39TH ANNUAL SCIENTIFIC MEETING

SEPT 11 - 13, 2025

Fable Hotel, 58 Queen Street, Auckland, New Zealand

NOSA Meeting
Thursday 11th & Friday 12th September

NeuroVision Training Day
Saturday 13th September

NOSA
Neuro-Ophthalmology Society of Australia

neurovision

Welcome to NOSA 2025 – Auckland, New Zealand

On behalf of the Council and Scientific Committee of the Neuro-Ophthalmology Society of Australia (NOSA), it is my great pleasure to welcome you to the 39th Annual Scientific Meeting, taking place here in Auckland from 11–13 September 2025 at the Fable Hotel.

We are privileged to host an exceptional lineup of international guest speakers:

- **Professor Ari Green** (UCSF) – a global leader in neuroimmunology, repair, and remyelination in multiple sclerosis
- **Professor Michael Barnett** (University of Sydney) – expert in neuroinflammation, imaging biomarkers, and Susac syndrome presenting the Asia-Pacific Keynote Lecture
- **Dr Sui Wong** (Moorfields Eye Hospital & St Thomas' Hospital, London) – renowned for her pioneering work in idiopathic intracranial hypertension and ocular myasthenia
- **Professor Grant Liu** (University of Pennsylvania) – one of the world's leading authorities in paediatric neuro-ophthalmology

This year's program reflects NOSA's breadth and energy — from neuroinflammation, idiopathic intracranial hypertension, genomics in optic nerve disorders, and the latest developments in efferent system disorders. The Walsh William case presentations return, alongside clinical updates.

We remain committed to fostering the next generation, with the NeuroVision Training Day on Saturday 13 September for registrars and fellows — an invaluable opportunity to learn directly from leaders in the field.

My sincere thanks go to our sponsors for their generous support, to our organising committee for their meticulous planning, and to each of you for travelling here to share your knowledge, questions, and ideas. NOSA's reputation for collegiality and intellectual generosity depends on you — our members, guests, and friends.

I look forward to an exciting, stimulating, and memorable few days together.

Professor Helen Danesh-Meyer CNZM FRSNZ MBChB MD PhD FRANZCO
President, Neuro-Ophthalmology Society of Australia

We are most grateful to our sponsors for the 2025 NOSA meeting

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General information

Fable Auckland
85 Queen St, Auckland CBD, New Zealand
Ph: 09 309 9979

Meeting rooms
The Conference will be held in the Sinclair Room located on the lower ground floor.
The posters can be viewed in the foyer between the Sinclar Room and Soda Suite.
The Exhibition & catering will be held in the Soda Suite on the lower ground floor.
The reception desk will be held in the foyer on the lower ground floor.

Name badges
Pleasure ensure to wear your name badge throughout the meeting which will be provided at the time of registration.

The registration desk will be open throughout the conference from 7:30am to 4pm on each day.

Conference Dinner
The conference dinner will be held at Cooke's restaurant at Fable hotel. Thursday 11th September at 6:30pm

For assistance throughout the conference, please contact Kath Poon
E: kath@kathpoonevents.com.au or **mobile:** 0402 891 804

RANZCO CPD allocation
The 2025 NOSA conference has been accredited as follows.

NOSA 2025	Category	CPD event name to select when claiming the hours in the CPD diary (for FRANZCO)	CAPE
Day 1	Education: 5 hours	Select *"Meetings, conferences, workshops & webinars"* for the Education hours. Select *"Other Reviewing Performance activities not listed"* for the Reviewing Performance hours.	Professionalism Ethical Practice
Day 2	Education: 2 hours		
	Reviewing Performance: 3 hours		
Day 1 + Day 2	Education: 7 hours		
	Reviewing Performance: 3 hours		

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gMG, generalised Myasthenia Gravis; **NMOSD**, Neuromyelitis Optica Spectrum Disorder.

Guest speakers

Professor Ari Green

Professor Ari Green. Dr. Ari J Green is a physician scientist and serves as Chief of the Division of Neuroimmunology and Glial Biology in the Department of Neurology at the University of California at San Francisco. His team consists of more than 20 laboratories and groups to investigate and develop cures for multiple sclerosis and other related diseases. Under his leadership the group has expanded its focus to investigate new methods for understanding infectious and inflammatory diseases of the brain and developmental and autoimmune disorders of the CNS. Dr. Green's laboratory is dedicated to the biological validation of potential biomarkers intended to measure remyelination and repair (as well as progression) in MS as a means to rapidly accelerate clinical trials for reparative treatments. His major scientific contributions include directing the first successful phase II clinical trial of a remyelinating agent for MS and finding ways to disentagle complex biological processes seen in MS so we can best prevent and overcome disability. His lab maintains an active interest in the developing and validating biomarkers for neurodegenerative disease. His team takes a multimodal approach including imaging, visual testing, electrophysiology, psychophysics, proteomics,and body-fluid biomarkers to deepen our understanding of the biological processes that drive neurodegeneration. He co-directs the Innovation Program in Remyelination and Repair with Dr. Jonah Chan. He trained as a Neuroimmunologist and Neuroophthalmologist after completing a residency in Neurology. He was named as the Debbie and Andy Rachleff Distinguished Professor of Neurology in 2009 and joined the Department of Ophthalmology at UCSF in 2011.

Guest speakers

Asia-Pacific Keynote Lecture

Prof Michael Barnett

Michael Barnett is the McLeod Professor of Neurology at the Brain and Mind Centre (BMC), University of Sydney; consultant neurologist and Director of the MS Service at Royal Prince Alfred Hospital (RPAH) Sydney; and Director of the MS Clinical Trials Unit at the BMC. He trained in neurology at RPAH and received further subspecialty training in Neuroinflammation and Neuro-ophthalmology at the National Hospital for Neurology and Neurosurgery, and Moorfields Eye Hospital, in London. He subsequently completed a PhD in MS pathophysiology at the University of Sydney; and has particular research interests in MS neuropathology and neuroimaging. He co-founded two successful med-tech companies, including the Sydney Neuroimaging Analysis Centre (SNAC), the first regulatory-compliant neuroimaging analysis / central MRI reading for MS clinical trials in the Southern hemisphere. He is a leader in the Brain and Mind Centre's Computational Neuroscience Team, which is spearheading the development and application of AI-derived algorithms and imaging biomarkers to further understanding of the mechanisms of neurological disease, improve diagnostic specificity and enhance treatment paradigms. He is also co-Director of the MS Australia Brain Bank and former Chairman of the PACTRIMS Scientific Program Committee.

Guest speakers

Dr Sui Wong

Dr Sui Wong is lead specialist Ocular Myasthenia and specialist IIH Service at Moorfields Eye Hospital and St Thomas' Hospital, London. Dr Sui H. Wong is a Neurologist and Neuro-Ophthalmologist based in London, United Kingdom. In addition to her clinical work as a medical doctor and physician, Dr Wong is an active neuroscience researcher, who translates pertinent and clinically relevant questions into research, to improve person-centred patient outcomes. Additionally, she has the qualifications and experience to consider a broader spectrum of lifestyle-specific interventions. Her person-centered approach to the clinical services she set up, e.g. the IIH clinical services, has been shortlisted for, and received, several awards (e.g. HSJ Acute Sector Innovation of the year 2020 shortlist; Royal College of Physician's Excellence in Patient Care Award 2020 longlist; the British Society of Lifestyle Medicine 2020 top conference abstract). Dr Wong's holistic approach in empowering patients has been recognised at the King's Health Partners Education Awards (Mind & Body 2019 shortlist) and in Dec 2021, the invitation from IIH UK patient charity to be their Patron.

Guest speakers

Professor Grant Liu

Professor Grant Liu Dr. Liu, a Professor of Neurology and Ophthalmology at the University of Pennsylvania Perelman School of Medicine, received his medical degree from Columbia University. He completed a neurology residency at the Harvard-Longwood Neurology Program and a neuro-ophthalmology fellowship at the Bascom-Palmer Eye Institute, University of Miami. Although he sees both adult and children with neuro-ophthalmic problems, his special interest is in pediatric neuro-ophthalmology. He was appointed Division Chief at Penn in 2012 and the Raymond G. Perelman Endowed Chair in Pediatric Neuro-Ophthalmology at the Children's Hospital of Philadelphia in 2015. His clinical research interests include optic pathway gliomas, pediatric pseudotumor cerebri syndrome, and optic neuritis in children. Along with Drs. Nicholas Volpe and Steven Galetta, Dr. Liu has written and edited the book, Liu, Volpe, Galetta's Neuro-ophthalmology: Diagnosis and Management (3rd edition 2019). The 4th edition will be published in 2027.

Guest speakers

A/Professor Yael Barnett

A/Professor Yael Barnett BSc MBBS FRANZCR is Director of Radiology and Head of MRI at St Vincent's Hospital (Sydney); and Consulting Neuroradiologist at Sydney Neuroimaging Analysis Centre (SNAC). Fellowship-trained in neuroradiology, A/Professor Barnett has particular expertise in the imaging of neuro-inflammatory disorders, both from a clinical and translational research perspective. Subspecialty interests also include imaging in neuro-ophthalmology, neuro-oncology and movement disorders. She co-established the first MRI-guided focussed ultrasound centre for the treatment of neurological disorders in Australia; and, in her clinical role at SNAC, has pioneered the integration of Artificial Intelligence into diagnostic neuroimaging workflows.

Save the date for the 2026 Meeting:
3-5 September, The Martin Family Auditorium,
Royal Victorian Eye & Ear Hospital, East Melbourne.

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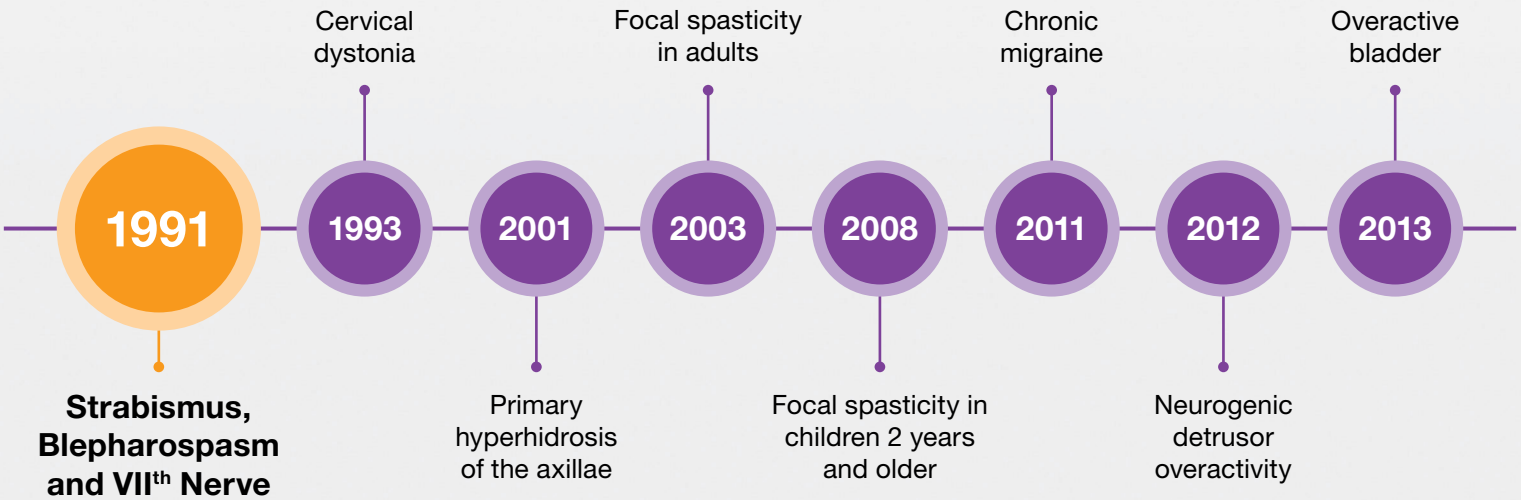

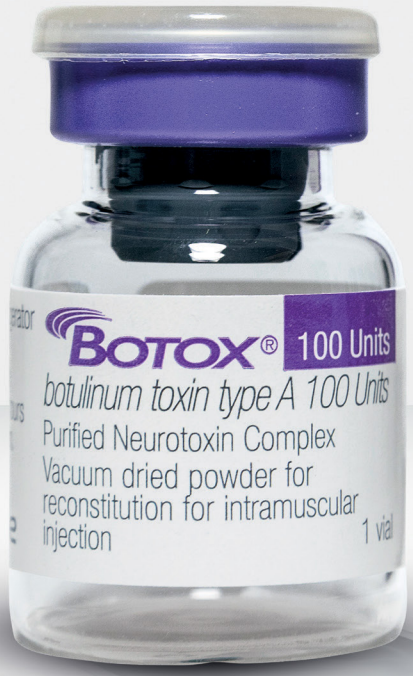

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References: 1. BOTOX® Data Sheet. **2.** Data on file. REF-131932. 2024 NZ registration dates. **3.** Naumann M *et al. Eur J Neurol* 2006;13(Suppl. 4):34–40. **4.** AbbVie Data on File, REF-76062.

