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Review Article

What Stroke Medicine, Ophthalmology, and Epileptology Each See in the Same Scan, and Why None of Them Sees All of It

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Abstract

Optical coherence tomography is performed several million times a year worldwide. Almost all of those scans are read for glaucoma or for macular disease, reported in that framework, and filed. On a substantial minority of them is a record of a cerebrovascular event, and nobody looks for it. This review is about that gap. We set out the anatomy that makes the retina genuinely, and not merely metaphorically, central nervous tissue, and then follow four clinical questions through it. Is retinal arterial occlusion a stroke, and if it is, why do so few services treat it as one? Does the retina keep a legible record of an occipital infarct, and can that record be dated? Can retinal images predict stroke before it happens, and does the prediction beat what the clinician already knows? And what, if anything, does the retina say about epilepsy, beyond the vigabatrin surveillance that is currently its only routine epileptological use? Our answers differ in confidence. The first is settled and simply not acted upon. The second is well established and, we think, under-exploited in a way that has consequences for the timing of visual rehabilitation. The third is oversold. The fourth is thin enough that we present it as a question rather than a finding. We propose a ten-item minimum retinal dataset intended to let these four literatures be combined, which at present they cannot be, and we state four predictions that would show us to be wrong. We also record a disagreement between two of us about the fourth of them, because it is not resolved and pretending otherwise would misrepresent the state of the evidence.

Keywords: *Oculomics; Optical Coherence Tomography; Retinal Artery Occlusion; Post-Stroke Epilepsy; Trans-Synaptic Degeneration; Visual Rehabilitation*

1. Introduction

Sudden painless loss of vision in one eye. Whether that patient is treated as having had a stroke depends, in most health systems, on which door they walked through.

Through the emergency department, there is a reasonable chance of a stroke code, carotid imaging, prolonged rhythm monitoring and a statin the same day. Through eye casualty, there is a reasonable chance of ocular massage, an outpatient Doppler in six weeks, and a discharge summary that never uses the word stroke.

This is not a knowledge problem. Since 2021 the American Heart Association has stated plainly that central retinal artery occlusion is a form of acute ischaemic stroke, that it constitutes a medical emergency, and that the stroke code needs two additions when it is suspected: funduscopy to confirm the diagnosis, and screening for arteritis.[1] The 2013 redefinition of stroke had already brought retinal infarction inside the tissue-based definition.[2] The statements exist. What is missing, in most hospitals, is a pathway that behaves as though they do.

We take that as the emblem of something wider. The retina is the only part of the central nervous system that can be photographed. Optical coherence tomography resolves its layers close to histological scale, non-invasively, in seconds, on machines already installed in nearly every ophthalmology department on earth, and colour fundus photography runs at population scale through diabetic eye screening. The neurological content of those images is, in routine practice, thrown away at the point of reporting.

Four separate literatures have grown up around this tissue. Acute retinal ischaemia belongs to stroke medicine and neuro-ophthalmology. Retrograde trans-synaptic degeneration belongs to neuro-ophthalmology alone. Oculomic risk prediction belongs increasingly to computer science. Retinal change in epilepsy belongs to a small group of epileptologists interested in one drug. Our contention is that these are four consequences of one anatomical fact, and that their separation is an artefact of how specialties are organised rather than anything about biology.

We should say at the outset what this review does not do. It is not systematic. It does not tell anyone to buy an OCT machine. It does not claim that retinal imaging should displace any existing investigation. The argument is narrower and, we hope, harder to dismiss: a scan that is already being performed should not be read by one specialty and discarded by three.

2. What the Retina Actually Shares with the Brain

“The eye is a window to the brain” has been repeated often enough to stop carrying content. The strength of everything that follows depends on the specifics, so it is worth being exact about what is shared and what is not.

The retina is not analogous to central nervous tissue. It is central nervous tissue. It arises as an evagination of the diencephalon. The optic nerve is a white-matter tract, myelinated by oligodendrocytes from the lamina cribrosa backwards, not a peripheral nerve. Retinal ganglion cells are projection neurons synapsing in the lateral geniculate nucleus, and like other central projection neurons they do not regenerate.[3] Ganglion cell loss therefore carries the interpretive weight of cortical neuronal loss, with the difference that it can be quantified in micrometres during a fifteen-minute clinic appointment.

