Mimickers of glaucoma

Jack Phu[1,2,3,4]

Honorary Lecturer, School of Optometry and Vision Science, UNSW
BOptom (Hons), BSc, MPH, PhD, FAAO, Diplomate (Glaucoma)

WAVE 2025

[1] School of Optometry and Vision Science, University of New South Wales, Australia
[2] Centre for Eye Health, University of New South Wales, Australia
[3] School of Medicine and Health, University of Sydney, Australia
[4] Concord Clinical School
jack.phu@unsw.edu.au

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Disclosures

- Research conducted in our laboratory is supported by National Health and Medical Research Council Ideas Grants
- No financial or commercial disclosures
- Centre for Eye Health is supported by Guide Dogs NSW/ACT

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Learning objectives

By the end of the talk, you will be able to:

- Understand the epidemiology of glaucoma and how it relates to the differential diagnosis process
- Develop a systematic approach to interpreting clinical information for differential diagnosis
- Apply longitudinal data to assist in the differential diagnosis process with a focus on glaucoma-specific progression indices

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The most important point in this talk:

Glaucoma is a diagnosis of exclusion!

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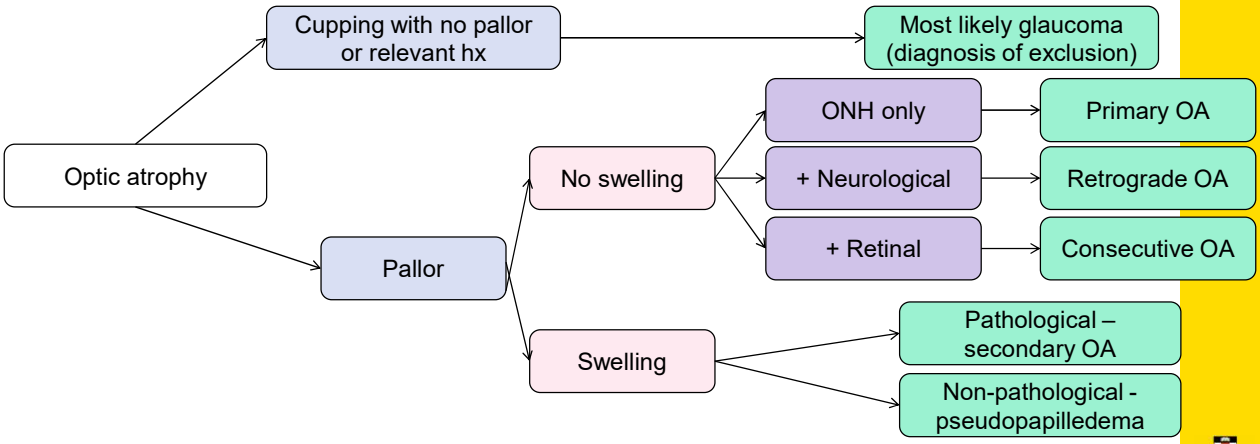
Types of optic atrophy



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PART 1: Primary optic atrophy

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Primary optic atrophy

- Optic atrophy **without swelling** preceding the atrophy
- Dx include:
  - Compressive
  - Retrobulbar optic neuritis (*)
  - ONH drusen
  - Toxic/nutritional
  - Traumatic
  - Hereditary optic neuropathy (*), dx of exclusion

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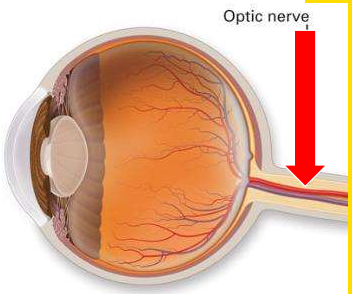
Compressive optic atrophy

- Pathophysiology: **direct compression** (ischaemic; mechanical compression) by extrinsic* lesion
  - Optic nerve glioma is a rare intrinsic cause; in children rhabdomyosarcoma
- Vulnerable sites
  - Near bone or vasculature, e.g. aneurysmal
  - Confined spaces, e.g. the optic canal



Neoplastic
- *Optic nerve sheath meningioma; intraorbital tumour (hemangioma, lymphangioma, metastatic lesion); sphenoid meningioma; pituitary tumour; craniopharyngioma*

Mechanical
- *Thyroid eye disease; haemorrhage; Paget disease; fibrous dysplasia; ophthalmic artery aneurysm; ectatic internal carotid artery*

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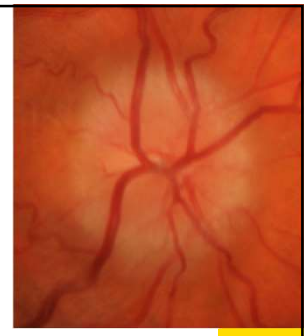
Retrobulbar optic neuritis

- Remember… this has **no swelling visible** on retinal examination despite inflammation
  - (Or may just be very subtly visible… 1/3 of cases of papillitis)
- Appreciable on other clinical signs/symptoms, e.g. pain on eye movements, contrast sensitivity/perimetric loss, colour vision loss
- Pathophysiology: inflammation of the optic nerve



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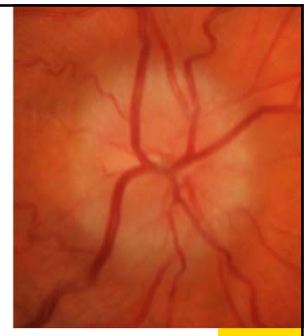
Retrobulbar optic neuritis

- Potential causes
  - Multiple sclerosis (~50%)
  - Infection
  - Other inflammatory disease
  - Toxic/nutritional causes
  - Diabetes
  - Trauma



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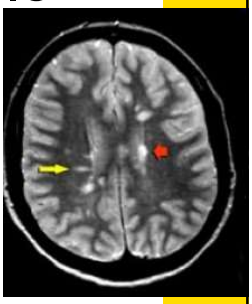
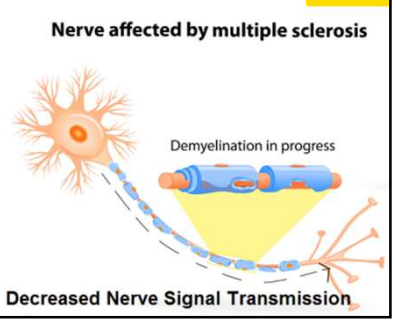
Retrobulbar optic neuritis and multiple sclerosis

- MS: neurodegeneration of the CNS, largely thought to be autoimmunity driven *(Trapp & Nave, 2008 Ann Rev Neurosci)*

- Specific pathophysiology
  - Inflammation leads to demyelination, gliotic scarring and axonal loss → optic atrophy + retinal lesions
  - White matter plaques (some grey matter) – anywhere along CNS
  - Essentially scar tissue formation





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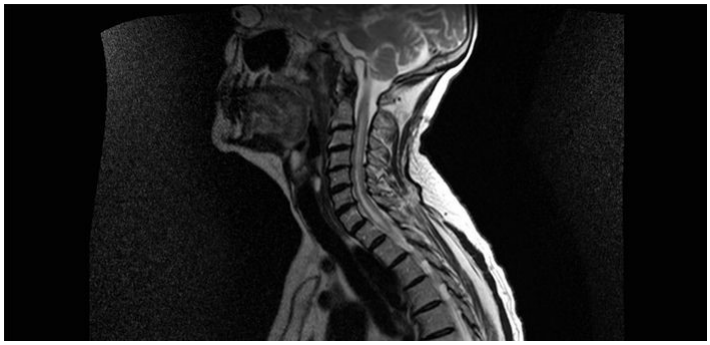
Retrobulbar optic neuritis and multiple sclerosis

- Remember **neuromyelitis optica** – a subtype of MS with additional spinal cord dysfunction



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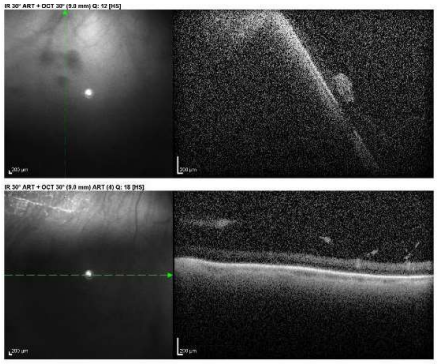
Retrobulbar optic neuritis and multiple sclerosis

- Do not forget the peripheral retinal examination
- Periphlebitis thought to be analogous to MS-related inflammatory events occurring in the brain *(Hogan et al 1971)*



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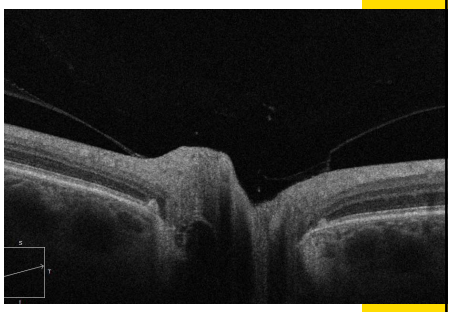
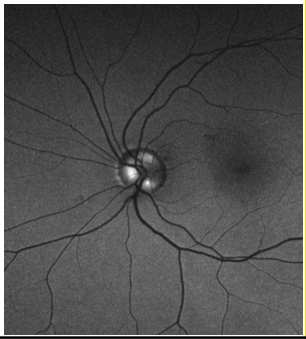
Optic nerve head drusen

- Pathophysiology: unknown; degenerative axonal byproducts? Calcific deposition in mitochondria and their subsequent coalescence
  - Natural history varies: from buried to manifest/surfaced drusen
- Probably under-diagnosed – clinical prevalence up to 25%





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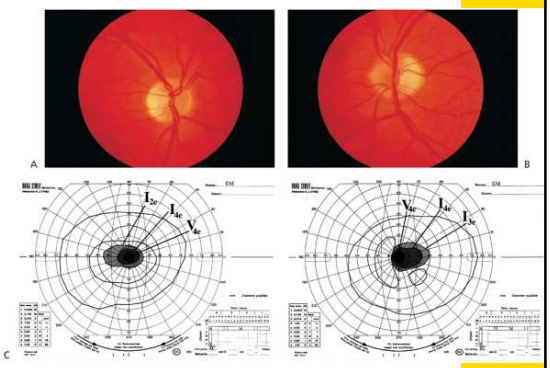
Toxic/nutritional optic neuropathy

- Bilateral and typically dose-dependent
- Typically identified from medical history
  - Alcohol
  - Antibiotics
  - Anti-malarials, e.g. hydroxychloroquine
  - Tuberculosis
  - Anti-arrhythmic, e.g. amiodarone
  - Sildenafil
  - Heavy metals, e.g. lead



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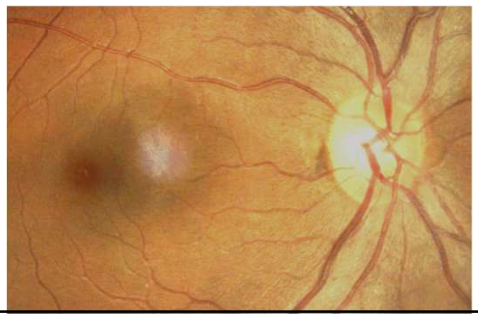
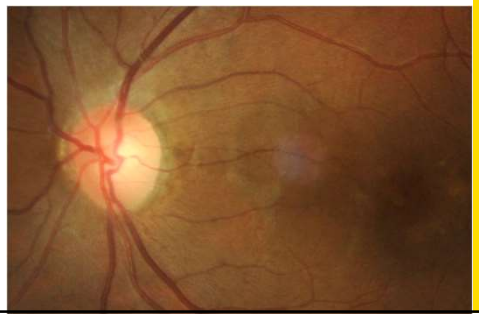
Traumatic optic neuropathy

- General pathophysiology: either direct or indirect trauma to the optic nerve
- Clinically likely to see other signs of trauma, e.g. iris sphincter damage, haemorrhages, detachments etc…





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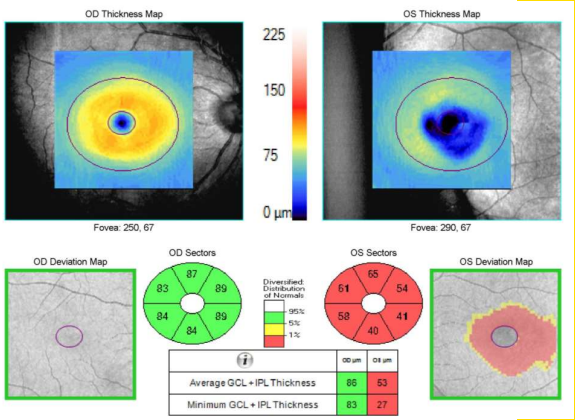
Traumatic optic neuropathy

- Clinically “random” appearance of neural loss – not a typical pattern, e.g. glaucoma



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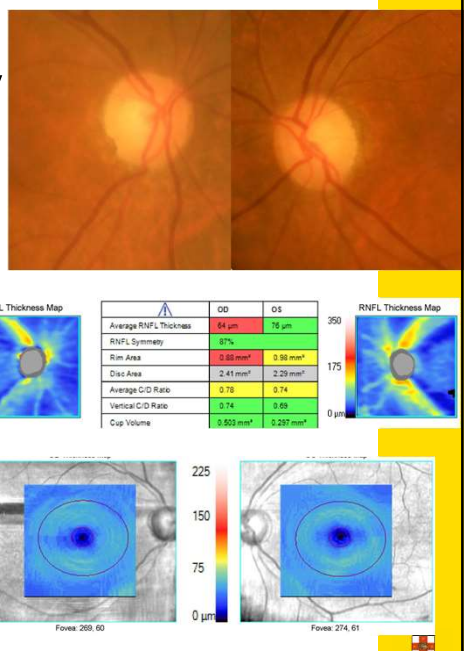
Hereditary optic neuropathy

- Two main forms
  - Autosomal dominant optic atrophy (DOA)
  - Leber’s hereditary optic neuropathy (LHON)
- Can be very slowly progressive
- Seems to disproportionately affect papillomacular bundle



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Quick T/F quiz

- Retrobulbar optic neuritis cannot be a primary optic atrophy due to intraretinal swelling
- Damage to the nerve due to intraocular surgery often results in a very ordered and clean optic nerve and retinal nerve fibre layer defect
- Advanced glaucoma is often accompanied by attenuation of the major retinal arteries and vasculature

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PART 2: Secondary optic atrophy

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Secondary optic atrophy

- Occurs **"secondary" to longstanding swelling of the ONH**
- General pathophysiology: primary condition that **causes swelling** and then **physical impingement on the nerve fibres**
- Examples:
  - Chronic papilloedema
  - Papillitis (diabetic, viral, Idiopathic)
  - Anterior ischaemic optic neuropathy (AION)
  - Compressive lesions in the anterior orbit (e.g. ON sheath meningioma)

