*January / February 2026*
**#82**

# the Ophthalmologist®

## Gut Feeling

Rethinking ophthalmology through the microbiome

10



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## We are excited to announce that The Ophthalmologist is now officially part of Conexiant Vision!

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This partnership allows us to expand our commitment to providing comprehensive, patient-centered eye care. By combining advanced diagnostic tools, treatments, and resources, we are positioned to deliver enhanced vision care that sets new standards in the field of ophthalmology.

We look forward to the exciting opportunities ahead as we continue to lead the way in vision care innovation. Stay tuned for more updates as we embark on this new chapter together!

# 2026: Priorities, People, Progress

*From Drivers of Change to emerging leaders in glaucoma, we continue to highlight how ophthalmology continues to evolve*

As we turn the page into a fresh year, I'm delighted to wish all our readers a very Happy New Year!

In 2025, The Ophthalmologist reminded us – through ground-breaking research, incisive commentary, and illuminating profiles – that this community never stands still. In 2026 we'll continue that journey, bringing you even sharper insight into the forces shaping modern ophthalmology.

One of our most eagerly anticipated features this year is the return of The Ophthalmologist Power List, reimagined for its 13th iteration as "Drivers of Change." This isn't just another tally of influence – it's a celebration of those individuals and teams whose work in the last 12 months has genuinely moved the needle on how eye care is delivered, experienced, and thought about globally.

Alongside this, we're excited to expand our Rising Stars program with a new focus on Glaucoma. Building on last year's spotlight on cataract and refractive innovators, this dedicated feature will shine a light on the next generation of clinicians and thinkers tackling one of ophthalmology's most pressing challenges.

Of course, the broader landscape of eye care continues to evolve with remarkable speed. From advances in AI-assisted diagnostics to novel gene therapies and digital care models reshaping patient pathways, 2026 promises to be a milestone year in both innovation and delivery. Studies and industry voices point to AI and remote care's growing roles, platforms that broaden access and tools that make diagnosis smarter and more personalized than ever before.

Here's to another year of discovery, impact and shared progress. Let's make 2026 unforgettable!

**Julian Upton,**
*Group Editor*

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# New Tool for Toddler Vision Testing

*WatDAT method measures recognition visual acuity in children as young as 18 months*

A team of researchers based at the School of Optometry & Vision Science at the University of Waterloo, Canada, have introduced and validated the Waterloo Differential Acuity Test (WatDAT) – a tool for measuring recognition visual acuity (VA) in children as young as 18 months. The study offers data on testability and early normative values, potentially reshaping how clinicians detect amblyopia and other early visual deficits.

"In young children under three years old… it is not [currently] possible to measure VA in a way that is equivalent to letter visual acuity, as measured in adults," explains Susan J. Leat, professor emerita at the School of Optometry and Vision Science and lead author of the study. "Since this type of visual acuity (called recognition visual acuity) is accurate and sensitive for detecting and monitoring eye disorders, we wanted to develop a test which would enable us to measure recognition VA as young as possible."

**Bridging a diagnostic gap**

Traditional recognition acuity tests – such as Kay Pictures or Patti Pics – typically require naming or matching, skills that develop around age three. Before then, clinicians rely on preferential-looking tests (e.g., Cardiff Acuity cards), which measure resolution visual acuity rather than recognition visual acuity and tend to overestimate visual function. WatDAT was designed to fill this gap by using an "odd-one-out" differential task that even toddlers of 18 months old and over could perform.

Children are shown four images (three identical distractors and one different target – a house among circles or a face among non-faces) and asked to identify the odd one out. This enables assessment of recognition VA without requiring the necessary language or matching skills of children to articulate their choice to the examiner.

**Superior testability in younger children**

The Waterloo team tested 57 children aged between 18 and 48 months, dividing them into "typical" and "atypical" developmental groups. They compared WatDAT's performance with Patti Pics and Kay Pictures, two widely used commercial tests.

The results showed:

- 96% of typical children successfully completed binocular testing with the WatDAT House/Circle (HC) version, compared with 75% for Patti Pics and 80% for Kay Pictures.
- Even monocular testing was feasible in more than half of participants – 56% for WatDAT HC and 54% for WatDAT Faces.
- 82% of children with ocular or systemic conditions (in the "atypical" group) were able to complete WatDAT binocularly.

These findings suggest WatDAT can reliably capture recognition VA at younger ages than current tools allow, helping clinicians to spot developmental or disease-related visual deficits earlier.

WatDAT produced VA values of around 0.21–0.27 logMAR in 18-48-month-olds, which is expected as children's VA is not fully developed at this age. In earlier adult validation of the WatDAT against ETDRS, WatDAT gave similar values to ETDRS VA and the differences between WatDAT, Patti Pics, and Kay were consistent in both studies. Also, WatDAT maintained excellent reliability, with over 90% of sessions in children rated as dependable.

Average testing time, including training, was roughly four minutes, comparable to existing methods. To keep the child engaged during this time, WatDAT's digital version includes a number of animated rewards throughout the process. "This helps to engage and motivate them, and it makes the whole test into more of a game," says Leat.

Credit: Adobestock.com

**Clinical and developmental implications**

The study authors emphasize that recognition acuity is a more sensitive indicator of visual deficits than resolution-based methods, and so should be used when possible to measure VA in young children. The WatDAT's ability to measure it in children under three years old represents a significant advance in pediatric screening.

The tool may be especially useful in screening for amblyopia, congenital cataracts, or high refractive errors – conditions where early detection is key to preventing long-term visual impairment.

In terms of commercialization, WatDAT prototypes are now being evaluated in various practices and clinics across Canada, as well as in the US and UK. "[We] are evaluating the test regarding how it works in an everyday clinic setting, and so far we have had good feedback," Leat says. "If and when it becomes commercialized, optometrists and ophthalmologists who are involved in pediatric eye care could purchase it and use it for measuring VA in children between 18-36 months and upwards. This would aid clinicians in detecting possible eye deficits, deciding when to start correction/treatment of those deficits and monitoring that treatment."

As pediatric ophthalmology moves toward earlier, behaviorally adaptable testing, the WatDAT could transform vision assessment in toddlers — bringing accurate, developmentally appropriate acuity measurement into everyday practice. Leat adds: "We hope that since the WatDAT will be an additional tool for pediatric eye care providers, it will improve the quality of eye care for young children and encourage more optometrists and ophthalmologists to provide eye care for this population."

## Image of the Month

Back in her home village, Kany feeds her donkey with a shy smile. Just two days after undergoing cataract surgery funded by Sightsavers, she is gradually reclaiming her surroundings, marking the start of a life with restored vision.

Credit: Sightsavers / Harandane Dicko

### QUOTE OF THE MONTH

*"As we enter this new era of care, our imaging techniques must evolve to meet the challenges of monitoring gradual disease progression."*

Eleonara M. Lad, MD, PhD, Duke University Medical Center, USA

## Pickleball Panic over Eye Injuries

*Pickleball surge fuels sharp rise in eye injuries, JAMA Ophthalmology study warns*

Credit: Adobestock.com

As pickleball cements its status as America's fastest-growing sport, ophthalmologists are sounding the alarm over an emerging public health concern: a steep rise in pickleball-related ocular injuries.

A cross-sectional study, conducted at Rutgers New Jersey Medical School, Newark, and published in JAMA Ophthalmology, has analyzed two decades of data from the National Electronic Injury Surveillance System (NEISS) to quantify the incidence and demographics of eye injuries linked to pickleball between 2005 and 2024.

The findings reveal that pickleball-related ocular injuries increased dramatically over the past four years, mirroring the sport's exponential growth in popularity.

With approximately 19.8 million players now participating in the sport, ophthalmologists are likely to see increasing cases in both emergency and outpatient settings. The study authors are calling for standardized guidelines for eye protection for this hugely popular sport.

