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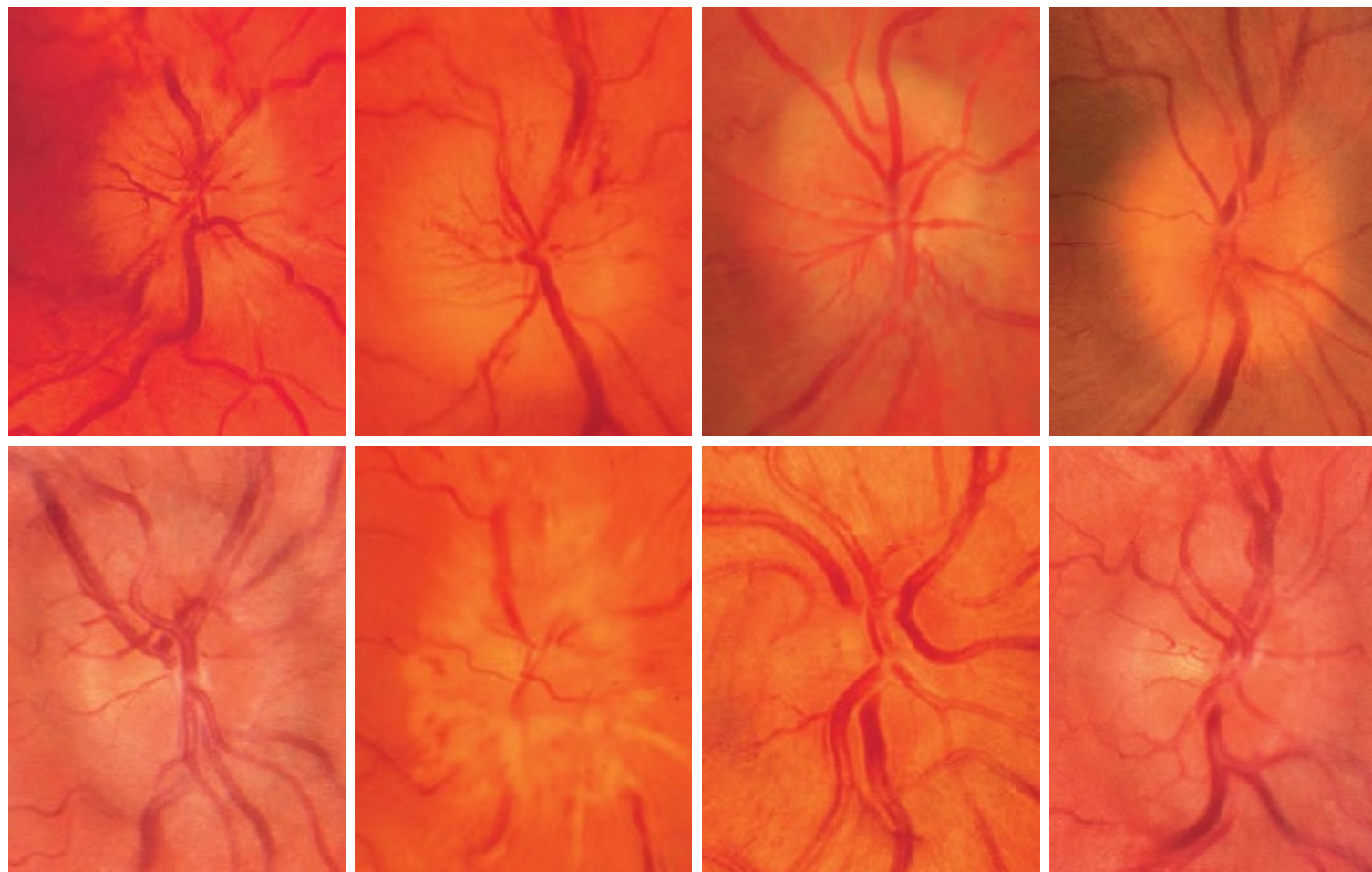


Neurology survival guide for ECOs

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Spot the diagnosis



Save me!



Kobayashi Maru

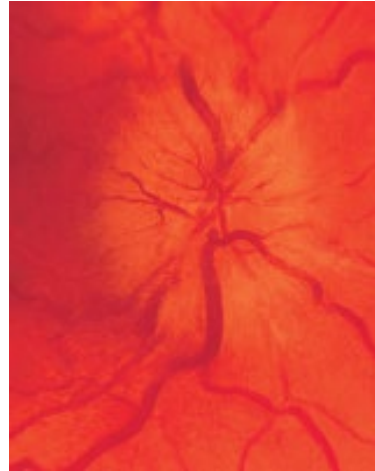
Spot the diagnosis



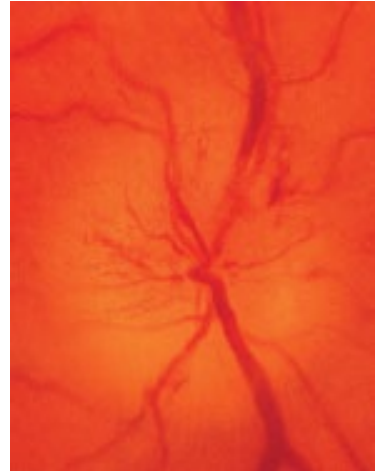
Save me!

Kobayashi Maru

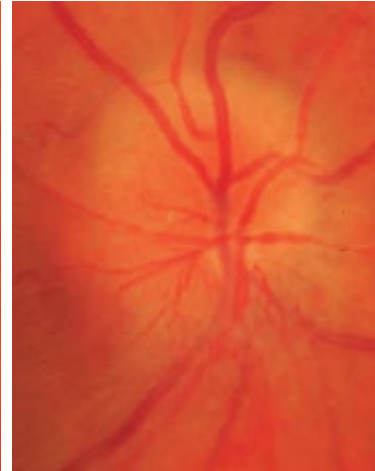
sarcoid
optic neuritis



Na-AION



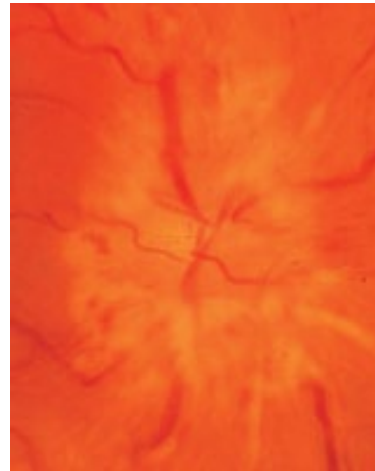
cat
scratch disease



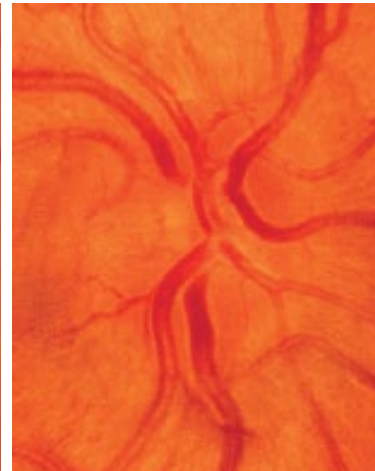
ON sheath
meningioma



lymphoma
infiltration
of optic nerve



papilloedema



Leber's
(LHON)



idiopathic
optic neuritis

Twenty 'rules' of survival



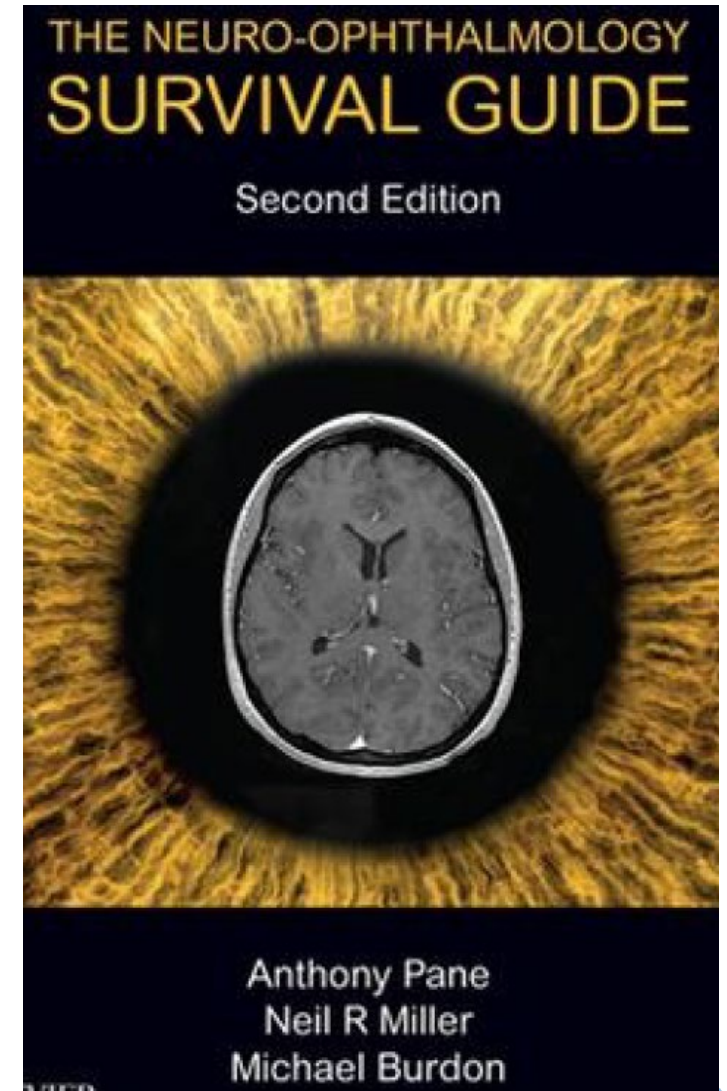
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USA



Dr Michael Burdon
Queen Elizabeth
Hospital Birmingham
UK



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Health2GO

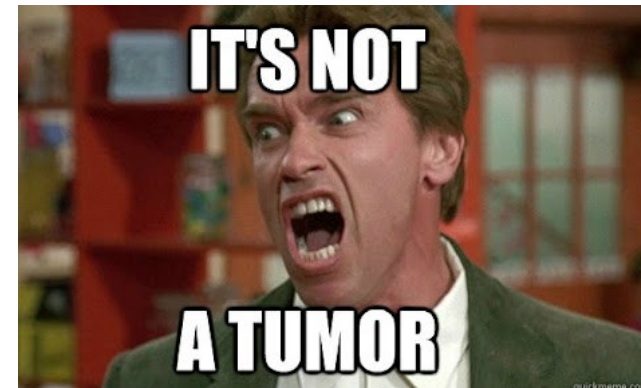
Twenty 'rules' of survival

- Rule #1. Beware the silent neuro-ophthalmic e.g. px with 'cataracts' or 'glaucoma'
- Rule #2. Every new px complaining of blurred vision should have a **confrontation, RAPD and perimetry** if abnormal
- Rule #3. You can never diagnose the cause of optic nerve dysfunction **just by looking** at the disc



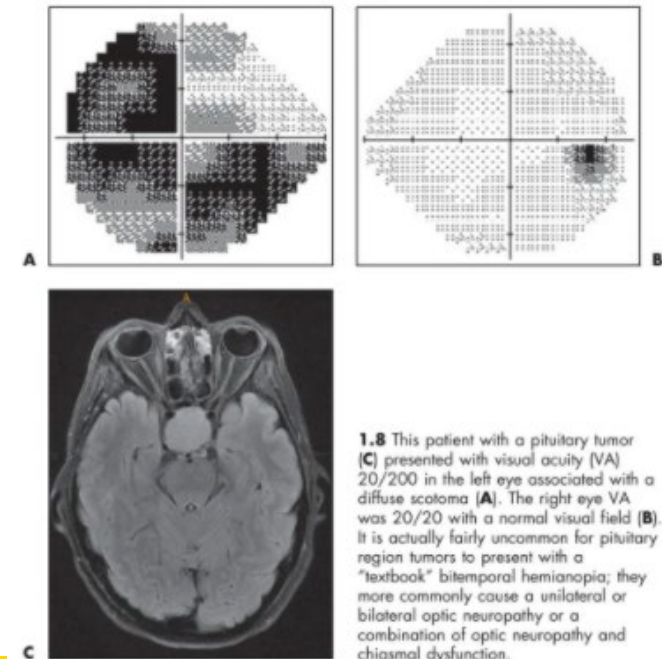
Twenty 'rules' of survival

- Rule #4. All px with **ACUTE** optic nerve dysfunction who do not meet all the clinical diagnostic criteria for either optic neuritis or AION require urgent referral
- Rule #5. All px with **CHRONIC ON** dysfunction who do not meet the clinical diagnostic criteria for glaucoma require urgent referral
- Rule #6. Amblyopia is a specific diagnosis – should not have ON disease and a **demonstrable amblyogenic factor**



Twenty 'rules' of survival

- Rule #7. When looking into an eye, is the level of vision explained by visible intraocular disease. **If not, is there disease behind the eye** e.g. unexplained poor vision, optic atrophy, disc cupping, VF loss
- Rule #8. Compressive optic neuropathy from orbital or brain tumour can present with **ANY optic disc appearance**



Twenty 'rules' of survival

- Rule #9. Just because normal looking eyes and normal RAPD – take all complaints of visual loss seriously. **Do not assume px has non-organic/'functional' vision loss** e.g. Streff syndrome, malingerers. Causes could be in brain.
- Rule #10. Bilateral disc swelling – **think papilloedema**. Same day MRI and MR venography to exclude tumour and dural venous sinus thrombosis
- Rule #11. Idiopathic intracranial hypertension is diagnosed based on SPECIFIC criteria – MRI + MRV + lumbar puncture



Twenty 'rules' of survival

- Rule #12. Beware of double vision – **don't assume longstanding. Acquired strabismus** should be treated with suspicion
- Rule #13. **Unexplained double vision** – needs to be referred for neuroimaging urgently if acute onset to rule out aneurysm, tumour or myasthenia
- Rule#14. Partial and complete CN3 palsy – **all require urgent MRI and MRA** to exclude aneurysm. Don't assume 'ischaemic' if pupil 'spared' or diabetic. For it to be ischaemic - need to meet ALL criteria for ischaemic CN3 (Slide 31)



Twenty 'rules' of survival

- Rule #15&16. All CN4 and CN6 require MRI to exclude brain tumour unless congenital (CN4) or ischaemic (CN6)
- Rule #17. Giant cell arteritis – **think of GCA in all over 50 yo with TIA** ('amaurosis fugax'), sudden and persistent vision loss, AION ON swelling, CRAO, transient or persisting double vision or *normal fundus* (retrobulbar ischemic ON)



Twenty 'rules' of survival

- Rule #18. MRI is superior to CT. Normal CT does not exclude serious disease. MRI should be requested with 'contrast' (typically gadolinium)– require specialist referral **not GP**
 - MRA/CTA – angiography to rule out aneurysms in **CN3 crises**
 - MRV/CTV – venography to rule out venous sinus thrombosis in **papilloedema**
 - Duplex doppler carotids to rule out ICA stenosis in **TIA**



Twenty 'rules' of survival

- Rule #19: Ophthalmic emergencies
- **Life threatening (in order of importance)**
 - **Double vision due to partial or complete CN3 palsy** which aneurysm cant be excluded
 - Bilateral ON swelling due to **brain tumour** or dural venous sinus thrombosis
 - Unilateral or bilateral **ophthalmoplegia**: think acute myasthenia, pituitary bleed, cavernus sinus thrombosis, carotid-cavernous fistula or orbital cellulitis
 - Ptosis or diplopia with **shortness of breath**, loss of swallow reflex: think severe myasthenia
 - Ptosis with ipsilateral small but reactive pupil from horner syndrome related to acute internal carotid dissection (**painful horners**)



Twenty 'rules' of survival

- Rule #19: continued (non-life threatening)
- Bilateral permanent sight threatening
 - Acute vision loss from **GCA**, pituitary apoplexy or neuromyelitis
- Unilateral sight threatening
 - Acute glaucoma, endophthalmitis, penetrating eye injury and sinus mucocele

Twenty 'rules' of survival

- Rule #20: Top 3 Common mistakes
 - Not suspecting possibility of orbital or brain disease as the cause of px 'eye' complaints
 - Not performing a thorough hx and examination
 - Not referring to a neuro-ophthalmologist early and urgently when necessary



Emergency and Urgent Definitions

- Emergency (<24h or asap)
 - ring up and ask to speak with the **registrar** *before* sending patient in **immediately**
- Urgent (24h – 6 days)
 - RVEEH Cat 1A, FMC Cat 1
 - Urgent <2 weeks (SA) and <4 weeks (Vic: Cat 1B)
- Routine (>3 months)

2. Clinic Timeframe Categories

The following table gives an indication of the timeframe within which patients of different acuity are expected to be seen.