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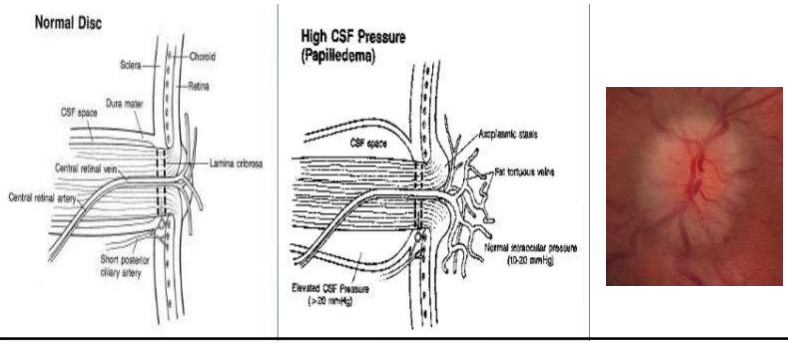
Papilloedema

- By definition: swelling secondary to **elevated intracranial pressure**
  - Other causes of **disc swelling** are **NOT papilloedema**
  - Incorrect to apply this to primary infection, infiltration or inflammation without increased intracranial pressure



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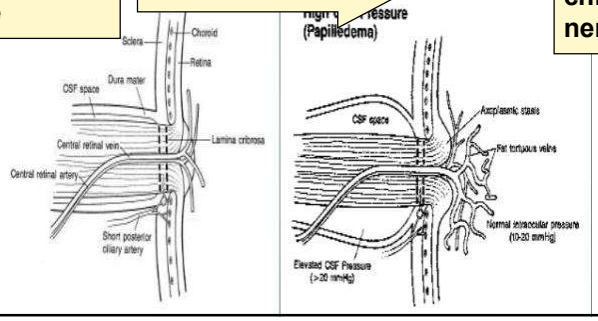
Papilloedema

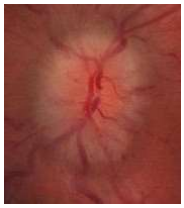
- Significance of **chronic** = long enough to cause damage

Orthograde axoplasmic flow stasis

Increased intracranial pressure



Chronicity therefore causes chronic mechanical damage to nerve and vasculature



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Papilloedema – Frisen grading

Grade	Rim	Parapapillary features
0	Slight blur of N, S, I inversely proportional to disc diameter	No tortuosity; intact; none (or rarely) obscuration of a major blood vessel
1	Nasal blurred, but no elevation	Greyish opacities and attenuation of RNFL; halo and folds present
2	All borders obscured; nasal elevation	Complete peripapillary halo
3	All borders obscured; nasal elevation	Obscuration of segments of blood vessels; halo present with “finger-like” extensions
4	All borders obscured; all elevated	Obscuration of all bloods; total obscuration of blood vessels
5	All borders obscured; all elevated; dome-shaped protrusions	Obscuration of all bloods; total obscuration of blood vessels; obliteration of optic cup

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Optic papillitis

- Any inflammation of the optic nerve head (note difference to retrobulbar optic neuritis and papilloedema)
  - Work up still similar: must exclude IIH; include CSF analysis (for infection)

Demyelinating disease	Autoimmune disease	Infectious/parainfectious	Inflammatory
MS; NMO Shilder's disease Encephalitis periaxialis concentrica	Sarcoidosis SLE Sjogren's syndrome Behcet's disease	Herpes zoster; VZV Lyme disease Syphilis TB Dengue fever Mumps Toxoplasmosis Measles Etc…	Sinusitis Post-vacciniation Hep B Rabies; tetanus Meningitis Etc…

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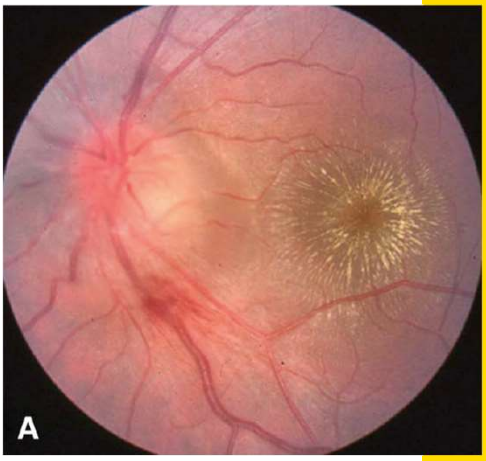
Neuroretinitis

- Inflammation affecting both optic nerve and retina
  - Characteristic "macular star" (localised to OPL)
- Aetiologies
  - Most common (especially young) – infectious (Bartonella – cat scratch)
  - Others: syphilis, Lyme disease, toxoplasmosis, toxocariasis etc
  - Otherwise, idiopathic



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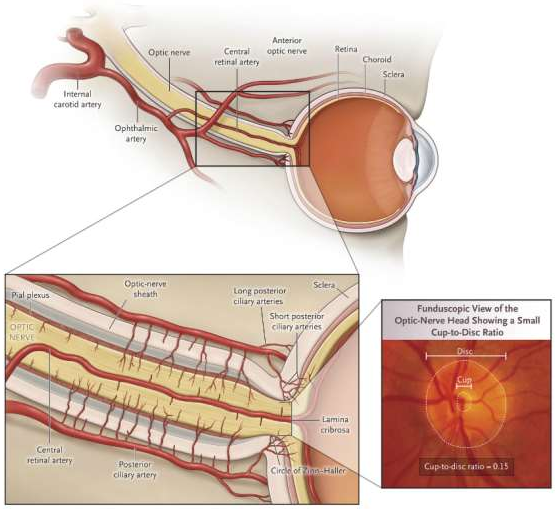
Anterior ischaemic optic neuropathy

- By definition: **ischaemic** attack of the optic nerve
  - Analogous to stroke in the eye – requires appropriate work up
- What about posterior ischaemic optic neuropathy?
  - Misnomer – merely the retrobulbar portions with swelling (by definition)



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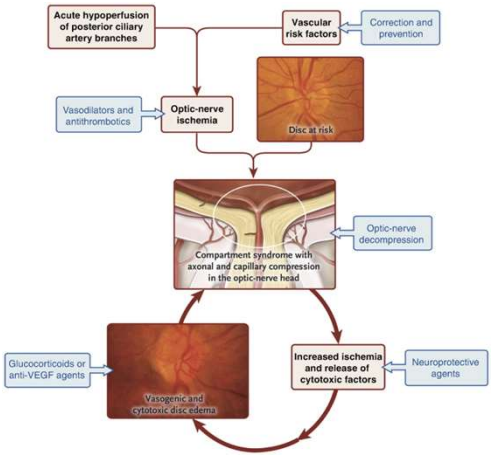
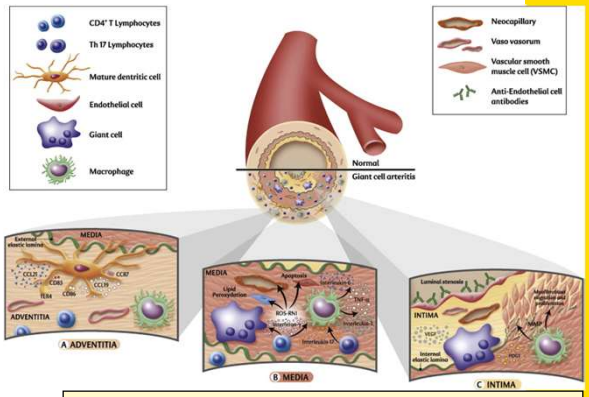
Anterior ischaemic optic neuropathy



Disc at risk for NAION – "compartment syndrome"



AAION does not require this condition… manifestation of systemic disease (key point here: vascular changes)

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Anterior ischaemic optic neuropathy

	AAION	NAION
Age of onset	Older (70s+) (mean 72 yrs)	Relatively younger (50-60s) (mean 52 yrs)
Gender	Female > male	Female = male
Associated symptoms	Jaw claudication, headache, scalp tenderness, myalgia, constitutional symptoms	<10% mild pain
Disc	• Pallid disc swelling • Cup of any size • Cup enlarges after oedema resolves	• Hyperaemic disc swelling • Small or absent cup – "disc at risk" • Cup unchanged or little change after oedema
VA	Generally worse than 6/60	Generally 6/9-12 or better (not bad)
Second eye	Involved in 1/3 (rule of thirds)	1/7 chance in 5 years
Retinal signs	Common: CWS	Uncommonly seen

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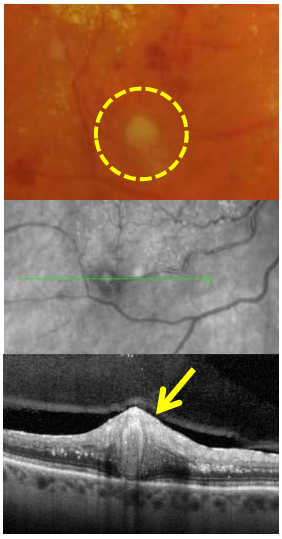
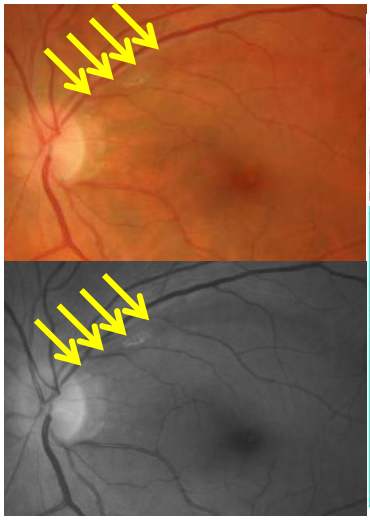
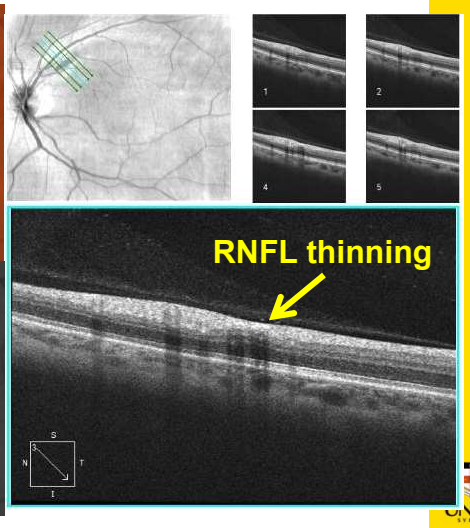
Other signs of retinal ischaemia







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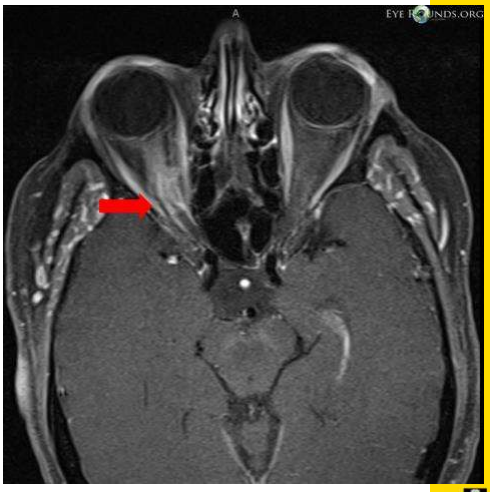
Optic nerve sheath meningioma

- Proliferation of meningothelial cells originating from arachnoid mater
- Grow circumferentially around the optic nerve **without invasion of nerve tissue**
- Pathology: mechanical compression



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Quick T/F quiz

- Arteritic anterior ischaemic optic neuropathy is accompanied by pain due to the inflammatory component
- Neuroretinitis is typically bilateral due to its systemic aetiology
- Disorders of axoplasmic flow at the level of the optic nerve head typically result in retrobulbar optic neuritis and are incompatible with secondary optic atrophy

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PART 3: Consecutive optic atrophy

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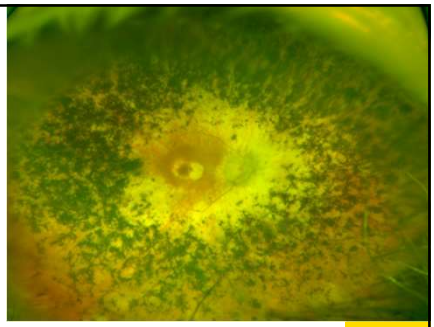
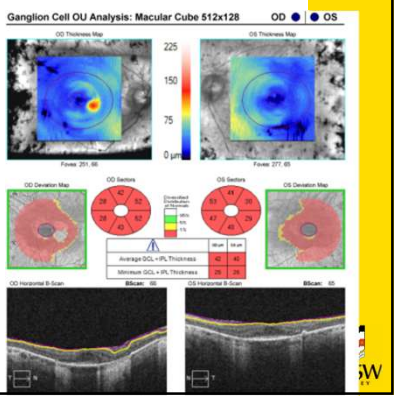
Consecutive optic atrophy

- "Consecutive" because it follows from other diseases of the inner retina or retinal blood supply

- Examples
  - Retinitis pigmentosa (RP); cone-rod dystrophies
  - Vasculitis
  - Retinal necrosis/neuroretinitis
  - Excessive photocoagulation
  - Retinal ischaemia





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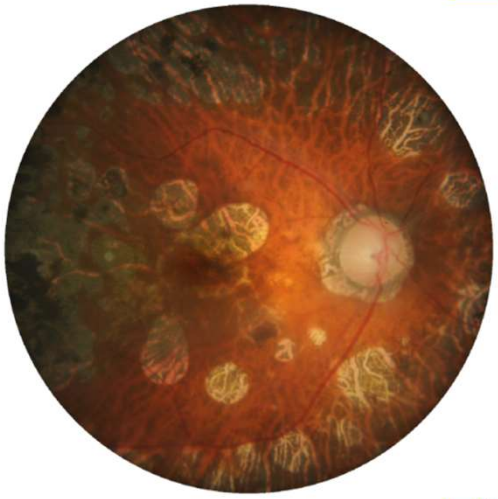
Consecutive optic atrophy

- General pathophysiology: ascending optic atrophy
  - Direct trauma to inner retina/vasculature