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Program

Day One – Thursday 11th September 2025

Session 1

Decoding Neurodegeneration: Visual Pathways as a Window to the Brain

Chairs: Celia Chen, Mitchell Lawlor

09:00	Helen Danesh-Meyer	Welcome
09:10	Ari Green	Remyelination and repair in multiple sclerosis: The Important understandings gleaned from studying the visual system
09:40	Paul Condron	Ultra-high contrast MRI. Making the invisible visible
09:50	William Schierding	Microstructural Changes in the Posterior Thalamic Radiations Following Traumatic Brain Injury: Insights into White Matter Integrity from Diffusion Tensor Imaging in the UK Biobank
10:00	Daniel Zhang	Neurodegeneration in Glaucoma: Microstructural MRI Evidence Beyond the Visual Pathway
10:10	Gil Newburn	Neuropsychiatry of neuro-ophthalmology Through the Lens of Traumatic Brain Injury.
10:20	Alex Sarossy	Assessing Reliability and Optical Fogging Effects in a Web-Based Contrast Sensitivity Test: A Pilot Study
10:30	**Morning Tea**	

Program

Day One – Thursday 11th September 2025

Session 2

IIH Plenary

Chairs: Benson Chen, Jesse Gale

11:00	Benson Chen	The Expanding Spectrum Idiopathic Intracranial Hypertension
11:15	Grant Liu	Paediatric Idiopathic Intracranial Hypertension
11:45	Shilpan Patel	Evaluating the Diagnostic Yield of Lumbar Puncture for Suspected Idiopathic Intracranial Hypertension: A Clinic-Based Audit
11:55	Jet Wright	IIH and aMRI
12:05	Paige Richter	A Systematic Review of Patient-Reported Outcome Measures (PROMs) and Qualitative Studies to Investigate the Hidden Burden of Idiopathic Intracranial Hypertension (IIH) on Quality of Life (QoL)
12:15	Sarah Morgan	Case series. Myeloproliferative neoplasm: a risk factor for IIH
12:25	Sujan Surendran	Paediatric patients with optic disc drusen and corresponding visual field defects
12:35	Sui Wong	Facilitating sustainable lifestyle changes and remission through a group clinic model
13:00	**Lunch**	

Program

Day One – Thursday 11th September 2025

Session 3

Optic Nerve Disorders in 2025: From Genomics to Guidelines

Chairs: Mark Paine, Suba Raviskanthan

14:00	Ari Green	Incorporating the optic nerve and visual testing into the diagnostic criteria in MS: What's changed in 2025
14:30	Antonia Kartika	Contrast Sensitivity in Metastatic Prostate Cancer Patients on Androgen Deprivation Therapy
14:40	Michael Wang	Prognostic factors for long term outcomes in optic neuritis: a prospective registry-based cohort study
14:50	Jesse Gale	Low serum pyrophosphate as a model for optic disc drusen.docx
15:00	Jack Maran	Bitemporal optic atrophy from retrograde transsynaptic axonal degeneration may signify cerebral injury in children
15:10	Shweta Singhal	Inherited optic neuropathies- a Multiethnic Asian study
15:20	David Choi	Efficacy of Plasma Exchange for Acute Optic Neuritis in MOG Antibody Disease
15:30	Rachael Niederer	Neurological sequelae of herpes zoster
15:40	**Afternoon Tea**	

Program

Day One – Thursday 11th September 2025

Session 4

Clinical Updates in Neuro-ophthalmology

Chairs: Neil Shuey, Blake Colman

16:05	Owen White	Visual Snow Syndrome: Fad, Functional, or Frontier?
16:20	Mitchell Lawlor	The Expanding Role of OCT in Neuro-ophthalmology
16:35	Celia Chen	OCTA in Neuro-ophthalmology
16:50	Cathy Zhong	Saccadic Abnormalities as Biomarkers: Insights from Electrophysiology and Deep Learning.
17:05	Blake Colman	Ocular Presentation of Stroke: A Diagnostic Pitfall
17:20	Suba Raviskanthan	Radiation Optic Neuropathy
17:35	**End of Day**	
18:00	**NOSA Board Meeting**	
18:30	**Conference Dinner** Cooke's restaurant, ground floor, Fable	

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Program

Day Two – Friday 13th September 2025

Session 1

Misguided Movements: Clinical Lessons from the Efferent System

Chairs: Owen White, Fiona Chan

9:00	Housekeeping	
09:05	Grant Liu	Eye movement disorders you do not want to miss
09:35	Sui Wong	Myasthenia Gravis
10:05	Anthony Fok	Can Structural Grading of Foveal Hypoplasia and Foveation. DurationPredict Visual Acuity in Infantile Nystagmus Syndrome?
10:15	Irna Novianti Irwan	When two conditions collide: Exploring Internuclear Ophthalmoplegia with see-saw nystagmus.
10:25	Mark Paine	Revisiting the directional selectivity of saccade abnormalities in Progressive Supranuclear Palsy
10:35	**Discussion**	
10:45 Morning Tea		

Program

Day Two – Friday 13th September 2025

Session 2 **Chairs:** Anthony Fok, Anneke Van der Walt		
11:15	Ari Green	The advent of body fluid biomarkers for studying neurological injury in 2025: From Neurofilament light to Proteomics - What have we learned
11:40	Sarah Hull	Hypovitaminosis A and visual loss in children with restricted diets
11:50	Oliver Burnett	Vision-Related Quality of Life in Friedreich's Ataxia
12:00	Alex Sarossy	Linezolid-induced Optic Neuropathy - A Case Series
12:25	**Asia-Pacific Keynote Lecture:** Michael Barnett	Susac Syndrome: Big Trouble in Little Vessels"
12:55	**Lunch**	

Program

Day Two – Friday 13th September 2025

Session 3

Walsh William Cases

Chairs: Helen Danesh-Meyer & Celia Chen

13:55	Anthony Yao	A massive thirst
14:10	Evelyn Turek	A Swell Case
14:25	Liana Dedina	Think poetically
14:40	Melissa Tien	Toxic relationships
14:55	Yunita Mansyu	A Silent infiltrator: Hidden in plain sight
15:10	**Afternoon Tea**	

Session 4

Chairs: Helen Danesh-Meyer & Celia Chen

15:30	Jessie Cai	Out of Sight, Out of Time
15:50	Liam Walsh	Out of Sight, Not Out of Mind: It starts with that sinking feeling
16:15	Ari Green	Clinical Trials in Remyelination
16:45	**Conclusion**	

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Reference: 1. Novartis SEE Impact Valuation Report 2024. Based on internal analysis of patient reach and societal impact across Australia and New Zealand. Data on File. Novartis New Zealand Limited, Auckland. Ph 0800 838 909.
Date of preparation: September 2025. NZ-01717.

9.40

Ultra-high contrast MRI. Making the invisible visible.

Presenter:
Paul Condron

Co-Authors:
Dan Cornfeld, Eryn Kwon, Samantha Holdsworth, Mark Bydder, Benson Chen, Ernie Willoughby, Heleln Danesh-Meyer, Graeme Bydder.

Ultra-high contrast (UHC) MRI is a term used to describe MR imaging that shows abnormalities with high contrast when little or no abnormality is seen on common conventional state of-the-art MR images. It is achieved by optimising sequences already available on most MRI systems, without the use of increased static or gradient magnetic fields.

Two inversion recovery sequences are optimised to exploit small changes in T1 in the middle Domain (mD) i.e., values of T1 between the two nulling T1s. Utilising the same tissue property (T1) twice or more in the same sequence rather than once we can provide up to a ten-fold increase in contrast compared to tradition approaches.

This approach has already identified obvious changes that are not depicted on T2 FLAIR and other conventional sequences in MS, mTBI, and Hypoxic injuries.

09:50

Microstructural Changes in the Posterior Thalamic Radiations Following Traumatic Brain Injury: Insights into White Matter Integrity from Diffusion Tensor Imaging in the UK Biobank

Presenter:
William Schierding[2,3,5]

Co-Authors:
Esha Varghese[1], William Tan[1], Eryn Kwon[3,4], Helen V Danesh-Meyer[2,3,5]

Institutions:
1. Department of Anatomy and Medical Imaging, University of Auckland, Auckland, New Zealand
2. Department of Ophthalmology, University of Auckland, Auckland, New Zealand
3. Mātai Medical Research Institute, Tairāwhiti Gisborne, New Zealand
4. Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand
5. Vision Research Foundation, Auckland, New Zealand

Visual disturbances are among the most persistent and debilitating symptoms following traumatic brain injury (TBI), often occurring in the absence of focal neurological findings or definitive diagnostic tests. Traumatic optic neuropathy, characterized by chronic and progressive retinal thinning, can persist for decades post-injury and suggests broader disruptions in visual processing pathways. However, the chronic pathophysiology beyond the retina remains poorly understood.

The posterior thalamic radiation (PTR), which transmits visual information from the lateral geniculate nucleus and pulvinar to the primary visual cortex, plays a critical role in higher-order visual processing. It has also been implicated in postural control deficits following TBI. However, most prior studies have been limited by small sample sizes.

To investigate potential structural changes in the PTR associated with TBI, we analyzed structural T1 and diffusion MRI data from the UK Biobank, comparing individuals with a history of TBI (n = 304) to a large control cohort (n = 43,216). TBI participants were slightly older (median age 57 vs. 56), more likely to be male (60% vs. 47%), and had higher rates of neurodegenerative and retinal diseases, including acute macular degeneration (6.3% vs. 3.8%), Alzheimer's disease (1.3% vs. 0.1%), and Parkinson's disease (1.3% vs. 0.3%). However, they only had a small, non-significant decrease in retinal nerve fibre layer thickness (25.5 vs. 26.5).

After adjusting for age, sex, and socioeconomic status, we identified significant microstructural alterations ($p < 0.01$, FDR-corrected) in both left and right PTRs across multiple diffusion metrics, including fractional anisotropy (FA), intracellular volume fraction (ICVF), isotropic volume fraction (ISOVF), and diffusivity measures (L1, L2, L3, MD, MO, OD). These findings suggest chronic white matter disruption in the visual pathways of individuals with TBI.

Our results underscore the importance of examining white matter structures beyond the retina in chronic traumatic optic neuropathy and demonstrate the value of large-scale neuroimaging datasets in detecting subtle but clinically relevant brain changes following TBI.

10:00

Neurodegeneration in Glaucoma: Microstructural MRI Evidence Beyond the Visual Pathway

Presenter:
Daniel Zhang[1],

Co-authors:
Eryn Kwon[2,3], Helen Danesh-Meyer[1,4], William Schierding[4,5]

1. Department of Ophthalmology, University of Auckland, Auckland, New Zealand
2. Mātai Medical Research Institute, Tairāwhiti Gisborne, New Zealand
3. Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand
4. Vision Research Foundation, Auckland, New Zealand

Objective: To investigate structural and microstructural brain changes associated with glaucoma using magnetic resonance imaging (MRI) with a specific focus on primary, secondary, and higher-order visual areas. We further assessed how these imaging markers relate to ophthalmological measures: retinal nerve fibre layer (RNFL) thickness, ganglion cell layer (GCL) thickness, and corneal-compensated intraocular pressure (IOPcc).

Methods: Participants were drawn from the UK Biobank and included individuals with diagnosed glaucoma (n = 1,465), and age- and sex-matched controls (n = 14,650). MRI data included T1-weighted structural imaging and diffusion MRI, incorporating diffusion tensor imaging (DTI) and neurite orientation dispersion and density imaging (NODDI). Linear regression models were adjusted for demographic, socioeconomic (Townsend Deprivation Index), and genetic variables (standardized PRS).

Result: Glaucoma was associated with reduced grey matter volume in the LGN (FDR p=1.55 x 10-3), Optic Chiasm (FDR p=3.46 x 10-4), intracalcarine cortex (FDR $p = 6.03 \times 10^{-4}$), and occipital pole ($p = 3.07 \times 10^{-5}$), with diffusion abnormalities in the posterior thalamic radiation (FA, FDR $p = 3.72 \times 10^{-11}$; MD, FDR $p = 4.19 \times 10^{-5}$). Secondary visual regions showed atrophy in the lateral occipital cortex (FDR $p = 2.15 \times 10^{-3}$), lingual gyrus (FDR $p = 1.21 \times 10^{-2}$), and occipital fusiform gyrus (FDR $p = 7.2 \times 10^{-4}$), and diffusion changes in the inferior fronto-occipital fasciculus (FDR $p = 1.03 \times 10^{-6}$) and inferior longitudinal fasciculus (FDR $p = 1.88 \times 10^{-9}$). In visual supporting structures and higher-order regions, the right putamen (FDR $p = 4.04 \times 10^{-2}$) (e.g., Paracingulate Gyrus, FDR $p = 1.14 \times 10^{-2}$) also correlated with glaucoma. These markers correlated consistently with both RNFL and GCL thickness. No significant associations were observed with IOPcc.