Category	Definition
Emergency	<p>A patient whose condition is identified from referral details as having an acute sight or life threatening condition where immediate medical or surgical intervention is required</p> <p><i>Discuss with the Admitting Officer in the Emergency Department – call switch on 9929 8666 – to confirm immediate referral to the Emergency Department</i></p>
Urgent: (within 1 week) Waiting list: Category 1A	<p>A patient whose condition is identified from referral details as having the potential to deteriorate quickly to the point that it may become an emergency.</p>
Urgent: (1 week to 30 days) Waiting list: Category 1B	<p>A patient whose condition is identified from referral details as having the potential to deteriorate quickly, with significant consequences for health and quality of life, if not managed promptly.</p>
Routine (30-90 days) Waiting list: Category 2	<p>A patient whose condition is identified from referral details as causing some pain, dysfunction or disability, but which is not likely to deteriorate quickly or become an emergency.</p>
Routine: (90-365 days) Waiting list: Category 3	<p>Patients whose condition is identified from referral details as being unlikely to deteriorate quickly and does not have the potential to become an emergency.</p>
Primary Care - not accepted	<p>Patients whose condition is identified from referral details as requiring primary care, and not reaching the threshold criteria for the hospital's specialist services. Refer to the Primary Care Management Guidelines.</p> <p>Patients over 45 years of age should have regular eye examinations with an ophthalmologist/optometrist every three years.</p>



Emergency and urgent referrals

(SA Health – Royal Adelaide Hospital & Victoria - RVEEH)

- Emergency by diagnosis
 - Acute angle closure glaucoma
 - Central retinal artery occlusion
 - Retinal detachment
 - Orbital cellulitis
 - Papilloedema (raised ICP)
 - Giant Cell Arteritis (AAION)
 - FB/penetrating/chem burns/trauma/transplant rejection

Emergency <small>All urgent cases must be discussed with the on call ophthalmology registrar.</small>	Category 1 <small>All urgent cases must be discussed with the on call ophthalmology registrar.</small>	Category 2 Target within 3 months	Category 3 Target 6-12 months
<ul style="list-style-type: none"> > Acute angle closure glaucoma > Central retinal artery occlusion > Chemical injury > Conjunctivitis >1/52 > Conjunctivitis with pain > Corneal ulceration > Flashes / floaters with field loss, > retinal detachment > Orbital cellulitis / acute dacryocystitis > Periocular herpes simplex/herpes zoster infection > Post-operative / post intra-ocular injection inflammation > Pupil changes > Red eye in contact lens wearer > Sudden loss of vision or diplopia > Sudden onset of blurred vision with headaches > Acute loss of visual field > Suspected penetrating eye injury > TIA / amaurosis fugax > Trauma to eye/orbit > Uveitis > Sudden onset of double vision 	<ul style="list-style-type: none"> > Acute dacryoadenitis > Diabetes if with recent visual loss > Distortion or vision loss in ARMD patient. > Elevated intraocular pressure ≥ 30 mmHg > Eye pain > Flashes / floaters without field loss > Proptosis with visual changes > Ptosis if pupil occluded > Severe light sensitivity > Squint if red reflex abnormal or lack of visual response > Known diabetic with drop in vision 	<ul style="list-style-type: none"> > Cataract with vision <6/12 in better eye > Elevated Intraocular pressure <30 mmHg > Eye discharge without redness > Eyelid problems: ectropion / entropion / ptosis. > Eyelid tumours > Glaucoma suspects high risk (high cup-disc ratio, glaucomatous visual field defect, shallow anterior chamber). > Proptosis without visual issues (Thyroid eye disease) > Severe dry eyes (Sjogren's, Rheumatoid arthritis) 	<ul style="list-style-type: none"> > Annual chronic disease review > Age related macular degeneration without visual distortion. > Blepharitis / dry eye > Cataracts with vision better than 6/12 in better eye > Glaucoma suspects low risk (normal cup-disc ratio, family history) > Routine keratoconus > Other eyelid lumps > Referral for ongoing care from elsewhere > Screening for eye disease > Systemic eye disease > Vision <6/12 for any reason other than refractive > Watery eyes



Neuro **things** that increase our index of suspicion

- **Unexplained** blurred vision symptoms or **field loss**
- **Transient vision loss**
- **Headaches**
- Double vision
- Ptosis
- Unequal **pupils**
- Colour vision (RG) loss



Blurry vision

- Not just VA
 - Many present with blurry vision but **relatively good VA (>6/12)**
 - ONTT showed 11% presented with perfect VA ($\geq 6/6$) (Beck et al., 2003)
- Onset
 - **Sudden** vs gradual
 - Worse with certain activity e.g. sports, postural change
- Monocular
 - Monocular: optic neuritis/tumour/**GCA**/OIS/Amaurosis fugax/retinal migraine
 - Rule out intermittent AACG, dry eyes, retinal disease



Blurry vision

- Binocular
 - Think migraine (most common)
 - **Papilloedema**
 - chiasmal/post-chiasmal lesions
 - neck/heart problems (embolisms/carotid dissection/vertebrobasilar insufficiency)
 - Uthoff sign (heat in MS)

Blurry vision

- Transient monocular (<24 hours)
 - Amaurosis fugax: vision loss 5-60 min(< 1hour), age >50, vascular risk factors, normal eye and vision.
 - Immediate danger: ICA dissection (painful horner syndrome) or GCA
- Fundus Normal, no pain, >60yo: Think of impending stroke: ICA stenosis
 - 10-20% within 90 days
 - 50% within 48 hours
 - Refer to GP for blood workup BP, diabetes, carotid doppler

Predicting stroke within 48 hours using ABCD²

Table 2. ABCD² tool

Age

60 years or older (1 point)

Blood pressure

Systolic ≥140 mmHg (1 point)

Diastolic ≥90 mmHg (1 point)

Clinical features

Any unilateral weakness (2 points)

Speech impairment without weakness (1 point)

Duration

60 minutes or more (2 points)

10–59 minutes (1 point)

Diabetes mellitus (1 point)

ABCD² >4 = high risk, ≤4 = low risk (maximum is 7)²³

Source: RACGP

True stroke:

Face droop, Arm weakness,
Speech difficulties, Time to call
000 (FAST)



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Blurry vision

- Transient **binocular**
 - Think postural hypotension in **young**
 - Think vertebrobasilar insufficiency from atherosclerosis in **elderly**
 - Heart disease e.g. patent foramen ovale or transient arrhythmia
- Chronic (>24 hours)
 - Stroke, tumour, brain aneurysm
- Scotoma type
 - whole monocular field: think amaurosis fugax/TIA
 - scintillating – ‘flashes’, ‘wavy’/’watery’, ‘sparkly’: think migraine
 - Usually starts centrally and **expands**



Visual field test

- Confrontation
 - Always do **simultaneous finger counting** (Anderson et al., 2009)
 - quicker and increases likelihood of detecting hemi-extinction (as opposed to hemi-anopia/neglect)



CLINICAL AND EXPERIMENTAL
OPTOMETRY

Optometry AUSTRALIA IN PARTNERSHIP WITH NZAO The New Zealand Society of Professional Optometrists

Free Access

Rapid confrontation screening for peripheral visual field defects and extinction


Andrew J Anderson, Neil H Shuey, Michael Wall

First published: 29 December 2008 | <https://doi.org/10.1111/j.1444-0938.2008.00280.x> | Citations: 7

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Volume 92, Issue 1
January 2009
Pages 45-48

Figures References Related Information

Recommended

[Binocular summation in the peripheral visual field](#)
Angela Whitaker, Shahina Pardhan
Ophthalmic and Physiological Optics

[043.25 ARE VISUAL FIELD DEFECTS IN NEGLECT PATIENTS SIMULATED BY THE PRESENCE OF THE FIXATION POINT IN STANDARD PERIMETRY?](#)
E. M. Mueller-Oehring, E. Kasten, D. A. Poggel, T. Schulte, B. A. Sabel
European Journal of Neuroscience



Visual field test

- Confrontation (finger counting)
 - Poor sensitivity 35%
 - 100% specificity

Effectiveness of testing visual fields by confrontation

Ranjeet J Pandit, Kevin Gales, Philip G Griffiths

Many tests are used to examine visual fields by confrontation, but such methods have not been thoroughly compared with an accepted reference standard. The choice of test might affect the identification of subtle defects in the visual field. We prospectively compared seven confrontation field tests with full-threshold automated static perimetry among 138 outpatients in an eye clinic. Our primary outcome was detection of a defect in the visual field. With automated perimetry, most field defects were small or shallow. Most confrontation field tests were insensitive in the identification of field loss. The most sensitive method was examination of the central 20° visual field with a small red target (73% [95% CI 63–82]). Assessment of the visual field should thus include such a test.

Lancet 2001; 358: 1339–40

Visual-field loss is routinely identified by testing visual fields by confrontation. Defects in the visual field are rarely

Automated fields	Patients*
Normal	49 (36%)
Abnormal	89 (64%)
Arcuate scotomast†	33 (24%)
Nasal step‡	13 (9%)
Concentric constriction	11 (8%)
Central scotomas	9 (7%)
Patchy central defects	8 (6%)
Altitudinal defects	6 (4%)
Isolated temporal defects	6 (4%)
Isolated nasal defects	2 (1%)
Bitemporal hemianopias	2 (1%)
Total	138 (100%)

*Number of patients with field defect in one or both eyes. One patient had an arcuate scotoma in one eye, and a nasal step defect in the other. †Points missed on automated perimetry in a region corresponding to the arcuate scotoma seen in patients with glaucoma. ‡Points missed on automated perimetry in a region corresponding to the nasal step defect seen in patients with glaucoma.

Table 1: Results of automated perimetry

Pandit et al., 2001 *Lancet*

	True positives	False positives	True negatives	False negatives	Sensitivity (95% CI)	Specificity
Confrontation test						
Description of examiner's face	39 (28%)	0	49 (36%)	50 (36%)	44% (33–55)	100%
Quadrant finger counting	31 (22%)	0	49 (36%)	58 (42%)	35% (25–46)	100%
Kinetic to finger	36 (26%)	0	49 (36%)	53 (38%)	40% (30–51)	100%
Kinetic to 20 mm white target	43 (31%)	0	49 (36%)	46 (33%)	48% (38–59)	100%
Kinetic to 20 mm red target	50 (36%)	0	49 (36%)	39 (28%)	56% (45–67)	100%
Red colour comparison	53 (38%)	0	49 (36%)	36 (26%)	60% (49–70)	100%
Central field test to 5 mm red target	65 (47%)	0	49 (36%)	24 (17%)	73% (63–82)	100%
All tests combined	68 (49%)	0	49 (36%)	21 (15%)	76% (66–85)	100%