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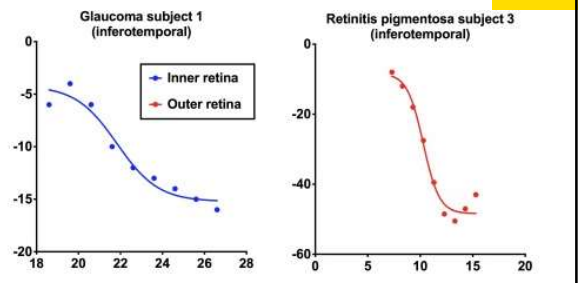
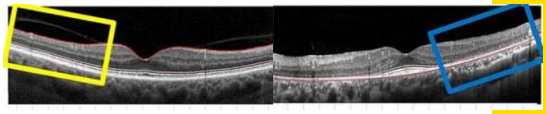
Consecutive optic atrophy

- Clinical manifestations → retinotopic visual field loss
- Gradients of change are present in terms of the structural losses occurring in disease
- Implication is a similar gradient of functional loss





Phu et al 2019 Front Neurosci

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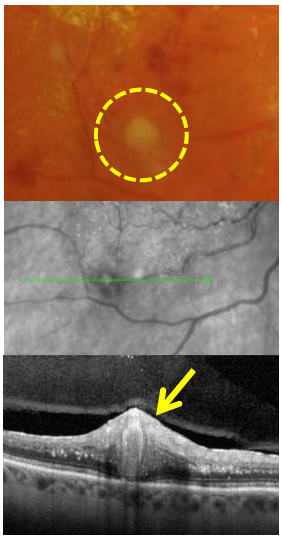
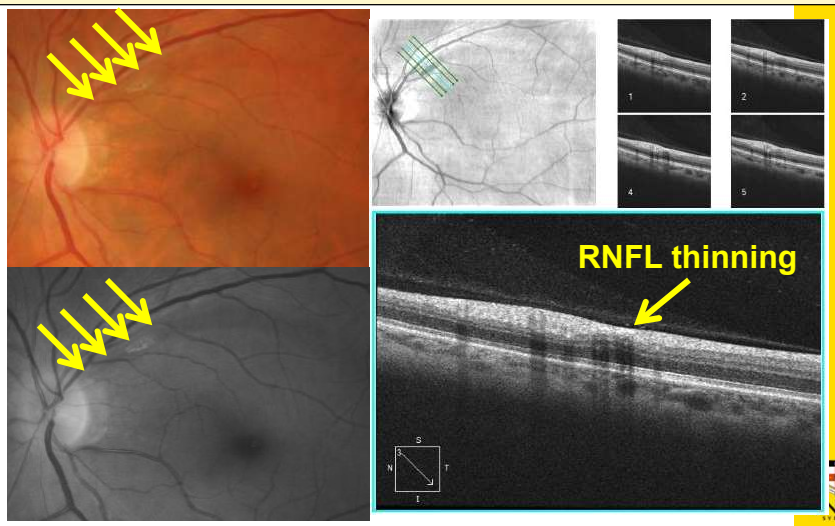
Other signs of retinal ischaemia

~8 weeks after resolution – microvascular infarct (neural loss)





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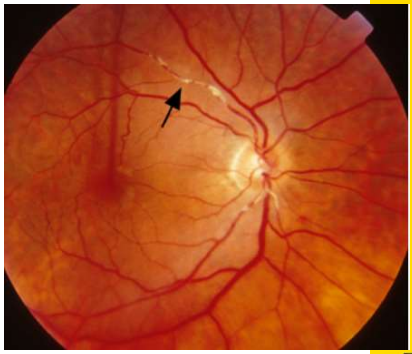
Examples: arterial plaques

Cholesterol (Hollenhorst): yellow, scintillating, more distal (smaller)

Calcific: similarly white, more proximal vs. cholesterol

Fibrin-platelet emboli: collections of embolic material



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Quick T/F quiz

- A cotton wool spot is a potential harbinger or sign of nerve fibre layer infarct and vascular investigations should be conducted
- Progressive neural degeneration should be accompanied by a very clear delineation of structural normality and abnormality
- Concordance is expected between gradients of structural and functional loss

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PART 4: Transynaptic retrograde degeneration

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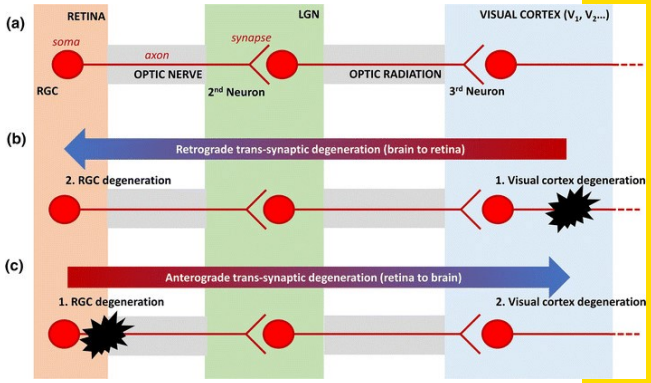
Retrograde degeneration

- Full name: trans-synaptic retrograde degeneration
- RNFL and GC loss due to post-LGN lesions
- Pathophysiology:
  - Well-defined specific apoptotic pathways
  - Pyramidal cell death → signals to V1



Jindhara et al 2012; Park et al 2013

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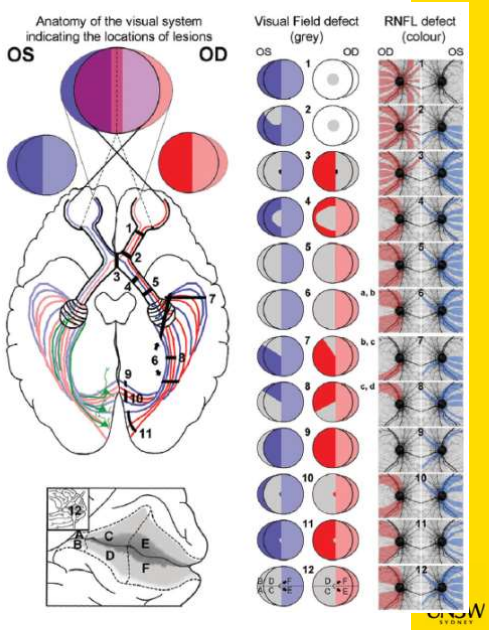
Retrograde degeneration

- Clinical characteristics: retinotopically specific



Zangerl et al 2017 Clin Exp Optom

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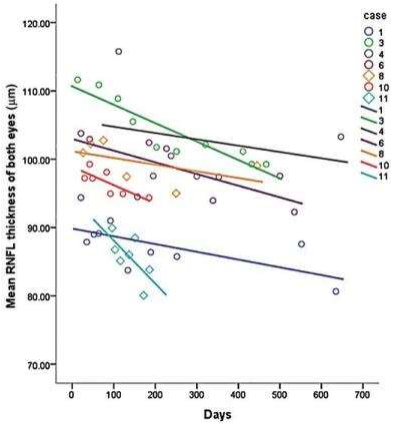
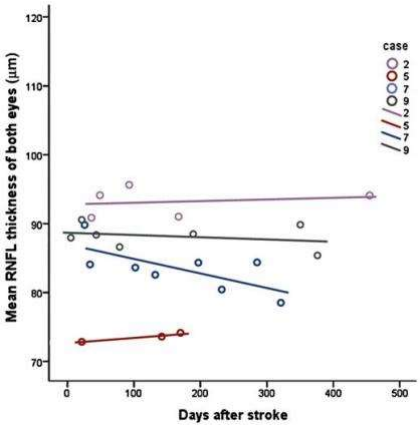
Retrograde degeneration

Importance of time



Importance of extent



Jindhara et al 2012 Brain

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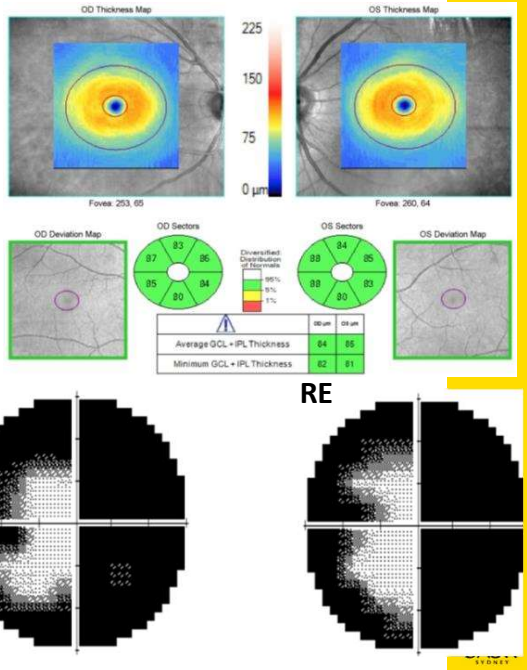
Retrograde degeneration

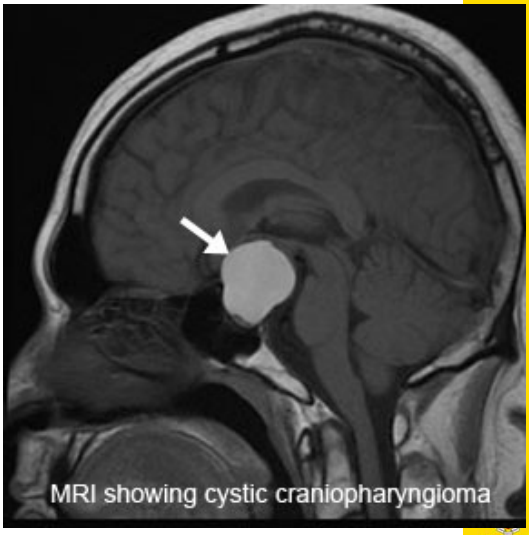


Remember: VF defects may precede structural deficits in retrograde degeneration - **Recent CVA** *(Zangerl et al., 2017)*

44

Pituitary lesion: not trans-synpatic

- Craniopharyngioma – lesion from above = visual field defect from below
- Opposite with pituitary tumour
- Binasal defects: rare – related to vascular tumour at pituitary



45

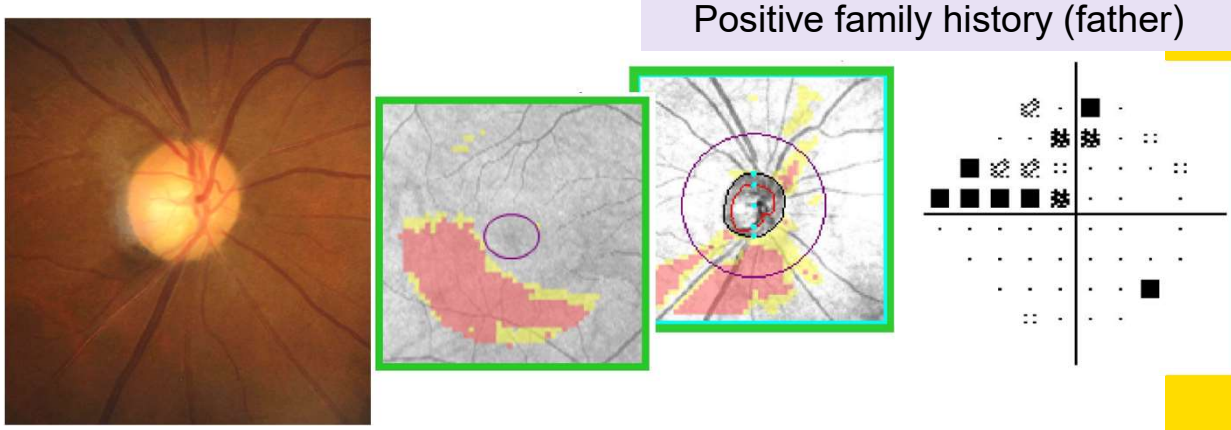
Quick T/F quiz

- Elevated intracranial pressure often causes visual field defects that respect the vertical midline
- The vertical midline is respected in both optic nerve and macular scans in retrograde degeneration
- Over time, people with stroke are likely to experience changes in vision and the visual field

46

"Typical" glaucoma

Other clinical features
IOP 25 mmHg
CCT 510 microns
Positive family history (father)



Key feature: **structure-function concordance** at the optic nerve head

47

What is the definition of glaucoma?

> *Glaucoma describes a group of ocular disorders of multi-factorial aetiology united by a clinically characteristic optic neuropathy with potentially progressive, clinically visible changes at the optic nerve head (ONH), comprising focal or generalized thinning of the neuroretinal rim with excavation and enlargement of the optic cup, representing neurodegeneration of retinal ganglion cell axons and deformation of the lamina cribrosa; corresponding diffuse and localized nerve-fibre-bundle pattern visual field loss may not be detectable in early stages; while visual acuity is initially spared, progression can lead to complete loss of vision; the constellation of clinical features is diagnostic.*

Emphasis is on **optic nerve head appearance**

Casson et al 2012 CEO

48

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Emphasis is on **progression towards vision loss**

Casson et al 2012 CEO

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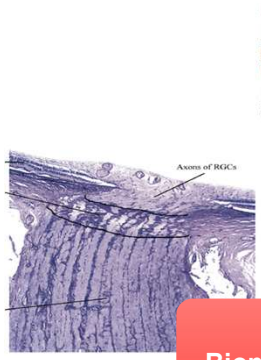
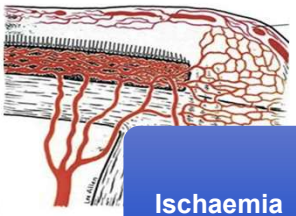
Epidemiology

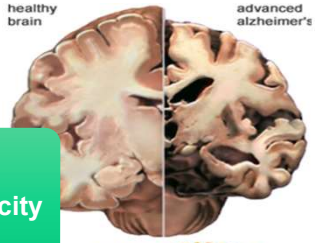
- Glaucoma is the most common optic neuropathy
- Prevalence ~3-5% - increasing with age *(Keel et al 2019 Br J Ophthalmol)*
- 50% of glaucoma remains undiagnosed, despite advances in technology *(Heijl et al 2013 Ophthalmology; Mitchell et al 1996 Ophthalmology; Varma et al 2004 Ophthalmology)*
- 50% of glaucoma is also **likely overdiagnosed and over-treated** *(Founti et al 2018 Acta Ophthalmol; Nayak et al 2011 Indian J Ophthalmol)*
  - Many differentials for glaucoma are managed differently
  - Implications for resource utilisation

50

Pathophysiology







Ischaemia

Neurotoxicity

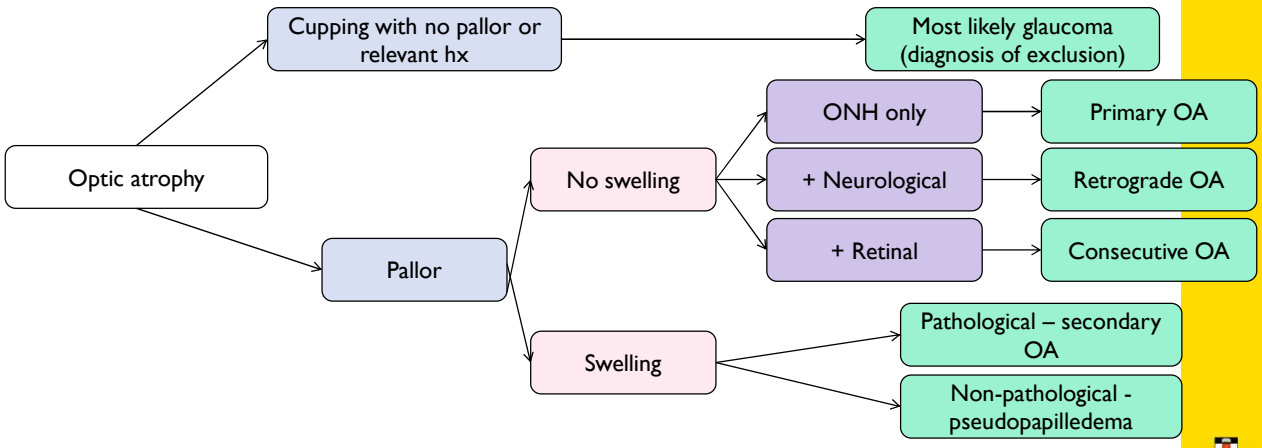
Biomechanical stress

Genetic Susceptibility

Glaucoma

51

Types of optic atrophy



52

The most important point in this talk:

Glaucoma is a diagnosis of exclusion!