# Multimodal Imaging for MacTel

*Employing multimodal imaging for the early detection of macular telangiectasia*

**By Eleonora M. Lad**

In my practice at Duke Eye Center, I have witnessed a remarkable transformation in our understanding of macular telangiectasia (MacTel). What was once considered a rare vascular condition affecting approximately 0.1% of the general population is now recognized as a potentially more common neurodegenerative disease involving Müller cell glia and photoreceptors (1, 2).

This conceptual shift has profound implications for how we approach MacTel, and the importance of early detection and accurate disease monitoring has never been greater. However, the early signs of MacTel are subtle and easily misdiagnosed during routine examination, often mimicking diabetic retinopathy, cystoid macular edema (CME), and other common macular conditions (3).

In this article, I will describe the imaging findings that are most helpful for early detection of MacTel, highlight how each modality contributes unique insights, and share how multimodal imaging has transformed and will continue to advance the diagnosis and management of this disease.

**Early signs of MacTel are easy to miss**

Most patients I encounter with MacTel arrive through referrals from general ophthalmologists or non-retina subspecialists who have noticed subtle macular changes they cannot definitively characterize. This referral pattern highlights the reality that MacTel is relatively easy to overlook during routine examination.

At initial presentation, patients typically have few or no symptoms despite ongoing disease progression. Clinical findings at this stage are subtle and easily missed — slight retinal graying or a faint opacity temporal to the fovea (4). Occasionally, right-angled venules or small crystalline deposits can provide additional clues (5). Many patients also present with type 2 diabetes mellitus and metabolic syndrome, conditions that can mask or confound the diagnostic process.

Given these challenges, multimodal imaging becomes not just helpful but essential. Each imaging modality reveals a different aspect of MacTel pathophysiology, and their combination transforms a difficult diagnosis into a much more straightforward one.

**Building the diagnostic picture with multimodal imaging**

In my practice, spectral-domain optical coherence tomography (SD-OCT) serves as the cornerstone diagnostic tool for MacTel. Whether performed before referral or as my first confirmatory step, SD-OCT reveals the disease's most characteristic early finding: cavitation within the ellipsoid zone (EZ) (Figure 1) (6, 7). This photoreceptor integrity loss, typically temporal to the fovea, manifests as a spectrum ranging from subtle disruption in early disease to pronounced cavitations in advanced cases.

The true value of OCT extends beyond mere visualization. Clinical trials have demonstrated a nearly one-to-one correlation between areas of photoreceptor loss on OCT and scotomas on microperimetry (8). This strong structure-function relationship provides confidence that what we observe on imaging translates directly to meaningful visual function changes.

While OCT provides the structural detail, fundus autofluorescence (FAF) offers further diagnostic clarity. In early disease stages, a hyperautofluorescent wedge-shaped pattern temporal to the fovea is highly suggestive of MacTel (Figure 2) (7, 9). I find FAF particularly valuable for two purposes: confirming the diagnosis when OCT findings are subtle, and tracking disease progression over time as hyperautofluorescent areas expand.

Fluorescein angiography (FA) serves as another valuable diagnostic tool, particularly for detecting baseline temporal foveal leakage and the presence

of choroidal neovascularization (CNV) (Figure 3) (2, 7). In my practice, FA helps not only confirm the diagnosis but also exclude alternative causes of macular pathology.

Finally, although not routinely integrated into my workflow, OCT angiography (OCT-A) can provide exceptional visualization of the hallmark telangiectatic vessels and right-angled venules (10, 11). These right-angle venules represent a three-dimensional finding that appears as vessels oriented perpendicular to the plane of view on fundus examination. In practices where OCT-A is readily available, this modality can further strengthen the diagnostic confidence.

Beyond detection, multimodal imaging enables monitoring of disease progression over time. In my practice, OCT remains my standard tool at every follow-up visit, while FAF and FA are useful at baseline and periodically thereafter to capture disease evolution.

**Next steps in MacTel**

Despite our sophisticated imaging capabilities, a critical gap remains in our lack of readily accessible tools to quantify disease progression in practice. The primary endpoint in clinical trials — EZ area loss — currently lacks a straightforward quantitative measurement tool like OCT devices in clinics, which hampers our ability to monitor disease change in meaningful ways. What we urgently need are automated systems that provide both qualitative mapping and quantitative metrics that we can display to patients, such as a color-coded map of the retina along with numerical readouts that would enable us to demonstrate the rate of their disease progression in a comprehensible manner.

Looking further ahead, I envision artificial intelligence (AI) algorithms that leverage clinical trial databases to predict functional outcomes from structural findings. These tools could generate reports demonstrating how patients' scotomas may or may not be progressing, and would represent a powerful advance in our ability to demonstrate the effects of this insidious disease on patients.

**Conclusion**

For general ophthalmologists, the key message is clear: when you encounter temporal juxtafoveal changes, consider MacTel and refer for specialized imaging evaluation. The subtle early signs that might be missed on routine examination can be definitively characterized through multimodal imaging.

As we enter this new era of care, our imaging techniques must evolve to meet the challenges of monitoring gradual disease progression. The future belongs to quantitative, AI-enhanced imaging platforms that can demonstrate disease changes in ways that are meaningful to both clinicians and patients.

Disclosure: Through Duke University, Dr. Lad received research funding from Neurotech.

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*"Despite our sophisticated imaging capabilities, a critical gap remains in our lack of readily accessible tools to quantify disease progression in practice."*

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# Seeing the Bigger Picture

*Why larger optical zones in KLEX might be the next step forward*

By Lena Beckers, with contributions from F. Kretz and D. Beckers

When I witnessed my first keratorefractive lenticule extraction (KLEX) years ago, I was amazed by the elegance and efficiency of the technique – a single femtosecond laser creating a lenticule that could be removed through a tiny incision, freeing patients from glasses or contact lenses with minimal discomfort. What an addition, I thought, from the excimer-driven days of PRK and LASIK, with their long recovery times and accompanying flap-related worries (1–4).

Yet even as we celebrated KLEX's precision and safety, one question kept coming up in our clinic: could we make good vision even better, particularly in young patients with large pupil sizes who struggled with night vision or glare?

This question led us down a path toward something deceptively simple: making the lenticule just a little bigger.

**From PRK to KLEX: a brief (and painful) evolution**

Refractive surgery has always been about refining light and reducing the side-effects of that refinement. PRK in the 1980s was revolutionary, but epithelial removal came with a price: slow recovery and potential haze for the patient (2). In the 1990s, LASIK changed the game with a corneal flap that sped healing but introduced biomechanical vulnerabilities like dislocations and dry eye (3,4).

Then keratorefractive lenticule extraction (KLEX) stepped onto the stage: a flapless, minimally invasive technique that uses a femtosecond laser to carve out and remove a thin lenticule from within the cornea (5). It preserves more corneal nerves and biomechanics (6), and shows less postoperative dry eye symptoms (7,8).

**The case for going big**

In standard KLEX, optical zones typically range from 6.0 to 7.0 mm (10,11). But as every refractive surgeon knows, the human eye is not one-size-fits-all. Some patients, particularly younger ones, have larger pupils, especially under scotopic conditions (13,19). When the pupil dilates beyond the treated zone, light enters through the untreated corneal periphery, leading to aberrations, halos, and reduced contrast sensitivity (14).

This realization sparked our interest in whether larger optical zones could bridge that gap – could we expand the treatment area to match the natural physiology of patients' pupils, keeping safety and long-term refractive stability intact?

**Putting it to the test**

To explore that question, we conducted a retrospective case series of 40 eyes from 20 patients, all treated with the VisuMax 800 (Carl Zeiss Meditec). We pushed beyond the standard parameters, performing KLEX with a 7.7 mm optical zone and a 7.9 mm cap diameter – to our knowledge, this was one of the largest reported configurations to date.

Our patient cohort had preoperative spherical equivalents ranging from –1.5 D to –4.75 D and astigmatism up to –3.0 D. Pupil size under photopic, mesopic, and scotopic conditions was measured, with scotopic pupils averaging around 7.0 ± 1.1 mm, consistent with published data in younger adults (19).