Table 2: Effectiveness of confrontation field tests*

sensitivity 35%



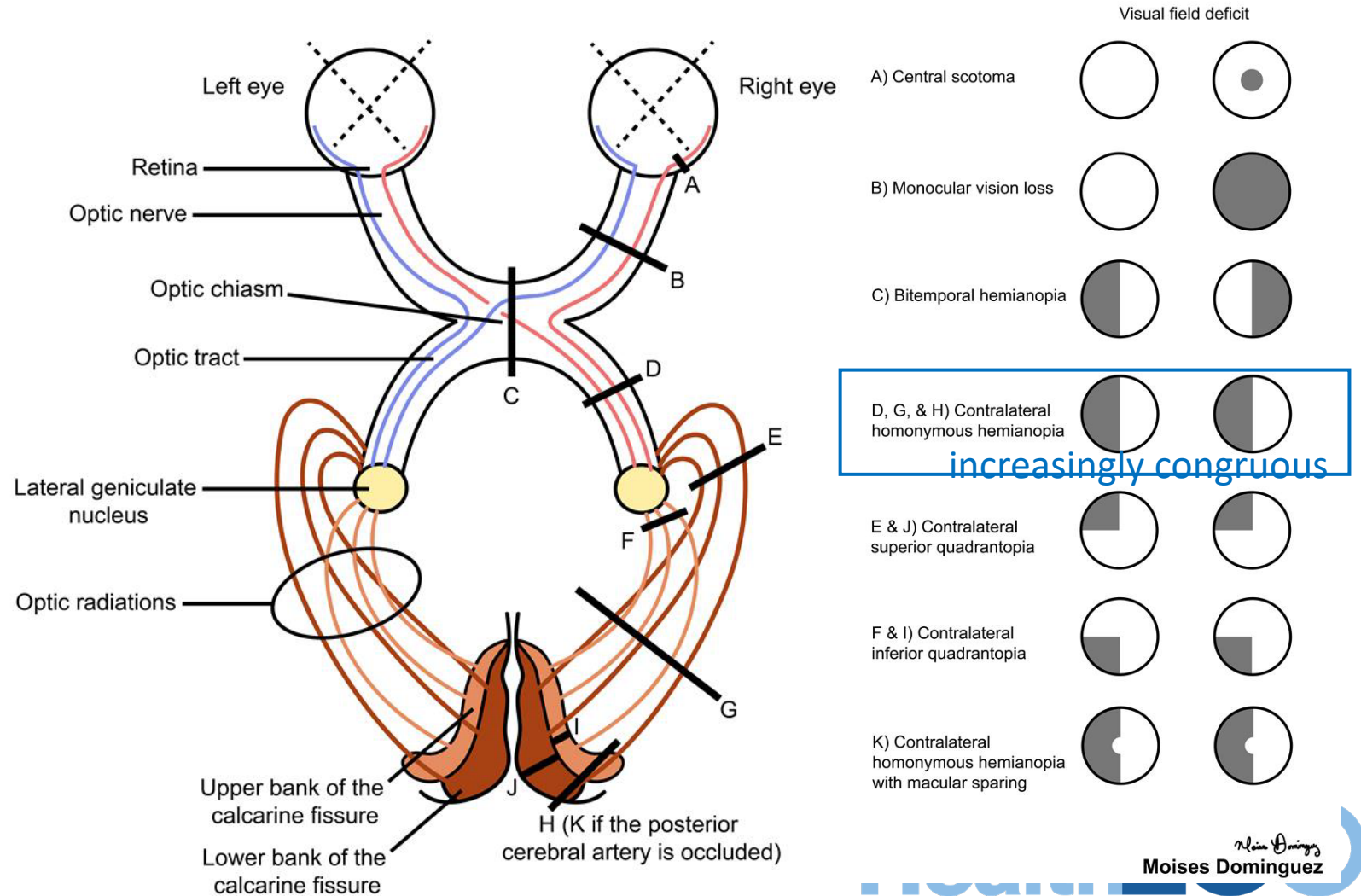
Visual field test

- Perimetry

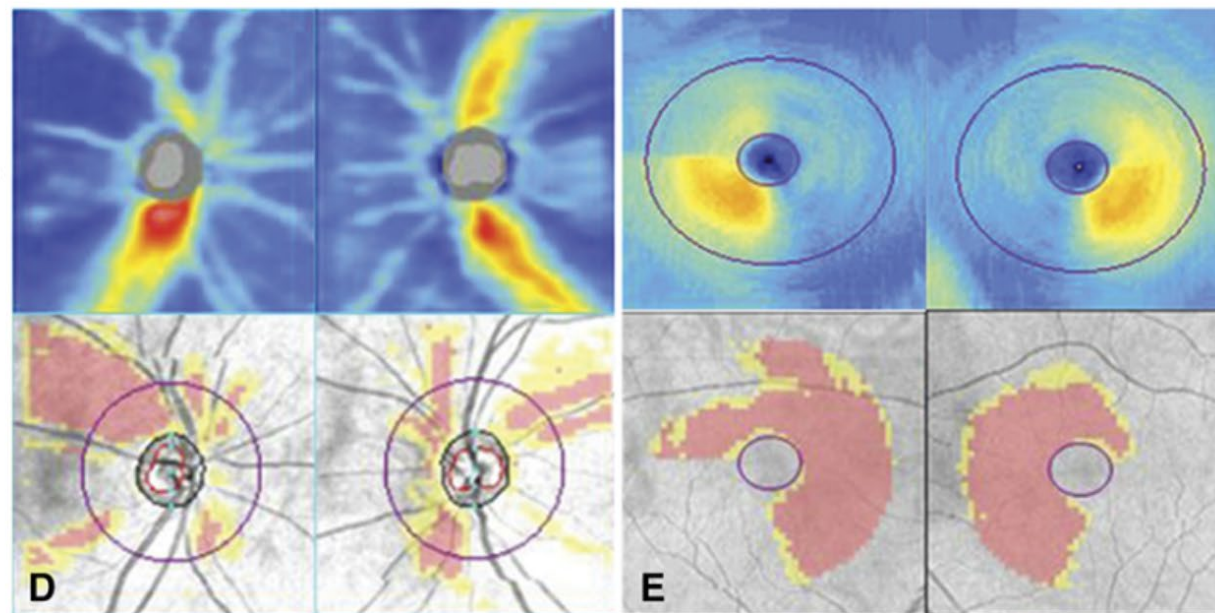
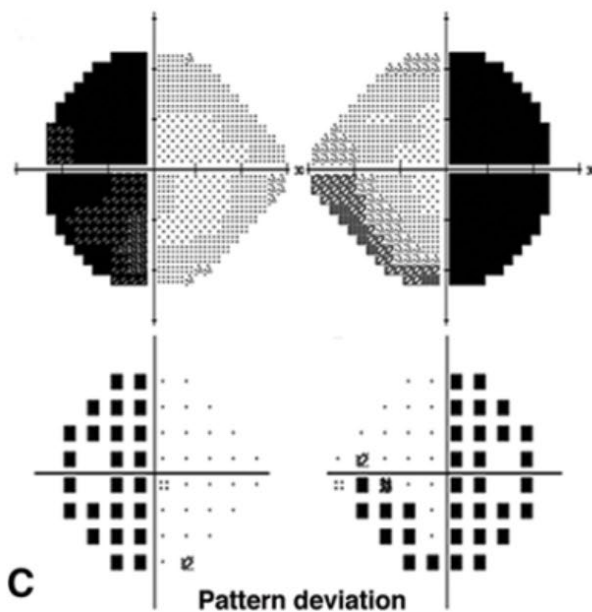
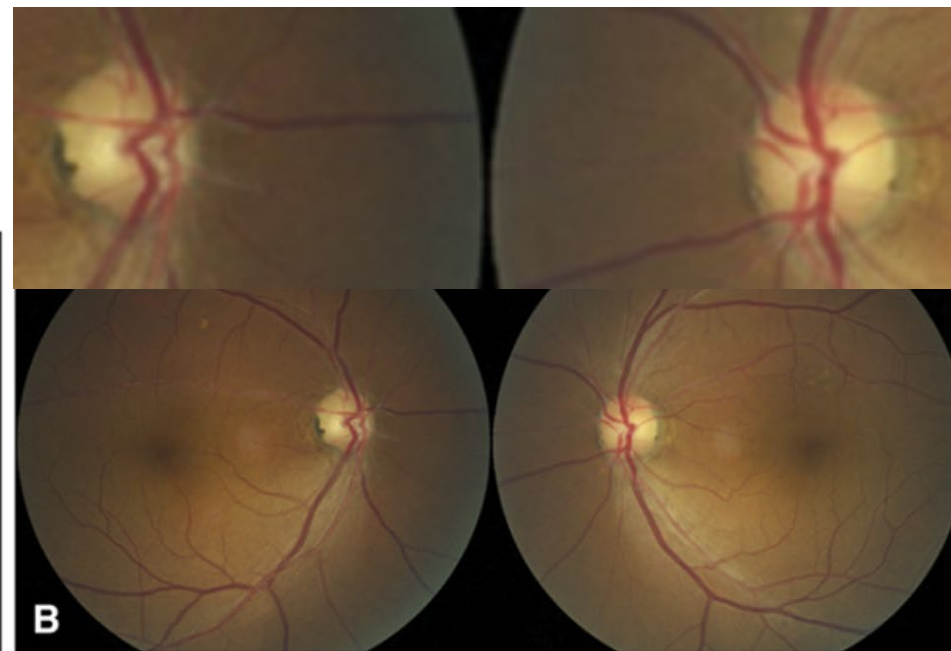
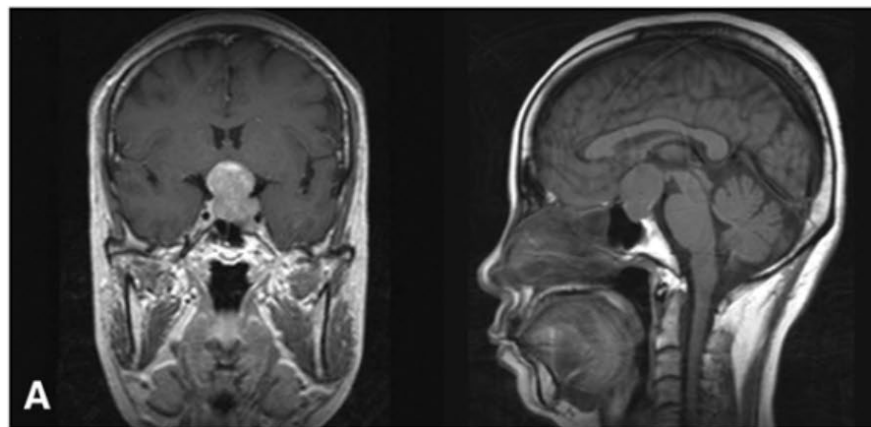
- Most commonly **pituitary tumour**
 - Binasal fibre death leads to classic **bow tie atrophy**



Visual Field Defects

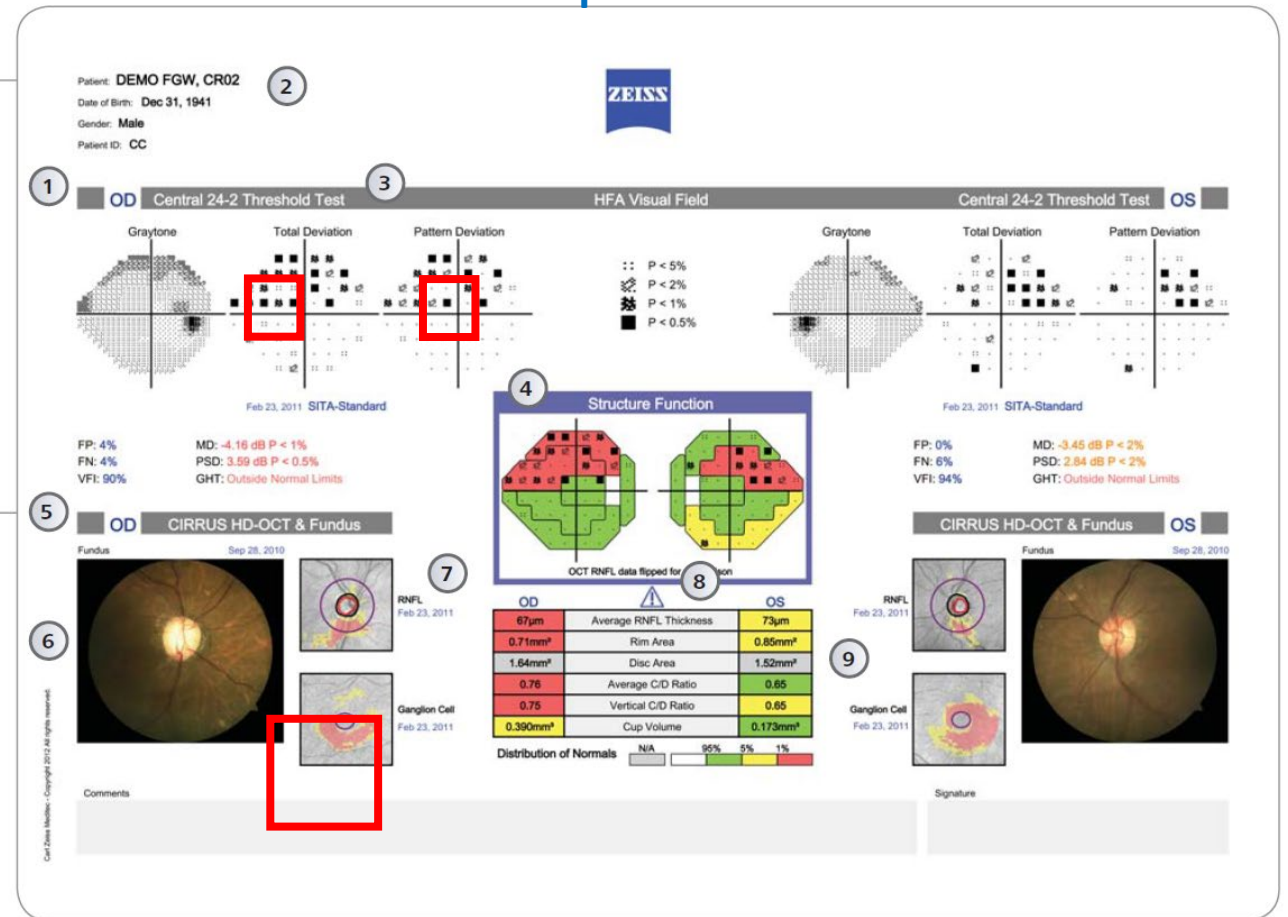


Visual field test



Visual field test

- Arcuate defects
 - Seen in multiple optic disc conditions
 - Optic neuritis
 - NA+A AION
 - Pituitary tumours (asymmetrical)
 - Crowded disc/ disc-at-risk
 - Optic disc drusen
- For **diagnosis of glaucoma**, **function must match structure**
- This includes **doing 10-2 for Mac GC-IPL/GCC loss** in the absence of RNFL loss



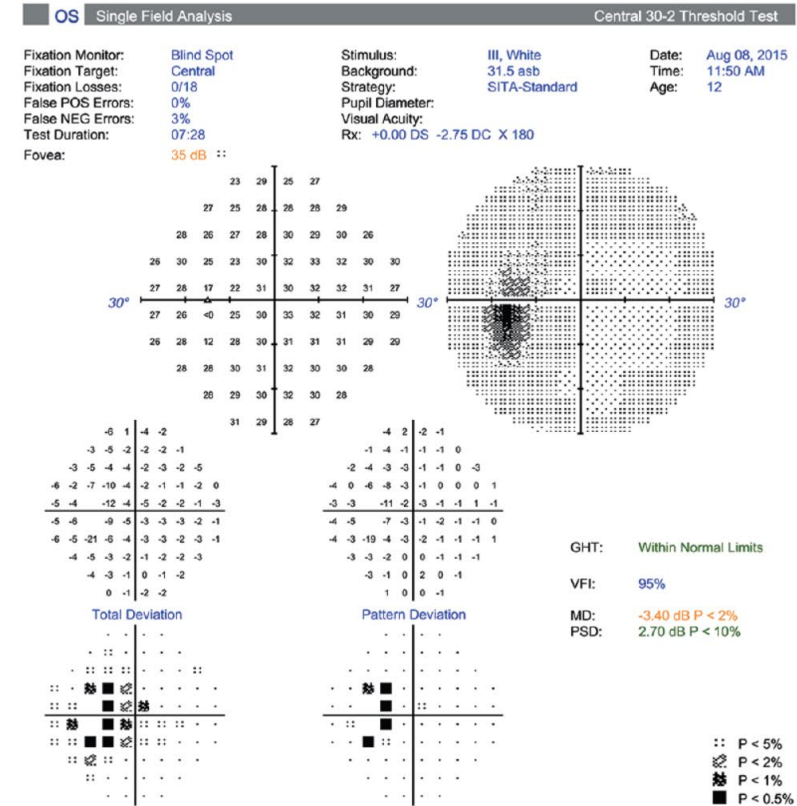
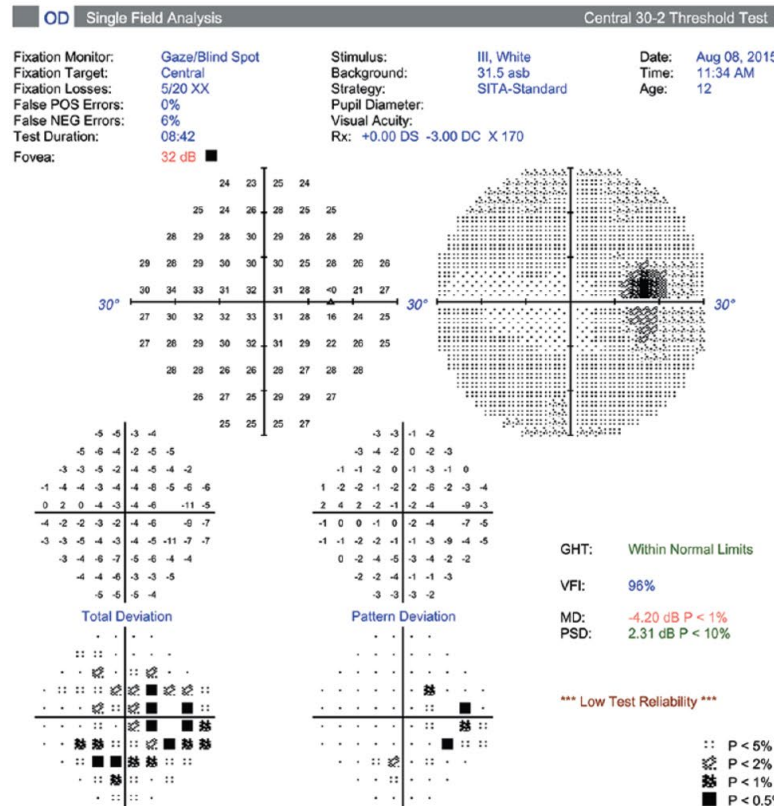
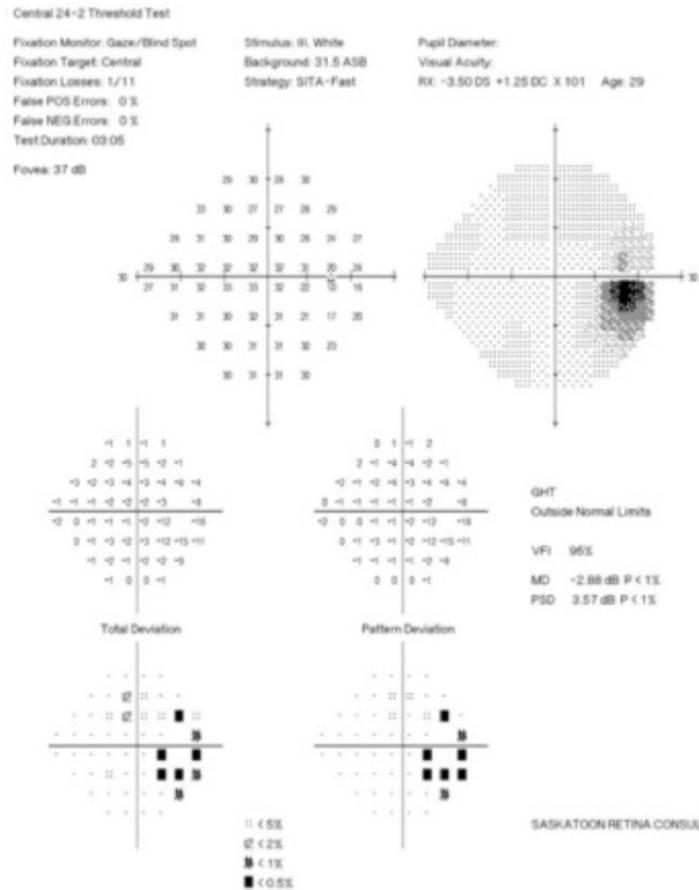
- 1 HFA Test Data
- 2 Patient Information
- 3 Visual Field Test Type
- 4 HFA and CIRRUS Combined Structure Function Section