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Coming Up - Interactive poll #1

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Poll 1. A history of trauma to the side of the head is most likely to lead to which type of optic neuropathy?

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PART 5: Clinical techniques for differential diagnosis

UNSW SYDNEY

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Clinical history

- Previous ocular insult
- Vascular/ischaemic challenge
- Other connective tissue disease
- Drugs

57

Clinical history

- Many systemic diseases have been “linked” to glaucoma
  - Cardiovascular (e.g. hypertension) *(Marshall et al 2020 Ophthalmology)*
  - Metabolic/endocrine (e.g. diabetes) *(Zhao et al 2015 Ophthalmology)*
  - Ischaemic stressors (e.g. obstructive sleep apnoea, migraine) *(Faridi et al 2012 Clin Exp Ophthalmol)*
  - Connective tissue (e.g. rheumatoid arthritis) *(Black et al 2016 PLoS ONE)*
  - Neurodegenerative *(Mancino et al 2018 Curr Neuropharmacol)*
  - Gastrointestinal? *(Gong et al 2020 Exp Eye Res)*

58

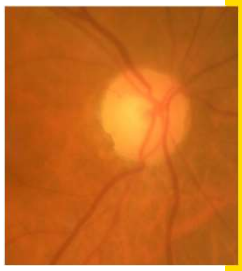
Entrance tests

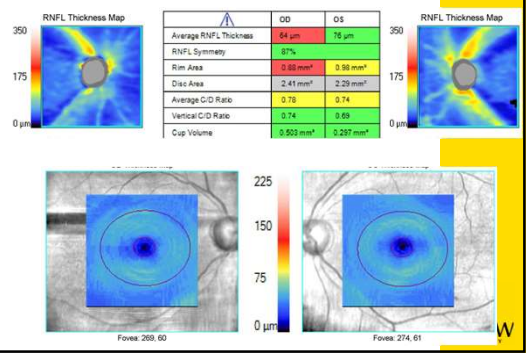
- Visual acuity
- Refractive error
- Pupillary reflexes

59

Entrance tests – visual acuity

- Account for any **reduction or changes** in visual acuity
- Examples
  - Refractive error
  - Cataract
  - Retinal diseases
  - Neurological diseases



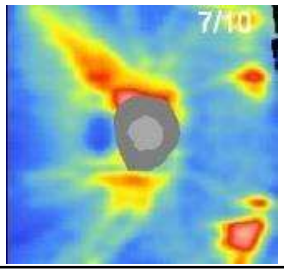


60

Entrance tests – refractive error

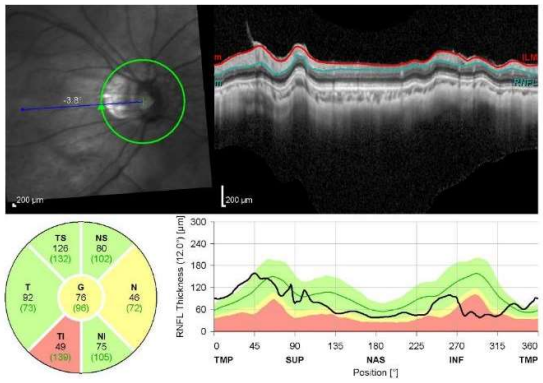
- Influence of refractive error
  - Vision changes
  - Visual field defects
  - Optic disc examination
  - OCT artefacts

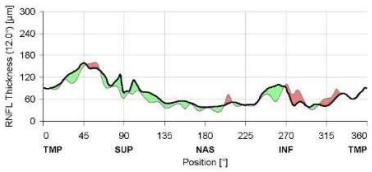
- Normative database comparisons



61

Entrance tests – refractive error





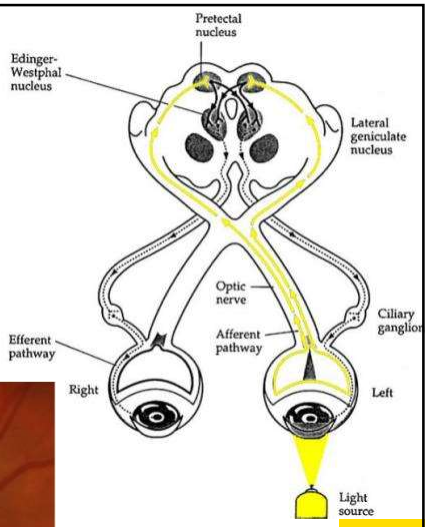
Classification

Outside Normal Limits

62

Entrance tests – pupil testing

- Represents a conduction deficit in the afferent pathway
- Typically unilateral and/or asymmetric disease
- Examples
  - AION
  - Optic neuritis
  - Contralateral optic tract



63

Optic nerve examination

- Maintain a systematic approach
  - Size
  - Cup width and depth
  - Rim: thickness and colour
  - RNFL reflectivity
  - Vasculature
  - Pertinent negatives (disc haemorrhages)

64

Optic nerve examination

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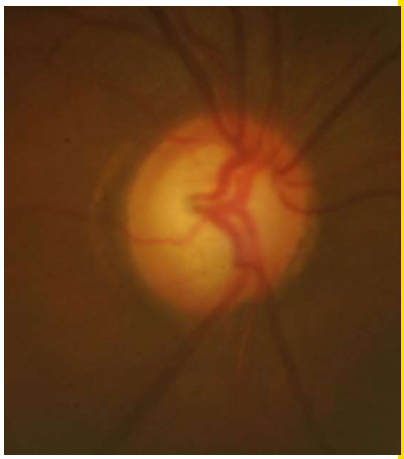

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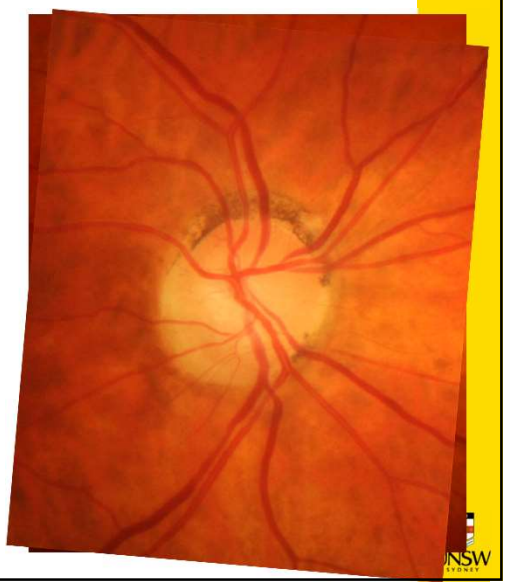
Optic nerve examination

- Stereoscopic viewing can assist with contour

- Flicker chronoscopy can help identify subtle rim changes – options?
  - In-built software (e.g. Kowa)
  - Photoshop (has scaling and alignment)
  - Powerpoint
  - Online GIF generator



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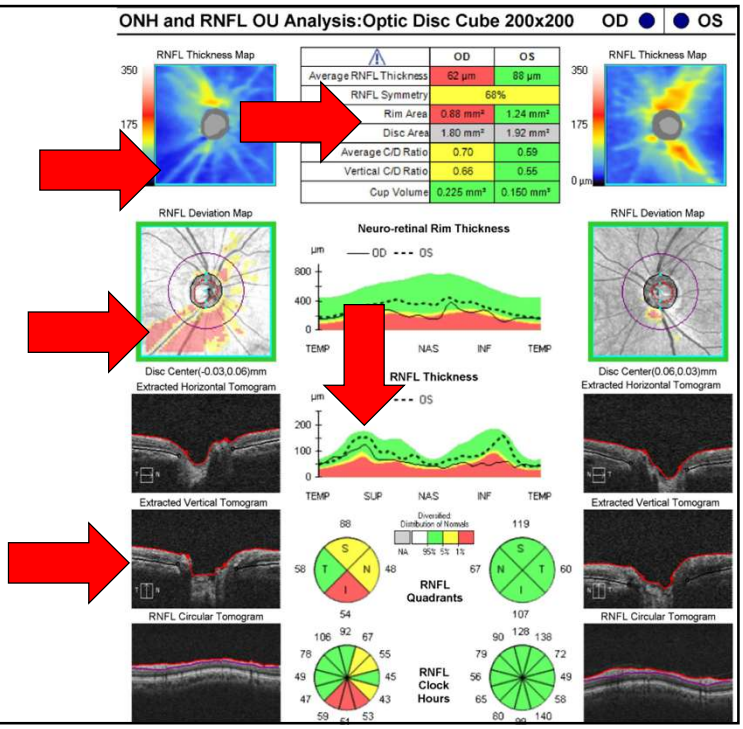
Ocular imaging

- Look for patterns of structural defects – are they consistent with glaucoma?
  - Asymmetry
  - Contiguity
  - “Wedge” or “arcuate”
  - Corruption?



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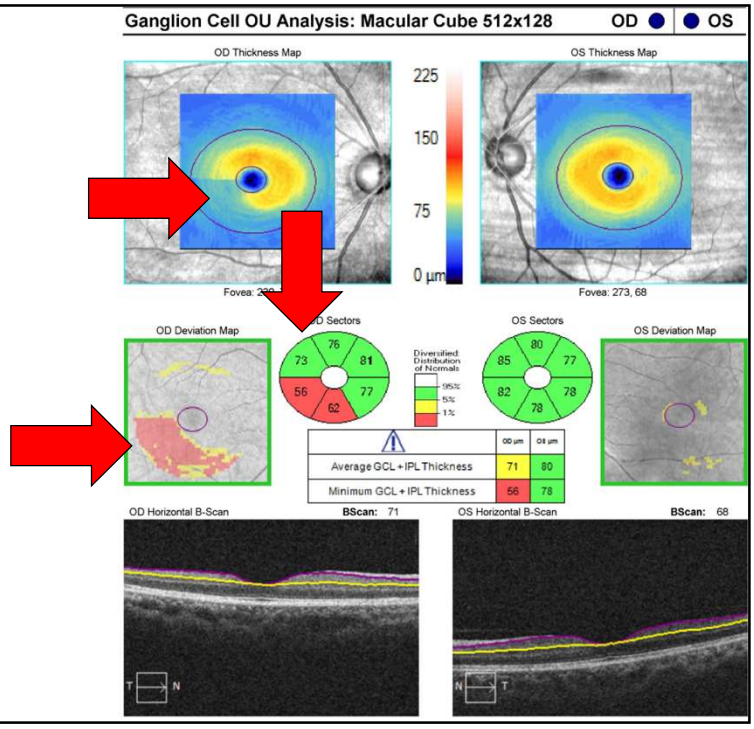
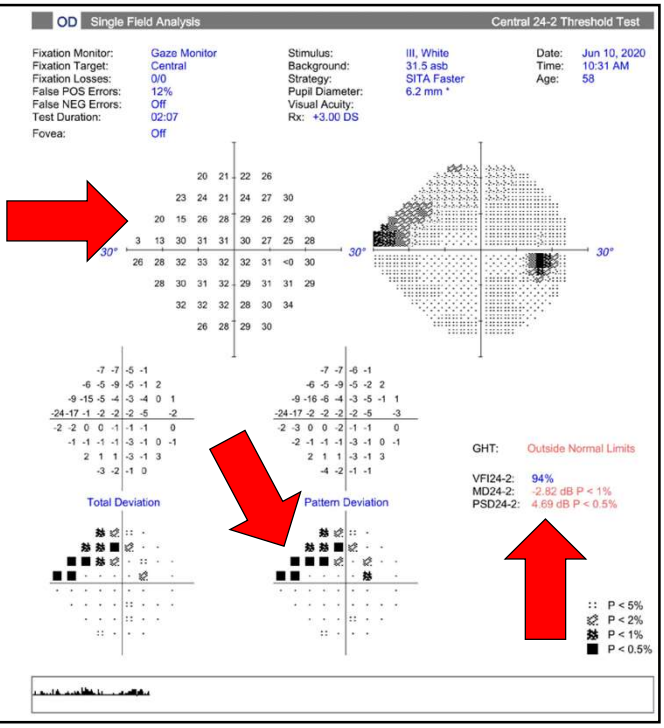


68

SAP

- Look for patterns of visual field defect
  - RNFL patterns: nasal step, arcuate, paracentral
  - Deep, then wide
  - Look for “criteria” for failing the test



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Poll 2. Which of the following represents best practice for reporting intraocular pressure for glaucoma?