Three months after surgery, 100% of eyes were within ±1.0 D of target, 97.5% within ±0.5 D, and all patients achieved 20/20 vision, with 25% reaching 20/16. These findings mirror recent real-world VisuMax 800 outcomes showing excellent safety and predictability (15).

Contrast sensitivity under mesopic conditions remained stable compared to normative data (16). No significant complications or adverse effects were observed.

**Why it matters**

If the refractive outcomes and contrast sensitivity remain unchanged, what's the true payoff of enlarging the lenticule? It comes down to the effective optical zone: the real area of corneal reshaping that contributes to clear vision. Studies have shown that the effective zone is typically smaller than the programmed one, shrinking by 1.4–1.7 mm due to biomechanical and epithelial remodeling (17–18).

So a programmed 6.5 mm zone may translate to an effective area closer to 5 mm, an area smaller than many young patients' pupils. By expanding the programmed optical zone to 7.7 mm, we aim to preserve an effective zone that

actually matches or exceeds the patient's scotopic pupil.

**Who stands to benefit**

While larger lenticules could theoretically benefit many patients, certain groups stand out:

- Younger patients (<40 years), who naturally have larger pupils (19)
- High myopes, in whom regression risk is higher (20–22)
- Occupationally dependent patients, such as pilots, drivers, or others working in low-light conditions

For these individuals, enlarging the optical zone could make a meaningful difference in everyday visual quality.

**Challenges and caveats**

Of course, no innovation comes without uncertainty. Larger lenticules mean more tissue removal, and so careful attention to residual stromal bed thickness remains essential. Surgeons must also ensure accurate centration (12) and consistent dissection to avoid any irregularities.

Our own study's limitations – a small sample size, short follow-up, and lack of postoperative aberrometry – highlight the need for longer-term evaluation to confirm biomechanical stability and determine whether regression truly decreases.

**Looking ahead**

As refractive surgeons, we often think of "customization" in terms of topography, wavefront, or nomograms. But perhaps it's time to add optical zone size to that list. Personalizing the lenticule diameter to each patient's pupil dynamics could represent the next subtle yet significant step forward in refractive surgery outcomes (10,11,17).

When I tell patients we are aiming to "make their SMILE bigger," they usually laugh. But the truth is: that might be exactly what their vision needs.

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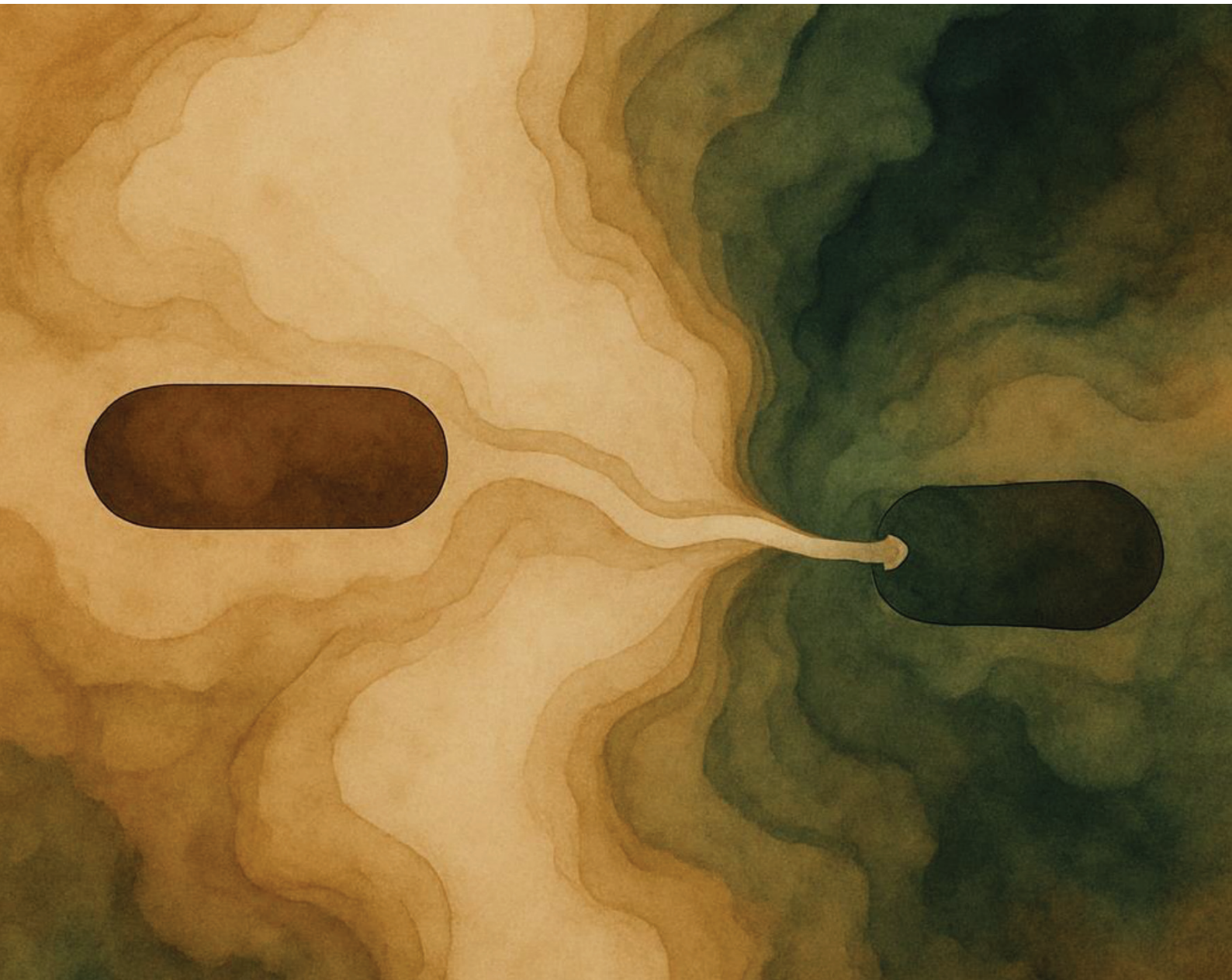
# Rethinking Ophthalmology

## *Through the Microbiome*

From microbial scarcity to immunological significance – rethinking the eye's interface with its microbial environment

By Farhad Hafezi, Mark Hillen



What if the next revolution in ophthalmology isn't engineered in a lab, but cultured in the gut? For centuries we've gazed through the cornea, peeled back retinal layers, and perfected intraocular technique – only to find ourselves staring into the microbial abyss. The eye, once thought sterile and immunoprivileged, is entangled in systemic crosstalk between microbial tenants and ocular health.

Welcome to the gut–eye axis – where the bacteria fermenting your lunch may be shaping meibomian glands, retinal vessels, and even glaucoma risk (1). It's as disconcerting as it is exhilarating: treating dry eye, age-related macular degeneration (AMD) or uveitis might one day mean prescribing kefir with cyclosporine. But the question isn't whether the microbiome matters – that ship has sailed. The real question is: how should ophthalmology respond?

## REWRITING THE ORIGINS: FROM VAN LEEUWENHOEK TO DYSBIOSIS (2)

It began, as many disruptions do, with curiosity and a crude lens.

In the 17th century, Antonie van Leeuwenhoek peered into water droplets and dental plaque with his handmade microscopes and discovered a universe. What he saw were the first glimpses of the microbial world: teeming, shape-shifting, complex. He could not have known that centuries later ophthalmologists would cite his name when discussing corneal inflammation, autoimmune uveitis, or intraocular flora (2). Yet here we are.

"Microbiome" is now scientific lingua franca – overused, underdefined, often blurred with "microbiota." Think of it as a dynamic ecosystem of microbes, molecules, and metabolites that hums within and upon us, shaping physiology at every level. The human gut harbors tens of trillions of organisms – bacteria, viruses, fungi, archaea, and phages. The balance between Firmicutes and Bacteroidetes has been linked to metabolic and mental health disorders, and ophthalmology has now entered the dialogue.