- 5 CIRRUS OCT Exam and fundus photo section
- 6 Optional fundus photo.
- 7 RNFL and Ganglion Cell Thickness Deviation Maps (GCL + IPL)
- 8 Symbol appears Δ if at least one parameter is near a normative limit.
- 9 CIRRUS Data Table with ONH Summary and RNFL Parameters



Visual field test

- Papilloedema – beware of these VFs



Headaches

- Have they had a **prior diagnosis of migraine** when they were <40 years old
- Is it suggestive of raised ICP?
 - **Worse in AM** (supine position all night)
 - Rule out obstructive sleep apnoea
 - Nausea/vomiting
 - Pulsatile tinnitus
- GH/PMH
 - Obese women of childbearing age (3xFs)
 - Hx of cancer, DVT
 - Medication causing pseudotumour cerebri

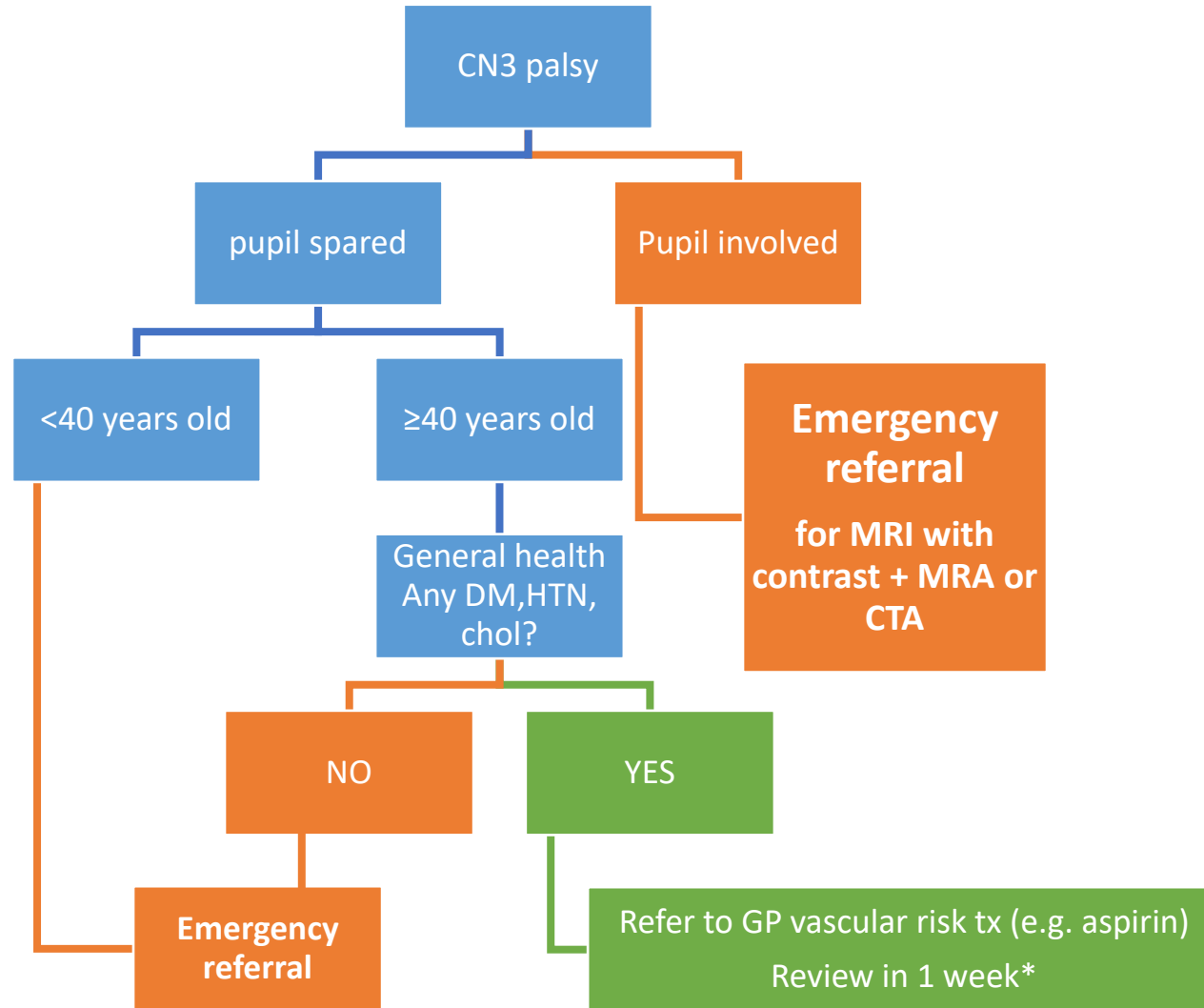


Pupils in 3rd nerve palsy

- Why do we care?
 - Most common cause is **ischaemic**
 - The most benign cause and often self limiting (except GCA)
 - Think **atherosclerosis, diabetes, hypertension and GCA**
 - **Up to 1/3** due to expanding/compressive **aneurysm** at junction of ICA and PCA
 - Can **kill px within hours to days if ruptured** hence regarded as **most urgent of all ophthalmic emergencies**

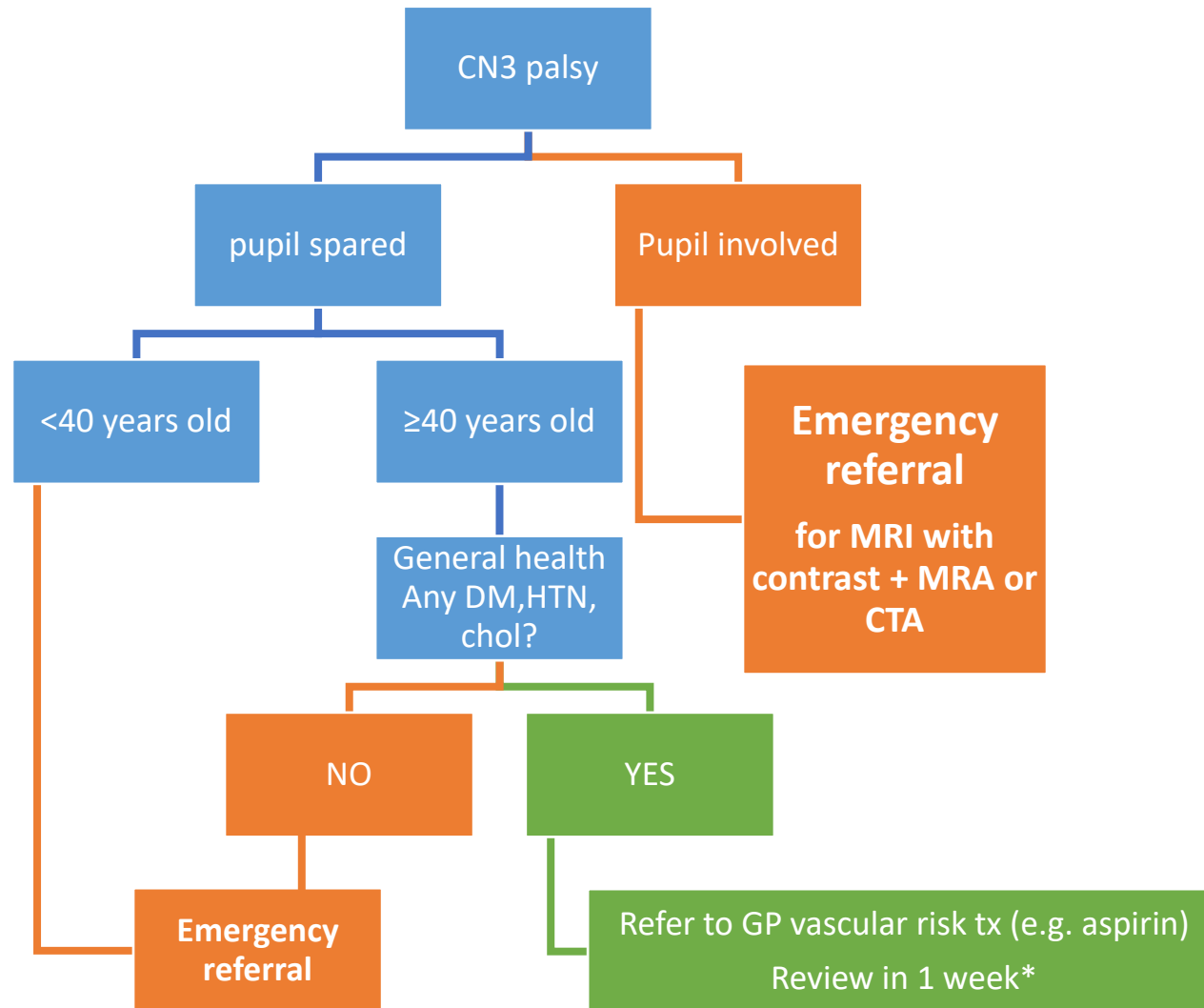


Pupils in 3rd nerve palsy flowchart



Courtesy of
Dr Mallika Prem Senthil
Ophthalmologist

Pupils in 3rd nerve palsy flowchart



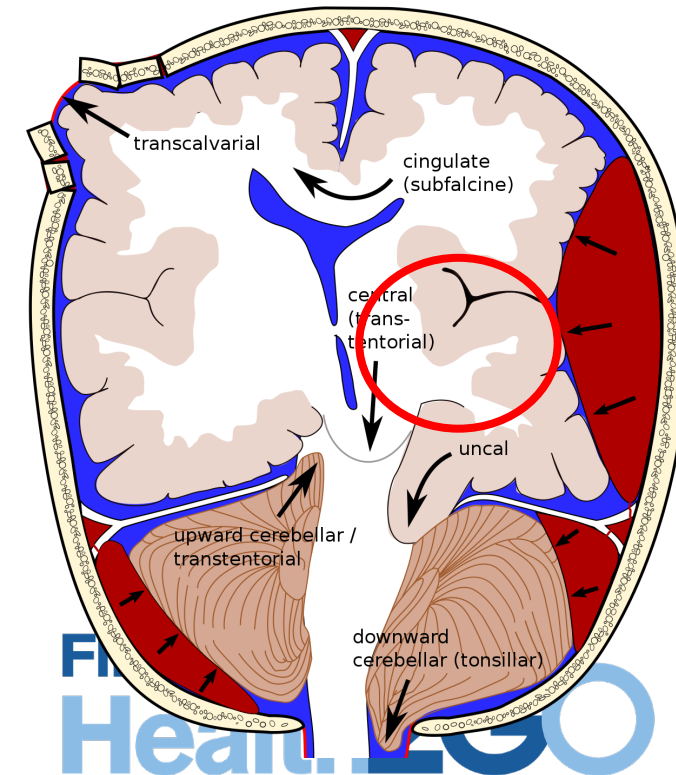
Ischaemic 3rd nerve palsy (must have the following)

- Time course: sudden diplopia or unilateral ptosis
- Ptosis becomes **complete*** within 24 hours
- Palsy must be **complete***
- No other nerve involvement, other side not involved
- No RAPD, normal confrontation
- No pain and no 'pins & needles'

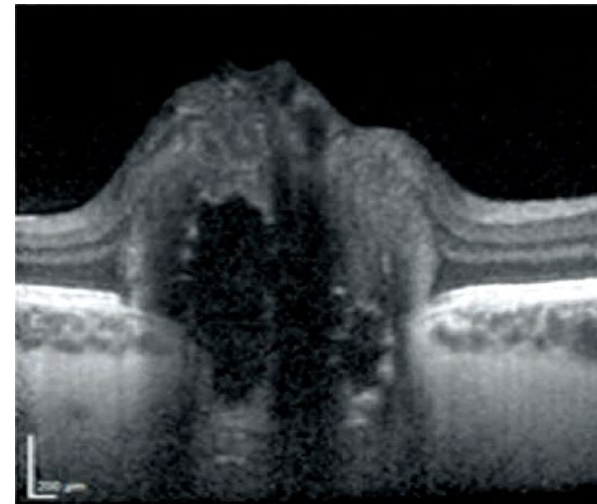
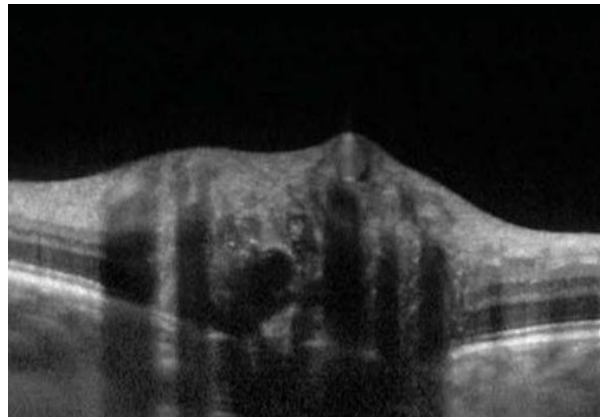
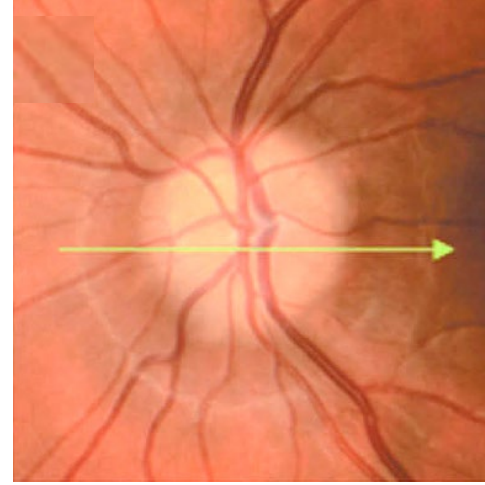
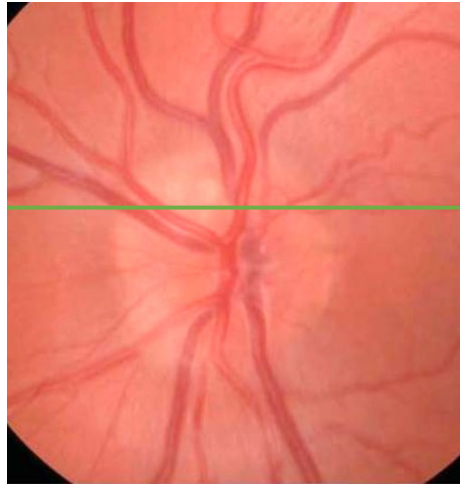
**this is because aneurysmal partial third nerve palsies may be "pupil sparing" early in their evolution*

