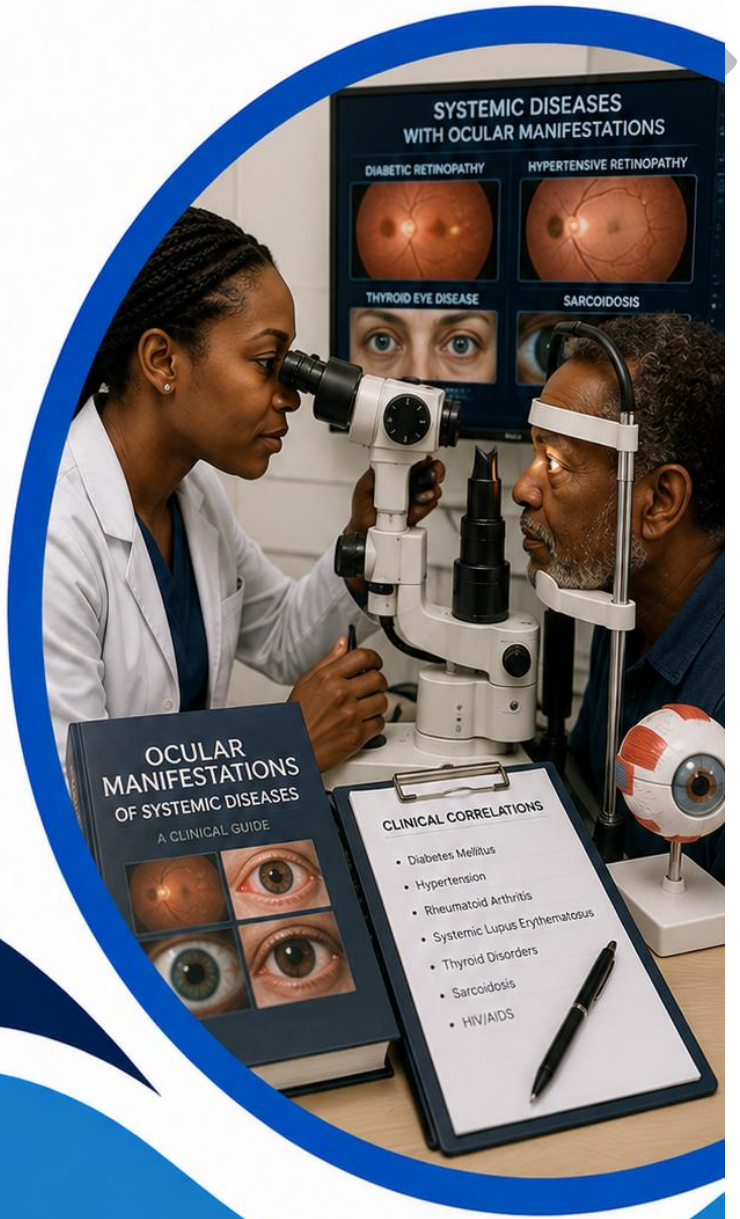
La-Net

OPT 611

OCULAR MANIFESTATIONS OF SYSTEMIC DISEASES



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Course code: OPT 611

Course title: Ocular Manifestations of Systemic Diseases

Course credit load: 1 Unit

Series 1: Retinal Anatomy, Vitreoretinal Symptoms, and Dilated Fundus Examination

- **Part 1.1: Anatomy and Function of the Retina and Macula**
  - Sub Part 1.1.1: Layers of the retina
  - Sub Part 1.1.2: The macula and fovea
  - Sub Part 1.1.3: Retinal vascular supply
- **Part 1.2: Symptoms Suggestive of Vitreoretinal Disorders**
  - Sub Part 1.2.1: Floaters and flashes
  - Sub Part 1.2.2: Field loss and distortion
  - Sub Part 1.2.3: Sudden versus gradual visual change
- **Part 1.3: Dilated Fundus Examination and the Normal Fundus**
  - Sub Part 1.3.1: Preparing for dilated fundus examination
  - Sub Part 1.3.2: Systematic examination technique
  - Sub Part 1.3.3: Normal fundus appearance