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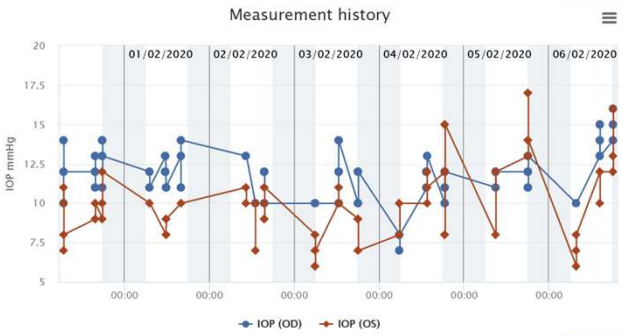
Intraocular pressure

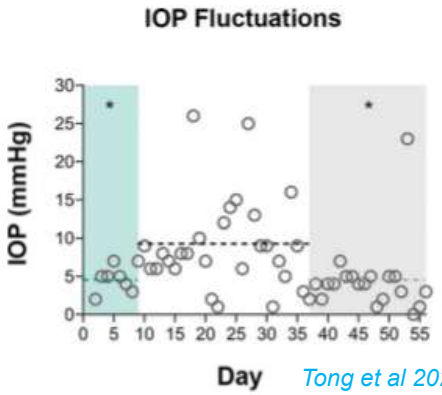
- NOT a defining feature for glaucoma
- Important for risk management and understanding intervention strategy
- Different measurement approaches



72

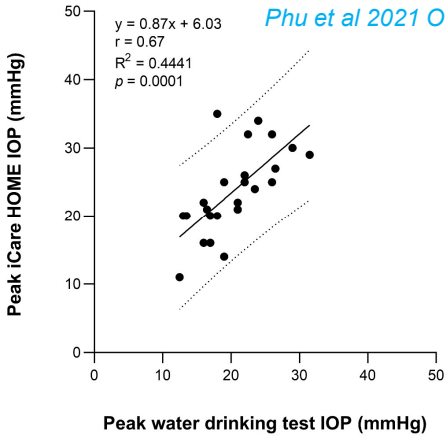
Intraocular pressure

- Diurnal fluctuations



Tong et al 2021 OVS

- Peak (osmotic stress)



Phu et al 2021 OVS

73

Intraocular pressure

- Used for identifying treatment/management paradigm
- NTG: less suitable for SLT
- High baseline pressures: SLT more likely successful

TABLE 2. Associations of Relevant Variables With ≥20% IOP Reduction at the Second Follow-up (6 mo Post-SLT) Using Univariate Analysis

Variable	IOP Reduction <20% (n=40)	IOP Reduction ≥20% (n=80; 66.67%)	Relative Risk (95% CI)	*P*
2.5	2 (5.41%)	6 (8.11%)	1.14 (0.74, 1.73)	0.6037
3.0	0 (0%)	1 (1.35%)	1.51 (1.32, 1.72)	0.4775
Pre-SLT IOP (mm Hg)	16.65 ± 4.32	21.75 ± 4.53		<0.0001
Pre-SLT maximum IOP	23.29 ± 6.38	26.47 ± 6.74		0.0176
Pre-SLT BCVA	0.23 ± 0.35	0.23 ± 0.29		0.9682

Martow et al 2011 J Glaucoma

74

Intraocular pressure reporting

- Typical recommendations is to report “as is”
- Corrected IOPs are not meaningful
- Some devices will output a modified IOP, e.g. the ORA
- Bayesian approach to interpretation

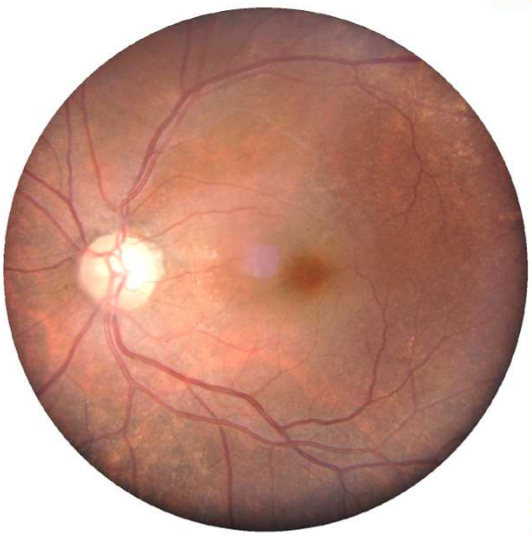
75

Case 1 – 72F

Medical history:
- Diabetes (5 years)
- Hypertension
- Hyperlipidaemia

Ocular history:
- Unremarkable cataract surgery
- Currently on Xalatan nocte

Clinical findings
- Vision normal
- IOP 15 mmHg
- Pachymetry 552 microns



76

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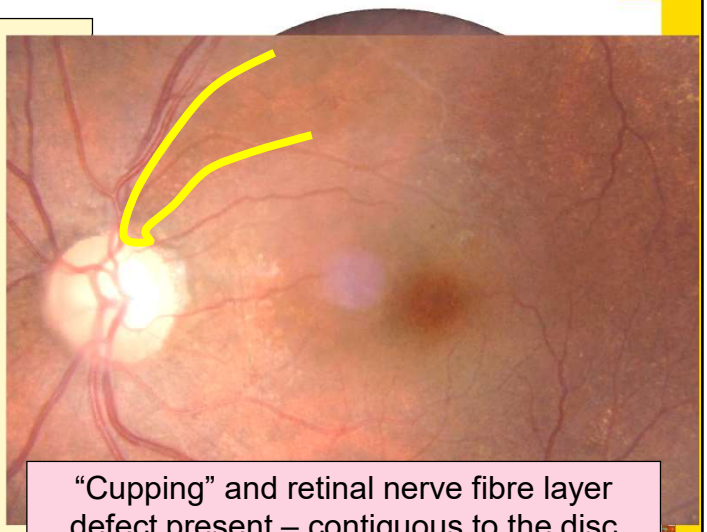

"Cupping" and retinal nerve fibre layer defect present – contiguous to the disc

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Poll 3. A patient with glaucomatous neuroretinal rim loss at the 1 o'clock position (superotemporal) of left eye will most likely have which visual field defect?

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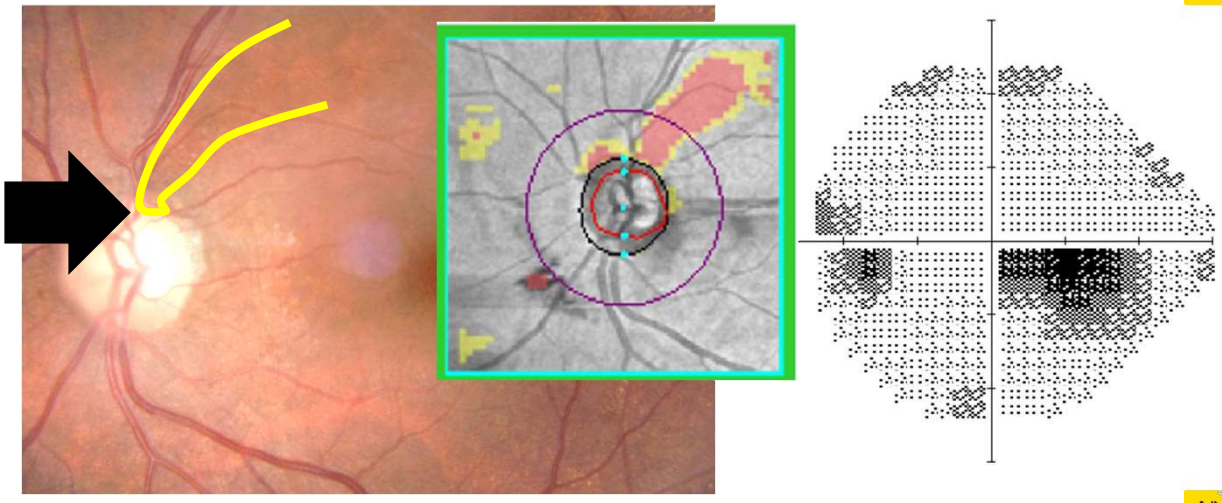
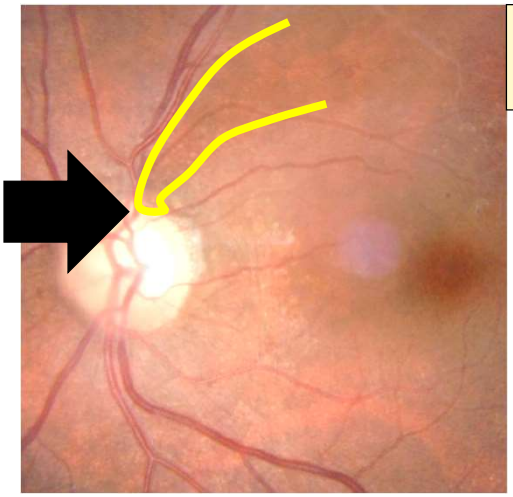
79

Case 1 – 72F

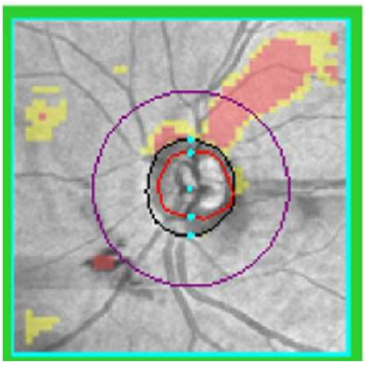


80

Case 1 – 72F

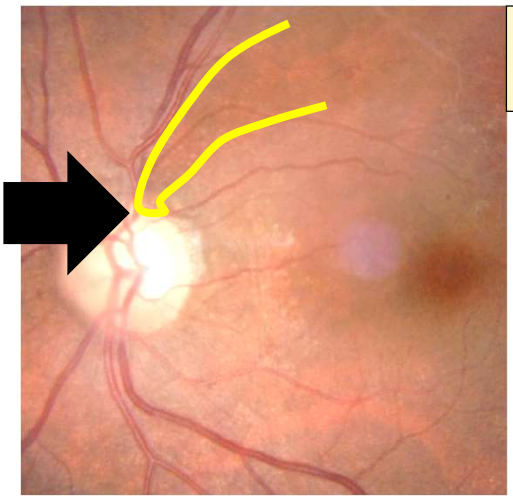


Optic nerve scan typical pattern (arcuate)



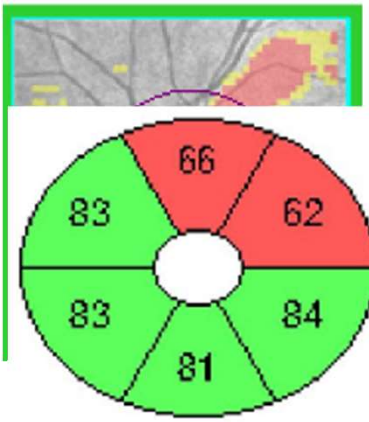
81

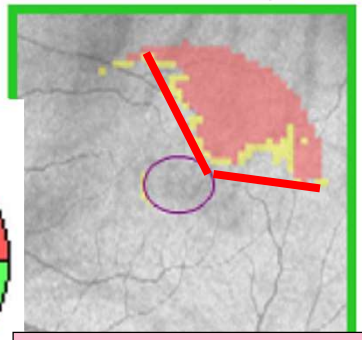
Case 1 – 72F



Optic nerve scan typical pattern (arcuate)

However, macula scan NOT typical

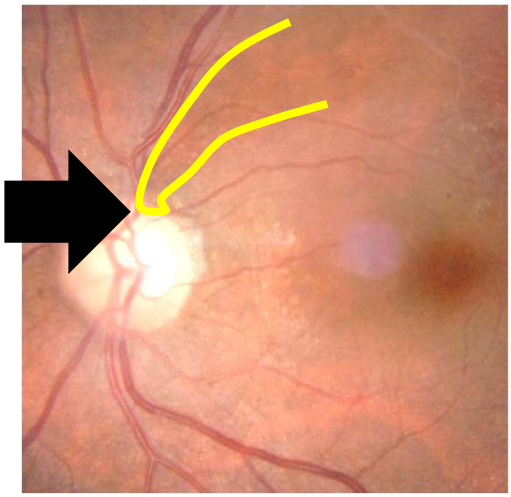


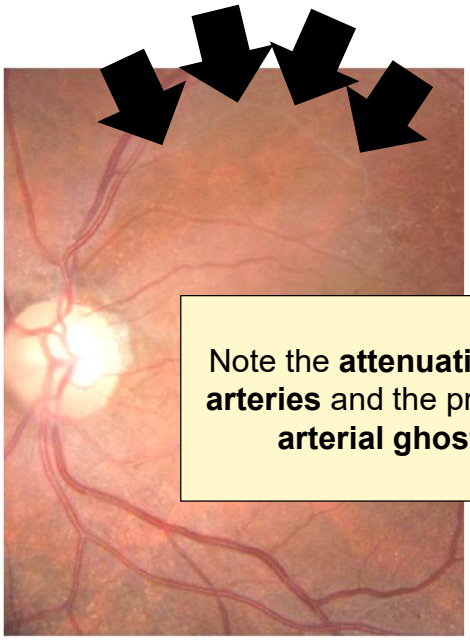


Incomplete arcuate… loss is too focal

82

Case 1 – 72F

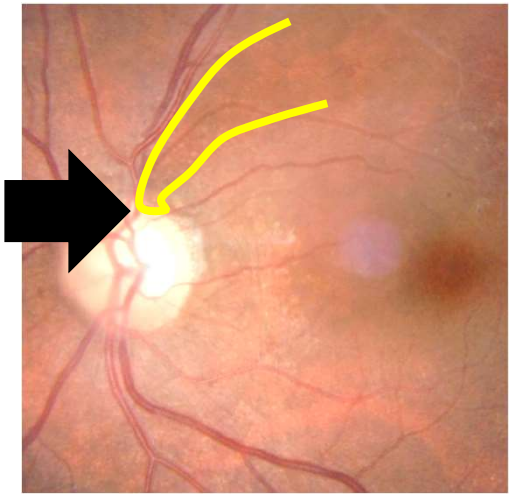


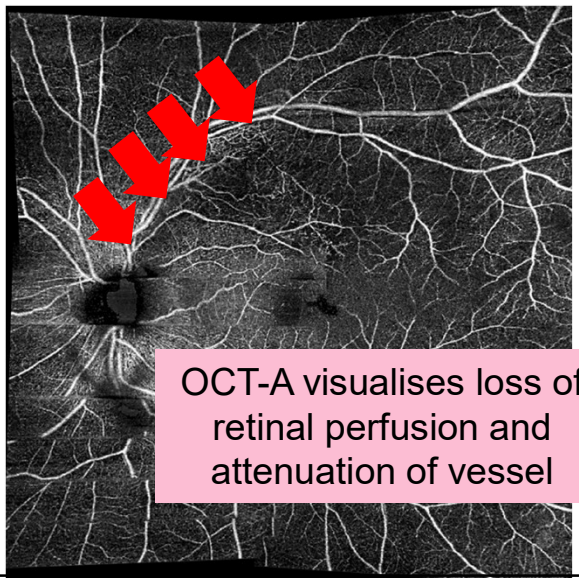


Note the **attenuation of retinal arteries** and the presence of an **arterial ghost vessel**