Discussion: MRI reveals structural brain alterations in both primary and secondary visual regions in glaucoma, with additional but less pronounced changes in higher-order cortical areas. These neuroanatomical changes correlate strongly with thinning of the RNFL and GCL, but not with IOP.

10:10

Efficacy of Plasma Exchange for Acute Optic Neuritis in MOG Antibody Disease

Presenter:
David Cho

Co- Authors:
Stefan Ivanov, Jane Shi, Daniel A. Scott, Helen V. Danesh-Meyer

Purpose: To evaluate the clinical efficacy and safety of plasma exchange (PLEX) for severe optic neuritis in patients with myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), and to assess associated visual, structural, and functional outcomes.

Method: MOGAD patients treated with PLEX for acute optic neuritis were retrospectively reviewed. Demographic data, attack characteristics, visual outcomes, OCT and visual field metrics, timing of PLEX, complications, and use of maintenance immunotherapy were extracted. Visual acuity (VA), retinal nerve fiber layer (RNFL) thickness, ganglion cell layer (RGC) volume, and visual field (VF) mean deviation were compared pre- and post-treatment. Relapse rates and time to relapse after PLEX were also evaluated.

Results: Twelve episodes of MOGAD optic neuritis were acutely treated with PLEX between 2012–2023. The median age at attack was 46 years. All patients were initially treated with high dose IV steroids; the most common indication for PLEX treatment was suboptimal improvement after high dose IV steroids treatment. Median pre-PLEX VA in the worse eye was ***Hand Movement (20/40-NPL)***, improving to ***20/30 (20/16-20/60)*** post-PLEX. Median VF mean deviation improved from ***–20.45 dB*** to ***–7.65 dB***. OCT measurements showed a change in RNFL from ***97 µm*** to ***58.7 µm***, and RGC from ***0.78 mm³*** to ***0.65mm³***. PLEX was initiated a median of 9 days after symptom onset, with a median of ***5 (2-7) sessions.*** Two CVL related complications were reported. Post-PLEX, six attacks (50%) relapsed despite all of them being on maintenance therapy, with median time to the first relapse of ***414 days.***

Conclusion: PLEX was associated with substantial improvement in visual acuity in MOGAD optic neuritis without major complications. While relapses are still common post-PLEX, most patients achieved favorable visual outcomes, especially as escalation therapy in severe or steroid-unresponsive cases.

10.20

Assessing Reliability and Optical Fogging Effects in a Web-Based Contrast Sensitivity Test: A Pilot Study

Presenter:
Chen Wang

Authors:
Chen Wang, Marc Sarossy

Institution:
Monash University

Email:
Wangchen01189@gmail.com
cwan0129@student.monash.edu

Purpose: Contrast sensitivity (CS) is an important visual function often omitted from routine assessments due to costly and specialised equipment. This pilot study evaluates the test-retest reliability and optical fogging effects of a free, open-source web-based CS test developed using R Shiny

Method: (n = 40 eyes) completed a CS function test across four conditions: Baseline, Retest (15 minutes post-baseline), Optical Fogging (+3.00D reading glasses to simulate blur), and Retest. CS was assessed across a range of optotype sizes (6/6 to 6/60) and contrast levels at a 3.1 m distance from the laptop screen.

Results: Test-retest reliability was high, with ICCs of 0.974 (95% CI: 0.941 - 0.988) for normal vision and 0.963 (95% CI: 0.916 - 0.983) for fogging. Optical fogging disproportionately reduced sensitivity for smaller optotypes (e.g 6/6), consistent with existing literature showing greater blur effects at higher spatial frequencies. The high 6/6 threshold may suggest display-related limitations.

Conclusion: The open source test is validated with high test-retest reliability and shows plausible results, showing promise to be an accessible alternative to traditional contrast sensitivity testing. The next steps include validation against commercially available CS function tests and enrolment of clinical cohorts.

11.00

The Expanding Spectrum of Idiopathic Intracranial Hypertension

Presenter: Benson S. Chen, PhD FRACP[1,2]

1. Department of Neurology, Te Whatu Ora Te Toka Tumai Auckland
2. Department of Medicine, University of Auckland
3. Department of Clinical Neurosciences, University of Cambridge

Idiopathic intracranial hypertension (IIH) is classically defined by the presence of headache and papilloedema in young women with raised intracranial pressure. However, recent clinical and radiological insights suggest that IIH encompasses a broader and more heterogeneous spectrum than previously recognised.

This presentation will explore atypical and under recognised presentations of IIH, including patients with asymptomatic radiological signs of intracranial hypertension and those presenting with isolated pulsatile tinnitus. These may represent forme fruste expressions of IIH, in which structural and physiological changes occur in the absence of classic symptoms. Spontaneous intracranial cerebrospinal fluid (CSF) leak and temporal meningoencephaloceles will also be discussed as related entities that may arise from or coexist with chronically elevated intracranial pressure.

Additional symptoms such as cognitive and psychological disturbances, along with less commonly recognised cranial neuropathies, may contribute significantly to morbidity but are frequently overlooked. The role of neuroimaging remains central, with features such as optic nerve sheath distension, empty sella, and transverse venous sinus stenosis aiding diagnosis, though not diagnostic in isolation. The evolving relationship between IIH and spontaneous intracranial hypotension challenges conventional diagnostic categories and highlights the need for a more nuanced approach.

Emerging evidence implicates factors such as glymphatic dysfunction and chronic disturbances in CSF homeostasis in the development of this expanding spectrum. A broader, mechanistically informed understanding of IIH may help ensure that patients with atypical presentations receive accurate attribution of symptoms and targeted care.

11.45

Evaluating the Diagnostic Yield of Lumbar Puncture for Suspected Idiopathic Intracranial Hypertension: A Clinic-Based Audit

Presenter:
Shilpan G Patel

Authors:
Shilpan G Patel, Kimberly Jeremiah, Joseph Donnelly, Benson S Chen

Institution:
Department of Neurology, Te Toka Tumai Auckland

Email:
shilpanp@adhb.govt.nz

Purpose: Lumbar puncture (LP) to confirm elevated cerebrospinal fluid opening pressure (CSF-OP) is a key component in diagnosing idiopathic intracranial hypertension (IIH). With a growing number of new and suspected IIH cases, referrals to neurology for LP are increasing. We aimed to review referrals to our LP clinic for suspected IIH, evaluate how initial clinical and imaging assessments correlated with LP findings, and identify patterns of misdiagnosis or over-referral where LP may have been unnecessary.

Methods: We identified all patients referred to our outpatient LP clinic between December 2024 and June 2025. Detailed information was collected for those referred with suspected IIH, including referral source, presenting symptoms, fundus examination, optical coherence tomography (OCT) findings, neuroimaging features, CSF-OP, and final diagnosis. Neuroimaging was reviewed by a neuroradiologist when available.

Results: Twenty-six patients (23 [88%] female; mean age 25 ± 8.3 years) were referred with suspected IIH. Most (22/26; 85%) were referred from ophthalmology. Prior to LP, 23 patients had documented disc oedema, and 19 (73%) had neuroimaging features suggestive of raised intracranial pressure. IIH was diagnosed in 18 patients (69%) with elevated CSF-OP. Among the 8 patients who did not meet diagnostic criteria, 7 had normal CSF-OP and 1 had an abnormal neurological exam. There was a strong positive correlation between average OCT peripapillary RNFL thickness and CSF-OP (Spearman's $\rho = 0.62$, $p=.002$). In several cases, referring clinicians appeared to misclassify optic disc appearances and overemphasize neuroimaging features.

Conclusion: Lumbar puncture remains a crucial but limited resource in the evaluation of IIH. While 69% of referrals were appropriate, our findings highlight the need to improve referral accuracy using a combination of careful clinical assessment, OCT, and neuroimaging interpretation.

11.55
Characterisation of IIH with q-aMRI and MRI-based flow methods

Presenter:
Jet WRIGHT[1,2]

Contributors:
Alireza Sharifzadeh-Kermani, Paul Condron, Sergio Dempsey, Vickie Shim, Helen V. Danesh-Meyer, Samantha J. Holdsworth, Eryn Kwon

Institution:
1. Mātai Medical Research Institute, Tairāwhiti Gisborne, New Zealand;
2. Auckland Bioengineering Institute, University of Auckland, Auckland, New Zealand

Intracranial dynamics are governed by the Monro-Kellie doctrine, which describes a pressure–volume equilibrium among brain tissue, blood, and cerebrospinal fluid (CSF) within the rigid cranial vault. Disruption of this balance underlies conditions such as idiopathic intracranial hypertension (IIH), a pressure disorder characterised by elevated intracranial pressure without overt structural causes. While previous research has explored CSF flow abnormalities in Chiari malformation, a comprehensive physiological profile of IIH remains limited. This study aimed to characterise IIH using a multimodal MRI approach combining quantitative amplified MRI (q-aMRI) for brain tissue displacement, four-dimensional flow MRI (4D Flow) for cerebral blood flow (CBF), and two-dimensional phase-contrast MRI (2D PC-MRI) for CSF flow analysis.

Suspected IIH cases and controls underwent high-resolution 3T MRI. Imaging protocols included whole-brain q-aMRI, volumetric CBF acquisition, and targeted CSF flow measurements at the aqueduct and C2 spinal level. IIH participants were scanned both before and after therapeutic lumbar puncture; controls were scanned once.

The methods used in this study were previously validated in an exercise cohort to observe differences in dynamics pre and post low intensity exercise. Whilst data for IIH is currently being analysed, the intention for this study is to better understand both the dynamic profile of IIH and how we may better diagnose the condition. We aim to improve the diagnosis and treatment pathway for IIH by creating a clinical toolbox to non-invasively diagnose the condition, reducing the burden for both patients and the healthcare system.

12.05

A Systematic Review of Patient-Reported Outcome Measures (PROMs) and Qualitative Studies to Investigate the Hidden Burden of Idiopathic Intracranial Hypertension (IIH) on Quality of Life (QoL)

Presenter:
Shilpan G Patel

Authors:
Paige Richter, Riddhima Gautam, Helen V. Danesh-Meyer, Benson S. Chen

Institution:
University of Auckland, University of Cambridge
Vision Research Foundation, Te Toka Tumai Auckland

Email:
prci068@aucklanduni.ac.nz
benson.chen@auckland.ac.nz

Purpose: Despite increasing recognition of the burden idiopathic intracranial hypertension (IIH) places on patients, there is limited understanding of how well existing patient-reported outcome measures (PROMs) capture their lived experiences. The purpose of this study was to identify and comprehensively evaluate studies capturing the experiences of individuals living with IIH, with a focus on PROMs and qualitative studies describing the patient experience.

Methods: A systematic literature review was conducted using a search strategy that combined four concepts: (1) IIH; (2) QoL and health status; (3) PROMs; and (4) qualitative research. Studies assessing the impact of IIH on any QoL domain using a PROM or qualitative methodology were included and appraised, using criteria based on the COSMIN checklist (for PROM studies) and the CASP checklist (for qualitative studies). Identified PROMs were grouped by the QoL domain or concept(s) they intended to capture.

Result: Of the 2255 studies identified, 33 studies were included. PROMs in IIH research clustered around 3 principal domains: headache-related burden, vision-related quality of life, and mental health (including emotional and psychological wellbeing). Commonly used PROMs included the Headache Impact Test (HIT-6) and the National Eye Institute Visual Function Questionnaire (NEI-VFQ-25). Many studies assessed health-related QoL using generic PROMs such as the 36-item Short Form Survey (SF-36). The psychometric performance of included PROMs were poorly described. No high-quality qualitative studies specifically exploring the lived experience or QoL impact of IIH were identified.

Conclusion: Existing PROMs in IIH research provide only a partial view of patient experience, with a narrow focus on symptoms like headache and vision loss. There is a clear need for co-designed research with people living with IIH to identify which aspects of life are most affected and how these should be meaningfully captured, whether through existing PROMs or the development of new outcome measures.

12.15

Case series. Myeloproliferative neoplasm: a risk factor for IIH.

