Your Programme

9:00-9:10	Welcome	Professor Dame Helen Danesh-Meyer
9:10-9:40	Keynote Address: Uncertainty, judgement & decision making in everyday practice.	Professor Keith Martin
SESSION 1	**THE DIAGNOSTIC GREY ZONE**	
9:40 - 9:50	Suspicious Discs: Who to watch and who to worry about	Dr Keliopy Matheos
9:50- 10:00	Structure-Function Mismatch: When OCT and Fields Disagree	Dr Graham Reeves
10:00-10:10	Normal tension glaucoma (what to do when there is progression at 10mmHg)	Dr Jesse Gale
10:10-10:20	High Pressure, Low Risk? Low Pressure, High Risk?	Dr Alex Buller
10:20-10:35	Panel Discussion	
10:35-11:05	**MORNING TEA**	
SESSION 2	**TESTING THE GREY ZONE**	
11:05-11:15	Sleep, Posture, and the Optic Nerve: Do Nocturnal Risk Factors Really Matter?	Dr Mark Donaldson
11:15-11:25	Corneal and Glaucoma: CCT and other relevant findings	Dr Jay Meyer
11:25-11:35	Emerging Risk Factors in Glaucoma: Signals, Speculation, or Something Real?	Dr Aaron Wong
11:35-11:45	Navigating the Glaucoma Maze - Controversies in Angle Closure Glaucoma	Dr Baswati Sahoo
11:45-11:55	Would Any of These Risk Factors Change What You Do?	Dr Michael Merriman
11:55-12:10	Panel Discussion	
SESSION 3	**MANAGEMENT IN THE GREY ZONE**	
12:10-12:20	When is Observation No Longer Safe?	Dr Antony Suter
12:20-12:30	Drops, SLT, or Watchful Waiting?	Dr Sonya Bennet
12:30-12:40	The Non-Adherent Patient: Risk or Reality?	Dr Hussain Patel
12:40:12:50	When to Escalate:Identifying the Surgical Window	Dr Divya Perumal
12:35-12:50	Panel Discussion	
12:50-13:40	**LUNCH**	
Session 4	**CASE BATTLES: THE GREY ZONE IN ACTION** **Five Patients, Five Decisions**	
13:40-13:55	Case	Dr Hannah Kersten
13:55-14:10	Case	Jason Xu
14:10-14:25	Case	Claire McDonald
14:25-14:40	Case	Adele Jefferies
14:40-14:55	Case	Inhae Park
14:55-15:30	**AFTERNOON TEA**	
SESSION 5	**RAPID FIRE CASES**	Professor Dame Helen Danesh-Meyer
15:30-16:30	Panel and Audience Discussion	Robin Yang
16:30-16:45	Gordon Sanderson Presentation/NZ Glaucoma Research	

Keynote Address: The Future of Glaucoma — Innovation, Uncertainty, and the Next Clinical Frontier – Professor Keith Martin

Glaucoma care is entering a period of rapid technological and conceptual change, driven by advances in imaging, artificial intelligence–based analytics, home monitoring, minimally invasive surgical techniques, and novel therapeutic targets. These developments promise earlier detection, more individualised risk stratification, and refined longitudinal surveillance. Yet they also introduce new forms of uncertainty, as clinicians confront expanding data streams, evolving algorithms, and clinical scenarios not yet fully addressed by guidelines or trial evidence.

This keynote examines how emerging tools may reshape diagnosis and management across the disease spectrum, from pre-perimetric glaucoma to advanced disease. Topics include AI-assisted optic nerve assessment, continuous or home intraocular pressure monitoring, remote perimetry, and structural progression analysis at unprecedented resolution. The session explores how these innovations may recalibrate traditional concepts such as target pressure, progression thresholds, and treatment timing, while highlighting the importance of rigorous validation and thoughtful integration into real-world practice.

The address concludes by considering the professional and ethical responsibilities that accompany technological progress. Issues of algorithmic transparency, medico-legal accountability, health-equity implications, and clinician over-reliance on automated systems are critically examined. Rather than presenting innovation as a solution to uncertainty, the keynote argues that the next era of glaucoma care will demand even sharper clinical judgement, combining new tools with disciplined scepticism, patient-centred decision-making, and an enduring willingness to navigate the grey zones with clarity and humility.