Introduction

The retina sits at a genuinely unique crossroads in medicine: it is the only place in the human body where blood vessels can be directly, non-invasively examined, offering a window onto systemic vascular and metabolic disease that no other single examination can match. **OPT 611: Ocular Manifestations of Systemic Diseases** builds on this idea throughout, and this opening Series lays the essential groundwork, the **anatomy and function** of the retina and macula, the **symptoms** that should prompt suspicion of vitreoretinal disease, and the practical skill of **dilated fundus examination** itself.

The aim of this Series is to equip you with a thorough understanding of the anatomy and functions of the retina and macula, symptoms suggestive of vitreoretinal disorders, and recognition of the normal fundus with the direct ophthalmoscope.

This Series begins with **anatomy and function of the retina and macula**, before turning to **symptoms suggestive of vitreoretinal disorders**. It concludes with **dilated fundus examination and the normal fundus**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 1.1:** describe the layers of the retina
- **SLO 1.2:** describe the macula and fovea and the retinal vascular supply
- **SLO 1.3:** list symptoms of floaters and flashes suggestive of vitreoretinal disorders
- **SLO 1.4:** describe field loss and distortion as vitreoretinal symptoms
- **SLO 1.5:** distinguish sudden from gradual visual change, and
- **SLO 1.6:** prepare for and perform dilated fundus examination and recognise the normal fundus.

1.1 Anatomy and Function of the Retina and Macula

1.1.1 layers of the retina

A layered neural tissue converting light into signal

The retina comprises ten distinct histological layers, from the outer **retinal pigment epithelium**, a single layer supporting photoreceptor metabolism and forming part of the blood-retinal barrier, through the **photoreceptor layer**, containing the rod and cone cells responsible for phototransduction, to the inner **nerve fibre layer**, where retinal ganglion cell axons converge toward the optic disc.

Between these outer and inner extremes lie the bipolar, horizontal, and amacrine cell layers, together forming an intricate processing network that begins shaping the visual signal well before it ever reaches the brain, meaning meaningful visual information processing genuinely begins within the retina itself rather than starting only at the visual cortex.

Fill in the blank: The retina comprises ten distinct histological layers, from the outer retinal pigment epithelium through the photoreceptor layer to the inner nerve fibre _____.

1.1.2 the macula and fovea

The specialised region responsible for sharp central vision

The **macula** is the specialised central retinal region, roughly 5.5 millimetres in diameter, responsible for high-resolution, detailed central vision and colour discrimination, with a yellowish pigmentation, from macular xanthophyll, that gives the region its name and its characteristic slightly darker fundoscopic appearance.

At the very centre of the macula lies the **fovea**, the point of thinnest retina and densest cone photoreceptor concentration, producing the sharpest visual acuity available anywhere in the visual field, with the foveal avascular zone, a small area free of retinal blood vessels, further optimising this central region for maximal optical clarity.

Fill in the blank: At the very centre of the macula lies the fovea, the point of thinnest retina and densest cone photoreceptor _____.

1.1.3 retinal vascular supply

A dual system supplying inner and outer retinal layers

The retina receives its blood supply from two genuinely distinct sources, the **central retinal artery**, a branch of the ophthalmic artery, supplying the inner retinal layers through its four major branch arterioles, and the **choroid**, a highly vascular layer immediately external to the retina, supplying the outer retina and photoreceptors by diffusion.

This dual supply arrangement means retinal disease affecting the inner and outer layers can present quite differently depending on which vascular system is primarily involved, a principle that underpins much of the fundus interpretation examined throughout the remainder of this course.

Fill in the blank: The retina receives its blood supply from two genuinely distinct sources, the central retinal artery supplying the inner layers and the choroid supplying the outer retina by _____.