Pupils in 3rd nerve palsy

- Other causes
 - Brain tumours esp pituitary tumours invading cavernous sinus
 - Raised ICP leading to uncal herniation
 - Infection (viral/postviral) and Inflammation (MS)
 - Trauma



Imaging a suspicious optic nerve

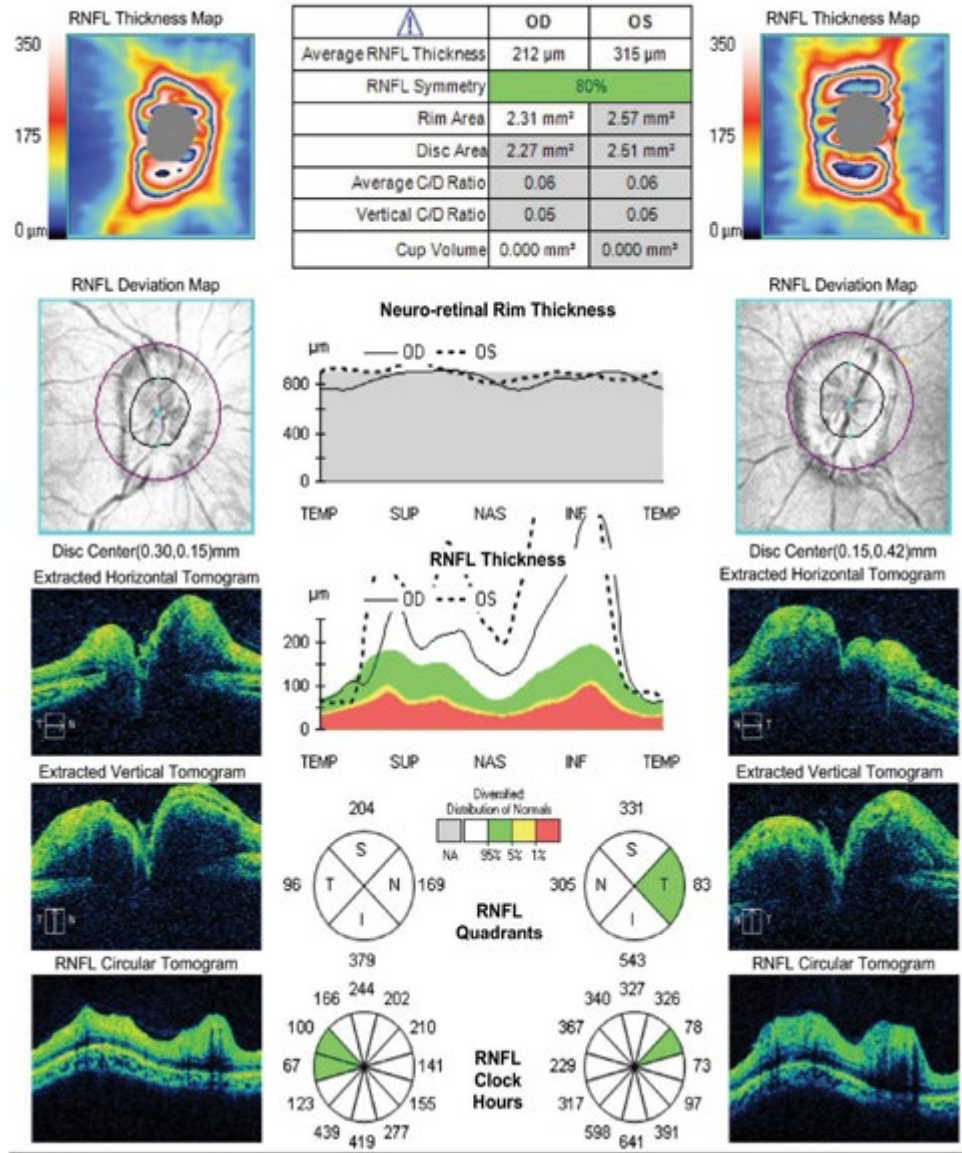


Imaging a suspicious optic nerve

- Optic Disc Drusen Consortium key concepts
 - Dilate px
 - Always use Enhanced Depth Imaging (EDI-OCT)
 - First described by Spaide et al., 2008
 - Penetrates anterior portion of optic disc, allows visualisation of deeper structures
 - **Volume scan** of optic disc **Radial scan** 6 lines 'star' with EDI
 - Alternatively: HD 5 line raster with EDI
 - Perform RNFL thickness scan – don't look for thinning, look for **thickening**
 - Perform macula scan – rule out macular disease & neuro disease



ONH and RNFL OU Analysis: Optic Disc Cube 200x200 OD ● ● OS



ODDC imaging recommendations

Before scanning	<p>Ensure optimal conditions</p> <ol style="list-style-type: none"> Dilate pupils before examination Measure corneal curvature and refraction for later transverse magnification adjustment (to ensure accurate measurements)*
Acquisition	<p>Visualize deeper structures</p> <ol style="list-style-type: none"> Use SD-OCT in EDI mode If no SD-OCT is available, adjust the distance from the OCT apparatus to the eye to get an inverted view of the optic nerve head for better visualization of deeper structures Type in corneal curvature value and refraction in the operator system*
Dense optic nerve head scan	<p>Identification, quantification and classification of ODD</p> <ol style="list-style-type: none"> Use EDI mode or invert scan Select high-resolution acquisition if possible Center a scan area of only $15 \times 10^\circ$ over the optic disc Scan with 97 sections in that area ($30 \mu\text{m}$ between each scan) Average at least 30 frames Perform the volume scan in both horizontal and vertical directions
Radial optic nerve head scan	<p>Assessment of scleral canal size</p> <ol style="list-style-type: none"> Use EDI mode or invert scan Select 20° 6-line radial scan (star pattern scan) Center scan at optic disc
Peripapillary scan	<p>Evaluation of RNFL thickness</p> <ol style="list-style-type: none"> Deselect EDI mode (if on) Select 12° peripapillary scan (circle scan) Center scan at optic disc
Macula scan	<p>Exclude macular pathology</p> <ol style="list-style-type: none"> Deselect EDI mode (if on) Center a scan area of $20 \times 20^\circ$ over macula. Scan with at least 25 sections ($240 \mu\text{m}$ between each scan) Average at least 9 frames
Autofluorescence	<p>Identification of autofluorescence positive ODD</p> <ol style="list-style-type: none"> Center the scan at the optic disc Average 100 frames



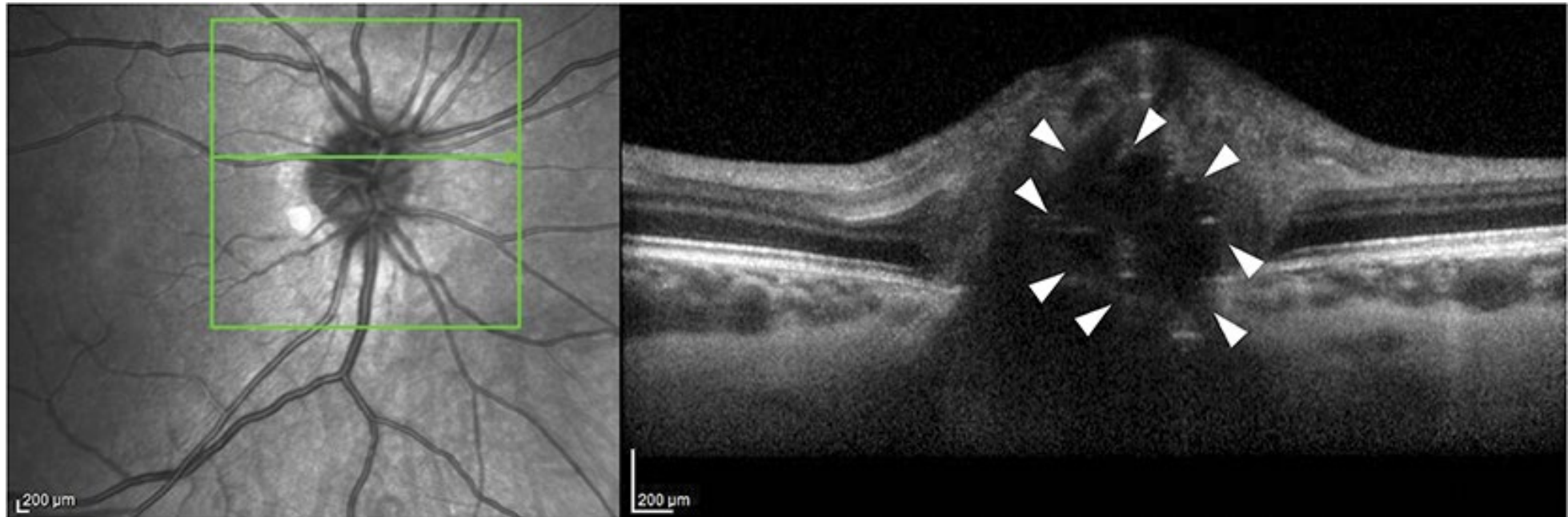
EDI-OCT makes optic disc drusen visible

Volume scan (no EDI)

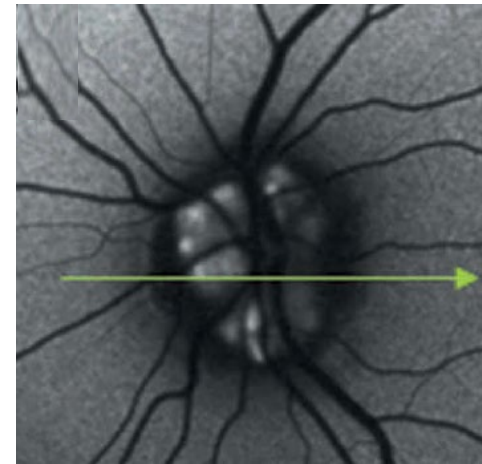
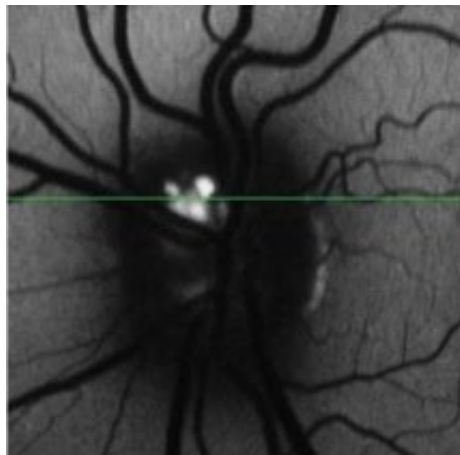
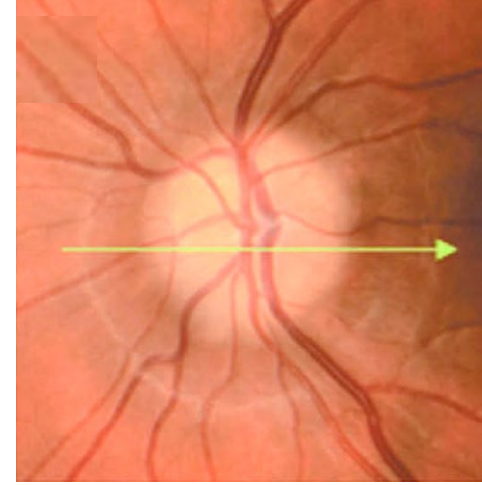
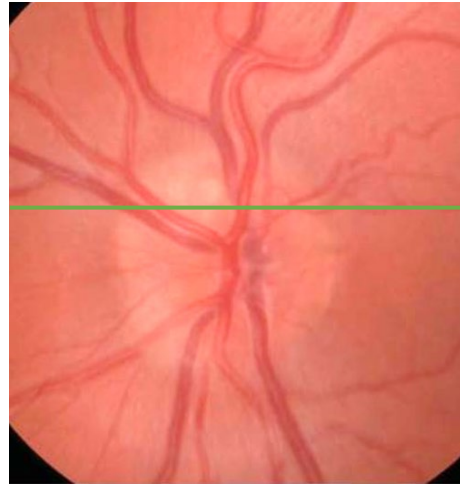


EDI-OCT makes optic disc drusen visible

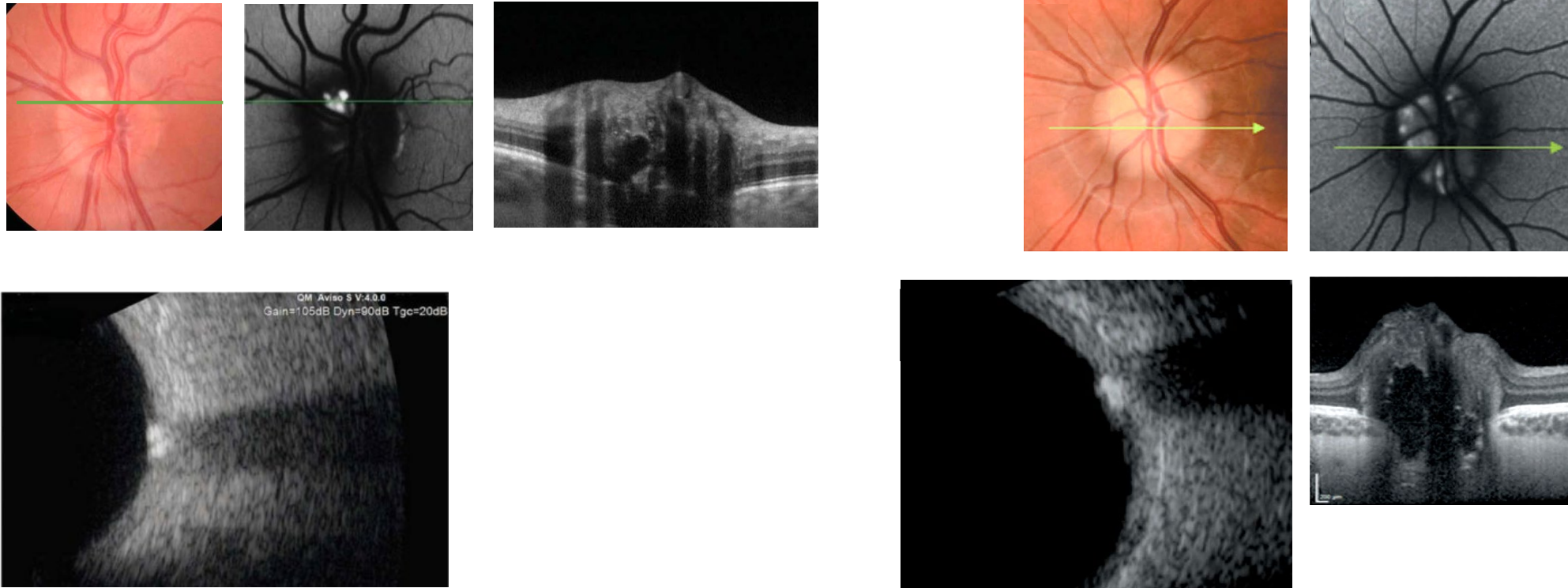
Volume scan (with EDI)