The vascular relationship is equally direct. The central retinal artery is the first intracranial branch of the ophthalmic artery, which is itself the first major branch of the internal carotid beyond the cavernous segment. In embolic terms the retinal circulation functions as a sampling port on the carotid. Retinal arterioles run at 100–300 µm, which is the calibre of the cerebral small vessels that cannot be imaged directly at all; the brain’s small-vessel bed can only be inferred from its consequences, whereas the retina’s can be photographed.

Metabolically the inner retina is among the most demanding tissue in the body per unit mass and tolerates ischaemia poorly. Primate work established irreversible damage after roughly 90–100 minutes of complete central retinal artery occlusion, a window tighter than anything assumed for cerebral cortex.[4]

Table 1 sets out the correspondences that matter at the bedside.

Table 1. Structural and functional correspondences across the retino-cerebral axis.

Domain	Cerebral counterpart	Retinal counterpart	Practical measurement
Projection neurons	Cortical pyramidal cells	Retinal ganglion cells	Macular GCL–IPL thickness (OCT)
White-matter tract	Optic radiation	Retinal nerve fibre layer	Peripapillary RNFL thickness (OCT)
Large-artery embolic target	MCA or PCA branch	Central or branch retinal artery	Fundoscopy; fluorescein angiography
Small-vessel bed	Lenticulostriate arterioles	Retinal arterioles and venules	Calibre, tortuosity, fractal dimension
Capillary density	Cerebral microvascular density	Superficial and deep capillary plexus	OCT angiography vessel density
Barrier	Blood–brain barrier	Blood–retinal barrier	Angiographic leakage
Downstream degeneration	Wallerian degeneration	Retrograde trans-synaptic degeneration	Homonymous GCL thinning (OCT)

Two limits belong here rather than buried in a limitations paragraph at the end.

The retina has no cortical lamination, no recurrent excitatory connectivity of the kind that makes neocortex epileptogenic, and nothing like the interneuronal diversity of cortex. Retinal findings in epilepsy are markers of a process happening elsewhere. They are not seizure onset zones and should never be discussed as though they might be.

And the ophthalmic circulation has collateral supply from the external carotid through the facial and superficial temporal systems, so the analogy with an end-artery cerebral territory is imperfect. This matters most when reasoning about time windows.

3. Retinal Arterial Occlusion

Of the four currents, this is the one where the evidence is least ambiguous and the practice gap widest.

Central retinal artery occlusion is a harbinger of further cerebrovascular and cardiovascular events, and the AHA statement frames it explicitly as an ischaemic stroke requiring the same secondary-prevention rigour as any other.[1] Retinal vascular occlusion carries a considerable burden of concurrent, clinically silent cerebral infarction on diffusion-weighted imaging.[5] That single finding is what converts the event from an ophthalmic misfortune into an index cerebrovascular event with a quantifiable recurrence risk.

Three things follow, and they are not equally well implemented.

The investigative pathway should be identical to that for any anterior-circulation ischaemic stroke: urgent carotid and intracranial vessel imaging, rhythm monitoring of adequate duration rather than a single ECG, and same-day attention to lipids, glucose and blood pressure. Patients who present to eye services frequently receive none of this inside the window where recurrence risk is highest.

Giant cell arteritis is the one cause in which immediate treatment reliably prevents catastrophic loss of the second eye. Inflammatory markers, temporal artery examination and a low threshold for corticosteroid belong in the first hour. This is the item a stroke physician, reasoning from a cerebrovascular differential, is most likely to omit; it is also the item an ophthalmologist is least likely to omit. That asymmetry is an argument for joint assessment rather than for either specialty owning the problem alone.

Whether intravenous thrombolysis helps within a narrow window remains genuinely unsettled, and we do not present it otherwise. The point worth making is procedural rather than therapeutic: the question cannot be asked at all of a patient whose onset time was never recorded, because they were triaged as an eye complaint.

A test for any unit reading this. What happens to a patient with sudden painless monocular visual loss who arrives at three in the morning on a Sunday? If nobody can answer in one sentence, the pathway does not exist, whatever the protocol folder says.