Dysbiosis – microbial disequilibrium – is no longer just gastrointestinal. It is tied to dry eye, Sjögren's syndrome, uveitis, glaucoma, even AMD. The gut doesn't just digest – it whispers to the retina, nudges immunity, tweaks cytokines, and tilts the balance between health and disease, and support access to healthcare, both globally and locally."

## THE LACRIOME: RETHINKING TEAR DUCT DISEASE ONE MICROBE AT A TIME

The nasolacrimal system has long been viewed as plumbing: tubes, valves, drainage. Blockage meant backflow; inflammation meant infection. Case closed.

Enter the lacriome – the microbial and molecular microenvironment of the lacrimal drainage system (2). Not a gimmick, but a recognition that tear ducts are ecosystems, vulnerable to microbial imbalance.

Primary acquired nasolacrimal duct obstruction (PANDO) was long blamed on idiopathic fibrosis or aging. But metagenomic studies (3–7) tell a different story: lacrimal sacs harbor rich biodiversity – Acinetobacter johnsonii, Porphyromonas catoniae, Escherichia coli, Haemophilus influenzae, and more. Culture methods once gave us little beyond Staphylococcus aureus, whereas sequencing reveals an entire community.

Silicone stents after dacryocystorhinostomy? Colonized too – by Pseudomonas, Corynebacterium, Citrobacter. Their genes weren't passive: metabolism, virulence, immune evasion – all active (5). Patients with failed dacryocystorhinostomy showed different microbial profiles to those whose DCR succeeded. Coincidence, or microbial sabotage disguised as fibrosis?

It's time to abandon the sterile pipeline model. The lacriome is an immune-modulating frontier. Could microbial screening guide surgery? Could manipulation reduce post-op failure? The paradigm is shifting, and ophthalmology must follow.

## MGD AND THE MEIBOMIAN MICROBIOME: WHERE OIL, SKIN, AND SOIL COLLIDE

You probably blame "gland obstruction" for meibomian gland dysfunction (MGD), while ignoring the microbial elephant on the lid margin. MGD affects over a third of the world's population (8), yet clinical narratives still reduce it to lipid deficiency, keratinisation, or hormones. True enough – but scratch beneath the surface and a microbial world emerges, shaping gland health, systemic inflammation, tear film stability, and surface immunity.

Cutibacterium, Corynebacterium, Staphylococcus – the regulars. But Zhao et al. (2020) (9) also identified Campylobacter jejuni and Enterococcus faecium in MGD, species linked to chemotaxis, immune evasion, and barrier disruption. Age shapes the microbiome too: youth favors Cutibacterium acnes, while older lids gravitate towards Corynebacterium and Neisseriaceae (10) – acne versus keratitis, coincidence or warning?

Soil adds another twist. A Finnish trial showed compost and moss exposure boosted skin microbial diversity within days (11). So could urban-driven biodiversity loss fuel atopic and ocular surface disease? Enter Nitrosomonas eutropha, a soil ammonia-oxidizer now tested for dermatitis – and perhaps MGD (12, 13). It consumes sweat ammonia, and releases nitric oxide: antimicrobial, anti-inflammatory, wound-healing.

Is this the future of MGD therapy – a bacterial balm instead of doxycycline? We've long prescribed antibiotics to silence microbes. Perhaps it's time to listen.

*"If dry eye disease (DED) were a person, it would be that chronically misunderstood patient – symptomatic, elusive, often dismissed as psychosomatic."*

## DRY EYE AND THE OCULAR SURFACE MICROBIOME: INFLAMMATION BY A THOUSAND CUTS

If dry eye disease (DED) were a person, it would be that chronically misunderstood patient – symptomatic, elusive, often dismissed as psychosomatic. But perhaps DED is more than a tear film problem. Perhaps it is microbial – a slow inflammatory dialogue where dysbiosis whispers and immunity screams.

The ocular surface is no microbial desert. Even one bacterium per 17 conjunctival cells can tilt the balance toward disease (14). In Sjögren's, non-Sjögren's DED, and even healthy individuals, the tear film microbiome diverges in ways both taxonomically intriguing and functionally decisive.

Song and Qi showed Sjögren's patients had more Actinobacteria, fewer Bacteroidetes, with Corynebacterium and Acinetobacter overrepresented (15, 16). These same taxa drive barrier dysfunction, chronic inflammation, and antigenic mimicry. The gut shows parallel disturbance: reduced Faecalibacterium, increased Prevotella, reversed Firmicutes/Bacteroidetes ratio – leading to elevated IL-6, Th17 skewing, and goblet-cell loss in germ-free mice (17).

Even the tear proteome reflects unrest. Cutibacterium acnes and Acinetobacter johnsonii express helicases and arsenical-resistance proteins, modifying host DNA repair and oxidative-stress responses (18).

DED is multifactorial, yes – but when gut and ocular microbiomes are simultaneously inflamed and depleted, the outcome is choreographed immunology. The uncomfortable question: by the time the Schirmer strip is dry and fluorescein pools, has the microbiome already dictated the course?

Portrait of Anthonie van Leeuwenhoek by Jan Verkolj (c1680). Credit: By Jan Verkolje - http://www.rijksmuseum.nl/collectie/SK-A-957, Public Domain, https://commons.wikimedia.org/w/index.php?curid=34320547

## AQUEOUS HUMOR AND THE INTRAOCULAR MICROBIOTA: THE MYTH OF STERILITY

For decades we've been taught that the anterior chamber is sterile and immunoprivileged.

But what if it never was?

In 2021, Deng et al. analyzed 1,000 aqueous humor samples collected under surgical sterility (19). With metagenomic sequencing and strict contamination controls, they found Cutibacterium acnes in >70% of samples – not as pathogen, but resident.

Others soon appeared: Enterococcus faecalis, Staphylococcus epidermidis, even anaerobes. Not post-op infections – present pre-incision. Controls were clean. The result was real. A supposedly sterile compartment wasn't sterile. Implications? First, these microbes may quietly regulate ocular immune tone – complement,

antigen presentation, T-cell trafficking. Second, they may explain unpredictable inflammation: why one patient develops fibrin after cataract surgery and another doesn't, despite identical technique.

And third – most provocatively – this may be a missing link in glaucoma pathogenesis. Cutibacterium acnes in aqueous fluid could prime immune responses or oxidative-stress pathways in the trabecular meshwork.

We've long trusted anterior chamber taps to reveal pathology. But perhaps we've overlooked baseline physiology – its microbiome. "Sterility" was a comforting fiction. Biology, as ever, is messier – and more interesting – than we imagined.

## UVEITIS AND THE GUT–RETINA AXIS: WHEN T CELLS TAKE THE BAIT

Autoimmune uveitis has always been a riddle wrapped in inflammation. Genes, MHC haplotypes, retinal antigens like interphotoreceptor retinoid-binding protein have been blamed. But what if the true trigger isn't in the eye – or even the bloodstream – but in the gut?

This is the gut–retina axis: once speculative, now backed by data. In the B10.RIII experimental autoimmune uveitis model, broad-spectrum oral antibiotics reduced not only gut bacteria but also retinal inflammation, T-cell infiltration, and cytokines (20).

Even single drugs like vancomycin or metronidazole cooled retinal disease. The gut was no bystander; it was an arsonist.

Germ-free mice sealed the case. R161H T-cell receptor transgenics rarely developed spontaneous uveitis without microbiota. Reintroduce commensals, and the retina flared (21).

This isn't infection; it's mimicry: gut bacteria expressing peptides resembling retinal antigens, priming naïve T cells for a misguided attack in the eye. T cells are being trained in the gut to strike the retina.

So why treat uveitis as a purely ocular disorder? If microbiota can induce or dampen retinal autoimmunity, gut-based immunomodulation isn't speculative – it's therapeutic. Yet probiotics, diet shifts, or microbiota-targeted therapies remain absent from clinical guidelines.

We're comfortable injecting steroids into eyes. But adjusting gut flora? Still fringe. The gut–retina axis dares us to look upstream. The question is: will we?