Presenter:
Sarah Morgan

Authors:
Sarah Morgan[1,2], Matthew Wright[3], William Morgan[4], Neha Irani[1,2,5]

Institutions:
1. Neurology department, Royal Perth Hospital, Western Australia
2. Ophthalmology department, Sir Charles Gairdner Hospital, Western Australia
3. Haematology department, Fiona Stanley Hospital, Western Australia
4. Lions Eye Institute, Perth, Western Australia
5. Neurology department, Joondalup Health Campus, Western Australia

Email:
Sarah.morgan@health.wa.gov.au

Purpose: Idiopathic intracranial hypertension (IIH) has been rarely associated with myeloproliferative neoplasms (MPN) in previous case reports. At times, this has been treated with cytoreductive therapy, with improvement in IIH. The relationship between IIH and MPN, however, remains unclear. We present a case series of four patients with IIH, with associated MPN. The clinical characteristics, potential relation to MPN, and treatments are discussed.

Method and results: Four patients were assessed in Western Australia, with IIH and MPN.
All presented with headache, papilloedema, and MRI features of IIH. All four had a preceeding diagnosis of MPN, or were diagnosed soon after IIH presentation, and all were JAK2-V617F positive. Three had essential thrombocythaemia, and one had polycythaemia vera. None had venous sinus thrombosis. Two out of four were associated with overweight, but there were no other risk factors for IIH. One patient presented with severe optic disc swelling, opening pressure of 40.5cmH2O, without overweight; treated with acetazolamide without improvement, followed by cytoreductive therapy and aspirin, with resolution of papilloedema, headache and thrombocytosis. Two patients had mild-moderate optic disc swelling; treated with standard IIH management of acetazolamide and weight loss, plus aspirin, and despite unchanged thrombocytosis, the IIH resolved. One patient had mild optic disc swelling; treated with topiramate and weight loss alone, and despite unchanged thrombocytosis, the IIH resolved.

Conclusion: MPN was identified as a risk factor for IIH in our case series, and in prior reports. Resolution of more severe IIH required cytoreductive therapy in one patient. In contrast, in three patients with milder IIH, resolution of IIH occurred following aspirin, and/or standard IIH management. Previously postulated mechanisms for this association include impaired CSF absorption at arachnoid granulations. We hypothesize increased resistance to CSF reabsorption due to MPN causes congestion of the glymphatic system, worsening the hydrodynamic cascade of IIH.

12:25

Paediatric patients with optic disc drusen and corresponding visual field defects

Presenter:
Sujan Surendran

Authors:
Dr Sujan Surendran, Dr Jeremy Mathan, Dr Shivanand Sheth

Institution:
Royal Children's Hospital Melbourne

Email:
jeremy.mathan@rch.org.au

Purpose: Herein the authors report on three patients clinically deemed to have optic disc drusen with corresponding visual field defects

Method: Three cases from the outpatient ophthalmology clinic at the Royal Children's hospital Melbourne's are described.

Results: Three cases are of girls aged 5, 6 and 8 who were all initially referral to the Ophthalmology service for optic nerve head swelling. All presenting cases were initially considered as having pseudopapilloedema or papilloedema. All patients examined to have disc drusen. MRI for 2 patients were unremarkable but one patient had an incidental chiari 1 malformation that was mild with no evidence of hydrocephalus. Lumbar puncture for one patient was elevated at 31cmH20 was normal in one patient and also normal in the patient with the Chiari 1 malformation who additionally went on to have an ICP monitor which was also normal. All patients were commenced on oral acetazolamide with demonstrable improvement in their OCT RNFL post oral acetazolamide. At clinical reviews approximately 5 years following their original presentation all patients demonstrated significant peripheral visual field defects.

Conclusion: We report on paediatric patients with disc drusen and visual field defects. These cases present a diagnostic and management challenge due to the possibility of dual pathology by virtue of either documented elevated CSF pressure or tomographic improvement on oral acetazolamide with or without raised CSF pressure. Topical antihypertensives may have a role in preserving optic nerve function in patients.

14:30

Contrast Sensitivity in Metastatic Prostate Cancer Patients on Androgen Deprivation Therapy

Presenter:
Antonia Kartika

Authors:
Antonia Kartika, Aaron Tigor Sihombing, Intan Ekarulita, Rusti Hanindyasari, Dianita Veulina Ginting, Prettyla Yolamanda

Institution:
Neuro-Ophthalmology Unit-Cicendo Eye Hospital, Urology Department-Hasan Sadikin Hospital , Faculty of Medicine Universitas Padjadjaran Bandung-Indonesia

Email:
antonia_kartika@yahoo.com

Purpose: To assess contrast sensitivity in patients with metastatic prostate cancer on androgen deprivation therapy (ADT).

Method: A cross-sectional study was performed on 2 patient groups; group 1 consists of 13 metastatic prostate cancer patients who received ADT and group 2 consist of 8 benign prostate hyperplasia (BPH) patients as control. Mars numeral contrast sensitivity test was performed in both groups.

Results: Mars numeral contrast sensitivity test was reduced significantly in both eyes of group 1 compared to group 2 (p=0.035).

Conclusion: Contrast sensitivity test was reduced in metastatic prostate cancer patients on ADT

14:40

Prognostic factors for long term outcomes in optic neuritis: a prospective registry-based cohort study

Presenter:
Michael T. M. Wang

Authors:
Michael T. M. Wang, Helen V. Danesh-Meyer

Institution:
University of Auckland

Email:
mwan759@aucklanduni.ac.nz

Purpose: To investigate the association between optic neuritis index episode parameters and long-term visual acuity (VA) recovery and retinal nerve fibre layer (RNFL) atrophy.

Methods: Prospective registry-based cohort study of all patients with first-episode optic neuritis referred to a tertiary Neuro-ophthalmology service in Auckland from 2011 onwards. Demographic, clinical, serological, and radiological data were collected.

Results: A total of 157 consecutive patients (95 females; mean±SD age, 39±18; mean±SD follow-up 4.8±5.3 years) were included. Thirteen (8.3%) patients had multiple sclerosis (MS), 27 (17%) patients neuromyelitis optica spectrum disorder (NMOSD), 48 (31%) patients myelin oligodendrocyte glycoprotein antibody disease (MOGAD). Corticosteroid therapy was administered in 85 (54%) patients, intravenous immunoglobulins in 5 (3%) patients, and plasmapheresis in 15 (10%) patients. Median (IQR) VA improved from 20/60 (20/30 to CF) at nadir to 20/20 (20/20 to 20/30) at final follow-up ($p<0.001$), while mean±SD RNFL decreased from 128±45 to 83±22µm ($p<0.001$). Multivariate regression analysis demonstrated that MOGAD and intravenous corticosteroid therapy were positive predictors of final VA recovery to 20/20 or better (all $p<0.05$); while advancing age, decreased nadir VA, NMOSD, longitudinally extensive optic neuritis, and active brain lesions on MRI were independently associated with poorer final VA (all $p<0.05$). Decreased baseline RNFL, NMOSD, MOGAD, and active brain lesions on MRI predicted long-term RNFL thinning below the first percentile (all $p<0.05$).

Conclusions: Overall, optic neuritis patients demonstrated favourable visual outcomes. Age, disease aetiology, nadir VA and RNFL, longitudinally extensive optic neuritis and active brain lesions on MRI might predict the subgroup with poorer long-term outcomes.

14:50

Low serum pyrophosphate as a model for optic disc drusen

Presenter:
Jesse Gale

Authors:
Jesse Gale[1,2], Yuanzhang Jack Jiao[2]

Institutions:
1. University of Otago Wellington, Wellington, New Zealand
2. Wellington Regional Hospital, Wellington, New Zealand

Purpose: Here we report several clinical observations which suggest low serum pyrophosphate is a contributing factor to the pathogenesis of optic disc drusen.

Methods: Three cases are described with overlapping features: angioid streaks, optic disc drusen (ODD), pseudoxanthoma elasticum (PXE), inherited anaemia, and non-arteritic anterior ischaemic optic neuropathy (NAION).

Results: In one case, a rare congenital anaemia was associated with angioid streaks and ODD. An animal model of thalassaemia shows how anaemia can downregulate the gene for PXE and result in similar phenotype. Another case had angioid streaks in association with fibrous dysplasia of the sphenoid bone (a new association), and bilateral NAION with possible tiny ODD. A third patient with genetic PXE and severe angioid streaks also developed NAION, but without ODD, suggesting a primary vasculopathy.

Conclusion: Together these cases highlight the role of pyrophosphate in ectopic calcification of the eye, a potentially therapeutic target. Our international collaborations aim to automate the detection and quantification of ODD, and the reflectivity of Bruch membrane, as outcomes and biomarkers for ongoing clinical trials.

15:00

Bitemporal optic atrophy from retrograde transsynaptic axonal degeneration may signify cerebral injury in children

Presenter:
Jack Jonathan Maran

Authors:
Dr Jack Jonathan Maran, Dr Cynthia Sharpe, Dr David Perry, Prof Helen Danesh-Meyer, Dr Sarah Hull

Institution:
Te Whatu Ora and University of Auckland

Email:
jjonathan1810@gmail.com

Purpose: The patterns of optic atrophy due to retrograde transsynaptic degeneration (RTSD) have not been well characterised in children. This study aimed to characterise optic atrophy in pediatric patients with focal intracerebral lesions.

Method: A retrospective review of children with optic atrophy and focal intracerebral lesions was conducted. Ophthalmic data were recorded, including visual acuity, colour vision, formal automated visual fields and optical coherence tomography (OCT) of the peripapillary retinal nerve fibre layer (pRNFL) and ganglion cell layer.

Results: Six patients (83.33% male) were included. The mean visual acuity (VA) of all eyes was 0.30 logMAR (20/40 Snellen), with no significant difference in the mean logMAR VA in the ipsilateral eye to the location of the lesion compared with the contralateral eye (0.30 vs 0.30, P = 1.000). Color vision (available in 5 patients) was normal in 2, mildly reduced in one and markedly reduced in 2. Bitemporal optic disc pallor was observed in 5 out of 6 patients. OCT data revealed that pRNFL thickness was most significantly diminished in the temporal (95% CI: -44.71 to -14.18 µm, P = 0.0021), inferotemporal (95% CI: -75.06 to -5.17 µm, P = 0.0294), and superotemporal (95% CI: -76.82 to -18.51 µm, P = 0.0055) sectors. Average pRNFL thickness was significantly reduced compared with normative data in both the ipsilateral (95% CI: -40.76 to -11.69 µm, P = 0.0003) and the contralateral eye (95% CI: -38.46 to -5.83 µm, P = 0.0063). When only nasal and temporal data were analyzed, mean pRNFL thickness was still diminished compared with normative data (95% CI: -33.01 to -9.77 µm, P = 0.0012).

Conclusion: Children presenting with optic atrophy, particularly with bitemporal optic atrophy, should have neuroimaging to exclude any underlying serious intracranial pathology.

15:10

Inherited optic neuropathies- a Multiethnic Asian study

Presenter:
Dr Shweta Singhal

Authors:
Yasmin Bylstra, Wen Kong Lim, Kanika Jain, Saumya Jamuar Jing Liang Loo, Shweta Singhal

Institution:
Singapore National Eye Centre

Email:
Shweta.singhal@singhealth.com.sg

Purpose:
Inherited optic neuropathies are underdiagnosed in the Asia-pacific region. This study aimed to understand the phenotype and prevalence of non-LHON inherited optic neuropathies in a multiethnic Asian (mixed Chinese, Malay and Indian) population presenting to a tertiary eye care centre in Singapore.

Method:
In this prospective study, all patients presenting to our neuro ophthalmology clinic since 2021 with bilateral pale discs and optic neuropathy for which no underlying compressive, toxic, traumatic or inflammatory cause was identified; and who tested negative for LHON primary mutations were recruited. Clinical phenotype data was collected and whole exome and mitochondrial DNA sequencing was performed.

Results:
Over 40 probands and family members were recruited in the study and solve rate was over 50%. OPA1 optic atrophy was the commonest mutation detected followed by WFS1. Many cases showed an optic atrophy only phenotype. Novel pathogenic nuclear and mitochondrial genomic mutations were also identified and interesting case phenotypes will be discussed.

Conclusion:
OPA1 and WFS mutations are prevalent and common causes of inherited optic neuropathies in the Asia pacific region. Increased index of suspicion and access to molecular testing will help correct diagnosis and management.

15:20

Neuropsychiatry of neuro-ophthalmology Through the Lens of Traumatic Brain Injury.

Presenter:
Dr Gil Newburn, Neuropsychiatrist

Authors:
Dr Gil Newburn

Institution:
Matai Medical Research Institute.