4. What the Retina Records After an Occipital Infarct

Here the direction of travel reverses. The brain lesion comes first and the retina keeps the record.

Damage to the optic radiation or striate cortex deafferents the lateral geniculate nucleus, and degeneration propagates backwards across the geniculate synapse to the retinal ganglion cell. Retrograde trans-synaptic degeneration was disputed for decades. Optical coherence tomography settled it.

The quantitative picture is now reasonably firm. Nerve fibre layer thinning after occipital or optic radiation injury follows a decelerating course when plotted against log elapsed time, steepest early.[6,7] Macular ganglion cell complex thinning has been detected as early as about 5.5 months after occipital stroke and was present in every patient studied at 2.5 years, with annual rates in the region of 2–6 µm.[8] Case series have shown ganglion cell complex thinning mapping precisely onto the homonymous field defect while the peripapillary nerve fibre layer and the disc appearance remained unremarkable.[9,10] That last detail is the practical one: a clinician looking at the disc, or at the peripapillary measurement alone, will miss this.

A 2026 systematic review confirms that the process is evident within the first months and preferentially affects the central macula, consistent with selective vulnerability of parvocellular-projecting ganglion cells.[11]

Three uses follow. The first two are convenient. The third, we think, matters.

Dating an old lesion. In a patient with homonymous hemianopia of uncertain duration, which is a routine problem in late-presenting or cognitively impaired patients, the degree of homonymous macular thinning gives an independent estimate of lesion age. A dense hemianopia with a completely normal ganglion cell layer is, on current evidence, unlikely to be more than a few months old.

Anchoring perimetry. Visual fields are effort-dependent, and early after stroke they are confounded by inattention, drowsiness, neglect and poor fixation. OCT is not. A structural signature that respects the vertical meridian is strong evidence that a perimetric defect is real and retrogeniculate rather than an artefact of participation.

A closing therapeutic window. If ganglion cell loss after occipital infarction is progressive and largely irreversible, then any intervention intended to restore or retrain vision in the affected hemifield depends on surviving retinal afferents, and those afferents are disappearing fastest in exactly the period when most services are still arranging outpatient follow-up.[11] Visual rehabilitation after stroke is conventionally offered late, in the chronic phase, on the reasoning that spontaneous recovery should be allowed to plateau first. The degeneration literature suggests that this reasoning may be precisely backwards. We are not aware of a trial that has tested it. Prediction 2 in Section 8 is that trial.

5. Oculomics

The third current concerns prediction, and it is the one where we part company with much of the published enthusiasm.

The term *oculomics* dates to 2020,[12] but the epidemiology underneath it is older: population cohorts established that retinal microvascular abnormalities are associated with incident stroke independently of conventional risk factors, and that vessel calibre carries prognostic signal.[13,14]

Deep learning changed the scale of what can be extracted. Convolutional networks infer age, sex, smoking status and blood pressure directly from fundus photographs.[15]

Retinal vessel calibre can be estimated automatically in a way that retains independent cardiovascular prognostic information.[16] In UK Biobank, a *retinal age gap* — image-predicted age minus chronological age — is associated with incident stroke.[17] Systems built on a broad panel of retinal biomarkers report areas under the curve around 0.83 in UK Biobank, with the additional and genuinely interesting capacity to estimate when the event is likely to occur.[18] Foundation models trained on very large unlabelled retinal datasets have improved generalisability across populations and devices.[19]

Now the cautions, which reviews in this space tend to compress into a final sentence.

Discrimination is not utility. An area under the curve of 0.83 in a research cohort says nothing about whether acting on the number changes an outcome. Almost none of this literature reports net reclassification against an established clinical score. Almost none reports decision-curve analysis. The question that matters is not whether a network can predict stroke from a photograph. It is whether it predicts stroke better than the information already sitting in the notes, in a patient for whom the answer would alter management.

Cohorts are not populations. UK Biobank participants are healthier, wealthier, whiter and older at recruitment than the populations carrying most of the world's stroke burden. Performance in South Asian, African and low- and middle-income settings, where stroke arrives a decade earlier and small-vessel phenotypes differ, is largely unestablished. Given that the principal promised advantage of retinal screening is deployability where CT and MRI are scarce, this is not a generalisability footnote. It is the central external-validity question, and it is routinely relegated to a limitations paragraph.