## GLAUCOMA AND THE MICROBIAL TRIGGER HYPOTHESIS: WHEN THE PRESSURE ISN'T JUST OCULAR

We've spent decades staring at the optic nerve, measuring cup-to-disk ratios, lowering IOP – as if that alone could halt glaucoma. Yet visual fields keep declining, and so could the damage begin not at the lamina cribrosa, but in the gut itself? Evidence says yes. In animals and humans, altered gut microbiota links to retinal ganglion cell loss, oxidative stress, and immune activation – even with normal IOP (22, 23). Here too, the microbiome may play a hidden role.

Helicobacter pylori, found in the trabecular meshwork and iris of POAG patients (24), has been tied to glaucoma across studies and meta-analysis (25). Not by invading the eye, but by priming systemic inflammation. Its heat shock proteins (HSPs), together with TLR4 and TLR9 polymorphisms, can trigger retinal autoimmunity (26).

Chen et al. showed transient IOP elevation in mice activated T cells against HSPs – an immune memory persisting after pressure normalized (27). Germ-free mice? No microbiota, no neurodegeneration. Glaucoma may be as much immune priming as mechanical stress.

Gut profiles in POAG reinforce the case: more Prevotella, Enterobacteriaceae, Escherichia coli; fewer SCFA-producers (23). A dysbiotic gut may be stoking neuroinflammation.

Here's the provocation: we already modulate glaucoma pharmacologically, but ignore a parallel axis. Should gut profiling, diet, or microbiota-directed therapies join treatment? Glaucoma has always been a thief of sight. Perhaps it has been an "inside" job all along?

## AMD AND THE MICROBIAL SIGNATURE OF DIET: A TALE OF DRUSEN AND DYSBIOSIS

For a disease named after age, AMD is showing its youthful side – at least through the microbiome. AMD has long been blamed on time, smoking, genetics, and complement dysregulation. But evidence suggests our microbial residents – shaped by diet, antibiotics, and lifestyle – may be just as guilty in forming drusen as any complement factor H polymorphism. The gut–retina axis is not metaphorical. It's metabolic.

Rowan et al. showed mice on high-glycemic diets developed AMD-like lesions, reversible with low-glycemic diets (28). The proposed mechanism: altered microbial metabolites – serotonin and SCFAs – modulating retinal inflammation and oxidative stress. Andriessen et al. found high-fat diets drove CNV with leaky vessels, microglial activation, and systemic inflammation; fecal transplants from healthy donors suppressed angiogenesis (29). Humans echo this. Zinkernagel et al. reported AMD patients enriched in Oscillibacter, Eubacterium ventriosum, and Ruminococcus torques – taxa tied to gut permeability and cytokine spillover (30).

Controls instead harbored SCFA-producing Bacteroides eggerthii. Even CFH genetics links back: carriers of risk alleles showed enrichment of Negativicutes, an obscure Firmicutes class (31).

AREDS hinted nutrition mattered. But zinc absorption, for

example, is mediated by microbial competition (32), suggesting supplement efficacy may depend more on gut ecology than dosage.

So what now? Should retina clinics prescribe fiber with lutein? Should probiotic profiling join AMD management? We've focused on drusen, but perhaps the deeper story lies in microbial fingerprints shaping retinal health long before OCT shows damage.

## DIABETIC RETINOPATHY AND MICROBIAL METABOLISM: SUGAR, INFLAMMATION, AND THE BACTERIAL MIDDLEMAN

We know the story of diabetic retinopathy (DR): hyperglycemia drives microvascular damage, ischemia, and neovascularization. But perhaps a co-author has been overlooked – one with its own metabolism: the gut microbiome.

Das et al. found patients with DR carried a distinct microbial signature – reduced Faecalibacterium and Roseburia (major butyrate producers) and increased Escherichia, Enterobacter, and Shigella, all pro-inflammatory and endotoxin-rich (33). These shifts lowered SCFA levels, increased gut permeability, and primed systemic inflammation to damage retinal vessels.

The pattern echoes Type 1 diabetes, where gut-derived bacterial amyloids may trigger autoimmunity through molecular mimicry – first against pancreatic β-cells, later affecting the retina (34). Contradictions exist. Huang et al. reported higher Bacteroidetes in DR (35) counter to its usual anti-inflammatory role. Even Lactobacillus and Bifidobacterium – probiotic "heroes" – were elevated.

Compensation? Or regional variation shaped by diet, drugs, and environment? These inconsistencies don't negate the link; they emphasize complexity. The microbiome is no static fingerprint but a dynamic organ, metabolically entangled in diabetes: fermenting carbohydrates, modulating insulin sensitivity, and influencing cytokines that cross the blood–retina barrier.

So here's the challenge: if metformin alters the microbiome, and metabolites like butyrate influence retinal inflammation, why aren't we tracking microbial signatures alongside HbA1c in trials? We monitor glucose obsessively. Perhaps it's time to measure microbial health with equal rigor.

## THE THERAPEUTIC FRONTIER: FECAL TRANSPLANTS, SYNBIOTICS, AND THE RISKS OF PLAYING GOD

It began with a fecal enema in 1958. Today, fecal microbiota transplantation (FMT) is FDA-approved for refractory Clostridioides difficile and is edging into new fields – even ophthalmology. Why? Because if gut dysbiosis can inflame the eye, rebalancing the gut might calm it. In small human studies, aqueous-deficient dry eye improved after FMT from healthy donors (36).

In mice, FMT restored goblet cells and ocular surface integrity (17, 37), while in AMD models, "old" microbiota transplanted into young mice accelerated retinal inflammation – reversed by the opposite transfer (38). This is medicine without molecules: not blocking cytokines, but reshaping ecosystems.

Yet the risks are sobering. FMT from patients with Behçet's or Vogt–Koyanagi–Harada disease into germ-free mice worsened uveitis (38, 39).

The microbes carried pathogenic potential. Like early blood transfusions, we're operating blind.

Probiotics and prebiotics – the consumer-friendly cousins – show mixed promise. Lactobacillus and Bifidobacterium improved mild DED, MGD, and vernal keratoconjunctivitis (40, 41). IRT5, a probiotic cocktail, reduced ocular inflammation in murine dry eye and autoimmune uveitis (42, 43). Yet results are inconsistent, and the risks, real: bloodstream infections, bowel ischemia, and even gene transfer of antibiotic resistance in immunocompromised hosts (44). The microbiome adapts, mutates, evolves.

So where does that leave us? We need rigorous, multiomics-driven human trials before racing to market synbiotics or FMT for ocular disease. Because manipulating the microbiome isn't prescribing a pill. It's akin to rewriting the rules of symbiosis.

## CONCLUSION: THE MICROBIOME IS WATCHING

It's tempting to dismiss the microbiome as just another "axis" fad – gut-brain, gut-lung, gut-eye. But that would be a mistake. The data are no longer suggestive; they are directive.

Across dry eye, MGD, AMD, uveitis, glaucoma, and even post-surgical fibrosis, microbial fingerprints keep appearing. Sometimes culprits, sometimes accomplices – always present, always evolving.

The question isn't whether the microbiome matters, but how we adapt to our new understanding. This means reframing how we undertake trials, as well as how we view inflammation and risk. Should every immunomodulation study collect stool samples? Should "ocular health" include microbial diversity scores? Should cataract consent forms acknowledge intraocular microbes?

Beyond the clinic lies a cultural reckoning. Medicine long equated health with purity and disease with contamination. The microbiome shows otherwise: health is symbiosis, and disruption – not invasion – often drives pathology.

The microbiome responds to what we eat, how we prescribe, how we sterilize, how we interpret inflammation. It remembers what we forget. How we account for this – and adjust how we assess and treat patients – might redefine what it means to see clearly.