83

Case 1 – 72F





OCT-A visualises loss of retinal perfusion and attenuation of vessel

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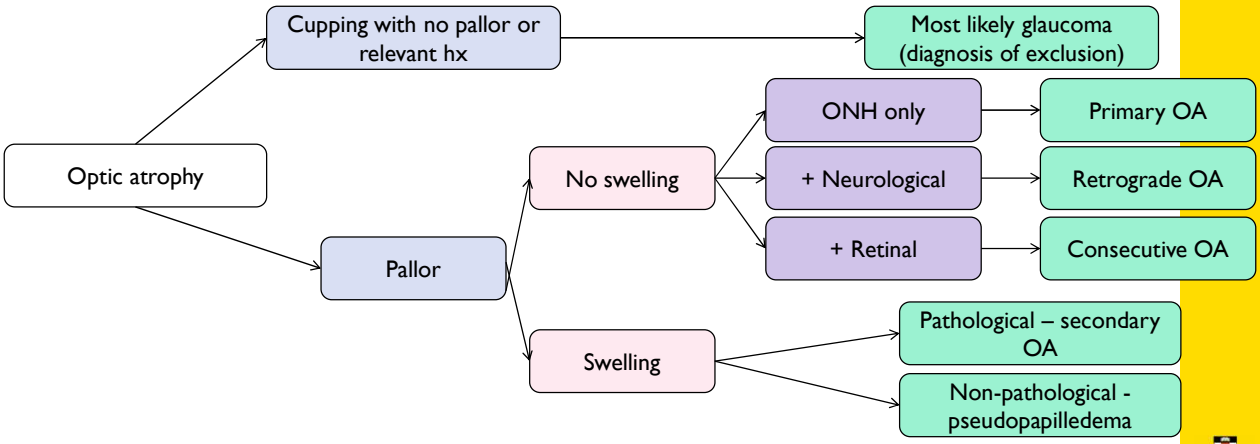
Types of optic atrophy



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Clinical examination: summary

- All aspects of the eye examination are important
- Use key tests to rule in or rule out glaucoma
- Glaucoma has characteristic features and patterns

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PART 5: Longitudinal clinical data

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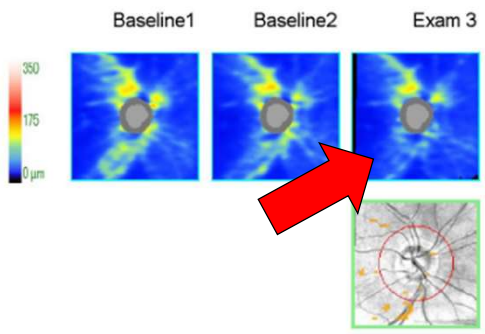
Longitudinal data

- Glaucoma is **progressive** but not all progression is equal
- Progression identification can be automated or manual
- Statistical versus clinical versus **patient relevance**?

88

Longitudinal data – structure



RNFL and ONH Summary Parameters

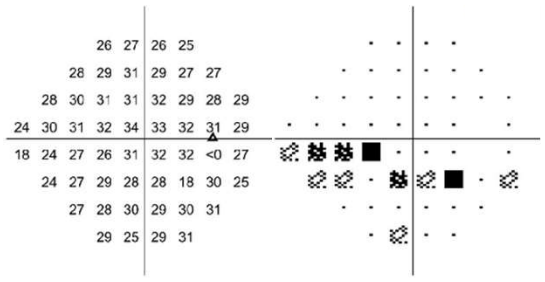
		Exam Date/Time	Serial Number	Registration Method	SS	Avg RNFL Thickness (µm)	Inf Quadrant RNFL (µm)	Sup Quadrant RNFL (µm)	Rim Area (mm²)	Average Cup-to-Disc Ratio	Vertical Cup-to-Disc Ratio	Cup Volume (mm³)
Baseline1:	1	16/08/2013 9:57:25 AM	4000-4912		9/10	78	89	89	1.15	0.66	0.67	0.136
Baseline2:	2	16/08/2013 9:58:04 AM	4000-4912	R2	8/10	77	85	90	1.16	0.66	0.66	0.140
Current:	3	16/07/2015 9:31:41 AM	4000-4912	R2	9/10	72	78	85	1.07	0.68	0.70	0.155

- Qualitative analysis: heat map, deviation map
- Quantitative analysis: thickness values

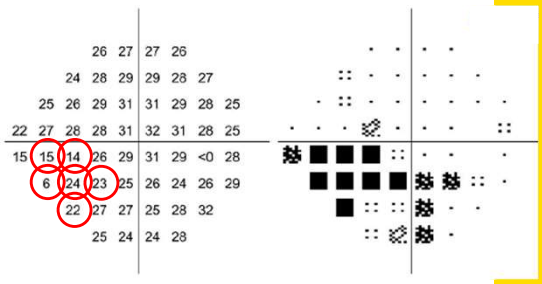
89

Longitudinal data – function

Baseline



Follow up



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Poll 4. Which combination represents the most likely scenario for a poor prognosis for glaucoma?

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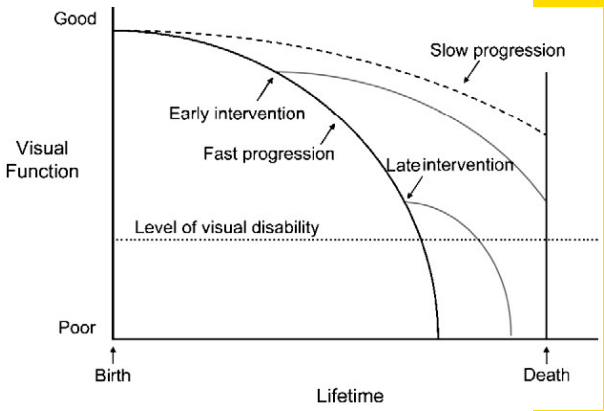
Longitudinal data

- Important for prognostication
- Detection of glaucoma depends on the **point in the natural history**



Caprioli & Zeyen 2009 J Glaucoma

93

Case 2 – 61F

- OHx: High myopia – vision otherwise normal
- FHx: Elder sister with severe, end-stage glaucoma
- MHx: systemic hypertension (treated); history of miscarriage and blood loss

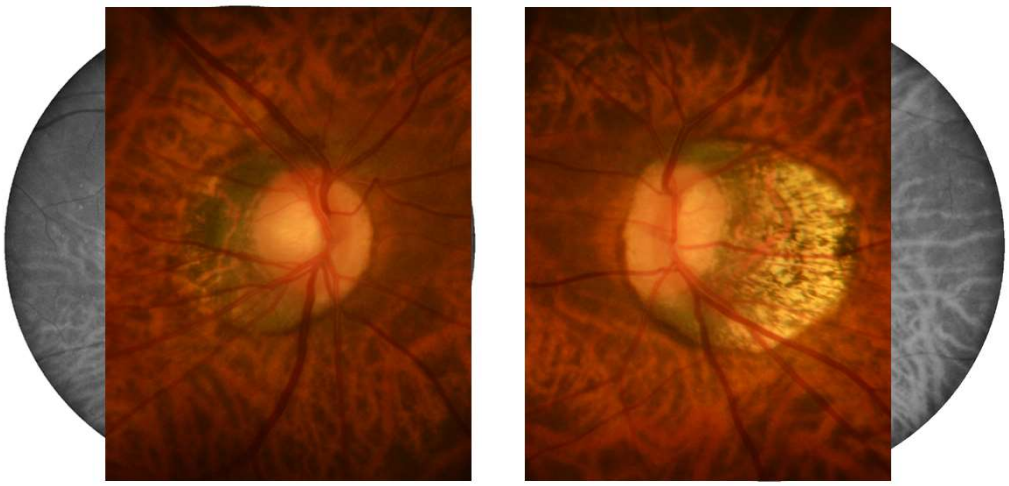
94

Case 2 – 61F

	Right eye	Left eye
Vision and Rx	-6.00/-2.00x95 (20/25)	-7.00/-1.75x95 (20/20)
Applanation IOP	14	14
CCT	550	556
Anterior segment	No cataract	No cataract
Gonioscopy	Wide open angles	Wide open angles

95

Case 2 – 61F



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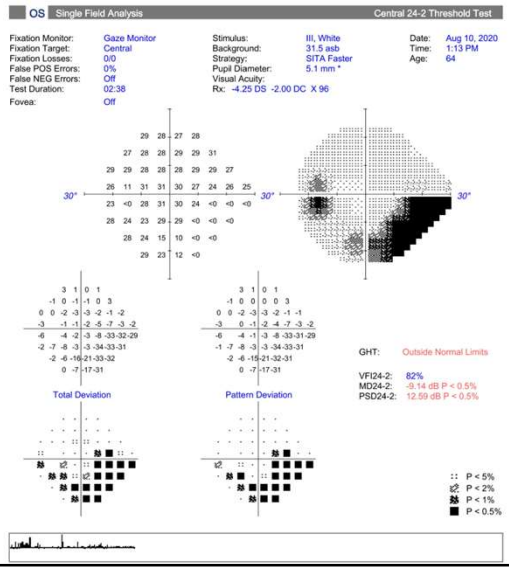
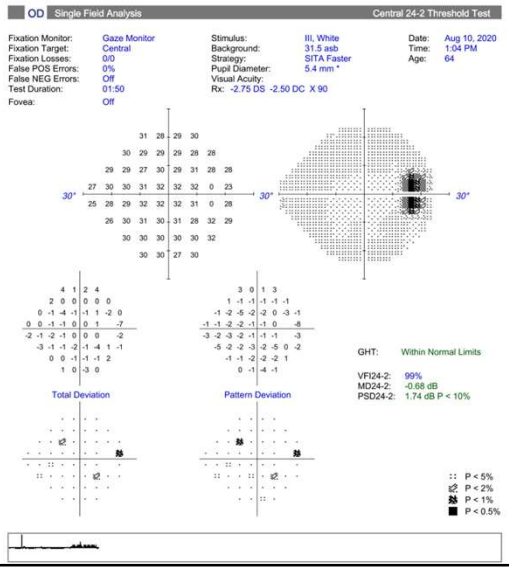
Case 2 – 61F

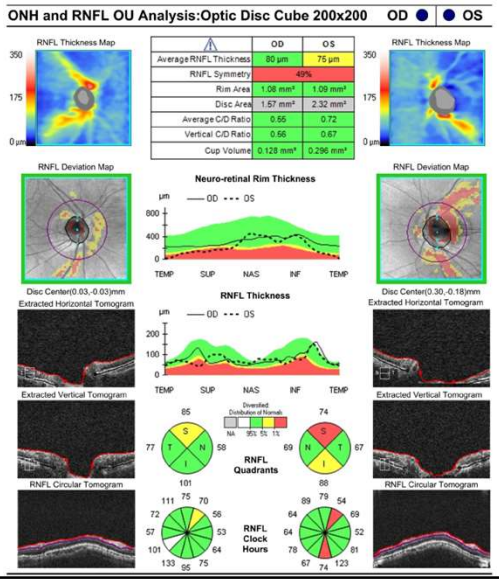




97

Case 2 – 61F

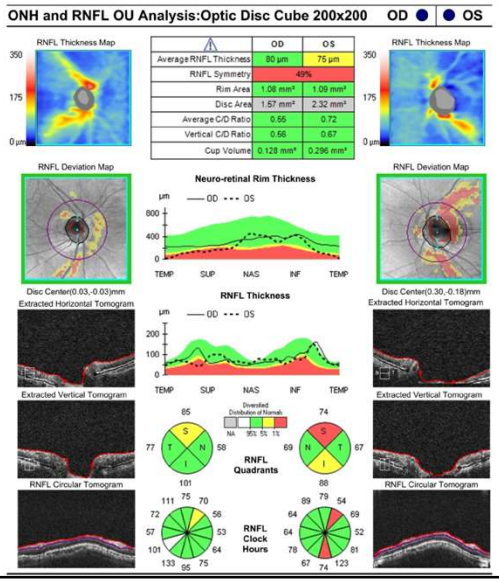
- Systematic approach
  - Segmentation and reliability?
  - Asymmetry?
  - Pattern of RNFL loss? Location?
  - Cup parameters?



98

Case 2 – 61F

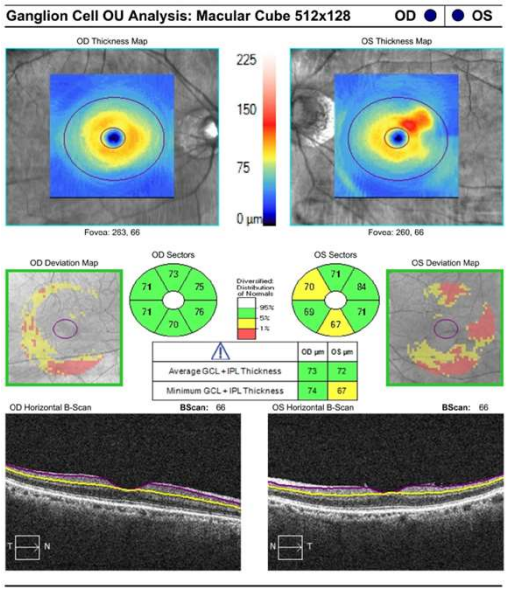
- Systematic approach
  - Segmentation and reliability? Poor due to myopia, but still relevant
  - Asymmetry? Yes
  - Pattern of RNFL loss? Location? Glaucoma-like
  - Cup parameters? Wider and deeper left eye



99

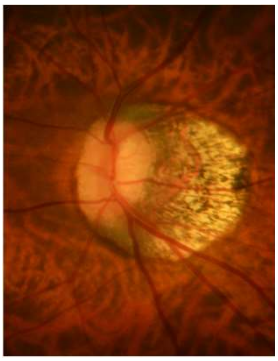
Case 2 – 61F

- Systematic approach
  - Segmentation and reliability? Poor due to left ERM – results likely invalid
  - Asymmetry?
  - Pattern of RNFL loss? Location?

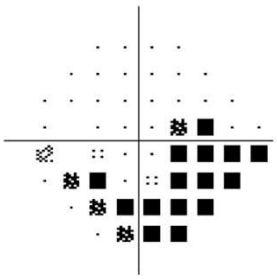


100

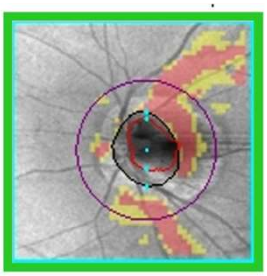
Case 2 – 61F



Myopic discs with superotemporal rim thinning



Inferior arcuate defect



Superotemporal and inferior RNFL thinning

Other clinical findings
- Low pressures
- Average CCT
- Open angles

Other risk factors
- Older age group
- Strong FHx
- Hypertension
- Blood loss?
- Myopia?