Explainability is not optional either. A model that predicts stroke without identifying which features carry the signal cannot be integrated with mechanism, cannot be audited for shortcut learning on camera model or demographic artefact, and cannot be defended to a regulator or to a patient who asks why. For clinical science, a system built on named measurable biomarkers — calibre, tortuosity, fractal dimension, arteriovenous ratio — is more useful than an end-to-end black box with marginally better discrimination.

6. Epilepsy, Where the Evidence Thins

The routine epileptological use of retinal imaging is drug surveillance, and it works.

Vigabatrin causes permanent concentric field constriction in a dose- and duration-dependent way. OCT-quantified peripapillary thinning tracks the field loss closely, with characteristic temporal sparing and early nasal and superior involvement.[20,21] Full-field electroretinography shows toxicity operating at several retinal levels, from photoreceptor to ganglion cell, and electrophysiological change can precede both structural thinning and perimetric loss.[22,23] For young children and adults with intellectual disability, in whom perimetry is often simply impossible, an objective effort-independent structural measure has been a genuine advance.

Beyond that, the evidence gets thin quickly.

Nerve fibre layer thinning has been reported in drug-resistant epilepsy independent of vigabatrin exposure,[24] which fits the broader picture of chronic epilepsy as a network disorder with widespread white-matter involvement rather than a purely focal one. But the studies are small, cross-sectional, incompletely replicated, and confounded by cumulative medication exposure, seizure-related trauma, comorbid vascular disease and the age structure of drug-resistant cohorts. We present this as a question worth asking, not as a finding.

Where the four currents meet is post-stroke epilepsy. Take a patient with an occipital or posterior-temporal infarct. That single patient carries an elevated risk of late unprovoked seizures; a homonymous field defect; progressive retrograde degeneration of the ganglion cells serving the affected hemifield; and, should epilepsy develop, probable exposure to antiseizure medication for years. All four are measurable on one scan. None of them is currently measured together.

The interaction is not academic. A patient with hemianopia who also needs antiseizure medication faces a compounded and poorly characterised threat to what vision remains, and to driving eligibility. We deal with that in the companion review.

7. Why Nothing Has Been Integrated

Four reasons, in ascending order of how easily they could be fixed.

Workflow. OCT lives in ophthalmology. Acute stroke lives in the emergency department and the stroke unit. Epilepsy lives in outpatient neurology. The scan is acquired in a building the stroke physician does not enter and stored on an archive the neurologist often cannot access.

Reporting convention. Commercial platforms report against glaucoma-oriented normative data and display sector maps designed for optic neuropathy. The homonymous, vertical-meridian-respecting pattern that signals retrograde degeneration is not something those displays make obvious. It has to be looked for on purpose, by someone who knows it exists.

No shared dataset. There is no agreed minimum set of retinal variables that a stroke or epilepsy study should record. Studies therefore report incommensurable subsets — peripapillary thickness here, macular sectors there, fundus-derived vascular indices somewhere else — and cannot be pooled even when they address the same question. This is a measurement-standardisation failure, and it has the same remedy as every other measurement-standardisation failure.

No owner. Ophthalmology owns the acquisition. Neurology owns the interpretation. Stroke medicine owns the consequences. Nobody owns the join.

The first three could be substantially addressed by a page of agreed definitions. The fourth requires somebody to decide it is their problem.

8. A Minimum Retinal Dataset

Table 2 sets out ten items. The design constraints were deliberate and restrictive: everything must come from one standard OCT acquisition plus a colour fundus photograph, on commonly available commercial equipment, inside a single clinic visit, without dilation in most patients, and at no cost beyond the scan already being done. Nothing requires OCT angiography, adaptive optics or a research-grade device. A standard that presumes equipment most units do not own is a standard nobody adopts.

Table 2. Minimum Retinal Dataset (MRD-10).