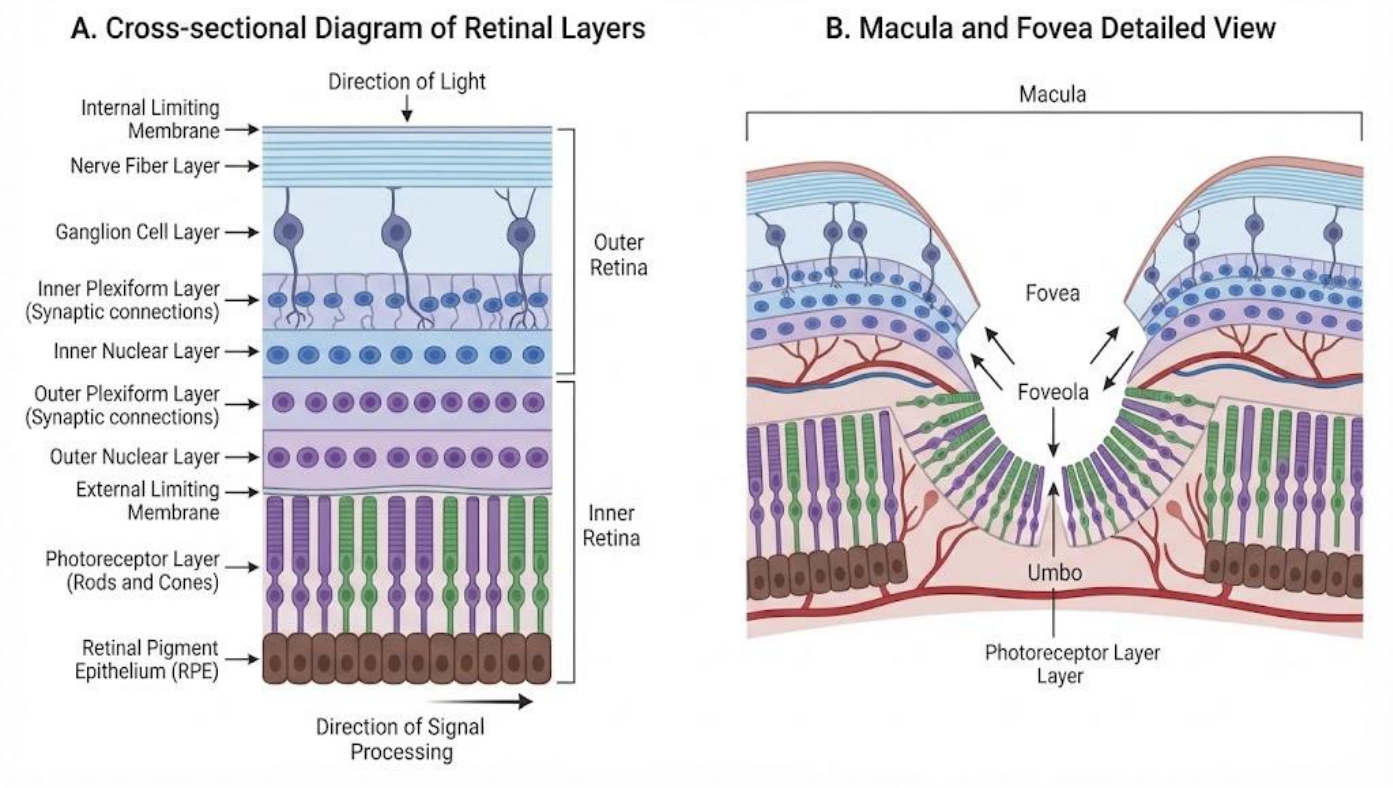

Figure 1.1: A cross-sectional diagram of the retina showing its ten layers alongside the macula and its central fovea.