Fundus autofluorescence (FAF)



Multimodal imaging + ultrasound



Ultrasound a-scan remains the gold standard for optic disc drusen

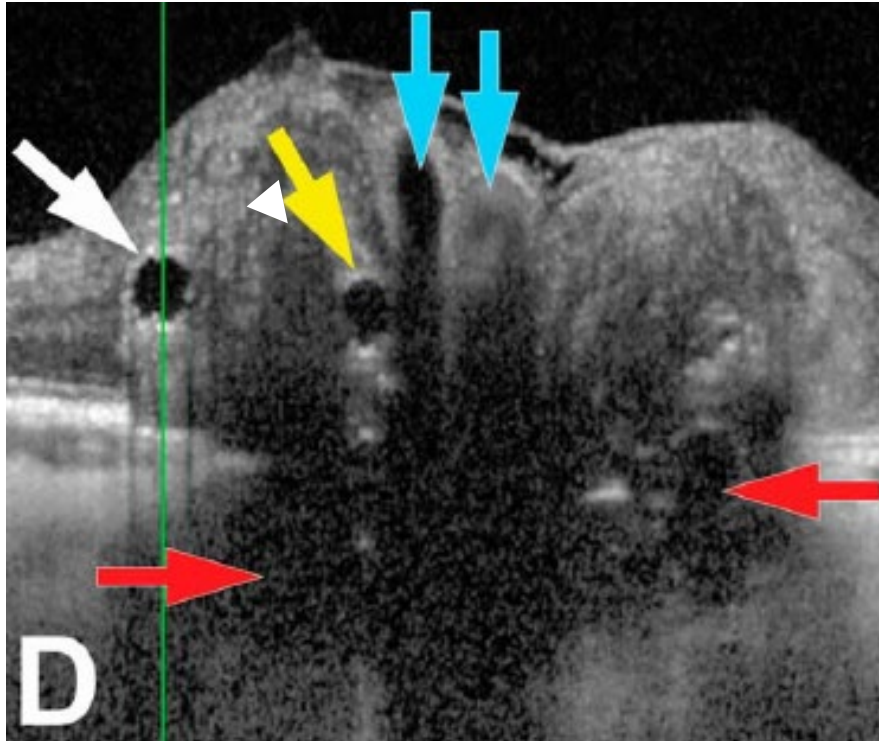


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Describe the features



ONH drusen appear as **hypore**reflective on OCT

- White arrow heads
- White arrow + yellow arrow
- Blue arrows = BV artefacts
- Red arrows = buried large drusen

Diagnosis: ONHD



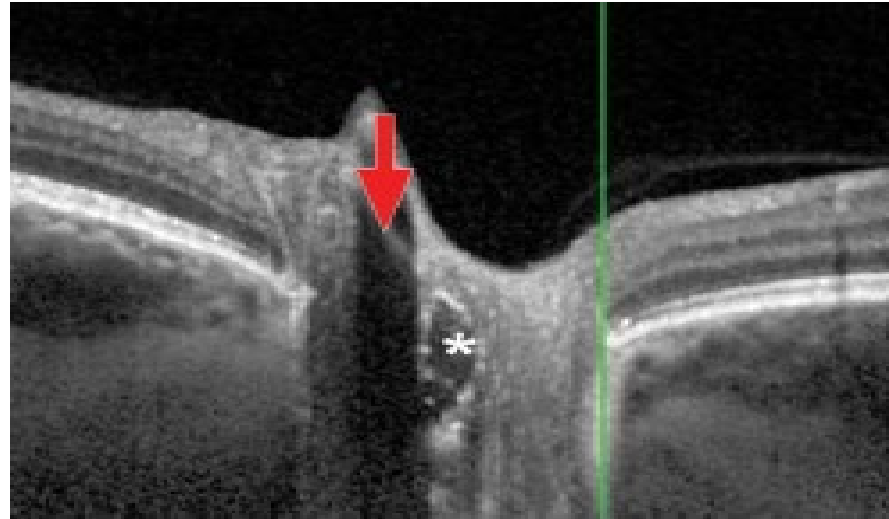
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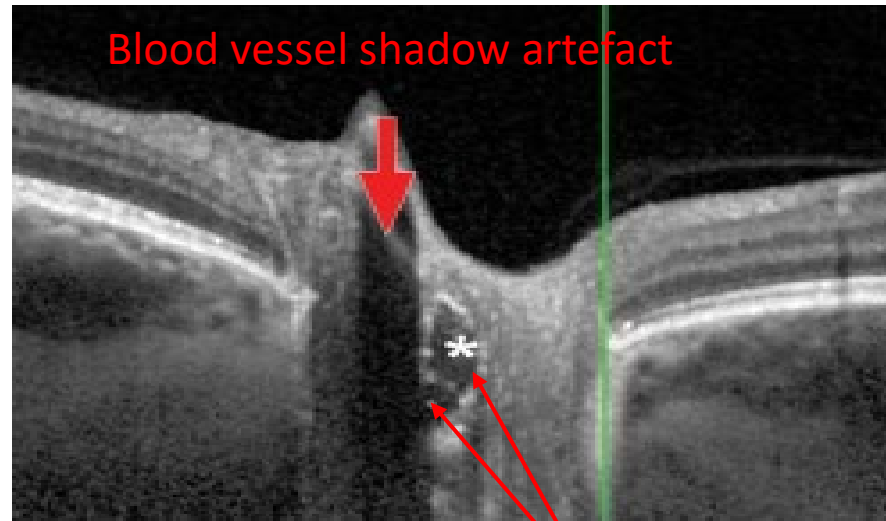
Bottom: Malmqvist 2018 ODD study consortium (red arrows = buried drusen), white + yellow = surface drusen blue = BV artefacts

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Describe the features



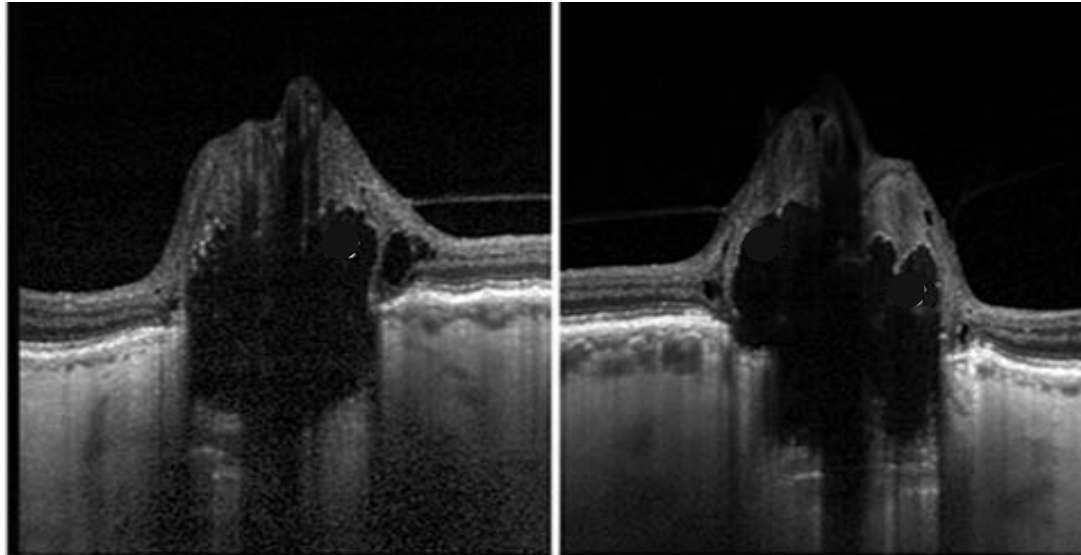
Describe the features



Diagnosis: ONHD

ON drusen with pore core signal (dark)
but in this case the presence of **conglomerates**
of multiple small ON drusen gives rise to hyperreflective foci

Describe the features

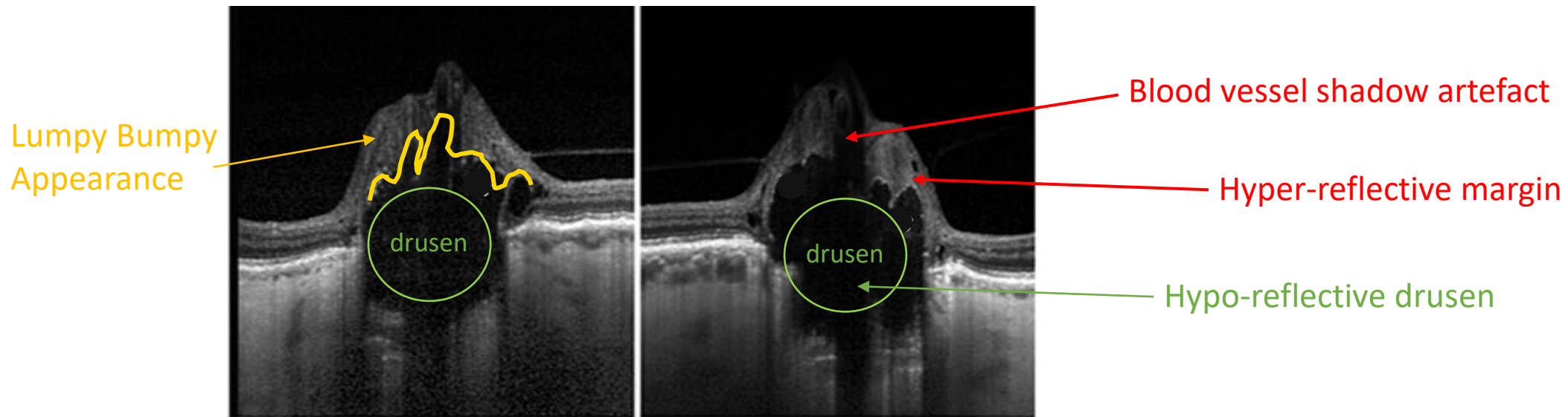


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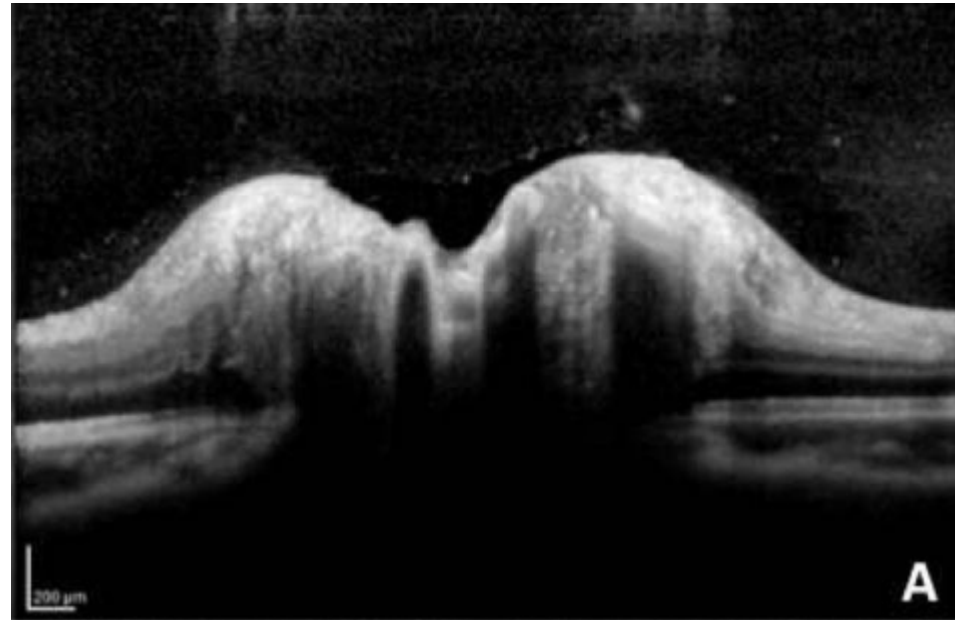
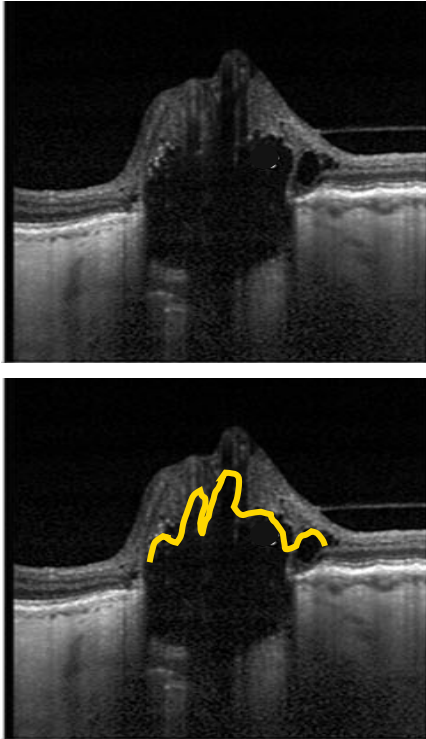
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Describe the features



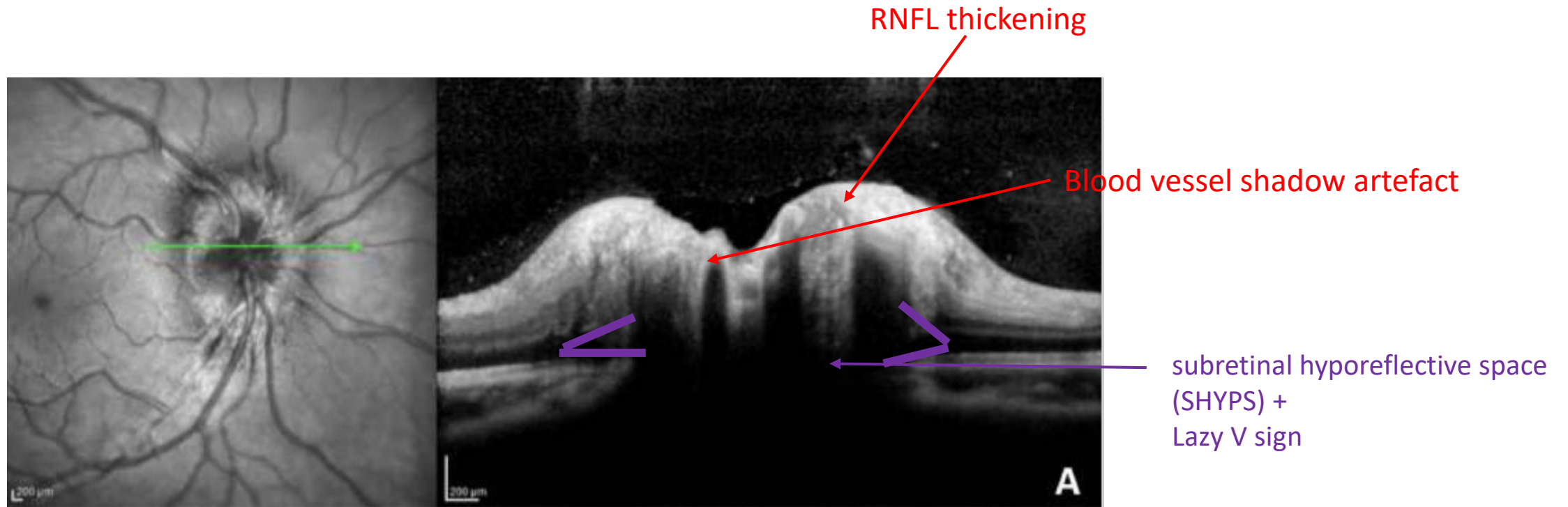
Diagnosis: ONHD

Describe the features



Is this Lumpy Bumpy appearance?