Suspicious Discs: Who to Watch, Who to Worry About - Dr Keli Matheos

This session addresses optic disc appearances that challenge traditional teaching and provoke diagnostic uncertainty. Common anatomical variants such as tilted discs, large physiological cups, and myopic changes are contrasted with early glaucomatous features, including neuroretinal rim thinning, focal notching, peripapillary atrophy, disc haemorrhages, and asymmetry. The influence of axial length, ethnicity, and imaging artefact on disc interpretation is also explored.

Emphasis is placed on longitudinal assessment as the cornerstone of management in equivocal cases. Serial photography, OCT metrics, and functional testing are integrated to refine risk stratification, with guidance provided on surveillance intervals, thresholds for escalation, and patient counselling when diagnostic certainty remains elusive.

Structure–Function Mismatch: When OCT and Fields Disagree – Dr Graham Reeve

Discordance between OCT findings and visual field results is common in routine practice and frequently generates clinical uncertainty. Biological explanations such as pre-perimetric disease and advanced-stage floor effects are considered alongside technical factors including segmentation error, scan misalignment, and media opacity. The relationship between disease

stage and test sensitivity is reviewed to clarify which modality may be most informative in specific clinical contexts.

A pragmatic approach to reconciling conflicting data is presented using case-based examples. Strategies for verifying results, repeating testing selectively, and integrating examination findings with imaging and functional measures are emphasised to prevent premature escalation or inappropriate reassurance.

Normal Tension Glaucoma – what to do when there is progression at 10mmHg? – Dr Jesse Gale

Normal-tension glaucoma (NTG) presents a persistent therapeutic dilemma when structural and functional progression occurs despite intraocular pressures (IOP) around 10 mmHg. Such cases challenge the traditional pressure-centric paradigm and require meticulous reassessment of diagnosis, risk factors, and rate of change before escalating therapy. This abstract reviews contemporary strategies for evaluating progression at so-called “floor” pressures, emphasizing confirmation of true progression with high-quality optical coherence tomography and visual-field trend analysis, exclusion of masqueraders and secondary optic neuropathies, and identification of contributory systemic factors such as nocturnal hypotension, sleep apnoea, migraine, vasospasm, and impaired ocular perfusion pressure. Attention is also given to corneal biomechanics, diurnal IOP fluctuation, medication adherence, and the limitations of single-digit tonometry in capturing true translaminar pressure stress.

When progression is confirmed, management must extend beyond incremental pharmacologic escalation. Options include achieving further IOP reduction toward episcleral venous pressure with maximally tolerated topical therapy, selective laser trabeculoplasty as adjunctive treatment even at low baseline pressures, and consideration of filtration surgery or minimally invasive subconjunctival procedures in carefully selected patients, balanced against the heightened risk of hypotony-related complications. Non-IOP-directed strategies—optimization of systemic blood-pressure regimens, treatment of sleep apnoea, avoidance of nocturnal antihypertensive over-treatment, and exploration of neuroprotective approaches—are increasingly recognized as integral to care. A structured, individualized algorithm combining rigorous confirmation of progression, aggressive yet judicious pressure lowering, and systemic risk-factor modification offers the best prospect of stabilizing vision in this challenging NTG subgroup.

High Pressure, Low Risk? Low Pressure, High Risk? – Dr Alex Buller

Intraocular pressure remains central to glaucoma management yet is an imperfect surrogate for disease activity and future visual loss. Factors contributing to differential susceptibility, including vascular dysregulation, optic nerve head anatomy, corneal biomechanics, and diurnal fluctuation—are examined to explain why progression may occur despite apparently satisfactory pressures.

Approaches to contextualising IOP within an overall risk profile are discussed, including target-pressure setting, longitudinal reassessment, and interpretation of clinic measurements alongside home or diurnal data. Clinical scenarios illustrate when aggressive lowering is justified and when observation may remain appropriate.

Session Two – Testing in the Grey Zone

Sleep, Posture, and the Optic Nerve: Do Nocturnal Risk Factors Really Matter? – Dr Mark Donaldson

Growing interest has focused on the possibility that glaucoma progression may be driven not only by daytime intraocular pressure but by nocturnal physiological stresses, including supine posture, head position, obstructive sleep apnoea, nocturnal hypotension, and impaired autoregulation of optic nerve head perfusion. Experimental and clinical studies suggest that IOP may rise in the lateral or prone position, while systemic blood pressure frequently falls overnight, potentially reducing ocular perfusion pressure at precisely the time when optic nerve vulnerability may be greatest. Emerging imaging and telemetry technologies now allow interrogation of 24-hour IOP profiles and sleep-related physiological changes, challenging traditional clinic-based paradigms of risk assessment.