Pilot questions 1.1

1. List the outer and inner retinal layers. (Linked to SLO 1.1)
2. Describe the macula and its characteristic pigmentation. (Linked to SLO 1.2)
3. Define the fovea and explain why it produces the sharpest visual acuity. (Linked to SLO 1.2)
4. Describe the dual blood supply of the retina. (Linked to SLO 1.2)
5. Explain the functional significance of the foveal avascular zone. (Linked to SLO 1.2)

1.2 Symptoms Suggestive of Vitreoretinal Disorders

1.2.1 floaters and flashes

Common symptoms with a genuinely wide range of significance

Floaters, small, mobile shadows perceived within the visual field, generally result from condensed collagen fibrils or cellular debris casting shadows on the retina as they drift within the

vitreous, most commonly reflecting benign, age-related vitreous liquefaction but occasionally signalling a genuinely more serious posterior vitreous detachment or retinal tear.

Flashes, brief flickering or lightning-like streaks of light, generally result from mechanical traction on the retina where the vitreous remains attached, with a sudden increase in either symptom, particularly when occurring together, warranting prompt ophthalmic assessment given the recognised risk of underlying retinal tear or detachment.

Fill in the blank: Flashes generally result from mechanical traction on the retina where the vitreous remains attached, with a sudden increase in either flashes or floaters warranting prompt _____ assessment.

1.2.2 field loss and distortion

Symptoms directly reflecting the site of retinal involvement

A **curtain-like** or shadow-like field defect, progressing across a portion of the visual field, represents a particularly concerning symptom suggestive of established retinal detachment, since it reflects the physical extent of retina that has already separated from its underlying supporting tissue.

Metamorphopsia, perceived distortion or warping of straight lines, together with **micropsia** or **macropsia**, perceived shrinking or enlargement of objects, generally indicates macular involvement, reflecting the disproportionate visual significance of even relatively small structural change within this specific, highly specialised central region.

Fill in the blank: Metamorphopsia, perceived distortion of straight lines, together with micropsia or macropsia, generally indicates involvement of the _____.

1.2.3 sudden versus gradual visual change

Onset speed as a useful initial diagnostic discriminator

Sudden, abrupt visual loss or field change generally reflects an acute vascular or structural event, such as retinal artery or vein occlusion, vitreous haemorrhage, or acute retinal detachment, each demanding urgent evaluation given the genuine potential for rapid, permanent visual consequence.

Progressive, gradual visual decline, by contrast, more typically reflects a slower underlying process, such as diabetic retinopathy or age-related macular degeneration, though this distinction should never be treated as absolute, since some genuinely serious conditions can also present with a comparatively gradual onset.

Fill in the blank: Sudden, abrupt visual loss or field change generally reflects an acute vascular or structural event, demanding urgent evaluation given the genuine potential for rapid, permanent visual _____.

Table 1.2: Relating vitreoretinal symptoms to underlying significance.

Symptom	Description	Level of concern
Occasional stable floaters	Long-standing, unchanging perception	Generally low
New flashes with a shower of floaters	Sudden onset, particularly together	High; assess urgently for tear or detachment
Curtain-like field defect	Progressive shadow across the field	High; suggests established detachment

Pilot questions 1.2

1. Describe floaters and their possible causes. (Linked to SLO 1.3)
2. Describe flashes and explain their underlying mechanism. (Linked to SLO 1.3)
3. Describe the significance of a curtain-like field defect. (Linked to SLO 1.4)
4. Define metamorphopsia, micropsia, and macropsia. (Linked to SLO 1.4)
5. Explain why sudden visual change generally demands more urgent evaluation than gradual change. (Linked to SLO 1.5)

1.3 Dilated Fundus Examination and the Normal Fundus

1.3.1 preparing for dilated fundus examination

Pupillary dilation for genuinely thorough peripheral views

Dilated fundus examination requires pharmacological pupillary dilation, generally using a combination of a mydriatic and cycloplegic agent, allowing a considerably wider view of the fundus than could ever be achieved through an undilated pupil, particularly important for adequately assessing the peripheral retina.