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GLAUCOMA

# Nurturing Innovation in Glaucoma Surgery

*How one ophthalmologist's decades-long experience led to the invention of an innovative supraciliary device*

By Philippe Sourdille

Over the last 50-plus years, I have observed and participated in many advancements in glaucoma surgery. But when I first took up a career analyzing the blebs of so-called filtering iridectomies, I would never have guessed that it would lead me to inventing an innovative supraciliary device. My invention is the result of a fascinating era of continuous technological and clinical advance, driven primarily by the development of microsurgery. Through magnification, for example, we gained improved visual access to the trabeculum, to Schlemm's canal, and to the detailed, minimal modifications of aqueous outflow anatomical supports. Surgeons went from creating a hole at the corneoscleral junction to guarded filtration, trabeculectomy, non-penetrating trabecular surgery, and microinvasive glaucoma surgery. We can now take ab interno approaches aimed at direct aqueous access to Schlemm's canal with trabecular stents or supraciliary devices to increase uveoscleral outflow. This advance came partially from John Edward Cairns' original idea in trabeculectomy – opening Schlemm's canal to use the existing system of outflow channels.

So, when I read about André Mermoud demonstrating different outflow supports in deep sclerectomy (1) – in particular supraciliary and suprachoroidal effusion for lowering postoperative IOP – I asked myself: "How about using a cilioscleral interposition device to ease the uveoscleral outflow?" Through scleral incisions, the device could be placed at 2 mm from the limbus, without entering the anterior chamber and without creating any subconjunctival filtration. The IOP-lowering support would be an increased supraciliary and suprachoroidal aqueous egress through the untouched ciliary band, with an additional access to intra and episcleral vessels.

In practice, of course, this initial idea presented some challenges, both technical and perceptual. Technically, it was difficult to implant the device without visual control; to control its positioning required UBM (ultrasound biomicroscopy) or OCT (optical coherence tomography). Perceptually, we faced skepticism from the broader glaucoma community about this new surgical technique.

But I was undeterred. I believed in what I was doing and enlisted support from a long-time industrial partner and friend, Olivier Benoit – a medtech and biotech entrepreneur. Together, we carefully determined the procedure's optimal indications. After a three-year clinical follow-up, indicators revealed that our approach is effective in treating mild and moderate glaucoma. We observed a 33 percent IOP decrease and 70 percent

fewer medical treatments. This three-year clinical follow-up (2) draws upon the combined results of two CIliatech studies: SAFARI I and SAFARI II with cohorts of 20 and 22 patients living with open angle glaucoma, non-controlled by medication; none had previously had glaucoma surgery. In a subsequent study (3), we enlarged the indications to angle closure glaucoma with the same surgical technique, which resulted in the same outcomes.

Our approach saw aqueous penetrate the ciliary muscle bundles through the ciliary band without a cyclodialysis cleft. This unique process needed further investigation as very few images of previous supraciliary devices are available in the literature. Successive UBM examinations were performed on all our patients during the follow-up to document the device's position, stability, and biointegration, with an independent UBM expert analyzing all the results and

*"After three years, another thing I am proud of is that none of our cases have needed reintervention"*

confirming the device's perfect location, stability and excellent biointegration.

We were anxious to see how glaucoma specialists reacted to this unexpected approach, and their reactions ran the gamut from skepticism to congratulations. They challenged us on the surgical technique, the peri and postoperative device position, and conjunctival involvement.

After three years, another thing I am proud of is that none of our cases have needed reintervention – but we must still consider the possibility of additional filtering procedures being needed over a patient's lifetime, and our concept is compatible with additional procedures (if eventually needed). The concept is proven and we are now working to make the surgical technique easier, more straightforward and faster.

The last 50 years have definitely seen improvements in the benefit–risk ratio of glaucoma surgery – to the significant benefit of patients. Our research continues because, as all innovators know, innovation never ends.

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The last 50 years have definitely seen improvements in the benefit–risk ratio of glaucoma surgery – to the significant benefit of patients. Our research continues because, as all innovators know, innovation never ends.

*Philippe Sourdille is a highly experienced glaucoma surgeon who has dedicated his entire 50+ year career to ophthalmology. Over the course of his career, he accumulated vast experience in various glaucoma surgical approaches, including trabeculectomy, ab interno excimer laser, visco-canalostomy, and deep sclerectomy. Philippe has held pivotal roles within the clinical field, including serving as chairman of the 'Clinique Sourdille' for 20+ years, and president of the Implant Society for five years. In addition, Philippe was one of the founders of the ESCRS (European Society of Cataract & Refractive Surgeons).*

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ANTERIOR SEGMENT

# A Tooth for an Eye: Restoring Sight When There is No Hope

*Dr. Alfonso Vasquez-Perez outlines the advances in osteo-odonto-keratoprosthesis (OOKP) in the UK*

By Julian Upton

Moorfields South has officially launched its new surgical pathway for the osteo-odonto-keratoprosthesis (OOKP) service at St. George's Hospital, London, marking a major milestone in the treatment of severe corneal blindness. The first patient successfully underwent stage 1 OOKP surgery in November 2024, in collaboration with maxillofacial surgeons from St. George's (Rahul Jayaram) and the Sussex Eye Hospital (Jim Herold). Later on, in March 2025 the patient underwent stage 2 OOKP surgery and recovered full vision.

OOKP – often called "tooth-in-eye" surgery – is used for patients with end-stage ocular surface disease.

In this interview, Alfonso Vázquez-Pérez – consultant ophthalmologist and clinical lead for the Moorfields OOKP Service – explains how the procedure has advanced, and underlines the multidisciplinary approach vital to its success.

## Can you briefly explain what OOKP is?

Dr. Alfonso Vasquez-Perez: Osteo-odonto-keratoprosthesis was developed to restore vision in people who aren't suitable candidates for traditional corneal transplants, often because their ocular surface is too damaged, dry, or scarred for a graft to survive. In the 1960s and 70s, Italian surgeons created an approach using natural tissues at a time when modern biomaterials didn't exist. They discovered that dentine from a tooth was one of the most durable materials available. The technique involves creating an artificial corneal device made from dentine and bone, which is placed in front of the eye carrying a small optical cylinder providing a transparent window for vision. It's a complex surgery that also requires reconstructing the ocular surface, which is why only a few centers worldwide perform it. Nonetheless, since the 1970s it has successfully restored full vision for many blind patients unsuitable for conventional corneal transplantation. The OOKP involves a multidisciplinary team including ophthalmic, maxillofacial, anesthesia and psychology specialists. The treatment consists of two surgeries: stage 1 and stage 2, separated by 3 months, and patients recover vision only after stage 2.

## Which patient profiles are most suitable for OOKP surgery?

Patients referred for OOKP are those who have no remaining options for vision restoration, with severe dryness of the ocular surface and who often have received multiple failed corneal transplants. The most common group includes people with severe bilateral chemical burns, or uncommon autoimmune conditions which destroy the ocular surface and leave it dry and heavily scarred. In these cases, standard transplants always fail, leaving artificial corneal devices – keratoprostheses – as the only option.

There are several keratoprosthesis designs, but two are most widely used: the Boston Type I, which still requires a reasonably healthy and moist ocular surface, and the Osteo-odonto-keratoprosthesis, which is designed specifically for patients who are completely blind in both eyes and have a dry, severely damaged surface where other treatments cannot succeed.

Typical indications for OOKP include people with autoimmune conditions such as Stevens–Johnson syndrome and ocular cicatricial pemphigoid, which cause scarring similar to that seen after chemical injury.

We also need to determine whether the patient's tooth is suitable, of course. We usually use the canines, which are better for this operation. But if a patient does not have a suitable tooth, sometimes we can't do it.

## What have been some recent innovations in OOKP?

One of the most significant developments has been in diagnosing and managing early complications. OOKP patients inevitably face ongoing issues, and glaucoma is the most common – affecting around 80–85% of patients.

Diagnosis used to be extremely difficult because the surgery reconstructs the eye's surface using buccal mucosa (inner cheek lining). This means the eye no longer looks or behaves like a normal eye: you can't measure intraocular pressure, and glaucoma drops don't penetrate the thick buccal mucosa and do not work, so treatment must be surgical.