This session critically examines the quality of evidence linking nocturnal factors to structural and functional progression, distinguishing compelling biological plausibility from reproducible clinical signal. Practical implications will be explored, including whether sleep apnoea screening is warranted in selected patients, whether head elevation or positional modification is advisable, and how antihypertensive regimens might influence nocturnal optic nerve perfusion. Attendees will leave with a nuanced understanding of which nocturnal factors deserve attention in contemporary practice, and which remain speculative.

Corneal and Glaucoma CCT and other relevant findings – Dr Jay Meyer

Corneal characteristics play a pivotal but often misunderstood role in glaucoma assessment and risk stratification. Central corneal thickness (CCT) influences applanation tonometry and has been associated with glaucoma susceptibility, yet simplistic numerical “corrections” to intraocular pressure risk misleading clinicians and obscuring true optic nerve vulnerability. This session reviews how CCT interacts with optic nerve head biomechanics, why thin corneas may confer increased risk independent of pressure measurement error, and how corneal parameters should be interpreted alongside disc appearance, imaging, and functional testing rather than in isolation.

Beyond thickness alone, emerging evidence regarding corneal biomechanics—such as hysteresis and viscoelastic behaviour—will be explored in relation to glaucoma progression and treatment response. The discussion extends to the impact of refractive surgery, keratopathy, and measurement variability on risk assessment and monitoring. By integrating corneal metrics into holistic clinical reasoning rather than relying on single surrogate values, this session aims to sharpen judgement in borderline cases and prevent false reassurance or unnecessary escalation in everyday practice.

Emerging Risk Factors in Glaucoma: Signals, Speculation, or Something Real? – Dr Aaron Wong

Beyond established determinants such as intraocular pressure, age, and central corneal thickness, a growing literature has proposed additional contributors to glaucoma susceptibility and progression, including vascular dysregulation, systemic hypotension, migraine, inflammation, cerebrospinal fluid pressure gradients, metabolic factors, and genetic susceptibility profiles. Advances in imaging, genomics, and large-scale biobank analyses have generated a proliferation of associations—some provocative, others contradictory-raising questions about how clinicians should interpret these findings at the bedside.

This lecture dissects emerging risk factors through a rigorous evidentiary lens, evaluating biological plausibility, study design, reproducibility, and effect size. Particular emphasis will be placed on separating causal pathways from epiphenomena, and on recognising where enthusiasm has outpaced proof. The goal is to equip clinicians with a framework for integrating new risk signals into clinical reasoning without over-medicalising uncertainty or prematurely altering management.

Navigating the Glaucoma Maze: Controversies in Angle Closure Glaucoma – Dr Baswati Sahoo

Primary angle closure disease encompasses a spectrum ranging from anatomical predisposition to acute crisis and established glaucomatous optic neuropathy, yet major controversies persist regarding classification, screening, and optimal intervention. Questions remain about the relative contributions of pupillary block versus plateau iris and lens vault, the role of gonioscopy versus imaging-based assessment, and how aggressively to intervene in asymptomatic individuals identified through opportunistic or population screening.

This session explores contemporary debates surrounding prophylactic laser iridotomy, early lens extraction, and long-term outcomes across different phenotypes of angle closure. Landmark trials and evolving guidelines will be critically reviewed, alongside unresolved questions regarding risk stratification and cost-effectiveness. Through case-based discussion, attendees will develop practical strategies for navigating anatomical complexity while avoiding both undertreatment and unnecessary intervention.

Would Any of These Risk Factors Change What You Do? – Dr Michael Merriman

The expanding catalogue of putative glaucoma risk modifiers poses a fundamental question for clinicians: when does epidemiological association translate into actionable management change? Vascular dysregulation, nocturnal hypotension, sleep apnoea, systemic medications, lifestyle factors, and genetic profiling all attract attention in the literature, yet the thresholds at which these considerations should alter surveillance intensity, therapeutic targets, or escalation strategies remain ill-defined.

This interactive session synthesises current evidence to determine which risk factors meaningfully influence prognosis and which should prompt modification of clinical pathways today. Through structured clinical scenarios, participants will consider whether to lower pressure targets, intensify monitoring, adjust systemic therapies, or pursue earlier intervention.

The emphasis will be on rational, evidence-anchored decision-making rather than reflexive response to emerging associations.