TBI is a common phenomenon with symptoms persisting in a significant proportion, including after so-called mild TBI. TBI is in essence a deafferentation syndrome with loss of connectivity impacting on network function across the brain. Vision is a process dependent on widely distributed connections across the CNS making it particularly susceptible to TBI, at a primary, secondary and tertiary level of neuropathological process (as with other deafferentation processes – e.g. multiple sclerosis). Visual function requires a coordinated set of functions involving end organ (eye) with the retinal extension of the CNS, delivery of this signal to the relevant processing systems which determine presence and via a series of developing processes building to an image which may enter consciousness, allowing executive processes to begin to act on the stimulus. TBI interacts at all levels of visual function to create a set of symptoms which interfere with the person's capacity to maintain mastery and control over themselves, their environment and their future. This commonly leads to an increasing set of secondary symptoms arising from the tertiary neuropathological processes initiated by stressors of living with their condition. These interacting factors will be discussed.

16:50

Saccadic Abnormalities as Biomarkers: Insights from Electrophysiology and Deep Learning.

Presenter:
Dr Cathy Zhong, MBChB, FRACP

Authors:
Dr Cathy Zhong

Institution:
Wellington Regional Hospital
The University of British Columbia

Email:
Cathy.s.zhong@gmail.com

Saccadic abnormalities have been characterised in various neurological disorders, but the extent to which they serve as a biomarker rests on our understanding of saccadic mechanisms. Using a novel approach combining electrophysiology and deep learning, we take advantage of the high temporal resolution to provide insight into the outstanding issues.

14:50
Neurological sequelae of herpes zoster

Presenter:
Dr Rachael Neiderer

Authors:
Dr Rachael Neiderer, PhD, MBChB, FRANZCO

Herpes zoster ophthalmicus is known to be associated with optic neuropathy in 1.5% of patients and cranial nerve palsies in 3.5%. Emerging data demonstrates other neurological sequalae associated with herpes zoster, particularly cerebrovascular accident (CVA) and dementia. This talk will review the evolving evidence and examine risk factors for neurological sequelae and their management.

10:00

Eye-tracking Technology in Myasthaenia Gravis

Presenter:
Akarsh Mathrani

Authors:
Matthew A. McDonald, Akarsh Mathrani, Jackie Low, Guy Neuberger, Helen V. Danesh-Meyer

Institution:
Vision Research Foundation

Email:
akarsh@vrf.org.nz

Purpose: Myasthaenia Gravis (MG) is an autoimmune condition characterised by impaired neuromuscular transmission at striated muscle junctions. Although well-established treatments exist, diagnostic challenges remain due to the heterogeneity of presentation and imperfect sensitivity of current diagnostic tools (e.g. serology and electromyography). The purpose of this study is to demonstrate the utility of eye tracking technology for the diagnosis of MG. Eye tracking offers a quick, non-invasive, and cost-effective tool which may characterise extraocular muscle dysfunction endemic to MG.

Method: In this study, we utilised an in-house infrared-based eye tracking device operating between 250-500 frames per second to collect and compare both fixation and smooth pursuit measures. The smooth pursuit task included both a non-accelerating and accelerating target to determine the point at which their pursuit broke down into poorly formed saccades (referred to as 'smooth pursuit breakdown'). MG patients (n=13) and healthy control subjects (HC; n=26) were age-similar and gender-matched from Auckland, New Zealand. MG participants had both positive serology and EMG findings. All eye tracking procedures were conducted in neuro-ophthalmology clinics alongside their standard clinical assessments.

Results: Preliminary findings reveal significant differences in eye tracking profiles between the two cohorts for both fixation and smooth pursuit tasks. MG subjects displayed earlier smooth pursuit breakdown with a lower amplitude error compared to HC subjects. Receiver operating characteristic analyses of this breakdown threshold presented an area under curve of 0.82, confirming good diagnostic viability.

Conclusion: This study highlights the potential of eye tracking as a diagnostic tool for both efficient and accurate identification of MG. These devices may offer a non-invasive and cost-effective means of monitoring both symptom severity and even recovery over time.

10:10

Can structural grading of foveal hypoplasia and foveation duration predict visual acuity in infantile nystagmus syndrome?

Presenter:
Anthony Fok

Authors:
Amanda Douglass B Optom, GCUT, PhD FHEA, Christopher Law BSc OD MPH, Madeline Baker M Optom, Matt J Dunn PhD MCOptom FHEA and Larry Abel PhD

Institutions:
Monash Health, Melbourne Health, Royal Victorian Eye and Ear Hospital, Deakin University, Cardiff University

Email:
Anthony.fok3@gmail.com

Purpose: Determining the impact of foveal hypoplasia and foveation duration on visual acuity in infantile nystagmus syndrome (INS) patients.

Participants: Twenty-five patients with INS (6 with albinism, 19 with idiopathic infantile nystagmus) were examined

Methods: Records were retrospectively examined for twenty-five patients diagnosed with INS based on clinical presentation and waveform recordings. Spectral-domain optical coherence tomography (OCT) of the macula records were graded for foveal hypoplasia using the Leicester grading scale. Eye movements were recorded with an Eyelink 1000 Plus and analysed using an automated program for analysing INS waveforms. The null location was confirmed by visual inspection. Foveation duration, foveal hypoplasia grade and visual acuity were analysed using simple and multilinear linear regression and correlation models. Results: Increasing foveal hypoplasia grades correlated with worsening logMAR visual acuity (Spearman's r = 0.61 p = 0.0011) and with shorter foveation duration (Spearman's r = -0.45 p = 0.036). Foveation duration showed no correlation with visual acuity either across all foveal grades analysed (p=0.57) and for low grades (grades 0, 1a and 1b) (p =0.99).

Conclusion: Severity of foveal hypoplasia is associated with worsening visual acuity and shorter foveation duration. Visual acuity was not affected by foveation duration in our case series of INS patients.

10:20

When Two Conditions Collide: Exploring Internuclear Ophthalmoplegia with See-Saw Nystagmus

Presenter:
Irna Novianti Irwan

Authors:
Irna Novianti Irwan[1,3], Yunita Mansyur[1,3], Muhammad Iqbal Basri[2,3]

Institutions:
1. Department of Ophtalmology, Hasanuddin University, Makassar, Indonesia
2. Department of Neurology, Hasanuddin University, Makassar, Indonesia
3. Wahidin Sudirohusodo Hospital, Makassar, Indonesia

Email:
irna.noviantiirwan@yahoo.com

Introduction: Internuclear ophthalmoplegia (INO) is characterized by the inability to adduct the eye on the ipsilesional side, while the contralesional eye shows nystagmus. This condition is often caused by lesions in the medial longitudinal fasciculus. See-saw nystagmus (SSN) involves alternating movements where one eye elevates and intorts while the other depresses and extorts. The occurrence of both conditions in the same patient commonly was rare. This study presents a case of a patient who developed both INO and SSN due to chronic small vessel disease

Case Illustration: A 47-year-old male presented to the hospital with an inability to move his left eye to the right gaze, diplopia and dizziness. His best corrected visual acuity was 20/25 in both eyes, and intraocular pressure was normal. Ophthalmic examination revealed seesaw nystagmus, with the right eye elevating and intorting while the left eye depressed and extorted. Both posterior segments were normal. MRI showed chronic small vessels disease. The patient diagnosed with internuclear ophthalmoplegia with bilateral see-saw nystagmus. He was referred to neurologist and treated with pharmacological therapy which led to significant improvement after six months.

Conclusion: This case highlights the clinical findings of both SSN and INO with risk factors of cerebrovascular disease, underscoring the likelihood of an acute brainstem stroke. The earlier diagnose and therapy, the better patient's outcome The diagnosis depends on clinical findings and neuroimaging. The patient showed significant improvement after six months of antiplatelet, antihistamines, and neuroprotective therapy.

Keywords: internuclear ophthalmoplegia, see-saw nystagmus, chronic small vessel disease, pharmacological therapy

10:30
Revisiting the directional selectivity of saccade abnormalities in Progressive Supranuclear Palsy

Presenter:
Dr Mark Paine

Authors:
Duy Duan Nguyen[1,2], Mark Paine[3], Soohyun Lee[1], Amir Fazlollahi[1,5], John D. O'Sullivan[3,6], Peter J. Nestor[1,2,4]

Institution:
1. Clem Jones Centre for Ageing Dementia Research, Queensland Brain Institute, The University of Queensland, St Lucia, QLD 4072, Australia
2. Cognitive Health Program, Mater Research Institute, The University of Queensland, QLD 4101, Australia
3. Department of Neurology, Royal Brisbane & Women's Hospital, QLD 4006, Australia
4. Department of Neurology, Mater Adult Hospital, Mater Misericordiae Limited, South Brisbane, QLD 4101, Australia
5. Department of Radiology, Royal Melbourne Hospital, The University of Melbourne, Melbourne, Victoria, Australia
6. UQ Centre for Clinical Research, Faculty of Medicine, The University of Queensland, Herston, Queensland, Australia

Email:
Mark.Paine@alexandraneurology.com.au

Purpose: To determine whether, as is commonly assumed in progressive supranuclear palsy (PSP), vertical saccades are disproportionately slowed relative to horizontal, and, whether aberrant saccade trajectory might differentially affect vertical or horizontal directions.

Method: N=20 PSP patients were compared to age-matched controls (n=82) and two degenerative groups (Alzheimer's disease, n=21; Lewy body disease, n=50). Eye movements were recorded using EyeLink videooculography (sampling rate: 1000 Hz). Participants completed 10 vertical (5 upward, 5 downward) and 10 horizontal (5 leftward, 5 rightward) visually guided saccade trials (15.3° amplitude; target duration: 500 ms). Velocity was normalized using a main sequence correction (Peak Velocity = Corrected V × √Amplitude). Trajectory was assessed by a novel misaligned angle metric, defined as the deviation between the actual saccade axis and the ideal axis (horizontal or vertical).

Results: Velocities were slowed in PSP however within-subject differences between horizontal and vertical did not differ significantly between groups (ANOVA, $p = 0.18$, $\eta^2 = 0.03$). Z-score analysis relative to controls also showed no preferential vertical slowing in PSP ($p = 0.50$; Cohen's $d = 0.22$). Vertical and horizontal velocities were strongly correlated in PSP ($r = 0.83$, $p < 0.001$). Vertical saccade trajectory was consistently more misaligned than horizontal across all groups (including controls), but this effect was significantly exaggerated in PSP ($p<0.005$). Horizontal trajectory did not differ between groups. No significant correlation was observed between velocity and trajectory.

Conclusion: Contrary to conventional belief, vertical saccades were not selectively impaired in PSP. Instead, comparable vertical and horizontal velocity reduction with exaggerated vertical misalignment suggest a more global saccadic system disruption. The observation of vertical saccade deficits in PSP may stem from horizontal saccades, though abnormal, being too fast to observe at the bedside combined with greater vertical trajectory disorganization, rather than a uniquely vulnerable vertical velocity.

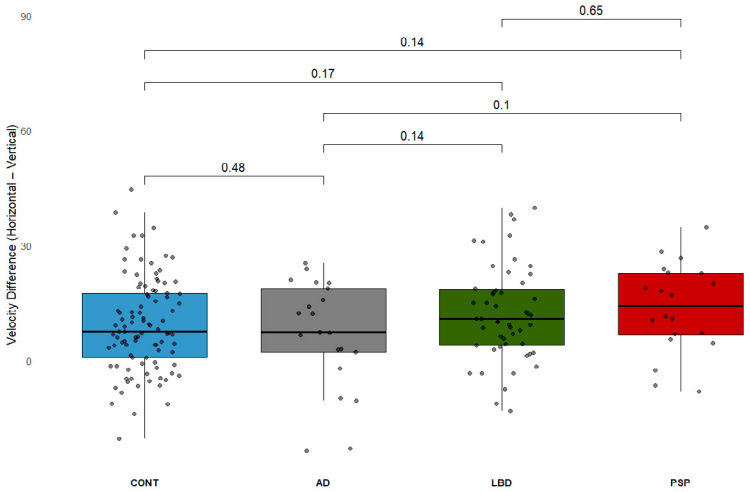

Figure 1. Saccade directionality difference (Horizontal – Vertical velocity) across groups with Bonferroni-corrected pairwise t-test p-values.