Patients should be counselled beforehand regarding the expected temporary blurring of near vision and light sensitivity, and, where relevant, the small recognised risk of precipitating acute angle-closure in a patient with pre-existing narrow anterior chamber angles, warranting a brief risk assessment before dilation is undertaken.

Fill in the blank: Patients should be counselled beforehand regarding the expected temporary blurring of near vision and light sensitivity following pupillary _____.

1.3.2 systematic examination technique

A consistent sequence supporting comprehensive assessment

A systematic examination sequence should identify the **optic disc** first, generally the easiest structure to locate, before following the major vessels outward from the disc to examine each retinal quadrant in turn, finally examining the macula directly, given its sensitivity to bright illumination.

Consistent application of this same sequence, regardless of the specific clinical indication for examination, reduces the genuine risk of inadvertently overlooking a significant finding, an especially important discipline given how much clinically relevant detail the fundus can contain.

Fill in the blank: A systematic examination sequence should identify the optic disc first, before following the major vessels outward from the disc to examine each retinal quadrant in _____.

1.3.3 normal fundus appearance

The essential baseline against which pathology is compared

The normal optic disc appears well defined, pink-orange, with a small central physiological cup, while normal retinal vessels demonstrate a smooth, gradually tapering course, arterioles narrower and brighter red than the correspondingly wider, darker venules, in an approximate two-to-three diameter ratio.

The background retina should appear a relatively uniform orange-red, with the macula appearing slightly darker and relatively avascular, and no visible haemorrhage, exudate, or abnormal vessel formation, this normal baseline forming the essential comparison point against which the systemic disease fundus signs examined throughout the remainder of this course are recognised.

Fill in the blank: The background retina should appear a relatively uniform orange-red, with the macula appearing slightly darker and relatively _____.

Table 1.3: Normal fundus appearance by structure.

Structure	Normal appearance	Comparison point
Optic disc	Pink-orange, well defined, small central cup	Baseline for disc oedema or pallor
Retinal vessels	Arterioles narrower and brighter than venules, ratio ~2:3	Baseline for arteriolar narrowing or vein occlusion
Background retina	Uniform orange-red, no haemorrhage or exudate	Baseline for haemorrhage, exudate, or new vessel formation

Pilot questions 1.3

1. Describe how a patient should be prepared for dilated fundus examination. (Linked to SLO 1.6)
2. State a recognised risk of pharmacological pupillary dilation. (Linked to SLO 1.6)
3. Describe a systematic sequence for fundus examination. (Linked to SLO 1.6)
4. Describe the normal appearance of the optic disc and retinal vessels. (Linked to SLO 1.6)
5. Describe the normal appearance of the background retina and macula. (Linked to SLO 1.6)

Series Summary

This opening Series covered the retina and macula's anatomy and function, its layered structure and dual vascular supply, the symptoms, floaters, flashes, field loss, and distortion, that should raise suspicion of vitreoretinal disease, and the practical skill of dilated fundus examination itself, from patient preparation through systematic technique to recognition of the normal fundus baseline.

This normal anatomical and examination foundation is essential preparation for the fundus signs of systemic vascular disease, hypertension and diabetes, examined in the next Series of this course.

Glossary of Terms

- **Retinal pigment epithelium:** the outer retinal layer supporting photoreceptor function and forming part of the blood-retinal barrier.
- **Macula:** the specialised central retinal region responsible for detailed, high-acuity vision.
- **Fovea:** the very centre of the macula, with the densest cone concentration and sharpest acuity.
- **Foveal avascular zone:** a small area at the fovea free of retinal blood vessels.
- **Choroid:** the highly vascular layer external to the retina, supplying the outer retina by diffusion.
- **Floaters:** small mobile shadows perceived in the visual field from vitreous debris or condensation.
- **Flashes:** brief flickering light perceptions from mechanical vitreoretinal traction.