Now, with improved imaging and more sensitive scans, we can monitor the optic nerve much more effectively and detect early glaucoma changes. In addition, we have developed an endoscopic surgical

approach, working with my consultant colleagues at Moorfields from vitreoretinal (Rob Henderson) and glaucoma (Nick Strouthidis). Using a tiny camera in an optic fiber, which is introduced inside the eye, we can directly visualize the optic nerve and retina, and perform procedures such as tube shunt surgery when needed. This ability to look inside the eye accurately and intervene safely is one of the major advancements in OOKP care.

The other innovation we're working on involves removing the vitreous jelly during OOKP stage 2 surgery. In the past, this was very difficult and risky. Ideally, these patients should undergo a conventional pars plana vitrectomy, but the OOKP optical window is only 3.5 mm wide. That makes it almost impossible for a retinal surgeon to safely visualize and treat the peripheral retina. With a new approach – partial open sky pars plana vitrectomy – we can now remove the vitreous safely during the second stage before the tooth device is placed. This offers long-term benefits by reducing the risks of retinal detachment, chronic vitritis, and glaucoma. The vitreous often blocks glaucoma drainage tubes, making them ineffective, so removing it is important.

We are also exploring alternative materials for the OOKP lamina because not all patients have suitable teeth. We are now starting a new approach using calvarial bone (from the skull) to make the artificial corneal device (lamina), which our maxillofacial colleagues use successfully in other procedures. This bone is strong, resistant, and readily accessible to harvest.

**Given that the procedure involves multiple surgical specialties, how do you coordinate between ophthalmic, maxillofacial, and psychological teams?**

This is a major undertaking, and it can't be delivered by an ophthalmology service alone. Fortunately, Moorfields has a strong network and operates at a network site at St George's Hospital in London – a large general referral center where the ophthalmology department is run by Moorfields. We had already collaborated with maxillofacial surgeons there on previous complex cases, often working alongside ENT and maxillofacial teams.

When the OOKP service in Brighton came to an end, it created an opportunity to rethink how we delivered the procedure. We had already been involved in other complex work, such as transferring nerves to restore sensation to the eye, so we had an established framework – or "umbrella" – for multidisciplinary collaboration.

I've continued working closely with these maxillofacial colleagues, for whom much of the required surgical work is routine. At Moorfields, the keratoprosthesis service also includes dedicated Boston Type I specialists, as well as retina and glaucoma surgeons specifically assigned to these patients. Bringing everyone together for this new project wasn't simple, but because the expertise already existed within the network, assembling the team and establishing the service ultimately proved less challenging than we had initially expected.

**What are your findings in terms of OOKP patients' visual outcomes, quality of life, and complication rates?**

The outcomes are remarkable. Most patients come to us with only light perception, often after years or even decades of blindness in both eyes. If the retina and optic nerve are healthy, these patients can often achieve vision as good as 20/20. They will still need glasses, but the optical media itself is completely clear, with no cataract or opacity allowing for excellent visual potential. However, the optical cylinder is quite narrow, so the field of vision feels restricted. Some patients may be borderline for driving.

If there is existing retinal or optic nerve disease, the final vision will be less than ideal but still they can have a significant visual improvement. The challenge is that, before surgery, the front of the eye is so scarred that we cannot see inside to properly assess the retina or optic nerve. We perform extensive testing – ultrasound scans, electrodiagnostics, and a detailed history – to estimate visual potential. If the results suggest any reasonable chance of improvement, we still offer the procedure, while explaining that pre-existing conditions such as glaucoma or retinal damage may limit the final outcome. Even so, most patients experience a meaningful improvement in vision that significantly enhances their daily functioning.

**How do you see OOKP fitting into the broader landscape of corneal and ocular surface reconstruction?**

We're working with the Department of Biomaterials at University College London, which develops biomaterials for many medical specialties, including bone and dental substitutes. Although OOKP can restore vision, it remains an invasive surgery because we must harvest a tooth and surrounding bone. A synthetic or highly biocompatible material that integrates well in the ocular surface could simplify the operation and reduce its complexity. We've begun early planning meetings with experts to explore this, and I'm hopeful that developing such a material will be a major contribution by the end of my career.

We're also exploring hybrid techniques. Alongside the Boston Type I keratoprosthesis, there is the synthetic Boston Type II device. For patients without suitable teeth, or older patients with weakened bones, we may be able to adapt the Type II device and combine it with buccal mucosa and the same vitreous-removal approach used in OOKP. Some centers have begun similar work, though not yet in Europe, and we believe it could be an important advancement. Traditional OOKP can last decades – some patients from 1999 still have 20/20 vision – but even a hybrid that provides five good years of sight for an older patient would be immensely valuable.

**Is there anything you'd like to add?**

I'd like to acknowledge all my multidisciplinary team members and especially to my "great ally" in this journey of setting up the OOKP Service – Rahul Jayaram, Consultant Oral and Maxillofacial Surgeon at St. George's Hospital.

RETINA

# Expanding ERG in Diabetic Eye Care

*Why handheld electroretinography fits perfectly within the diabetic retinopathy management workflow*

By Raj K. Maturi

Diabetic retinopathy (DR) is growing in prevalence — it affected an estimated 9.6 million people in the US in 2021, with nearly 2 million already in the vision-threatening stage (1). The pressure to identify and manage risk efficiently is increasing, but the debate about how best to achieve this is ongoing.

One test that is becoming more routinely used in clinics is electroretinography (ERG). However, when I speak with colleagues about incorporating ERG into DR management, a common concern arises: "Will this slow down my clinic?" That concern reflects reality a decade ago when ERG systems were larger and more technically demanding, whereas current handheld ERG devices are designed for efficiency. These modern tests typically take only a few minutes, can be delegated to staff, and do not require dilation. Moreover, in most cases these testing protocols integrate into clinic flow without any significant disruption.

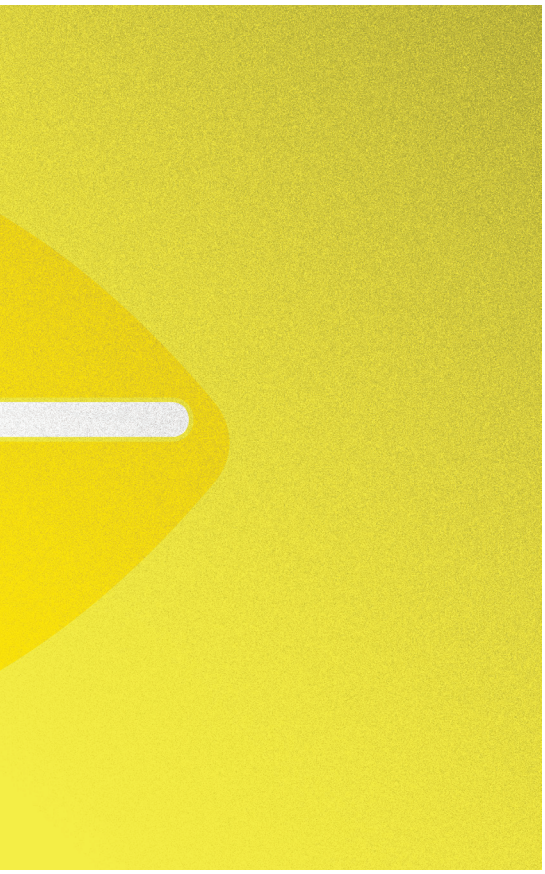
## Objective evidence for the patient

What makes ERG so powerful is its ability to detect functional changes in the retina that often precede structural ones (2, 3, 4, 5). Just as we track early cardiac biomarkers to prevent heart attacks, we need to embrace early functional markers in the eye, and ERG is ideally suited to play that role. It complements optical coherence tomography (OCT) and fundus imaging, especially in the preclinical stages when structural signs may be subtle or completely absent (2).