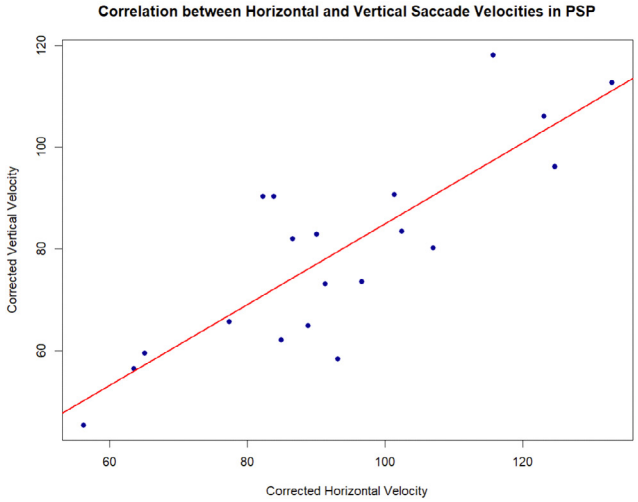

Figure 2. Correlation between Horizontal and Vertical Saccade Velocities in PSP Patients ($r = 0.830$, $p < 0.001$).

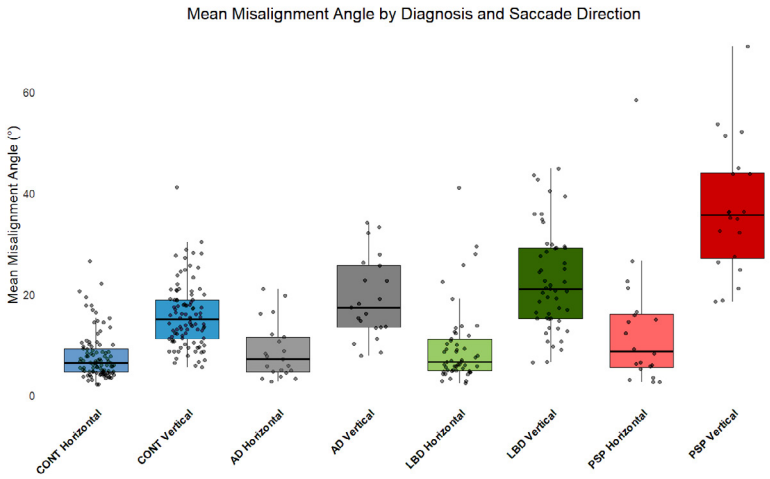

Figure 3. Boxplot of Mean misaligned angle of horizontal and vertical saccades across different groups. Note that the misalignment is greater for vertical compared to horizontal saccades in all groups (including controls) but this effect is exaggerated in PSP.

11:40

Hypovitaminosis A and visual loss in children with restricted diets

Presenter:
Sarah Hull

Authors:
Jane Shi, James Caldwell, Leo Sheck, Bobby Tsang, Rebecca Alekzander, Julia Escardo-Paton, Andrea L Vincent, Claire Spooner, Peter Heppner, Helen Danesh-Meyer, Sarah Hull

Aim: Hypovitaminosis A is a leading cause of preventable childhood blindness, especially in developing nations. Vitamin A is a fat-soluble essential micronutrient that serves vital functions in the visual system and in regulating bone resorption. We report on a series of four children with mixed nutritional and compressive optic neuropathy and provide a review of the literature.

Methods: A retrospective observational study of four males (ages 9-12), three with autism spectrum disorder who presented with loss of vision and multiple vitamin deficiencies including hypovitaminosis A.

Results: Patients presented with unexplained visual loss or a change in visual behaviour. All patients had severely restricted diet comprising of predominantly carbohydrates. Two of the four cases demonstrated optic nerve pallor at initial presentation with marked optic atrophy developing in all patients over time. Electrophysiology available in two patients demonstrated optic nerve dysfunction with preserved retinal function. Extensive investigations revealed profound deficiency in multiple vitamins including vitamin A (<0.1-0.2 µmol/L, normal = 0.9-1.7 µmol/L). Three patients also had low vitamin B12 (90-111 pmol/L, normal = 170-800 pmol/L) with normal folate. All four cases had radiological evidence of skull base thickening indicative of low vitamin A. Genetic testing did not find any relevant pathogenic variants.

Conclusions: Hypovitaminosis A is a crucial form of nutritional deprivation that results in significant visual loss with potential hyperostosis and optic nerve compression exacerbating nutritional optic neuropathy. Additional micronutrient deficiencies usually co-exist and may contribute. Extra vigilance in vitamin replacement is required of clinicians with patients with autism who have restricted diets.

11:50
Vision-Related Quality of Life in Friedreich's Ataxia

Presenter:
Oliver Burnett

Authors:
Oliver Burnett, Juno Collins, Balaje Vivekanandan, Richard Roxburgh, Joseph Donnelly, Benson S. Chen

Institutions:
Department of Medicine, University of Auckland
Department of Neurology, Te Toka Tumai Auckland

Email:
oliver.burnett@auckland.ac.nz

Purpose: Friedreich's ataxia (FRDA) is the most common autosomal recessive hereditary ataxia. While cerebellar involvement gives rise to gait ataxia, FRDA also affects peripheral nerves, the heart, and the visual system, each of which may contribute to the patients' experience of the condition. In this study, we explored the relationship between visual function, disease severity and vision-related quality of life (VRQoL) in people with FRDA.

Method: This was a cross-sectional observational study of individuals with FRDA in Aotearoa. Participants attended a research clinic where they completed standardised questionnaires and underwent detailed neurological examination and measurement of visual acuity. VRQoL was assessed using the National Eye Institute Visual Function Questionnaire (VFQ-25), the 10-Item Neuro-Ophthalmic Supplement, and the Impact of Visual Impairment Scale (IVIS). Disease severity was assessed using the Scale for the Assessment and Rating of Ataxia (SARA) and the Friedreich's Ataxia Rating Scale - Activities of Daily Living (FARS-ADL). The association between vision-related scores and disease severity-related scales were explored with Spearman's ranked test; $P<0.05$ was considered statistically significant.

Results: Seventeen participants were included (58% female). Mean±standard deviation scores at baseline were 82.7±15.8 (VFQ-25), 91.6±19.1 (10-item Neuro-Ophthalmic Supplement), 2.3±3.4 (IVIS), 21.3±8.8 (SARA), and 20±5.9 (FARS-ADL). No significant correlation was found between SARA and VRQoL scores. In contrast, FARS-ADL was strongly negatively correlated with VFQ-25 (-0.77, $p = 0.0003$), and positively correlated with IVIS ($r = 0.70$, $p = 0.0052$). FARS-ADL and SARA were moderately correlated ($r = 0.64$, $p = 0.0061$).

Conclusion: Patients with FRDA report a high level of VRQoL as determined by the VFQ-25 and VFQ-10. However, as FARS-ADL scores increase, VRQoL decreases. These findings raise the possibility that commonly used VRQoL measures in FRDA may reflect broader aspects of disease burden beyond visual impairment and should be interpreted with caution in this population.

12:00

Linezolid-induced Optic Neuropathy - A Case Series

Presenter:
Alexander Sarossy

Authors:
Alexander Sarossy[1, 2], Rachael C. Heath Jeffery[3], Fred K. Chen[3], Marko Hawlina[4], Lea Kovac[4], Marc Sarossy[1, 2]

Institution:
1. Alfred Hospital, Melbourne, Victoria
2. Monash University, Clayton, Victoria
3. Lions Eye Institute, Perth, Australia
4. Ljubljana University, Ljubljana, Slovenia

Purpose: To report a case series of linezolid-induced optic neuropathy with clinical features resembling Leber hereditary optic neuropathy (LHON)

Method: we describe three patients who developed bilateral optic neuropathy while receiving linezolid. Patients underwent comprehensive neuro-ophthalmological assessment including visual acuity, perimetry, colour vision testing, optical coherence tomography and mitochondrial DNA analysis.

Results: Three patients (2 female, 1 male, ages 28-66 years) developed bilateral sequential painless central vision loss after 2-9 months of linezolid 600mg daily therapy for antibiotic-resistant microbial infections. Clinical presentation closely resembled LHON with cecocentral scotomas, severe dyschromatopsia, peripapillary telangiectasia, thickening of nerve fibre layers and preferential papillomacular thinning on OCT imaging. Visual acuities in affected eyes ranged from 6/18 to counting fingers at presentation. One patient carried a variant of uncertain significance in OPA3 gene. Another case was found to be homoplasmic for m.11778G>A. The third patient presented with asymmetric involvement (6/18 right eye, 6/6 left eye) which remained stable. Following immediate linezolid cessation upon recognition of toxicity, two patients experienced gradual partial visual recovery over 3-8 months, while visual function remained static in the patient with asymmetric presentation.

Conclusion: Linezolid-induced optic neuropathy demonstrates remarkable clinical and imaging similarities to LHON, reflecting their potentially shared mitochondrial toxicity mechanism. The antibiotic is a rarely used but important therapy for mycobacteria and antibiotics-resistant gram-positive organisms. Ophthalmologists should work with their physician colleagues to ensure screening is performed regularly as prompt recognition and immediate drug withdrawal may improve visual prognosis, though recovery remains unpredictable and often incomplete.

13:55
A massive thirst

Presenter:
Anthony Yao

Institution:
Royal Victorian Eye and Ear Hospital

A 37-year-old man presented with acute vision loss in his left eye, on a background history of 3 weeks of worsening bilateral proptosis and conjunctival injection, associated with subjective fevers, chills and headache.

Examination showed VA of RE 6/12 LE LP, with dense LE RAPD. Intraocular pressure was RE 15mmHg LE 14mmHg. Exophthalmometry measured RE 25mm LE 32mm. Motility was limited in the RE -1 all gazes, and LE -4 elevation, -1 depression, -2 dextroversion, -1 laevoversion. Light and red saturation were severely reduced in the LE.

Further questioning revealed a preceding 12-month history of polydipsia and polyuria, with the patient drinking up to twenty-four litres of water a day…

14:10
A Swell Case

Presenter:
Evelyn Turek

Recent literature has questioned the necessity of diagnostic lumbar puncture in patients with typical features of idiopathic intracranial hypertension (IIH) without red flags. We present a case of a 37-year-old woman with a history of IIH, previously treated with acetazolamide and weight loss. Fifteen years later, she re-presented with recurrent pulsatile tinnitus, transient visual obscurations, and bilateral optic disc swelling. Would you have requested a lumbar puncture in this scenario? In this case, we did… and the results were not what we expected

14:25
Think Poetically

Presenter:
Liana Dedina

Authors:
Dr. Liana Dedina, Prof. Celia Chen

In 2025, a 54-year-old female was referred to the neuro-ophthalmology clinic for evaluation of bilateral optic disc oedema. The patient reports occasional headaches but denies any associated tinnitus or visual disturbances. Her medical history is significant for hypertension.

Her ophthalmic history includes incidental detection of bilateral optic nerve swelling by an optometrist 2 years earlier, when she was referred to the eye clinic. Investigation at the time included MRI head and MRV, which were unremarkable. Lumbar puncture was also conducted. Opening pressure was 14 mm Hg. CSF composition showed raised proteins with other paraments being within normal limits.

14:40
Toxic Relationships

Presenter:
Dr. Melissa Tien

A 38-year-old Chinese male presented to the Ophthalmology department complaining of an increase in right eye floaters. He was found to have right intermediate uveitis. Fundus fluorescein angiography revealed vascular leak in both eyes, right more than left. Serological screening for autoimmune and infective causes were negative. Two-weeks after, the patient presented again with binocular horizontal and vertical diplopia. A neuro-ophthalmic consult was obtained to determine the cause of diplopia. Left eye inferior rectus weakness with superior rectus overaction was found. Additionally, there was partial ptosis on the left. No pupillary abnormalities were found. Neuroimaging was obtained and diagnostic tests were performed.

14:55

A Silent Infiltrator: Hidden in plain sight

Presenter:
Yunita Mansyur

Institutions:
1. Department of Ophthalmology, Hasanuddin University, Makassar, Indonesia
2. Wahidin Sudirohusodo Hospital, Makassar, Indonesia
3. EC Orbita Makassar, Indonesia

A 12-year-old boy presented with a rapidly progressive bilateral visual decline and complete ophthalmoplegia, accompanied by systemic symptoms including persistent headache, nausea, and lethargy. Ophthalmologic evaluation revealed bilateral ptosis, afferent pupillary defects, and total ocular motility impairment, with vision reduced to light perception. Initial laboratory investigations pointed toward anemia and a profound systemic inflammatory response.

As the clinical picture expanded, additional findings emerged, connecting ocular symptoms to a much deeper pathology with unexpected systemic involvement and rapid progression. What initially appeared as an isolated ophthalmic complaint developed into a multidisciplinary investigation involving neuro-ophthalmology, pediatrics, radiology, and pathology.