Session 3 – Management in the Grey Zone

When Is Observation No Longer Safe? – Dr Antony Suter

Deciding when to initiate or escalate treatment is among the most consequential judgements in glaucoma management. Clinical features prompting intervention—including reproducible progression, optic disc haemorrhage, high-risk phenotypes, and patient-specific visual demands—are examined.

Balancing the risks of overtreatment against the irreversible consequences of delayed action is explored through clinical reasoning frameworks. Documentation standards, patient communication, and reassessment thresholds are highlighted as essential components of defensible decision-making.

Drops, SLT, or Watchful Waiting? – Dr Sonya Bennett

Initial management of ocular hypertension and early glaucoma is increasingly nuanced, with expanding data supporting topical therapy, selective laser trabeculoplasty as first-line treatment, and carefully monitored observation in low-risk individuals. Randomised trials have challenged traditional stepwise paradigms, while real-world considerations such as adherence, access to care, adverse effects, and patient preference exert growing influence on treatment choice.

This lecture reviews the comparative efficacy, durability, safety, and cost-effectiveness of these strategies across different patient profiles. Attention will be given to identifying candidates most likely to benefit from early intervention, those in whom surveillance is reasonable, and how baseline risk modifies treatment thresholds. Attendees will gain a pragmatic framework for tailoring first-line therapy to biology, behaviour, and health-system realities.

The Non-Adherent Patient: Risk or Reality? – Dr Hussain Patel

Poor adherence to topical glaucoma therapy is widely cited as a major cause of disease progression, yet accurately measuring real-world medication use remains challenging. Electronic monitoring, pharmacy refill data, and patient-reported outcomes consistently demonstrate that persistence with drops declines over time, influenced by regimen complexity, side effects, dexterity, health literacy, and psychosocial factors. However, the magnitude of risk attributable specifically to non-adherence—and how best to mitigate it—remains debated.

This session examines the evidence linking adherence patterns to visual field and structural outcomes, and evaluates interventions designed to improve persistence, from regimen simplification and sustained-release technologies to behavioural strategies and digital monitoring. The discussion will move beyond blame toward system-level solutions, helping clinicians identify at-risk patients early and deploy realistic, effective countermeasures.

When to Escalate: Identifying the Surgical Window – Dr Divya Perumal

Determining the optimal timing for surgical intervention remains one of the most consequential decisions in glaucoma care. Escalating too late risks irreversible vision loss, while premature surgery exposes patients to unnecessary complication and long-term management burdens. Advances in imaging, progression analysis, and minimally invasive surgical techniques have reshaped thresholds for intervention, yet consensus on defining the ideal "surgical window" remains elusive.

This lecture integrates structural and functional progression metrics, rate-of-change analyses, and risk-prediction models to guide timely escalation. Comparative outcomes of trabeculectomy, tube shunts, and newer procedures will be reviewed in the context of disease stage, life expectancy, and target pressure requirements. Attendees will leave with a principled, patient-centred approach to recognising when maximal medical therapy is no longer enough—and when decisive surgical action offers the best chance of preserving vision.

Session Four Case Battles: The Grey Zone in Action

Five Patients, Five Decisions – Dr Hannah Kersten, Jason Xu, Claire McDonald, Adele Jefferies and Inhae Park

This interactive session presents complex real-world cases that resist straightforward categorisation or management. Competing pathways of observation, escalation, and investigation are explored, highlighting the uncertainties inherent in clinical practice.

Facilitated discussion focuses on how expert clinicians articulate reasoning, weigh competing risks, and justify divergent approaches. The session reinforces the legitimacy of plural defensible strategies when guided by rigorous clinical judgement.

Session Five – Rapid Fire Cases

The Future of Glaucoma: New Tools, New Uncertainties – Professor Dame Helen Danesh-Meyer

Emerging technologies including artificial intelligence, home monitoring, and novel imaging platforms are reshaping glaucoma detection and surveillance. Potential benefits for earlier diagnosis, risk prediction, and longitudinal assessment are considered.

Challenges introduced by these innovations—such as data overload, false positives, algorithmic opacity, and medico-legal uncertainty—are critically examined. Maintaining clinician oversight and interpretive responsibility is emphasised as these tools enter routine practice.