#	Item	Specification	Why
1	Device and software	Manufacturer, model, software build, scan protocol	Thickness values are not interchangeable between devices; pooling without this is invalid
2	Timing	Days from index stroke or seizure to scan	Degeneration is time-dependent; an undated scan cannot be interpreted
3	Peripapillary RNFL	Global and six-sector, both eyes, µm	Standard axonal measure; sensitive to vigabatrin and to chronic degeneration
4	Macular GCL–IPL	ETDRS sector thickness, both eyes, µm	Earliest and most specific signal; often abnormal when RNFL is normal
5	Hemiretinal asymmetry	Nasal minus temporal hemimacular difference, each eye, referenced to the vertical meridian	Turns item 4 into a directional test of retrogeniculate origin

#	Item	Specification	Why
6	Image quality	Manufacturer signal strength; exclusion threshold stated in advance	Media opacity is common at stroke age; unreported quality invalidates comparison
7	Segmentation audit	Proportion of B-scans needing manual correction, and whether corrected	The commonest silent error in the field
8	Vascular indices	Arteriolar and venular equivalents, AV ratio; tortuosity and fractal dimension where software allows	Small-vessel proxy; the oculomic signal
9	Ocular comorbidity	Glaucoma, diabetic retinopathy, AMD, prior surgery, refraction, axial length where available	The dominant confounders of every measure above, and routinely omitted
10	Concurrent perimetry	Strategy, reliability indices, mean deviation — or a statement that fields were not done, and why	Structure without function cannot be interpreted; unreliable fields must be declared, not silently included

Two items will be the first to be dropped, and both should be defended.

Item 7 is the least interesting and the most important. Automated retinal layer segmentation fails more often than its users assume, particularly with epiretinal membrane, media opacity or poor fixation — which is to say, in the eyes of older stroke patients. An uncorrected segmentation error of a few micrometres is indistinguishable in the output from a real biological signal of the size reported throughout the degeneration literature. A study that does not report its segmentation audit has not measured what it says it measured.

Item 9 will be resisted on grounds of effort. It should not be. Glaucoma and diabetic retinopathy are common in stroke populations, both produce exactly the ganglion cell and nerve fibre thinning that degeneration studies attribute to the infarct, and both share the vascular risk factors that caused the stroke in the first place. Confounding here is not a theoretical concern. It is the default condition of an unadjusted analysis.

MRD-10 is not a guideline and does not tell anyone what to do with the numbers. It is a shared vocabulary, without which the four literatures above cannot be combined even in principle.

9. Four Predictions, and a Disagreement

Prediction 1 — Structure gates rehabilitation response. In patients with homonymous hemianopia after occipital or optic radiation infarction, baseline macular GCL–IPL thickness in the affected hemiretinae, measured within six weeks, will predict response to visual restitution or compensatory training, independently of lesion volume, age and time since stroke. *Refuted by:* no association between baseline hemiretinal thickness and change in perimetric mean deviation or reading speed at six months. Roughly 120 participants would give reasonable power for a moderate association, allowing for 20% attrition and 10% exclusion on image quality.

Prediction 2 — Early beats late. Visual training begun within eight weeks of occipital infarction will produce greater functional gain than identical training begun after six months. *Refuted by:* no difference between arms. A randomised delayed-start design of about 200 participants. This is the expensive one and the consequential one: current service configuration assumes the opposite and no adequately powered trial has tested it. The delayed arm must receive standard care throughout and must not be denied anything of established benefit.

Prediction 3 — Retinal small-vessel phenotype predicts post-stroke epilepsy. Among survivors of cortical ischaemic stroke, reduced retinal fractal dimension and increased arteriolar tortuosity at baseline will be associated with incident post-stroke epilepsy over five years, independently of established clinical predictors. *Refuted by:* no independent association after adjustment, or no gain in discrimination when added to an existing model. This should be tested by nested analysis within a cohort that already has baseline retinal imaging, and success should be pre-specified as a meaningful C-statistic gain rather than a significant coefficient.

Prediction 4 — Incremental value over clinical scores. Retinal deep-learning stroke risk scores will produce clinically meaningful net reclassification over an established clinical score, in a population cohort not used for development. *Refuted by:* no net reclassification improvement, or improvement confined to a stratum where management would not change.

A disagreement we have not resolved. AD regards Prediction 3 as the most likely of the four to fail and included it chiefly because stating a prediction one expects to lose is a discipline the field needs. VK disagrees, and thinks the case is stronger than that: cortical small-vessel burden is associated with post-stroke epilepsy through several plausible routes, including chronic blood–brain barrier compromise, and a retinal proxy for that burden has a reasonable mechanistic claim rather than merely a statistical one. We have left both positions in the text. Neither of us can settle it without the study, and we would rather a reader saw the disagreement than a manufactured consensus.