15:30
Out of Sight, Out of Time

Presenter:
Jessie Cai

A 68-year-old man with a history of atrial fibrillation on dabigatran, hypertrophic cardiomyopathy, hypertension, and type 2 diabetes presented 90 minutes after sudden, sequential, painless vision loss. This occurred first in the right eye, then in the left a few minutes later. He denied new onset headache or jaw claudication, and no focal neurological deficits were noted on examination

15:50

Out of Sight, Not Out of Mind: It starts with that sinking feeling

Presenter:
Liam Walsh

A 59-year-old woman presented to eye clinic with a two to three week history of mildly blurry vision, bilateral optic disc swelling(right worse than left) and right optic nerve dysfunction

She also reported a year-long history of severe frontal–occipital headaches and cognitive fogginess. Her background included a recent diagnosis of achalasia. Further questioning and diagnostic testing revealed a possible cause for her findings.

Poster Presentations

From Eye Dysfunction to Neurological Disease: A Clinical Diagnosis of Miller Fisher Syndrome in a Child	Asvika Anis Anwar
Incidence of IIH in Victoria	Mina Botrous
Structural and functional biomarkers of visual dysfunction in MOG antibody-positive optic neuritis: a scoping review	Fiona Chan
A Phase 1A Single Ascending Dose Study of PYC-001; a peptide conjugated oligonucleotide designed to treat OPA1 mutation-associated Autosomal Dominant Optic Atrophy	Clare Fraser
A Devastating Cascade: Papilledema Secondary to Brain Tuberculoma, Hydrocephalus, and Tuberculous Meningoencephalitis	Dianita Ginting
The Reverse Relative Afferent Pupil Defect: A Prospective, Consecutive Study	Hannah Kamgarpour
When the Eyes Speak First: Role of AChR Antibody in Detection of Juvenile Ocular Myasthenia Gravis	Dzakiyyah Marsuqah Nasrullah
Progression from Ocular to Generalised Myasthenia Gravis: Insights from the MGBase Registry	Yong Lin Wang
Recurrent Arachnoid Cyst with Optic Pathway Compression – A rare cause of intermittent visual loss	Edward Yates

Poster Abstracts

From Eye Dysfunction to Neurological Disease: A Clinical Diagnosis of Miller Fisher Syndrome in a Child

Presenter:
Asvika Anis Anwar

Authors:
Asvika Anis Anwar[1], Yunita Mansyur[1,3], Marlyanti Nur Rahmah[1,3], Urfianty[2],

Institution:
1. Ophthalmology Department, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
2. Pediatric Neurology Department, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
3. Hasanuddin University Hospital, Makassar, Indonesia

Email:
Asvikaanis08@gmail.com

Introduction: Miller Fisher Syndrome (MFS) is a rare variant of Guillain-Barré Syndrome (GBS) characterized by a triad of ophthalmoplegia, ataxia, and areflexia. It is often preceded by an infection and can present with various neurological and ophthalmic symptoms.

Case Illustration: We report the case of a 6-year-old boy who developed bilateral ptosis and total ophthalmoplegia following an upper respiratory infection. Over the following days, he experienced progressive weakness in both legs, leading to significant balance disturbances. Neurological examination revealed ophthalmoplegia, ataxia, and areflexia, aligning with the classical presentation of MFS. Fundoscopy revealed bilateral papilledema, an uncommon finding in MFS. Electromyography (EMG) showed motor-predominant axonal polyneuropathy, supporting the diagnosis. The patient was treated with a five-day course of intravenous immunoglobulin (IVIG) therapy, which resulted in complete recovery of ocular motility and muscle strength within 6 months.

Discussion: The diagnosis was established based on the characteristic clinical triad and supported by EMG findings. The presence of bilateral papilledema was unusual but has been reported in rare MFS cases, suggesting possible CNS involvement or altered CSF dynamics. The patient's history of swimming in a school pool and subsequent respiratory symptoms aligns with the known pathogenesis of MFS, which often follows respiratory tract infections. Although anti-GQ1b antibody testing was unavailable, the clinical presentation and excellent response to immunotherapy strongly supported an autoimmune etiology consistent with MFS.

Conclusion: This case highlights the importance of early recognition of MFS in pediatric patients, especially when presenting with acute neurological and ocular symptoms. The presence of papilledema expands the clinical spectrum of MFS. Timely diagnosis and treatment with IVIG resulted in a favorable outcome, emphasizing the benefit of immunomodulatory therapy in accelerating recovery despite the generally self-limiting nature of the condition.

Keywords: Miller Fisher Syndrome, Guillain-Barré Syndrome Variant, Ophthalmoplegia, Papilledema

Poster Abstracts

Incidence of IIH in Victoria

Presenter:
Mina Botrous

Authors:
Dr Mina Botrous, A/Prof Anneke Van der Walt, A/Prof Clare Fraser, A/Prof David Darby, A/Prof Marc Sarossy

Institution:
Royal Victorian Eye and Ear Hospital / The Alfred

Email:
m.botrous@alfred.org.au

Background: Idiopathic intracranial hypertension (IIH) is strongly associated with obesity, and its incidence is expected to rise alongside increasing obesity rates. While this trend has been documented internationally, Australian data remain scarce. This study aimed to evaluate the incidence of IIH in Victoria between 2010 and 2023, hypothesising a significant increase over time.

Methods: A single-centre retrospective study was conducted, identifying all new IIH cases (514) diagnosed within the study period using standardised diagnostic criteria (Freidman criteria) Clinical records, neuro-imaging results, and lumbar puncture opening pressures were reviewed to confirm diagnoses. Annual incidence rates per 100,000 population were calculated using Australian Bureau of Statistics (ABS) population data for Victoria.

Results: The annual number of new IIH cases rose markedly from 3 cases (0.1096 per 100,000) in 2010 to 108 cases (3.2692 per 100,000) in 2023, reflecting a 30-fold increase in incidence over the 13-year period. This upward trend paralleled rising obesity rates in Australia.

Conclusion: The incidence of IIH in Victoria increased significantly between 2010 and 2023, mirroring global trends and underscoring the disease's association with obesity. These findings highlight the need for targeted public health interventions to address obesity-driven IIH and establish Australian-specific management guidelines. Further multi-centre studies are warranted to validate these results and explore regional variations.

Poster Abstracts

Structural and functional biomarkers of visual dysfunction in MOG antibody-positive optic neuritis: a scoping review

Presenter:
Fiona Chan

Authors:
Fiona Chan, Clare Fraser, Fabienne Brilot, Russell C Dale, Sudarshini Ramanathan

Institution:
Vision Research Foundation

Email:
Fiona.chan12@outlook.com

Introduction: Myelin-oligodendrocyte glycoprotein antibody-associated disease(MOGAD) commonly manifests as optic neuritis(ON). This review will evaluate the literature on MOG-Ab+ ON visual dysfunction, with a focus on ophthalmic structural biomarkers and ancillary outcome measures.

Methods: PubMed was systematically searched using the terms "optic neuritis", "visual outcomes" and "myelin-oligodendrocyte glycoprotein" between 2004 to January 2025. High-contrast visual acuity(HCVA), low-contrast visual acuity(LCVA), contrast sensitivity, visual fields(VF), electrophysiology, optical coherence tomography (OCT)/angiography, magnetic resonance imaging(MRI) and quality of life(QoL) were evaluated.

Results: 35 studies and 2235 patients were included. 89% of studies reported HCVA.OCT and MRI were reported in 63% and 46% of studies, respectively. MOG-Ab+ ON had moderate-severe HCVA loss at nadir but good HCVA recovery. MRI features characteristic to MOG-Ab+ ON include longitudinally extensive lesions +/- perineural enhancement. OCT in acute MOG-Ab+ ON revealed moderate-severe peripapillary retinal nerve fibre layer (p-RNFL) swelling. However severe axonal loss was noted in chronic outcomes, despite preservation of HCVA, highlighting a recognised structure-function discordance.31% reported on VF,17% on LCVA,and 14% on VEP, all of which demonstrated abnormalities even in patients with normal HCVA. Only one study reported on contrast sensitivity or QoL.

Conclusion: Our findings suggest current clinical assessments may underestimate visual dysfunction in MOG-Ab+ ON, and its impact on patient QoL. Prospective, longitudinal multimodal evaluation should be a key focus for future research, to identify the most sensitive biomarkers to be incorporated into routine clinical practice. This will better identify patients at risk of adverse visual outcomes in MOG-Ab+ ON, guide clinical management and improve QoL.

Poster Abstracts

A Phase 1A Single Ascending Dose Study of PYC-001; a peptide conjugated oligonucleotide designed to treat OPA1 mutation-associated Autosomal Dominant Optic Atrophy

Presenter:
Clare Fraser

Authors:
Clare Fraser, Doron Hickey, Aishwarya Kundu, George Mitchell, Sri Mudumba

Purpose: ADOA is a blinding eye disease caused by haploinsufficiency of the OPA1 gene in the retinal ganglion cells (RGCs). There are no therapeutic options for patients with OPA1-associated autosomal dominant optic atrophy (ADOA). PYC-001 is an intravitreally injected investigational drug designed to upregulate OPA1 protein expression, thereby addressing the underlying cause of ADOA. A Phase 1 Single Ascending Dose (SAD) first-in-human study was initiated to evaluate the safety and tolerability of PYC-001.

Method: An open-label, SAD study recruited 9 participants with genetically confirmed OPA1 mutation-associated ADOA in 3 dose cohorts (3, 10 and 30 µg). Subjects each received a unilateral intravitreal injection of PYC-001 in the worse affected eye and are followed for incidence, severity and relatedness of adverse events. Dose escalation was based on review by a safety review committee (SRC) using 4-week and incremental data collected after each cohort dosing. Safety and tolerability are evaluated based on adverse event (ocular and non-ocular) reporting, including clinical chemistry parameters, measures of visual function, functional vision and imaging. Exploratory efficacy was assessed using visual acuity.

Results: No drug related adverse events, and no intraocular inflammation were observed at any visit following injection of PYC-001. Encouraging early improvements in visual acuity were observed in eyes treated with PYC-001 from week 4 onwards.

Conclusion: Single intravitreal injections of PYC-001 were safe and well tolerated at all doses assessed to date. The safety and efficacy profile following single injection of PYC-001 supports progression into repeat dose clinical studies in 2025.

Poster Abstracts

A Devastating Cascade: Papilledema Secondary to Brain Tuberculoma, Hydrocephalus, and Tuberculous Meningoencephalitis

Presenter:
Dianita Veulina Ginting

Authors:
Dianita Veulina Ginting, Antonia Kartika Indriati, Rusti Hanindya Sari, Prettyla Yollamanda

Institution:
Neuro-Ophthalmology Division, National Eye Center Cicendo Eye Hospital, Bandung Indonesia

Email:
ditaveulina@gmail.com

Introduction: Tuberculous meningoencephalitis (TBM) represents one of the most severe manifestation of central nervous system (CNS) tuberculosis, frequently leading to increased intracranial pressure (ICP) due to complications such as hydrocephalus and intracranial tuberculomas. Papilledema is a neuro-ophthalmological sign of elevated ICP, and its recognition is paramount to prevent irreversible visual loss.

Purpose: This abstract presents a complex clinical case to illustrate the severe and cascading neurological and ophthalmological manifestations of central nervous system (CNS) tuberculosis. The primary objective is to highlight papilledema as a critical and potentially devastating sign of elevated intracranial pressure (ICP) stemming from complications such as brain tuberculomas and hydrocephalus, and to emphasize the urgent need for a timely, multidisciplinary diagnostic and therapeutic approach to prevent irreversible visual and neurological deficits.

Method: Case Report

Result: A patient presented with a constellation of acute symptoms, including intractable headache, persistent nausea, and progressive bilateral vision loss. A funduscopic examination revealed papilledema with bilateral optic disc edema. Brain MRI showed extensive pathology, including multiple ring-enhancing lesions consistent with tuberculomas and abscesses, diffuse meningeal enhancement indicating meningoencephalitis, and hydrocephalus. Lumbar puncture confirmed the diagnosis of tuberculous meningitis. The patient was treated with a multi-drug anti-tuberculous regimen, high-dose corticosteroids, and underwent ventriculoperitoneal shunt placement to relieve the ICP. The papilledema resolved post-treatment, but permanent visual function deficits persisted.

Conclusion: This case serves as a powerful reminder that papilledema can be a crucial initial indicator of severe, life-threatening CNS infections like tuberculosis. The rapid progression from tuberculoma formation and hydrocephalus to vision-threatening papilledema underscores the critical need for a high index of suspicion and prompt investigation. This case highlights that a swift and collaborative management strategy involving ophthalmology, neurology, and neurosurgery is paramount to reduce ICP, mitigate neurological damage, and preserve visual function in such complex and devastating clinical scenarios.