- **Metamorphopsia:** perceived distortion or warping of straight lines, suggesting macular involvement.
- **Curtain-like field defect:** a progressive shadow across the visual field suggestive of retinal detachment.
- **Dilated fundus examination:** examination of the retina following pharmacological pupillary dilation.
- **Mydriatic:** a drug used to dilate the pupil for fundus examination.
- **Physiological cup:** the normal central depression within the optic disc.

Concept Check Questions [Series 1]

Objective questions

1. The retina comprises ten distinct histological layers, from the outer retinal pigment epithelium through the photoreceptor layer to the inner nerve fibre _____.
2. The retina receives its blood supply from the central retinal artery and the choroid, which supplies the outer retina by _____.
3. A sudden increase in flashes or floaters, particularly when occurring together, warrants prompt _____ assessment.
4. Metamorphopsia, perceived distortion of straight lines, generally indicates involvement of the _____.
5. The background retina should appear a relatively uniform orange-red, with the macula appearing slightly darker and relatively _____.

Short-answer questions

6. Define the fovea and explain why it produces the sharpest visual acuity.
7. Describe the dual blood supply of the retina.
8. Describe the significance of a curtain-like field defect.
9. Distinguish sudden from gradual visual change in terms of likely underlying cause.
10. Describe a systematic sequence for fundus examination.

Long-answer questions

11. Discuss the layers of the retina and the anatomy of the macula and fovea. (Linked to SLO 1.1, SLO 1.2)
12. Discuss the retinal vascular supply. (Linked to SLO 1.2)

13. Discuss floaters, flashes, field loss, and distortion as symptoms of vitreoretinal disorders. (Linked to SLO 1.3, SLO 1.4)
14. Discuss sudden versus gradual visual change. (Linked to SLO 1.5)
15. Discuss preparation for, technique of, and normal findings on dilated fundus examination. (Linked to SLO 1.6)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Layer.
2. Diffusion.
3. Ophthalmic.
4. Macula.
5. Avascular.

Short-answer questions

6. The very centre of the macula; it has the densest cone concentration, producing the sharpest visual acuity.
7. The central retinal artery supplies the inner retinal layers; the choroid supplies the outer retina by diffusion.
8. It reflects the physical extent of retina already separated from underlying tissue, suggesting established retinal detachment.
9. Sudden change generally reflects an acute vascular or structural event; gradual change more typically reflects a slower degenerative or metabolic process.
10. Identify the disc first, follow vessels outward through each quadrant, then examine the macula last.

Long-answer questions

11. **Basic response:** The retina's ten layers span from the outer pigment epithelium through the photoreceptors to the inner nerve fibre layer, with the macula representing the specialised central region for detailed vision.

Value-added response: The fovea, at the macula's centre, achieves the sharpest acuity through its dense cone concentration and avascular zone.

12. **Basic response:** The retina is supplied by both the central retinal artery, feeding the inner layers, and the choroid, feeding the outer retina by diffusion.

Value-added response: This dual supply explains why retinal disease can present quite differently depending on which vascular system is primarily affected.

13. **Basic response:** Floaters and flashes reflect vitreous change or traction, generally benign but occasionally signalling retinal tear or detachment.

Value-added response: Field loss and distortion, including curtain-like defects and metamorphopsia, carry specific localising value toward peripheral or macular involvement respectively.

14. **Basic response:** Sudden visual change generally reflects acute vascular or structural events demanding urgent evaluation.

Value-added response: Gradual change more typically reflects slower processes, though this distinction should never be treated as absolute.

15. **Basic response:** Dilated examination requires pharmacological pupillary dilation with appropriate patient counselling, followed by a systematic disc-vessels-macula sequence.