Fluorescein angiography (FA) might show peripheral nonperfusion at a certain stage, but before those vascular changes become apparent, ERG can provide an early signal that offers us an opportunity to intervene. In situations where the necessity of FA is uncertain, ERG can provide additional information that supports our decision-making. Because it is non-invasive and relatively quick to perform, ERG may help determine whether FA is immediately warranted, or whether it can be deferred. In this way, ERG does not replace FA, but rather complements it by refining the timing and context in which dye-based studies are pursued. For example, if a patient with a long-term diabetes history comes in without hemorrhages or microaneurysms but shows functional decline on ERG, that may be the pivotal moment to emphasize better systemic control. It gives patients tangible information — objective evidence that their retina is changing even if their vision seems fine for now.

I was pleased to see this sentiment supported in a recent longitudinal prospective study published in Ophthalmology Science (6). The study evaluated 56 parameters from four testing modalities — ERG, ultra-widefield fluorescein angiography (UWF-FA), optical coherence tomography angiography (OCTA), and fundus photography (FP) — to determine which modality best predicted progression to vision-threatening complications (VTC) such as proliferative DR, diabetic macular edema (DME), or the need for treatment.

What stood out most was that the RETeval DR Score, derived from ERG and pupillometry, emerged as the single strongest predictor of progression. A DR Score of 26.9 was associated with a relative risk of 5.6 for developing VTC ($p < 0.0001$). That's higher than any of the structural parameters evaluated. While these structural imaging parameters also showed statistical significance, their



predictive power was lower. The best predictive parameters from UWF-FA, OCTA and FP were:

- UWF-FA total ischemia index ≥ 0.125: RR = 5.3 ($p < 0.0001$)
- OCTA foveal avascular zone (FAZ) area ≥ 0.295 $mm^2$: RR = 3.6 ($p < 0.05$)
- Fundus photograph–based DRSS ≥ 47: RR = 2.1 ($p < 0.05$)

It's also important to note that ERG is now included in the American Academy of Ophthalmology's Preferred Practice Pattern® Guidelines for Diabetic Retinopathy (7). These guidelines are the benchmark for evidence-based care, and their acknowledgment of ERG reflects a growing consensus that functional testing should be part of comprehensive DR assessment.

## Information we can act upon

In my clinical practice and research, I've always been driven by a simple goal: to identify risk earlier so we can intervene sooner and preserve vision. For many years we've relied almost exclusively on structural findings to guide DR management, but structure alone doesn't tell the whole story. When fundus signs are already advanced, we've missed our chance to intervene proactively. The sweet spot for ERG is earlier — when it can serve as a wake-up call and a guide for tailored follow-up.

In terms of risk stratification, ERG gives us a way to personalize follow-up intervals. Imagine two patients with mild or moderate nonproliferative diabetic retinopathy (NPDR). If one shows completely normal full-field ERG results, I may feel comfortable extending their next visit out to 18 months. But if the other shows marked functional deficits, I'd likely bring them back in six months. The functional data guides my decisions in a way that fundus appearance alone cannot.

This is a significant advancement that has practical implications not only for retina specialists like myself, but also for general ophthalmologists and optometrists. For optometrists, it offers a reliable and efficient way to identify patients who may need earlier referral. For general ophthalmologists, it supports more informed decisions about when to monitor and when to treat. And in retina clinics, ERG can help triage patients more accurately and explain prognosis more clearly. When a single, objective number can provide that kind of predictive clarity, it's a tool worth paying attention to.

## A turning point in diabetic eye care

We've learned from other chronic disease specialties — cardiology, nephrology, endocrinology — that early intervention matters. The same holds true for DR. Yet we still often wait until structural damage becomes visible before we act; that's a missed opportunity. As a retina specialist involved in the DRCR Retina Network and the Mary Tyler Moore Vision Initiative, I've spent years exploring how we can improve outcomes for DR patients. One technology starting to stand out — particularly for its potential to reshape how we evaluate, stratify, and motivate patients — is electroretinography (ERG).

As these devices are placed in more clinics and as we begin testing ERG as part of the Mary Tyler Moore Vision Initiative, I hope we'll see a shift in how we think about diabetic eye care. ERG may not have been part of standard DR care a decade ago, but the data — and our clinical experience — are catching up fast. It's time to re-assess our protocols. It's not just about managing damage — it's about anticipating it. With the right tools, we can detect risk earlier, intervene more wisely, and perhaps even change the course of this disease.

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# A Truly Blessed Career

*Sitting Down With… Professor David Gartry, Senior Consultant Ophthalmic Surgeon to Moorfields Eye Hospital, London and Honorary Professor, City, University of London*

**Can you speak about why you first decided to become a doctor?**
When I was 12, I had a near fatal injury falling from a tree and a wonderful general surgeon at the Sunderland Royal Infirmary saved my life. Mr Banerjee was just amazing. When he discharged me from the hospital many weeks later he asked, "What would you like to do when you grow up?" Well, what was I going to say to that? "I want to be a doctor!" I replied without hesitation.

**What are some of the main obstacles you have faced in your career?**
Honestly, I really don't feel that I've encountered any obstacles at all. I've had a truly blessed and privileged career. When I tell people about the various posts I was appointed to and their timing, how I was in the right place at the right time with the right background for each individual step of my career, I consider that I've been blessed.

**Can you describe one of your proudest professional moments?**
By far the greatest moment in my professional career was being appointed as a consultant to the Corneal, Cataract and Refractive Services at Moorfields in July 1995. I remember that day very well. Back then consultant jobs at Moorfields were very hard to get and I was very pleased – and relieved - to have been appointed. I remember going home and telling my wife, who said, "We both knew you were going to become a consultant somewhere, but I didn't think it would be at Moorfields!"

**Looking forward, are there any specific developments you would still like to see in the cataract/refractive space?**
With laser surgery on the cornea, I think it's reached a very high pinnacle already. One thing worth mentioning is that – with all of the algorithms, the high-tech lasers, the fantastic tracking systems and the wavefront aberrometry analysis we now have – I still can't see us getting around the problem of individual corneal wound healing. Because no matter what you do with the most amazing technology available, how do you know exactly how an individual cornea is going to heal? And basically, you don't.

'Back in the day' our original publications – in the late '80s and early '90s – highlighted the considerable variability in refractive outcomes because of the relatively unpredictable way in which individual corneas healed. Since then, we've done a lot of work to try to control this variable, including algorithm adjustment using multivariate analysis and vastly improved laser technology, but ultimately we're left with a biological substrate, and I think there's not much further to go with that. If we are going to get even better results with predictability approaching 100 percent, I believe we will have to rely on a completely different type of technology – another paradigm shift. Unfortunately, I'm not able to predict what that would look like.

*"I've had a truly blessed and privileged career. Looking at the way it all panned out, I really couldn't have made it up!"*

**What are your thoughts on the integration of artificial intelligence into laser eye surgery?**
With all of the imaging that we do in Medicine and Surgery today, anything with a digitized image can now be analyzed to the nth degree using AI. And then, of course, machine learning – and now deep learning – will allow us to find subtle differences in images that we previously might have missed. This is very important in screening, early diagnosis and management, and in the treatment of corneal, cataract, refractive and retinal conditions AI comes into its own. There's been a lot of debate in both the House of Lords and the Houses of Parliament about AI, and I think they all agree that it's an amazing technology. The general consensus is that it just needs good regulation.

**What advice would you give to ophthalmologists starting out on their career journeys?**
Be very enthusiastic in everything you do and in all branches or subspecialties of Ophthalmology, and keep an open mind as to what you might like to do later in your career. And then, perhaps getting involved in clinical studies – anything that your consultant is working on or interested in – and helping with those studies. This may even be retrospective surveys (yes – I know!) - I did quite a few of those in the past.

**Do you have any hobbies outside of ophthalmology?**
My dad – rest his soul – gave me his old 1930s Dakora camera when I was 10 or 11, and I've been keen on photography ever since. When we did the first excimer laser trials in 1988/89 at St Thomas', I did all the photography of the pre-op and post-laser patients. This included standardized flash photography to record very subtle anterior stromal haze in the lasered corneas, which wasn't very easy to see in all patients by just looking at the slit lamp. So my photography experience came in very handy then.

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