Case Abstracts:

Pseudoexfoliation Progressing to Glaucoma
Pseudoexfoliation syndrome presents a common yet unpredictable pathway to glaucomatous damage, characterised by fluctuating intraocular pressure, asymmetric involvement, and rapid

structural change in some patients. This case explores the challenges of identifying early conversion from ocular surface or anterior segment findings to true optic neuropathy, including interpretation of pressure variability, optic nerve appearance, and evolving OCT and visual field metrics.
Discussion focuses on recognising high-risk features for progression, determining appropriate surveillance intervals, and selecting the optimal timing and modality of intervention. Participants will consider the implications for medical therapy, laser trabeculoplasty, and surgical escalation in a phenotype often associated with treatment resistance and accelerated disease course.

Non-Glaucomatous Optic Neuropathy Mimicking Glaucoma
Not all cupped or pale optic nerves represent glaucoma, and failure to recognise alternative optic neuropathies may lead to delayed diagnosis of serious neurological or inflammatory disease. This case presents a patient with apparent glaucomatous features but atypical progression, prompting reconsideration of the underlying diagnosis.
Attention is given to red flags such as disproportionate visual acuity loss, colour vision deficits, field patterns respecting the vertical meridian, and neuro-imaging clues. The case challenges participants to identify when to step outside the glaucoma algorithm and pursue alternative investigations.

Ocular Hypertension: When Risk Becomes Disease
Ocular hypertension occupies a quintessential grey zone, with many patients remaining stable for decades while others progress to irreversible optic nerve damage. This case explores risk stratification using pressure magnitude, corneal properties, disc appearance, family history, and demographic modifiers.
Discussion centres on balancing surveillance against early intervention, setting target pressures, and communicating probabilistic risk to patients. Participants will weigh the consequences of treatment burden against the hazards of delayed therapy in an asymptomatic population.

Progressive Glaucoma Despite Apparent Control
Some patients demonstrate continued structural or functional loss despite apparently adequate intraocular pressure lowering, forcing clinicians to re-examine assumptions about adherence, measurement accuracy, and disease biology. This case examines potential explanations including pressure fluctuation, nocturnal spikes, vascular factors, and treatment failure.
Participants will debate escalation strategies ranging from intensification of medical therapy to laser or surgical intervention. The case highlights the importance of reassessing targets, verifying progression, and revisiting systemic risk factors when glaucoma refuses to behave predictably.

Normal Tension Glaucoma: Low Pressure, High Stakes
Normal tension glaucoma challenges traditional pressure-centric models of disease, raising questions about vascular dysregulation, perfusion pressure, and individual susceptibility of the optic nerve. This case explores diagnostic confirmation in the setting of statistically normal pressures and subtle early damage.
Discussion focuses on defining progression, setting appropriate target pressures, and managing systemic contributors such as hypotension or sleep apnoea. Participants will consider when aggressive pressure lowering is justified and how to monitor response in a phenotype prone to slow but relentless decline.

Pigment Dispersion Glaucoma: Variable Course, Variable Risk
Pigment dispersion syndrome and its glaucomatous sequelae display a highly variable natural history, with intermittent pressure spikes and periods of stability complicating management decisions. This case examines clinical findings such as Krukenberg spindle, iris transillumination defects, and trabecular pigmentation, alongside evolving optic nerve and visual field changes. The discussion addresses risk of progression, indications for laser or medical therapy, and the uncertain role of prophylactic interventions. Participants will debate how aggressively to treat fluctuating disease and how to individualise follow-up in younger, often asymptomatic patients.

2025 Gordon Sanderson Scholar – Robin Yang

Most glaucoma eyedrops are comprised of two components: the active ingredient and a preservative. Benzalkonium chloride (BAK), a quaternary ammonium molecule, is the most commonly used ophthalmic preservative. There is a recognised harm from the preservative in the eye drops. The most common being ocular surface irritation. However, there is also preliminary evidence that the preservatives in the eye drops may cause inflammation and damage to the trabecular meshwork. As a result, preservative free eye drops have been developed and are readily available in most developed countries. New Zealand is one of the only countries in which there is no access to preservative free eye drops because it is not accepted by PHARMAC that patients receive a significant benefit.

Closing Address: Practising Comfortably in Uncertainty – Professor Dame Helen Danesh-Meyer

Uncertainty is intrinsic to glaucoma care rather than a failure of expertise. Reflection on cognitive bias, evolving evidence, and patient-centred reasoning reinforces the role of humility and adaptability in clinical decision-making.

The closing address synthesises the day's themes, encouraging clinicians to tolerate ambiguity while remaining vigilant for change. Cultivating disciplined judgement rather than reflexive intervention is presented as central to high-quality glaucoma care.