Poster Abstracts

The Reverse Relative Afferent Pupil Defect: A Prospective, Consecutive Study

Presenter:
Hannah Kamgarpour

Authors:
Hannah Kamgarpour, Lloyd Kopecny, Amy Tsoi, Aadhavi Vasanthan, Grace Borchert, Ashish Agar, Ian Francis

Institution:
University of Notre Dame Sydney, Australia

Email:
Hannahkamgar08@gmail.com

Purpose: This study was designed to identify and determine the frequency and functionality of the Reverse Relative Afferent Pupil Defect (RAPD) in a series of intravitreal injections (IVI) given for retinal vascular disorders. As background, all Neuro-ophthalmologists will have seen hundreds if not thousands of RAPDs, and therefore Reverse RAPDs, since every RAPD is associated with a Reverse RAPD. In orbital apex lesions with third nerve involvement and a paretic pupil with associated optic nerve involvement, an RAPD may be difficult to identify. In this situation, identification of a Reverse RAPD will demonstrate afferent visual pathway dysfunction ipsilateral to the lesion.

Methods: This was a prospective, unmasked, single-surgeon, single-centre, no-exclusions study. Each patient undergoing an IVI of a VEG-F inhibitor had their intraocular pressure (IOP) and pupillary responses evaluated immediately prior to the IVI. Immediately following the IVI, the presence of a Reverse RAPD was sought, along with the post-IVI IOP.

Results: In this study, 411 eyes undergoing IVIs were evaluated. The eye to be injected had its pupil dilated for pre-injection fundoscopy and OCT assessment. Afibercept (Eylea) was injected in 97% of cases and Faricimab (Vabysmo) in 3%. Immediately following the IVI, a Reverse RAPD was present in 97.6% of all cases. While median pre-IVI IOPs were 10mmHg (5-22mmHg), IOPs elevated in all patients following IVI, with a median IOP of 50mmHg (31-82mmHg). When asked, all patients reported immediate significant visual reduction.

Conclusion: The marked IOP elevation and presence of a Reverse RAPD immediately following IVIs indicate substantial compromise of the ocular circulation. Future studies could further evaluate the optic nerve appearance, duration of the elevated IOP and the visual loss post-IVI. An explanation is deserved as to why this almost complete shutdown of ocular circulation does not appear to compromise future visual function.

Poster Abstracts

When the Eyes Speak First: Role of AChR Antibody in Detection of Juvenile Ocular Myasthenia Gravis

Presenter:
Dzakiyyah Marsuqah Nasrullah

Authors:
Dzakiyyah Marsuqah Nasrullah[1], Yunita Mansyur[1,3], Marlyanti Nur Rahmah[1,3], Urfianty[2],

Institution:
1. Ophthalmology Department, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
2. Pediatric Neurology Department, Faculty of Medicine, Hasanuddin University, Makassar, Indonesia
3. Hasanuddin University Hospital, Makassar, Indonesia

Email:
marsuqahnasrullah@gmail.com

Introduction: Juvenile ocular myasthenia gravis (JOMG) is a rare autoimmune neuromuscular disorder in children characterized by weakness of the extraocular muscles, often presenting solely with ptosis and ophthalmoplegia. Unlike adult-onset MG, JOMG frequently lacks systemic involvement and may present diagnostic challenges due to its variable clinical course and the limited sensitivity of bedside tests such as the ice pack test. Early identification remains critical, as delayed diagnosis may result in visual developmental complications such as amblyopia. This case underscores the importance of a comprehensive diagnostic approach, especially when conventional screening tests yield inconclusive results.

Objective: To report a pediatric case of Ocular Myasthenia Gravis (OMG) with negative ice pack test, confirmed through serological testing, and successfully managed with pyridostigmine.

Case Illustration: An 8-year-old girl presented with a 3-month history of bilateral ptosis and restricted eye movements following a febrile illness. Despite no other systemic symptoms, her eye movement was limited in all directions. The ice pack test was negative, and brain MRI showed no abnormalities. Serologic testing revealed positive acetylcholine receptor (AChR) antibodies. The diagnosis of ocular myasthenia gravis was established. The patient was started on pyridostigmine leading to significant clinical improvement in both ptosis and ocular motility within one month.

Conclusion: This case illustrates the challenges in diagnosing Ocular Myasthenia Gravis (OMG) in children, particularly when symptoms such as ptosis and limited eye movements are present. While the ice pack test can be a helpful first step, its reliability in pediatric cases is limited, and a negative result should not rule out the diagnosis. A thorough, individualized diagnostic process grounded in careful clinical evaluation and supported by additional tests is key to identifying Juvenile Ocular Myasthenia Gravis (JOMG) early and accurately. In this patient, the presence of AChR antibodies was pivotal in confirming the autoimmune disease, allowing for timely and appropriate treatment.

Keywords: Opthalmoplegia, Ptosis, Myasthenia Gravis, Juvenile Ocular Myasthenia Gravis, AChR antibody, Pyridostigmine.

Poster Abstracts

Progression from Ocular to Generalised Myasthenia Gravis: Insights from the MGBase Registry

Presenter:
Yong Lin Wang

Authors:
Yong Lin Wang, Jamie Formosa, Michael Hayes, Paul Sanfilippo, Elisabeth Chroni, Jeannine Heckmann, Katherine Buzzard, Stefan Blum, Matteo Foschi, Pamela McCombe, Gary Cutter, Carolina Barnett-Tapia, Helmut Butzkueven, WenWen Zhang, Mahima Kapoor, Mastura Monif, Stephen Reddel, Anneke van der Walt on behalf of the MGBase Investigators

Institution:
Alfred Health, Monash University

Email:
Yong.wang1@monash.edu

Aim: Ocular myasthenia gravis (OMG) is frequently perceived as a localized and potentially less severe form of myasthenia gravis (MG), but a substantial proportion of patients progress to generalized MG (gMG). Identifying predictors of such progression remains an unmet need. This study aims to characterize the clinical trajectory of patients with ocular-only MG and identify factors associated with progression to generalized disease. We examined whether current prednisolone dose is associated with the hazard of generalisation, leveraging real-world data from MGBase, an international multi-centre MG registry.

Results: We identified 140 patients with OMG with a median follow-up of 38.5 months. Mean age at onset was 52.6 years; 51 patients (36%) were female. 54 patients (38.6%) progressed to gMG. The analytic dataset for the time-to-event analysis contained 1006 risk intervals with 53 events. Relative to being off prednisolone, low dose prednisolone did not increase the hazard of generalisation (HR = 0.91, 95% CI 0.42-1.94, p = 0.80). High-dose prednisolone increased the HR (HR = 2.01, 95% CI 0.97-4.14) without reaching significance (p = 0.06). Higher age-at-onset (per-year HR = 1.03, 95% CI 1.02-1.05, p < 0.001), female sex (HR = 1.86, 95% CI 1.04-3.31, p=0.029) and presence of thymoma (HR = 3.89, 95% CI 1.38–10.96, p = 0.01) were associated with greater generalisation risk. Antibody status did not increase generalisation risk.

Conclusion: In this time-to-event analysis, the current prednisolone dose was not associated with a statistically significant reduction in the hazard of generalisation. Findings for age, sex and thymoma align with known risk factors, and the rate of generalisation is consistent with cohorts described elsewhere.

Poster Abstracts

Recurrent Arachnoid Cyst with Optic Pathway Compression – A rare cause of intermittent visual loss

Presenter:
Dr. Edward Yates

Authors:
Dr. Finn Ghent, Dr. Megha Kaushik, Dr. William Yates

Institution:
Royal Prince Alfred Hospital, John Hunter Hospital Neurosurgical department, Royal Prince Alfred Hospital department of Ophthalmology, Liverpool Hospital department of Ophthalmology, John Hunter Hospital Department of Ophthalmology

Email:
edzyates@gmail.com

Arachnoid cysts are cerebrospinal fluid-filled structures typically found incidentally, with symptomatic presentations being rare in adulthood. We present a unique case of a 71-year-old male with recurrently enlarging arachnoid cyst-induced left-sided hemianopia, which resolved spontaneously without surgical intervention.

The patient presented with a 4-month history of left-sided visual disturbances and headache, raising concerns for a mass lesion or stroke. Notably, he had a similar presentation two decades prior, diagnosed via MRI with a right middle cranial fossa arachnoid cyst with suprasellar extension of 12 x 11 mm, which resolved with reduction of lifestyle stress. His past medical history included a partial lobectomy for a benign lesion and hypertension managed with irbesartan. Clinical evaluation revealed a right superior visual defect and left hemianopia, with MRI demonstrating an interval increase in cyst size (suprasellar component: 17 x 17 mm, sellar component: 26 x 18 mm), causing chiasmal compression. The mechanism what causes arachnoid cysts to grow is unclear; however, the most likely answer is either a reflection of arachnoid mater or other tissue causing a one-way valve disrupting drainage of cerebrospinal fluid. The patient was offered neurosurgical intervention, with a sub-frontal approach craniotomy with endoscopic fenestration, but he opted for conservative management. Three weeks later, both his headache and hemianopia recovered, confirmed by follow-up visual field testing. The resolution of symptoms with no intervention is not well understood and will need follow-up imaging to further characterise the mechanism. This case underscores the unpredictable nature of arachnoid cysts, challenging the prevailing surgical-first approach in symptomatic patients. The absence of reliable predictive tools for spontaneous resolution necessitates further research into long-term conservative management strategies.

This case highlights the importance of interdisciplinary collaboration in guiding management of symptomatic arachnoid cysts, particularly in ophthalmology and neurosurgery. It also underlines the need for further studies to determine optimal management strategies in cases of cyst-induced visual impairment with spontaneous resolution.

NeuroVision Training Day
Saturday 13th September

Session 1: Introduction		
09.00-09.10	Introduction and welcome	Celia Chen, Elle Nguyen, Anneke Van der Walt
09.10 – 09.30	Visual fields and OCT in neuro-ophthalmology	Megha Kaushik
09.30 – 09.50	MRI: visual pathway	Andrew Smith
09.50 – 10.30	Optic neuritis – MS, NMO, MOG	Ari Green
10.30 – 11.00	**Morning Tea**	
Session 2: Swollen discs		
11.00 – 12.00	Idiopathic Intracranial Hypertension and raised intracranial pressure	Anneke van de Walt Jenny Hepschke
12.00 – 13.00	**Lunch**	

NeuroVision Training Day
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Session 3: Optic neuropathies		
13.00 – 13.20	Toxic / nutritional optic neuropathies	Sumu Simon
13.20- 13.40	Giant Cell arteritis	Mitchell Lawlor
13.40-14.00	Compressive optic neuropathy	Anthony Fong
14.00-14.20	Anisocoria	John Leaney
14.20-14.40	Pediatric cases	James Smith
14.40 – 15.10	**Afternoon Tea**	
Session 4: Miscellaneous		
15.10 – 15.30	Higher visual processing issues	Sui Wong
15.30 – 15.50	Functional visual loss	Neil Shuey
15.50-16.10	Inherited optic neuropathies	Helen Danesh Meyer
16.10-16.30	Non-arteritic anterior ischaemic optic neuropathy	Elle Nguyen
16.30 – 16.50	Transient visual loss and retinal arteriole occlusion	Celia Chen
16.50	**Close and thanks**	

NeuroVision Training Day
Faculty

Prof. Celia Chen	Consultant Neuro-Ophthalmologist	Clinical Professor of Ophthalmology University of South Australia
Dr Anthony Fong	Consultant Neuro-Ophthalmologist	Department of Ophthalmology Royal Brisbane and Women's Hospital Princess Alexandra Hospital Gold Coast University Hospital
Dr Jenny Hepschke	Consultant Ophthalmologist	Southern Ophthalmology, Kogarah NSW
Dr Megha Kaushik	Consultant Neuro-Ophthalmologist	Royal Prince Alfred Hospital Liverpool Hospital Sydney, Australia
Dr Mitchell Lawlor	Consultant Neuro-Ophthalmologist	Sydney Eye Hospital Royal Prince Alfred Hospital Sydney, Australia
Dr John Leaney	Consultant Neuro-Ophthalmologist	Liverpool Hospital Royal Prince Alfred Hospital Sydney, Australia
Dr Elle Nguyen	Consultant Neurologist and Neuro-Ophthalmologist	Alfred Health and Royal Victorian Eye and Ear Hospital Melbourne, Australia
Dr. Neil Shuey	Consultant Neuro-Ophthalmologist	St Vincent's Hospital and Royal Victorian Eye and Ear Hospital, Melbourne, Australia
Dr Sumu Simon	Consultant Neuro-Ophthalmologist	Neuro-ophthalmology Unit Department of Ophthalmology Royal Adelaide Hospital
Dr James Smith	Consultant Paediatric Ophthalmologist and Head of Department RNSH	Westmead Childrens Hospital Royal North Shore Hospital Sydney, Australia
Dr Anneke Van de Walt	Consultant Neurologist	Neuro-ophthalmologist Monash University