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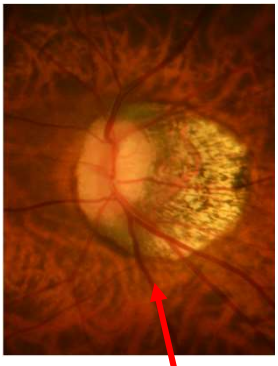
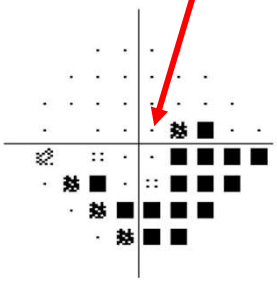
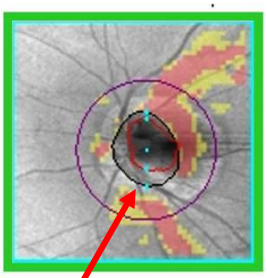
Case 2 – 61F

Low IOP



No central VF defect





Unreliable OCT

Other clinical findings
- ~~Low pressures~~
- Average CCT
- Open angles

Other risk factors
- Older age group
- Strong FHx
- Hypertension
- ~~Blood loss?~~
- ~~Myopia?~~

Very myopic disc – hard to judge

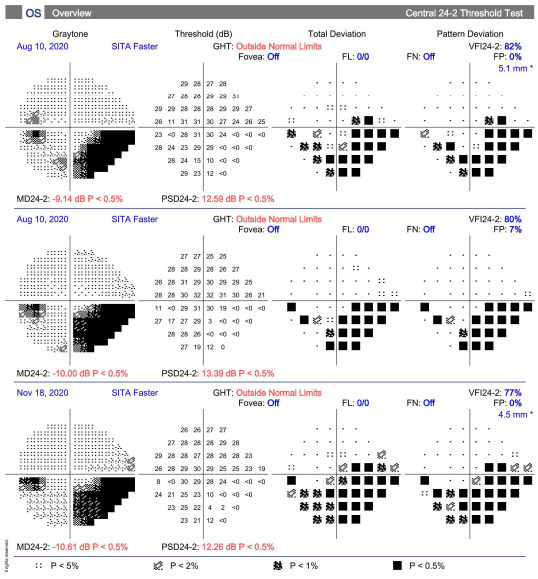
Acute, not chronic

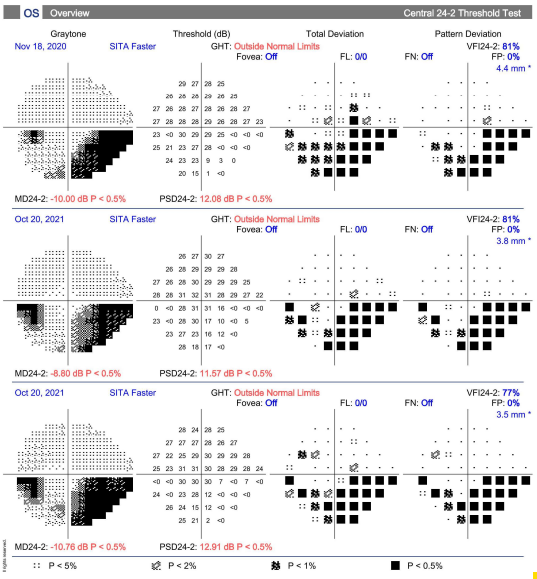
Confounding (not risk?) factor

102

Case 2 – 61F

No functional progression over 14 month period





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Longitudinal analysis: summary

- Glaucoma is a condition defined by progression (even if slow)
  - Many comparable optic neuropathies are relatively static
- Use structural and functional techniques to characterise progression rates
- Remember: IOP and risk factors do not define glaucoma

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PART 6: Ancillary clinical testing

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Additional clinical tests?

- Other clinical tests may assist in differential diagnosis
- Ophthalmic imaging
  - OCT-angiography
  - Fundus autofluorescence
- Laboratory tests, e.g. blood tests (toxic/nutritional optic neuropathy)
- Neuroimaging (MRI preferred for brain tissue, but ? access)

106

Case 3 – 66M

- No issues with vision and no ocular symptoms
- Personal ocular history unremarkable
- Family history of glaucoma (father)
- Medical history: treated hypertension

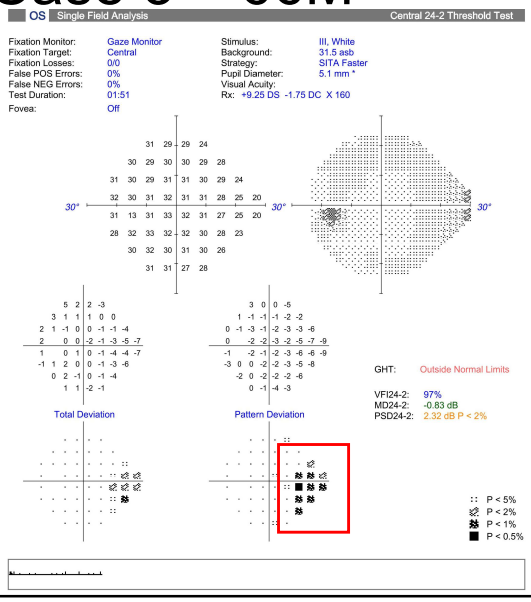
107

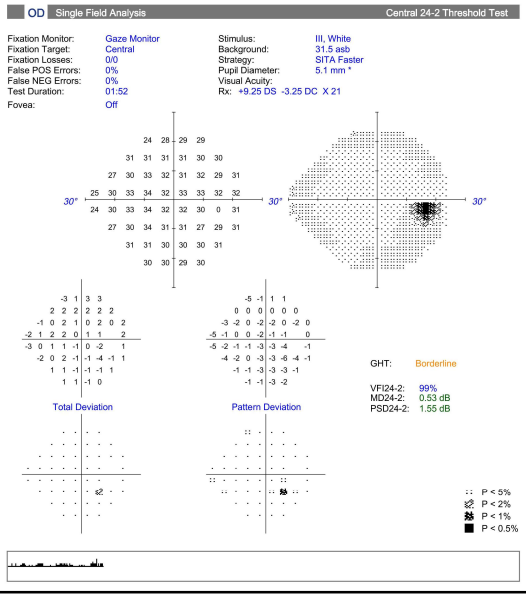
Case 3 – 66M

	Right eye	Left eye
Acuity and refraction	+0.50DS (20/20)	+0.25/-0.25x10 (20/20)
Applanation pressures (mmHg)	15	15
Corneal thicknesses (microns)	545	551
Pupils	No RAPD	
Anterior segment and gonioscopy	Wide open angles, no secondary glaucoma risk factors	

108

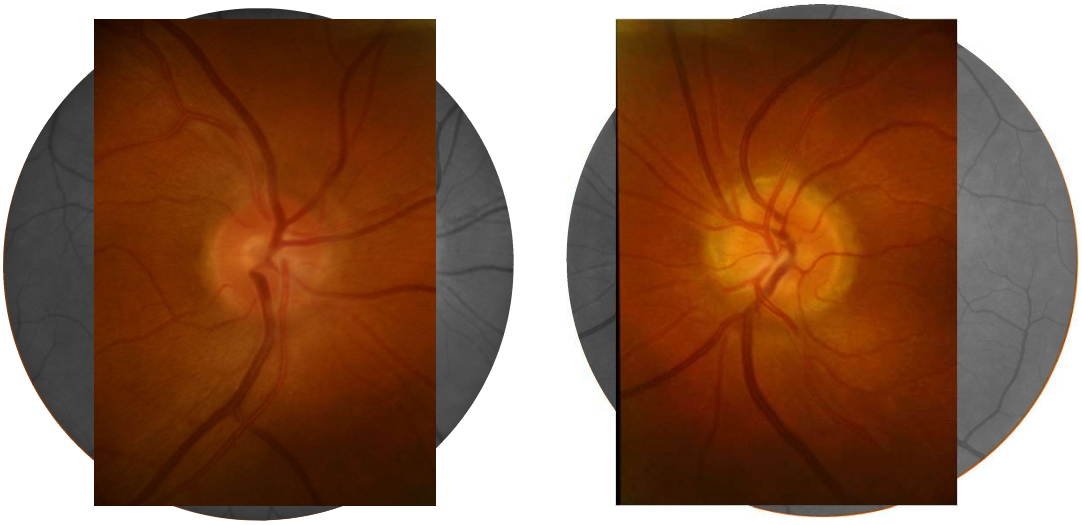
Case 3 – 66M





109

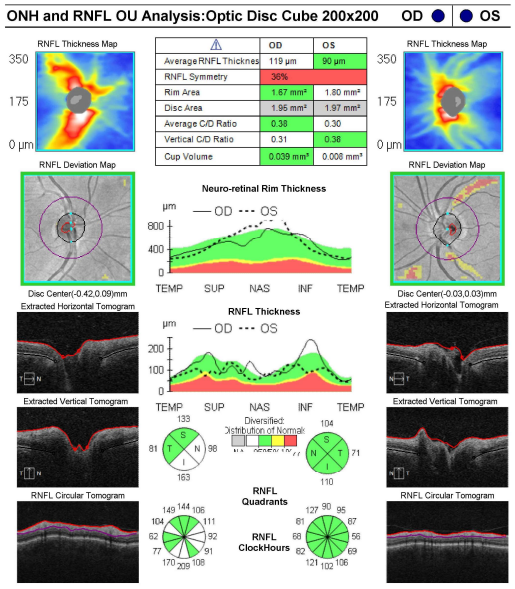
Case 3 – 66M



110

Case 3 – 66M

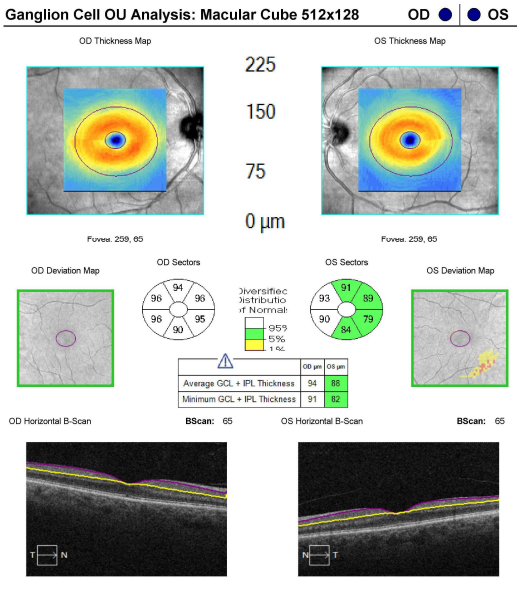
- Systematic approach
  - Segmentation and reliability?
  - Asymmetry?
  - Pattern of RNFL loss? Location?
  - Cup parameters?



111

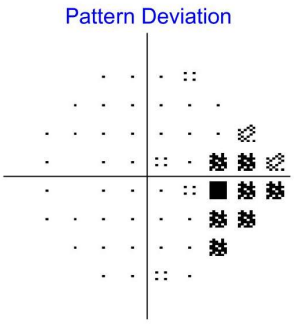
Case 3 – 66M

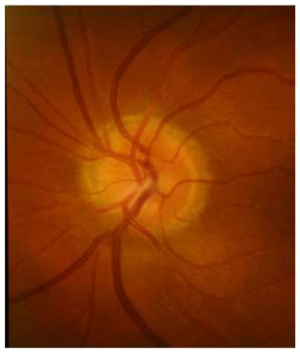
- Systematic approach
  - Segmentation and reliability?
  - Asymmetry?
  - Pattern of RNFL loss? Location?

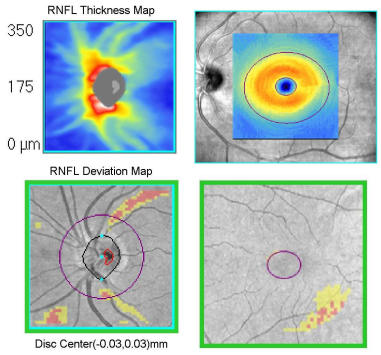


112

Case 3 – 66M







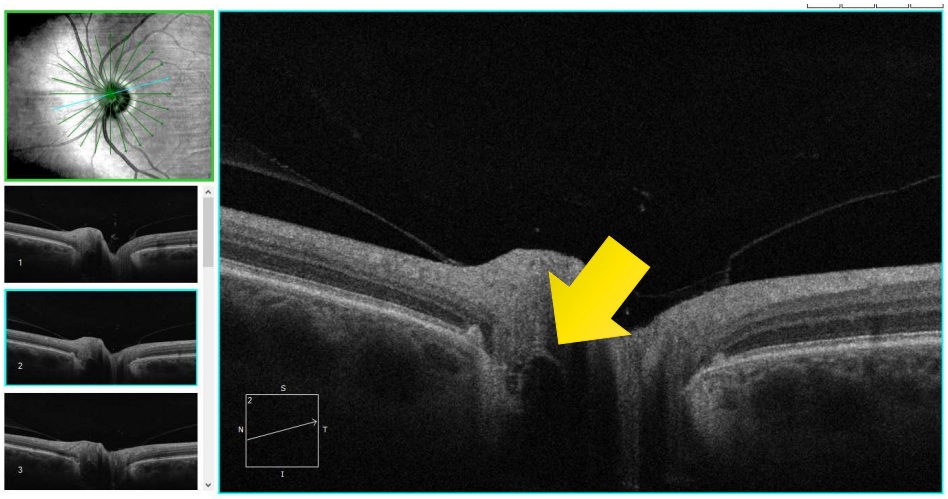
Superonasal and inferonasal defects

Shallow cup with **intact** neuroretinal rim

Superotemporal and inferotemporal OCT thinning

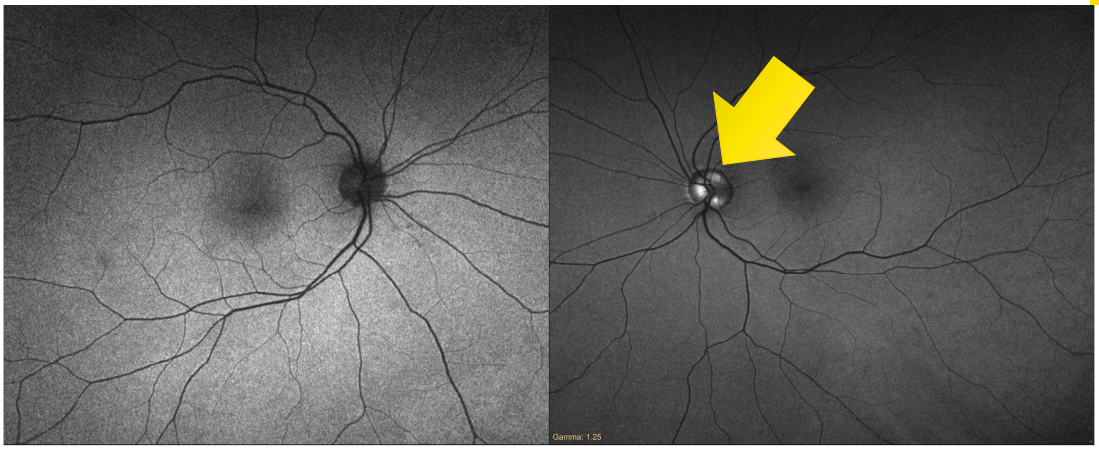
113

Case 3 – 66M



114

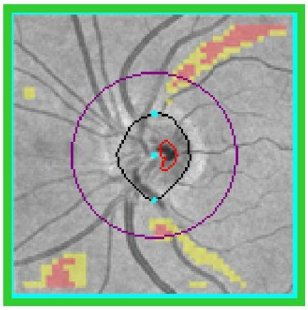
Case 3 – 66M



115

Case 3 – 66M

- Additional ophthalmic imaging reveals the presence of **optic nerve head drusen**
- Explains the **shallow cup** but **deep RNFL loss**