Value-added response: Recognising the normal appearance of each structure provides the essential baseline against which systemic disease fundus signs are later compared.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Outer: retinal pigment epithelium and photoreceptor layer; inner: nerve fibre layer, among others such as bipolar and ganglion cell layers.
2. The specialised central retinal region for detailed vision, with a yellowish pigmentation from macular xanthophyll.
3. The very centre of the macula, with the densest cone concentration; it produces the sharpest acuity of anywhere in the visual field.
4. The central retinal artery supplies the inner layers; the choroid supplies the outer retina and photoreceptors by diffusion.
5. It optimises central optical clarity by keeping vessels away from the point of sharpest vision.

Part 1.2

1. Small mobile shadows from vitreous debris or condensation, generally benign but occasionally reflecting detachment or tear.
2. Brief flickering light from mechanical traction on the retina where the vitreous remains attached.
3. It reflects the extent of already-detached retina, suggesting an established retinal detachment.
4. Metamorphopsia is distortion of straight lines; micropsia and macropsia are perceived shrinking or enlargement of objects.
5. Because it more typically reflects an acute vascular or structural event with genuine potential for rapid, permanent consequence.

Part 1.3

1. Counselling regarding temporary near blurring and light sensitivity following pharmacological pupillary dilation.
2. Precipitating acute angle-closure in a patient with pre-existing narrow anterior chamber angles.
3. Identify the disc first, follow vessels outward through each quadrant, then examine the macula last.
4. The disc is pink-orange with a small central cup; vessels show arterioles narrower and brighter than venules, ratio approximately 2:3.
5. A relatively uniform orange-red, with the macula slightly darker and relatively avascular.

References and Attributions [Series 1]

- Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
- Riordan-Eva, P., & Augsburger, J. J. (Eds.). (2017). Vaughan and Asbury's General Ophthalmology (19th ed.). McGraw-Hill.
- American Academy of Ophthalmology (2021). Retina and Vitreous, Basic and Clinical Science Course Section 12.
- Khurana, A. K. (2019). Comprehensive Ophthalmology (7th ed.). Jaypee Brothers Medical Publishers.

Figures, Plates, Tables, and Boxes

- Figure 1.1: A cross-sectional diagram of the retina showing its ten layers alongside the macula and its central fovea. AI-generated using the prompt: "A single clean, accurate

cross-sectional anatomical diagram of the retina with labelled horizontal layers from retinal pigment epithelium to nerve fibre layer, alongside a second labelled panel showing the macula with a central foveal depression, textbook histology diagram style, neutral background, no photographic realism, no other panels."

- Table 1.2: Relating vitreoretinal symptoms to underlying significance. AI-generated using the prompt: "Create a clean clinical reference table titled Relating Vitreoretinal Symptoms to Underlying Significance, listing symptom, description, and level of concern. Ophthalmology reference table style with outside border, no photographic elements."
- Table 1.3: Normal fundus appearance by structure. AI-generated using the prompt: "Create a clean clinical reference table titled Normal Fundus Appearance by Structure, listing structure, normal appearance, and comparison point. Ophthalmology reference table style with outside border, no photographic elements."

Course code: OPT 611

Course title: Ocular Manifestations of Systemic Diseases

Course credit load: 1 Unit

Series 2: Fundus Signs of Systemic Vascular Disease: Hypertension and Diabetes

- **Part 2.1: Hypertensive Retinopathy**
  - Sub Part 2.1.1: Arteriolar narrowing and arteriovenous nicking
  - Sub Part 2.1.2: Copper and silver wiring
  - Sub Part 2.1.3: Haemorrhages and exudates in hypertensive retinopathy
- **Part 2.2: Diabetic Retinopathy**
  - Sub Part 2.2.1: Microaneurysms and dot-blot haemorrhages
  - Sub Part 2.2.2: Hard exudates and cotton wool spots
  - Sub Part 2.2.3: Proliferative diabetic retinopathy
- **Part 2.3: Disc Oedema and Integrating Fundus Findings**
  - Sub Part 2.3.1: Recognising disc oedema
  - Sub Part 2.3.2: Causes of disc oedema relevant to systemic disease
  - Sub Part 2.3.3: Integrating fundus findings in systemic disease assessment

Introduction

Hypertension and diabetes are, between them, responsible for a substantial proportion of every fundus abnormality encountered in general clinical practice, and each produces a genuinely characteristic, recognisable pattern of retinal change well before either condition has necessarily declared itself through any other clinical sign. This Series takes the normal fundus baseline established previously and applies it directly, working through the specific vascular, haemorrhagic, and exudative changes of **hypertensive** and **diabetic retinopathy**, before turning to **disc oedema**, a finding relevant across both conditions and several others besides.