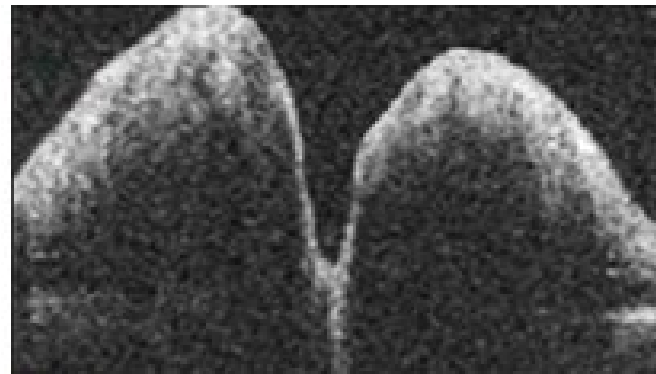
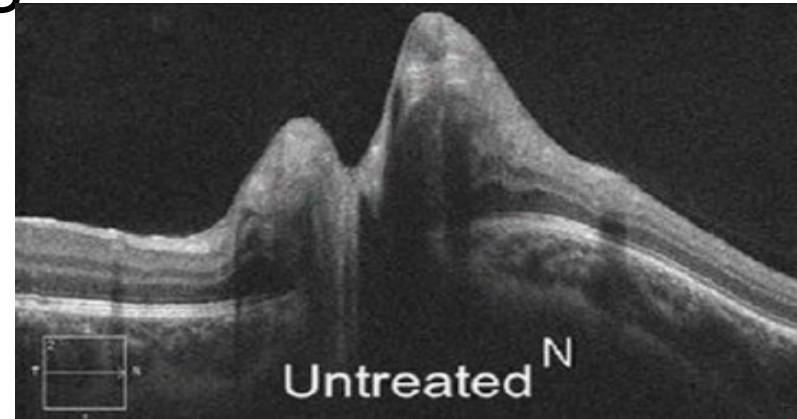
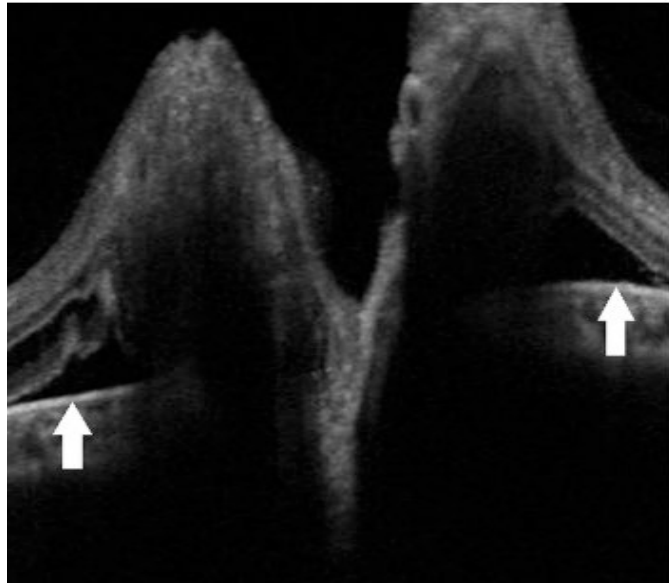
Describe the features



Diagnosis: PAPHILLOEDEMA

Is this Lumpy Bumpy appearance?
NO. Internal contours are smooth.

Describe the features



subretinal hyporeflective space (SHYPS) + lazy V sign??

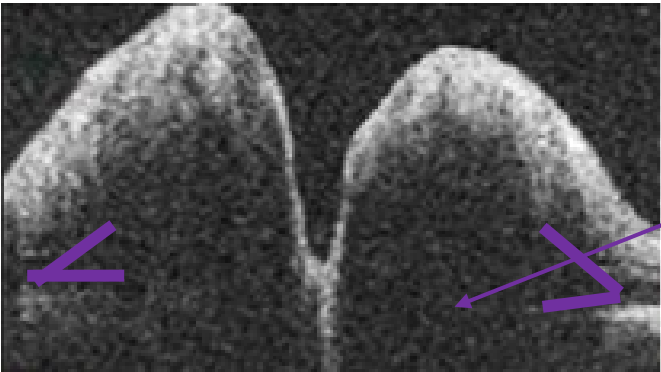
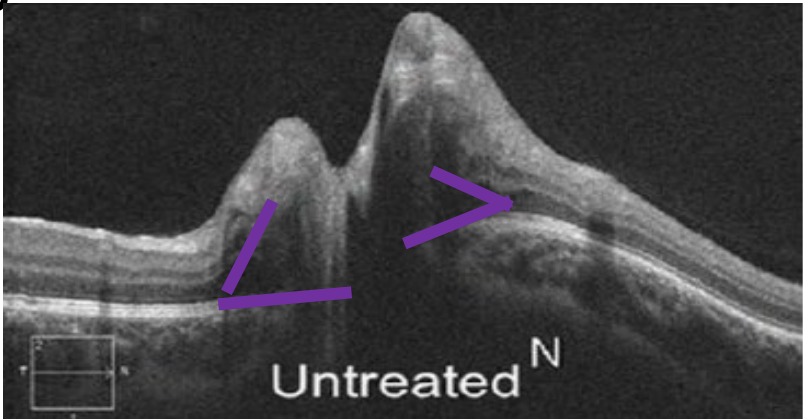
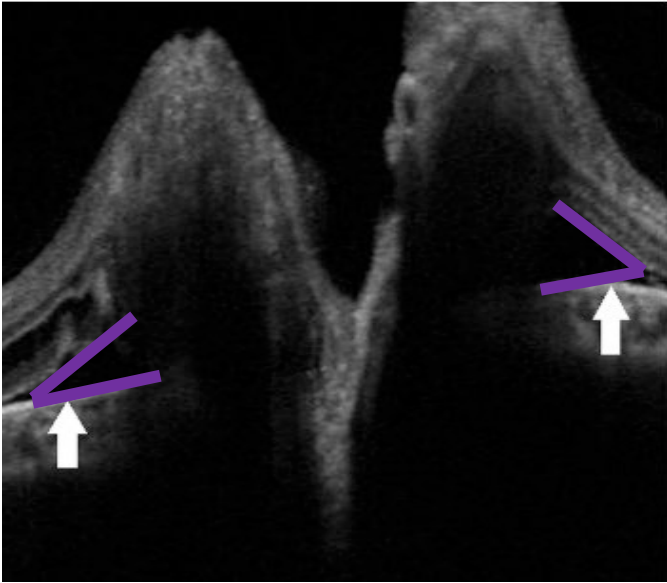


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Describe the features

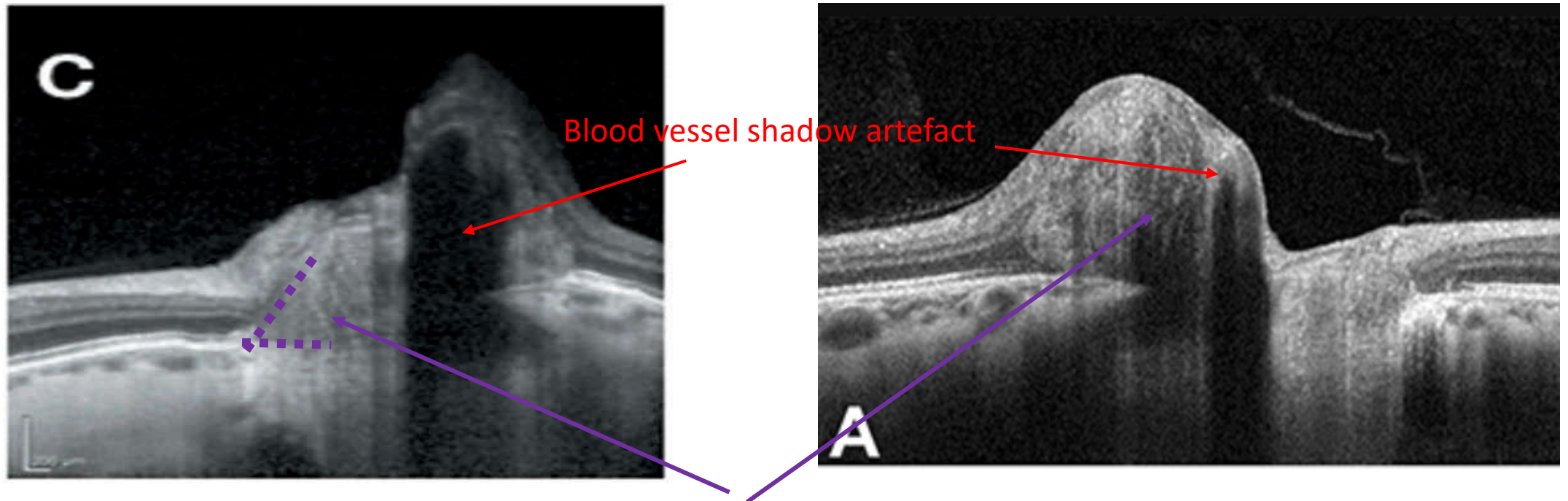


subretinal hyporeflective space (SHYPS) + lazy V sign

Is seen only when standard OCT is used
Artefact from thickened RNFL

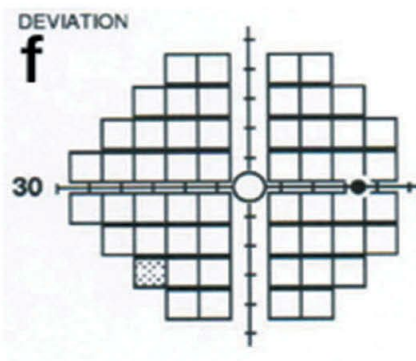
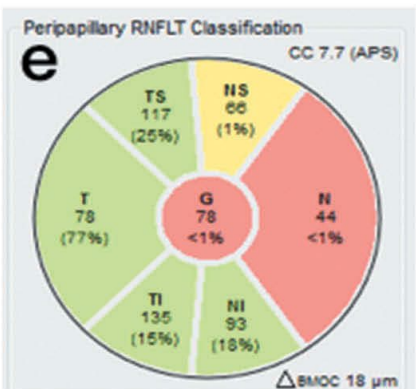
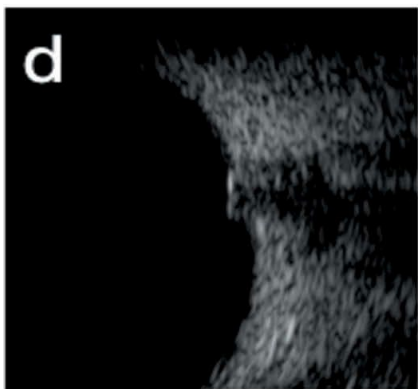
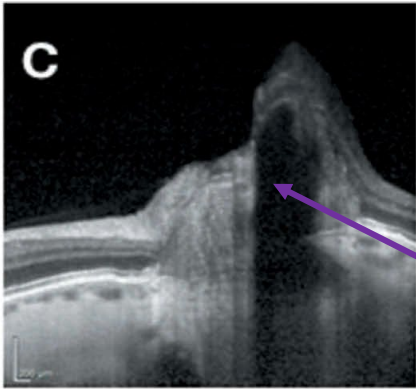
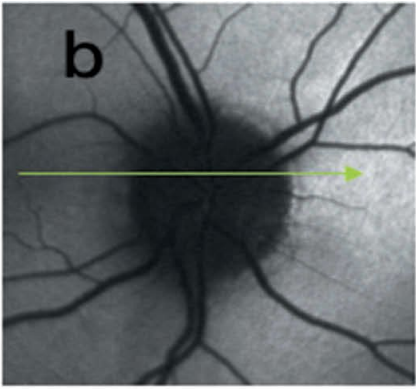
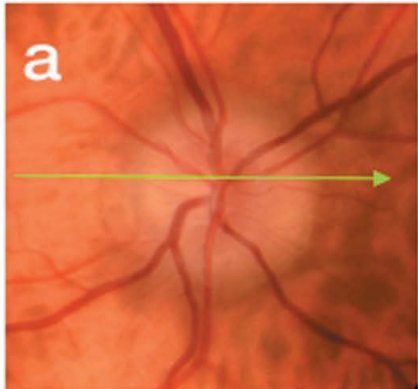
Diagnosis: PAPHILLOEDEMA

Describe the features



subretinal hyporeflective space (SHYPS) + lazy V **DISAPPEARS** on EDI

Describe the features



subretinal hyporeflective space (SHYPS) + lazy V **DISAPPEARS** on EDI in some instances

Lazy V sign not indicative



N = 83 (Optic disc oedema/papilloedema)
N = 117 (ODD)

Lazy V sign for optic disc oedema detection:
Sensitivity = 54%
Specificity = 63%



Utility of the OCT lazy V sign in diagnosing optic nerve edema in a pediatric population

Hilliary E. Inger, Hersh Varma, Mea A. Weaver, Catherine O. Jordan, Rachel E. Reem, Mary Lou McGregor, Shawn C. Aylward, David L. Rogers

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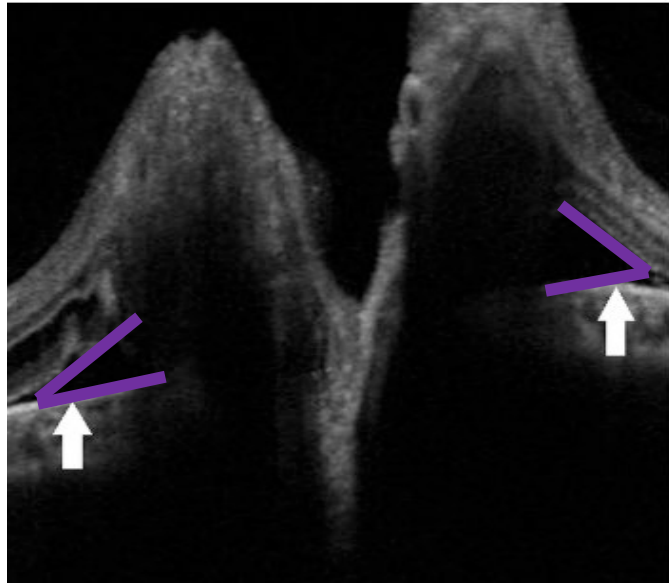
Introduction

A subretinal hyporeflective space between the sensory retina and the RPE/choriocapillaris, known as the “lazy V,” has been described on optical coherence tomography (OCT) in patients with optic nerve edema (ONE). The purpose of this study is to determine if the lazy V can distinguish pediatric ONE from optic nerve head drusen (ONHD) and determine if it is visible at all grades of ONE.

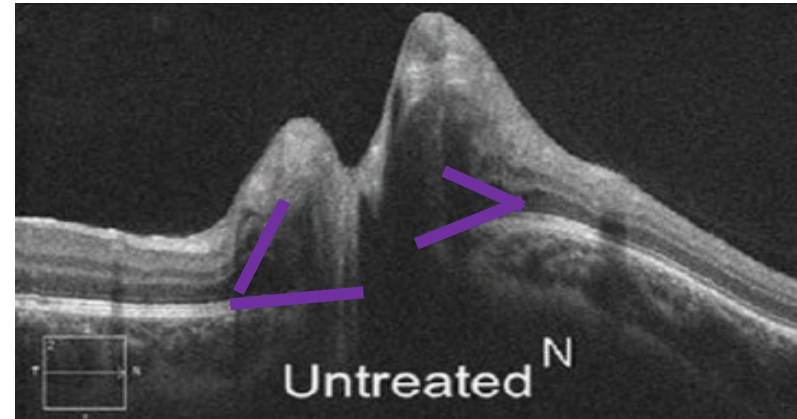
Methods

Spectral-domain OCT optic nerve scans of 83 eyes with ONE and 117 eyes with ONHD were collected retrospectively. Four masked pediatric ophthalmologists independently reviewed the images for the presence of the lazy V.