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Case 4 – 70F

- Opinion on narrow angles
- No ocular symptoms
- No medications; no relevant medical history
- Previous MVA 20 years ago – passenger side

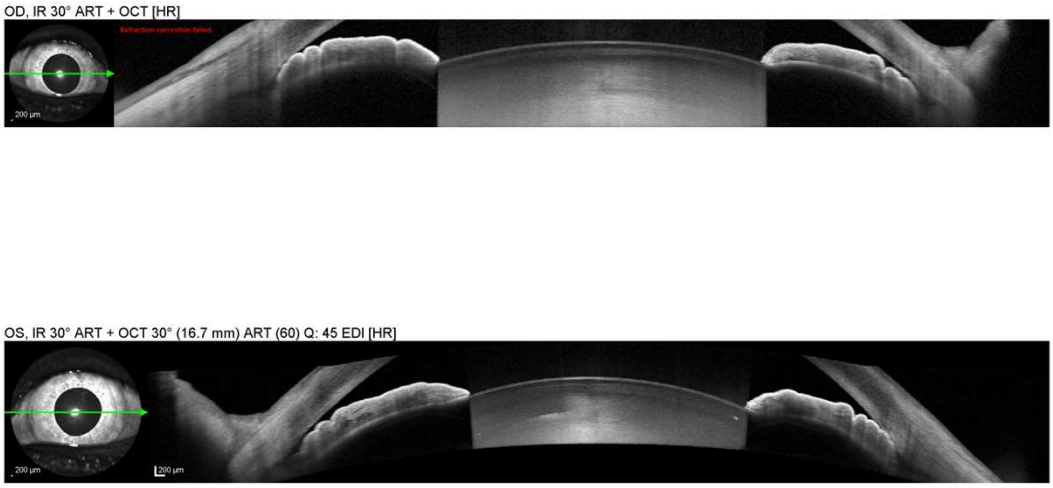
117

Case 4 – 70F

	Right eye	Left eye
Acuity and refraction	Plano 20/30	Plano 20/25
Applanation pressures (mmHg)	16	14
Corneal thicknesses (microns)	529	545
Pupils	No RAPD	
Anterior segment and gonioscopy	Narrow angles; gr 2+ NS	

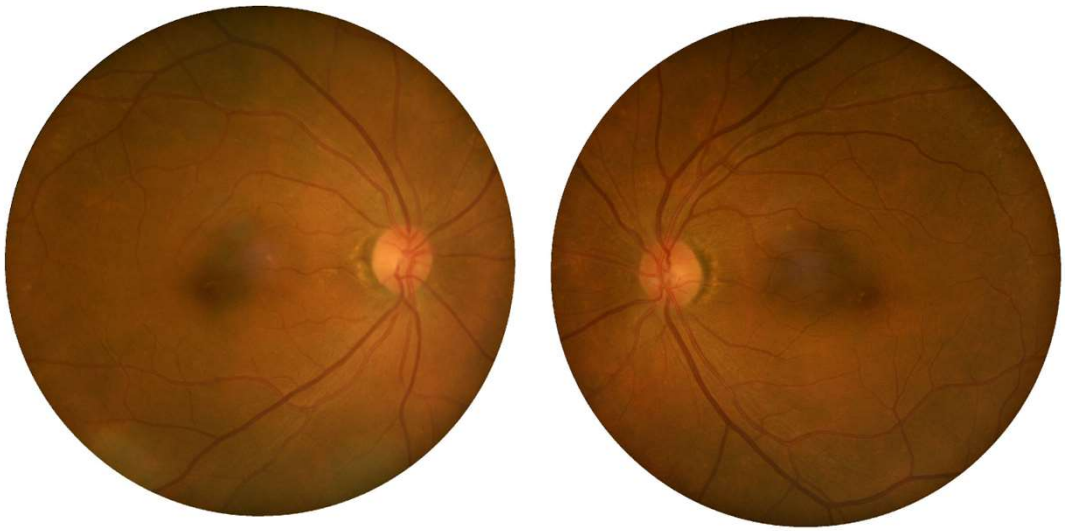
118

Case 4 70F



119

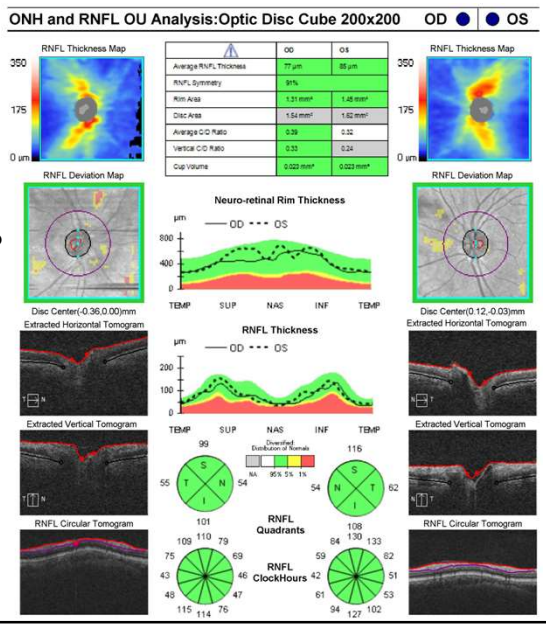
Case 4 – 70F



120

Case 4 – 70F

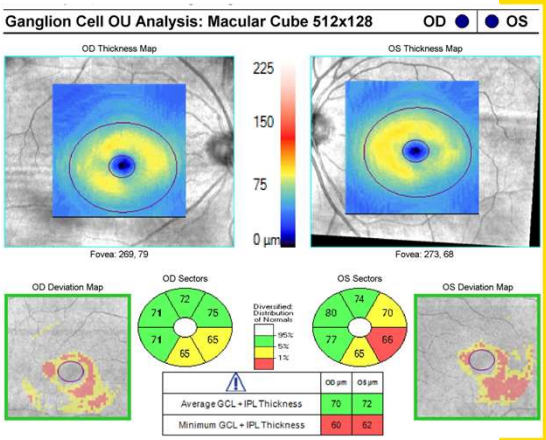
- Systematic approach
  - Segmentation and reliability?
  - Asymmetry?
  - Pattern of RNFL loss? Location?
  - Cup parameters?



121

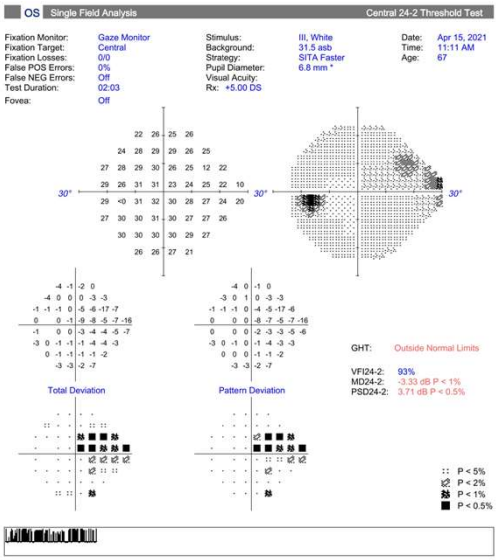
Case 4 – 70F

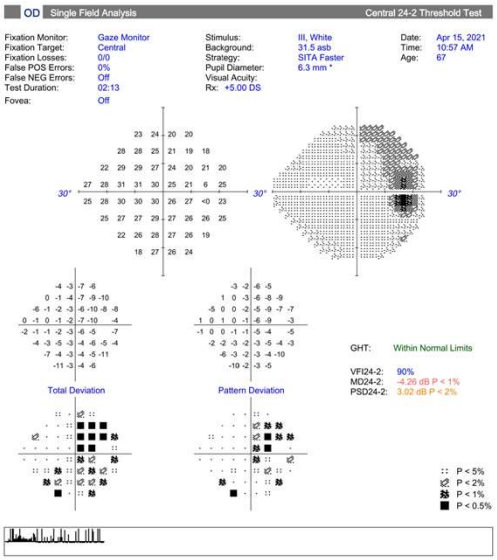
- Systematic approach
  - Segmentation and reliability?
  - Asymmetry?
  - Pattern of RNFL loss? Location?



122

Case 4 – 70F





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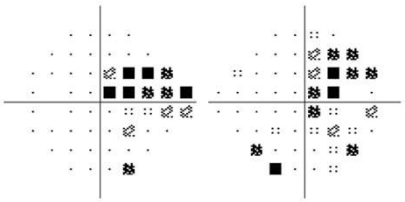
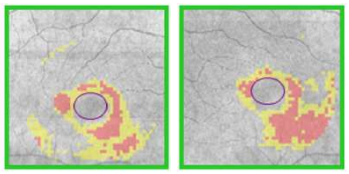
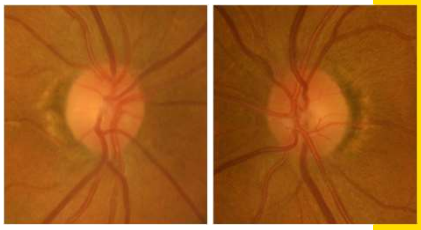
Case 4 – 70F







Symmetric VF defects

Symmetric OCT macula defects

Not cupped

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Coming Up - Interactive poll #5

Have Your Device or Browser Ready

Virtual Delegates – Scroll Down On Your Device To Interact Polls

Or go to www.slido.com & enter code: 3450 959

Or Scan QR Code:

125

slido

Poll 5. Next most appropriate management step for a patient with history of head trauma who has a homonymous right superior quadrantonopia on examination?

ⓘ Start presenting to display the poll results on this slide.

126

Case 4 70F

MRI BRAIN AND SKULL BASE

CLINICAL HISTORY:
MVA on the left side with muscle weakness. Bilateral superior right quadrantanopia.

TECHNIQUE:
3T high resolution imaging of the brain, skull base and orbits.

COMPARISON:
No previous similar imaging.

FINDINGS:
There is gliosis and encephalomalacia over the left temporal lobe from prior insult, presumed trauma. A few non-specific high FLAIR/T2 signal intensities in the bilateral white matter compatible with mild chronic microvascular ischaemia. No acute infarction or haemorrhagic sequela. There is no abnormal intracranial enhancement.

There is slightly more prominent CSF within the right optic nerve sheath, although the optic nerve volumes are preserved and the bilateral orbits are otherwise unremarkable. No intra-orbital mass lesion or abnormal enhancement. The pituitary gland is not enlarged measuring approximately 4 mm in height. No compression, shift or mass effect on the optic chiasm.

The bilateral mastoid air cells and paranasal sinuses are clear.

IMPRESSION:
Old post-traumatic insult to the left temporal lobe. Otherwise, no acute intracranial or intraorbital pathology.

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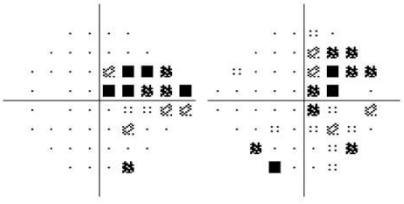
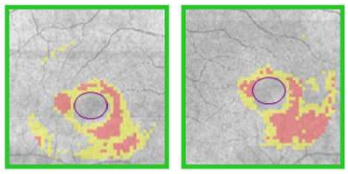
Case 4 – 70F

- Symmetry of VF and OCT findings suspicious for lesions beyond the retina
- Neuroimaging and radiology is the gold standard for lesions beyond the eye





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Ancillary testing: summary

- Other ocular imaging techniques may reveal characteristic findings in non-glaucomatous disease
- Have a high index of suspicion in cases of visual field and OCT symmetry for lesions beyond the eye

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The most important point in this talk:

Glaucoma is a diagnosis of exclusion!

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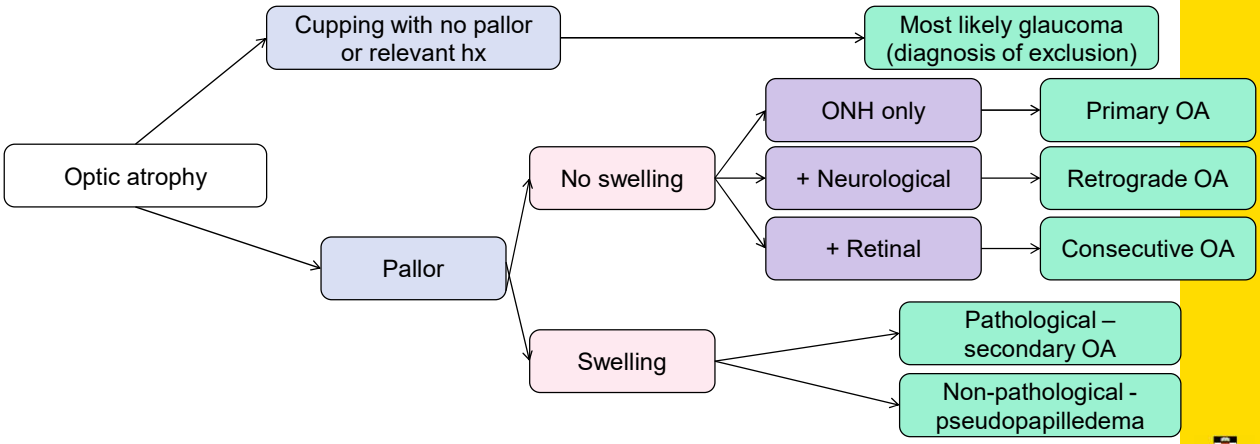
Types of optic atrophy



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Thank you – questions?

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