or chronic red eye (Oph01)
Acute trauma to the eye (Oph02)
Child with red swelling around one eye (Oph10)
Diplopia (Oph06)
Pupil abnormality (Oph08)
Sudden loss of vision and headache (Oph05)
Sudden painless loss of vision (Oph04)
Headache, morning stiffness and shoulder pain (Rh06)

Eye Banking & Corneal Transplantation in New Zealand
Gradual deterioration in visual acuity over time (Oph07)

Glaucoma
Gradual deterioration in visual acuity over time (Oph07)
Sudden loss of vision and headache (Oph05)
Watery eye in an infant (Oph03)

Ocular Surface Inflammation and Allergy
Watery eye in an infant (Oph03)
Itching child (Derm01)
Facial swelling and itchy rash (Derm04)

Oculoplastics overview
Child with red swelling around one eye (Oph10)
Diplopia (Oph06)
Gradual deterioration in visual acuity over time (Oph07)
Watery eye in an infant (Oph03)
Skin tumours (Derm03)

Ophthalmoscopy
Annual eye screen for patient with diabetes (Oph11)
Infant with an altered light reflex (Oph12)
Sudden loss of vision and headache (Oph05)
Sudden painless loss of vision (Oph04)
Sudden onset severe headache (ED07)
Child or adolescent with a headache (Paed10)
Space occupying lesion / progressive unilateral weakness (N11)
Head trauma (ED09)
Paediatric Ophthalmology and Strabismus
Diplopia (Oph06)
Infant with an altered light reflex (Oph12)
Infant with strabismus (Oph09)
Pupil abnormality (Oph08)
Watery eye in an infant (Oph03)

Pupil Abnormalities
Pupil abnormality (Oph08)
Diplopia (Oph06)
Infant with strabismus (Oph09)
Sudden loss of vision and headache (Oph05)
Altered level of consciousness in an adult (N04)

Signs of Retinal Diseases
Annual eye screen for patient with diabetes (Oph11)
Gradual deterioration in visual acuity over time (Oph07)
Sudden painless loss of vision (Oph04)

The Acute Red Eye
Acute or chronic red eye (Oph01)
Acute trauma to the eye (Oph02)
Child with red swelling around one eye (Oph10)

Uveitis & Floaters
Pupil abnormality (Oph08)
Inflammatory low back pain (Rh07)
Reactive arthritis (Rh01)
Swollen and tender joints (Rh02)
Section 5. Contact Details

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