10. Limitations

This is a narrative review with all the weaknesses of the form. No systematic search, no pre-specified eligibility criteria, no formal risk-of-bias assessment; the selection of evidence reflects our reading and our priors.

The four literatures we have drawn on are not of equal maturity, and readers should weight our claims unevenly rather than uniformly. Section 3 rests on an AHA scientific statement. Section 6 rests on a handful of small cross-sectional studies. Treating those as equivalent because they appear in the same review would be a misreading we have tried to make difficult.

MRD-10 has not been piloted, and its feasibility in a hyperacute setting is asserted rather than shown. Item 10 may well prove impractical in the first days after stroke.

Our sample size figures are indicative, based on assumed effect magnitudes, and are not completed formal power calculations. Any of these studies would need pre-registration with a specified analysis plan before those numbers were used for anything.

Finally, the authors are a stroke physician, an ophthalmologist, a general medical trainee and an epilepsy fellow. The breadth is deliberate. It also guarantees that a specialist in any one of the four areas will find our treatment of theirs less complete than they would like.

11. Conclusion

The claim here is not that retinal imaging is underused. It is being used constantly, at enormous scale, on precisely the population in which stroke and post-stroke epilepsy occur. The neurological content of those images is being discarded at the point of reporting.

Acute retinal arterial occlusion is a stroke. Occipital infarction leaves a durable, datable, quantifiable signature in the macula. Retinal microvascular architecture carries independent information about future cerebrovascular events, though how much of it is incremental to what we already know remains unproven. Chronic epilepsy and its treatment write themselves onto the same tissue, faintly, in ways we do not yet read well. Each of these is separately established or separately plausible. None is routinely acted upon alongside the others.

What is missing is not an instrument or a discovery. It is a shared measurement vocabulary and a decision about whose job this is. We have offered the first as MRD-10 and set out four ways of showing us wrong. The second is not something a review article can settle.

Put at its narrowest: a patient with an occipital infarct will probably have an OCT scan in the next five years for some unrelated ophthalmic reason. On that scan will be a record of their stroke, an estimate of its age, a structural correlate of their disability and possibly a marker of their epilepsy risk. It will be read for glaucoma and filed. Nothing prevents it from being read for both.

Declarations

Author Contributions

CRediT taxonomy. **AD:** Conceptualisation; methodology; writing — original draft; writing — review and editing; supervision. **SK:** Conceptualisation; investigation (ophthalmic imaging content); writing — original draft (Sections 2 and 8); writing — review and editing; validation. **GG:** Investigation; data curation (literature identification and screening); writing — original draft (Section 5); writing — review and editing. **VK:** Conceptualisation; investigation (epileptological content); writing — original draft (Section 6); writing — review and editing; validation. All authors read and approved the final version and agree to be accountable for all aspects of the work. The disagreement recorded in Section 9 is stated with the consent of both parties.

Ethics Statement

This is a review article. It does not report primary research involving human participants, human material, human data or animals. No ethical approval and no patient consent were required, and no identifiable patient information appears. The studies proposed in Section 9 would each require prospective ethical approval in the jurisdiction where undertaken; the safeguards we consider necessary for the delayed-start design in Prediction 2 are stated there.

Consent for Publication

Not applicable.

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Conflict of Interest Statement

There was no conflict of interest.

Data Availability Statement

No new primary datasets were generated or analysed. All evidence discussed is available in the cited literature. MRD-10 (Table 2) is released without restriction to support independent adoption and evaluation.

Use of Artificial Intelligence

There was NO use of AI in preparing any portion of this paper.

Abbreviations

AHA, American Heart Association; AMD, age-related macular degeneration; AV, arteriovenous; CRAO, central retinal artery occlusion; ETDRS, Early Treatment Diabetic Retinopathy Study; GCL–IPL, ganglion cell layer–inner plexiform layer; MCA, middle cerebral artery; MRD-10, Minimum Retinal Dataset; OCT, optical coherence tomography; PCA, posterior cerebral artery; RNFL, retinal nerve fibre layer.

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