Describe the features (*do's and don'ts*)

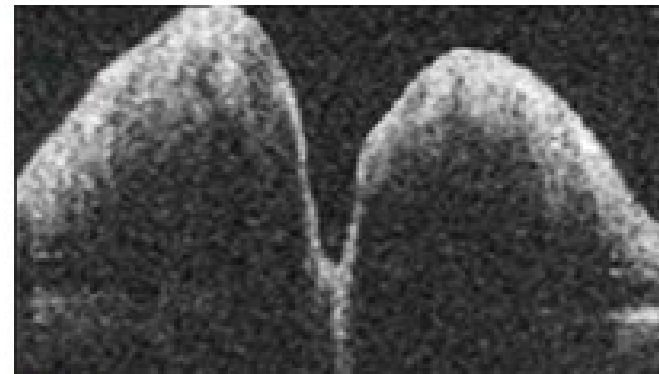
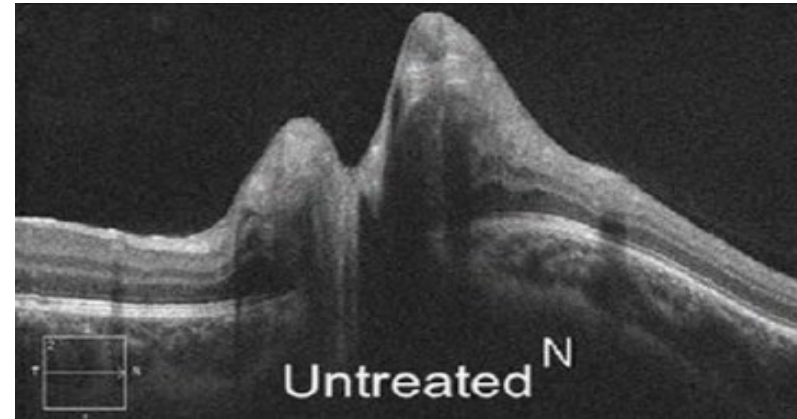
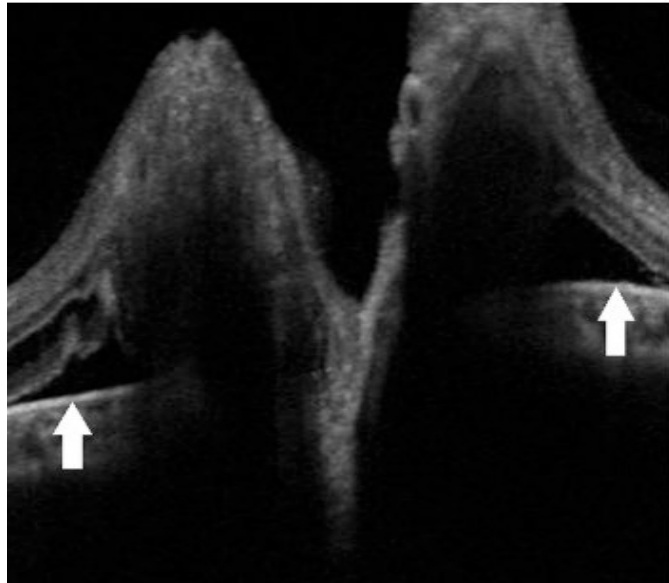


Diagnosis: PAPHILLOEDEMA



1. **Don't** use lazy V sign
2. **Don't** use SHYPS

Describe the features (*do's and don'ts*)



1. Posterior bulging of the RPE-Bruch complex
2. Thickening of the RNFL

Diagnosis: PAPHILLOEDEMA

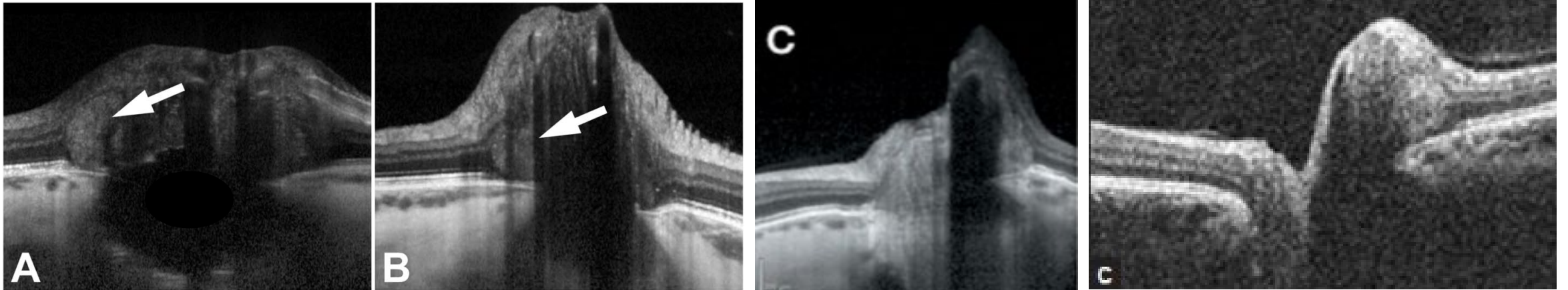


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What is common between these images?

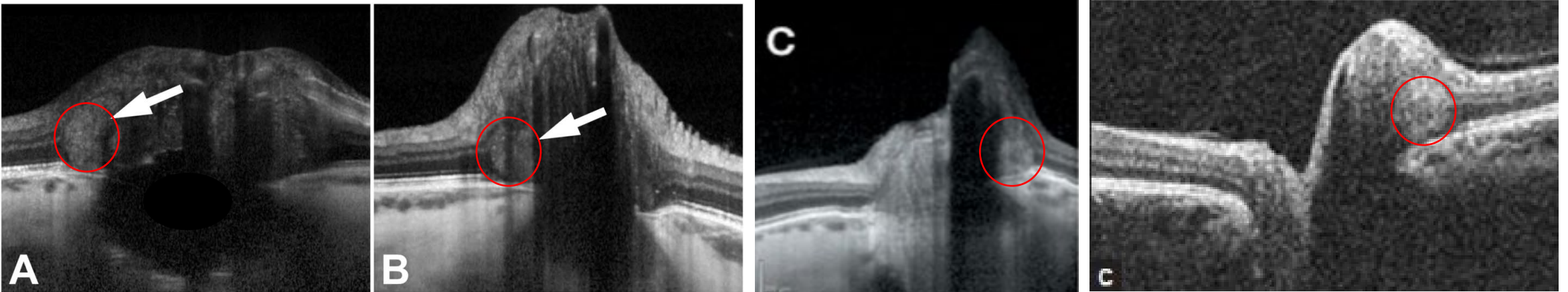


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What is common between these images?



Peripapillary hyperreflective ovoid mass-like structure (PHOMS)

- Once thought to be a key sign of ON drusen, represents the drusen itself or the precursor

What is your DX??

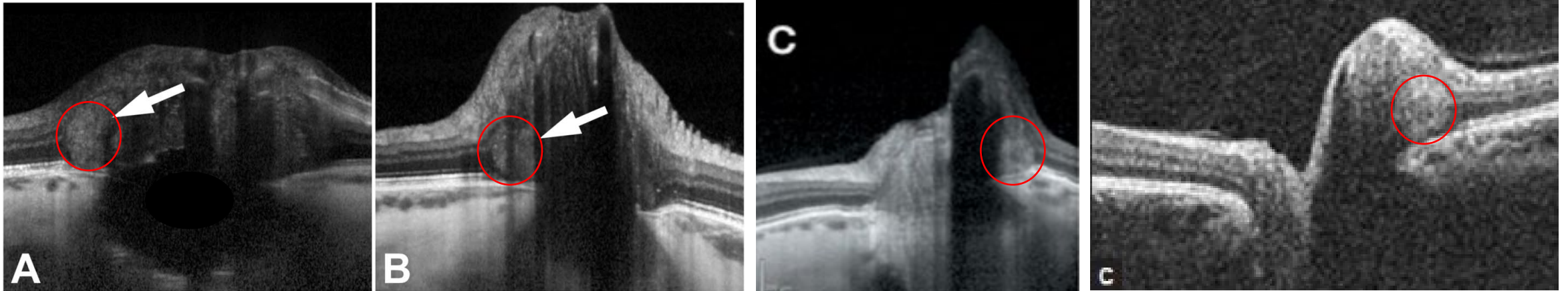


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What is common between these images?



ONHD

papilloedema

papilloedema

ONHD

PHOMS now thought to represent displacement of RNFL axons,
Not the actual ON drusen itself. Whether it's a precursor still up for debate..
Because its seen very often in ONHD



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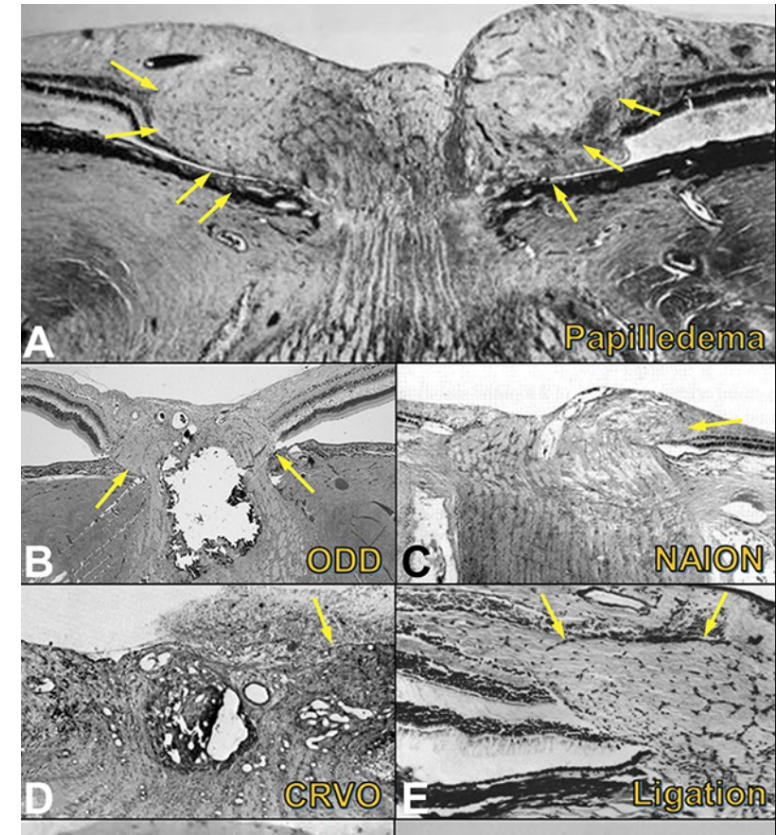
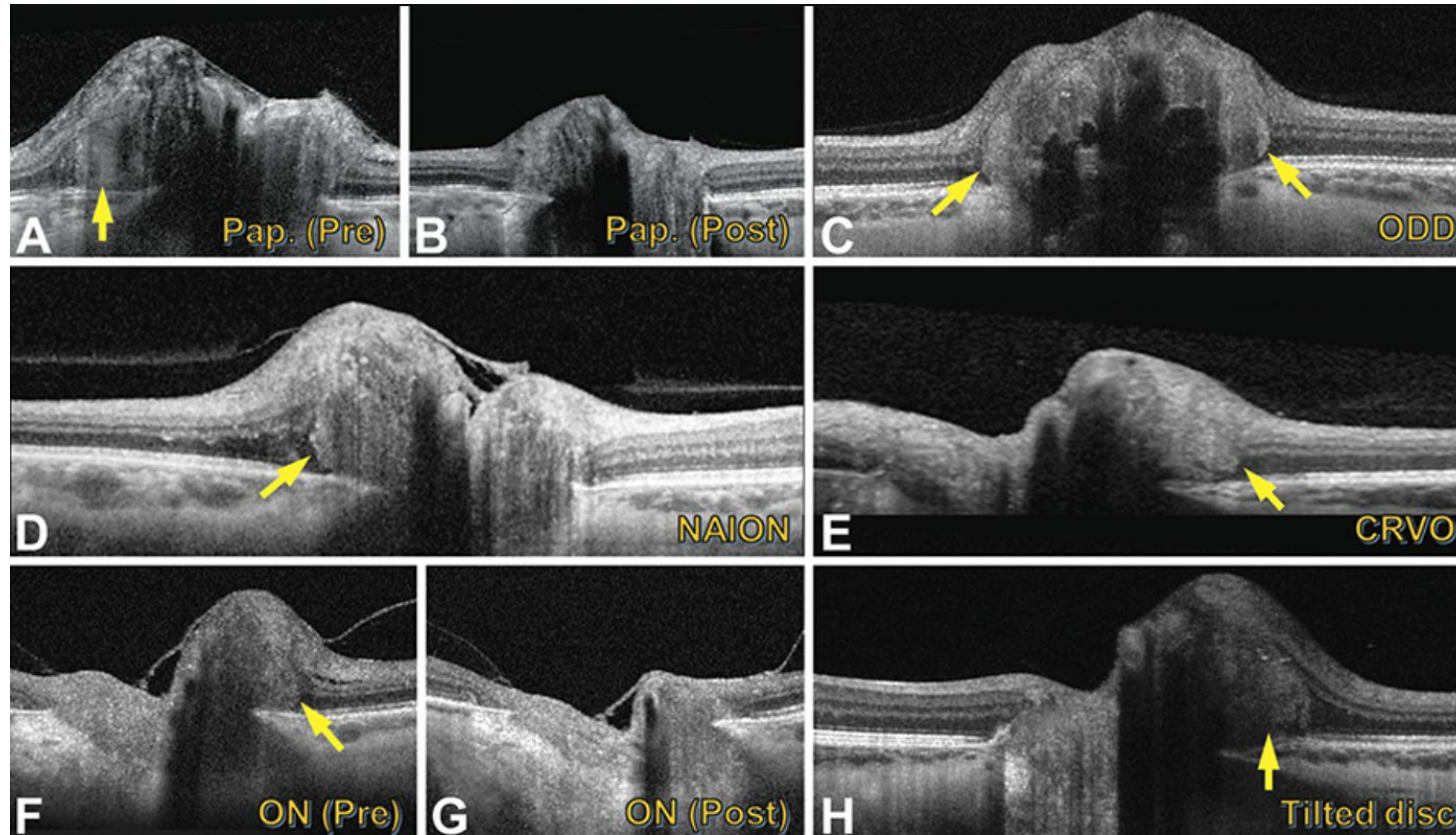
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PHOMS are simply RNFL fibres herniated due to axoplasmic stasis

Peripapillary Hyper-reflective Ovoid Mass-like Structure (PHOMS): An Optical Coherence Tomography Marker of Axoplasmic Stasis in the Optic Nerve Head

Fraser, J. Alexander MD; Sibony, Patrick A. MD; Petzold, Axel MD, PhD; Thaug, Caroline MB, ChB, DPhil; Hamann, Steffen MD, PhD [Author Information](#)
 Section Editor(s): Costello, Fiona MD, FRCP(C); Prasad, Sashank MD



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Imaging a suspicious optic nerve

- Summary

- Straight lines = BV artefact/shadowing
- Always use Enhanced Depth Imaging (EDI-OCT)
- Volume/3D scan alone is **not enough** (pre-tester/optom asst)
 - – must **do radial or raster** in addition
- Optic disc drusen – **HYPO/BLACK**, hyper/bright superior margin
- **Papilloedema** – SHYPS, Lazy V and PHOMS not good anymore - use RPE bowing and RNFL thickness