The aim of this Series is to equip you with a thorough understanding of recognising retinal arteriolar narrowing, copper and silver wiring, disc oedema, and retinal exudates and haemorrhages on dilated fundoscopy.

This Series begins with **hypertensive retinopathy**, before turning to **diabetic retinopathy**. It concludes with **disc oedema and integrating fundus findings**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 2.1:** recognise arteriolar narrowing and arteriovenous nicking in hypertensive retinopathy
- **SLO 2.2:** recognise copper and silver wiring and haemorrhages and exudates in hypertensive retinopathy
- **SLO 2.3:** recognise microaneurysms, dot-blot haemorrhages, and hard exudates in diabetic retinopathy
- **SLO 2.4:** recognise cotton wool spots and features of proliferative diabetic retinopathy
- **SLO 2.5:** detect disc oedema on dilated fundoscopy, and
- **SLO 2.6:** describe causes of disc oedema relevant to systemic disease and integrate fundus findings in overall assessment.

2.1 Hypertensive Retinopathy

2.1.1 arteriolar narrowing and arteriovenous nicking

Early, subtle vascular changes from chronic pressure elevation

Chronic hypertension produces generalised or focal **arteriolar narrowing**, a reduction in the normal arteriole-to-venule diameter ratio, generally the earliest recognisable fundus sign of hypertensive vascular change and, for that reason, one of the more subtle and easily overlooked findings on routine examination.

Arteriovenous nicking, focal compression or displacement of a venule at the specific point where an arteriole crosses over it, reflects thickening of the arteriolar wall compressing the shared adventitial sheath, and represents a further useful, moderately specific sign of chronic hypertensive change.

Fill in the blank: Arteriovenous nicking reflects thickening of the arteriolar wall compressing the shared adventitial sheath at the point where an arteriole crosses a _____.

2.1.2 copper and silver wiring

Vessel wall thickening progressively obscuring the blood column

Copper wiring describes a broadened, coppery-orange central light reflex along the arteriole, resulting from increasing arteriolar wall thickening that partially obscures the underlying blood column, generally representing a moderately advanced stage of hypertensive vascular change.

Silver wiring, a further stage beyond copper wiring, describes an even more pronounced, silvery-white light reflex, reflecting such severe arteriolar wall thickening that the blood column is almost entirely obscured, generally indicating genuinely long-standing, severe hypertensive vascular disease.

Fill in the blank: Silver wiring describes a pronounced, silvery-white light reflex, reflecting such severe arteriolar wall thickening that the blood column is almost entirely _____.

2.1.3 haemorrhages and exudates in hypertensive retinopathy

Findings marking a more severe, acute-on-chronic stage

Flame-shaped haemorrhages, reflecting bleeding within the superficial nerve fibre layer, and **cotton wool spots**, representing localised retinal nerve fibre layer infarction, generally indicate more severe hypertensive retinopathy, particularly relevant in the context of acutely, significantly elevated blood pressure.

In the most severe cases, **malignant hypertension** produces disc oedema, discussed further later in this Series, together with a macular star, a stellate pattern of hard exudate deposition around the fovea, reflecting genuinely dangerous, acutely elevated blood pressure requiring urgent systemic management.

Fill in the blank: In the most severe cases, malignant hypertension produces disc oedema together with a macular star, a stellate pattern of hard exudate deposition around the _____.

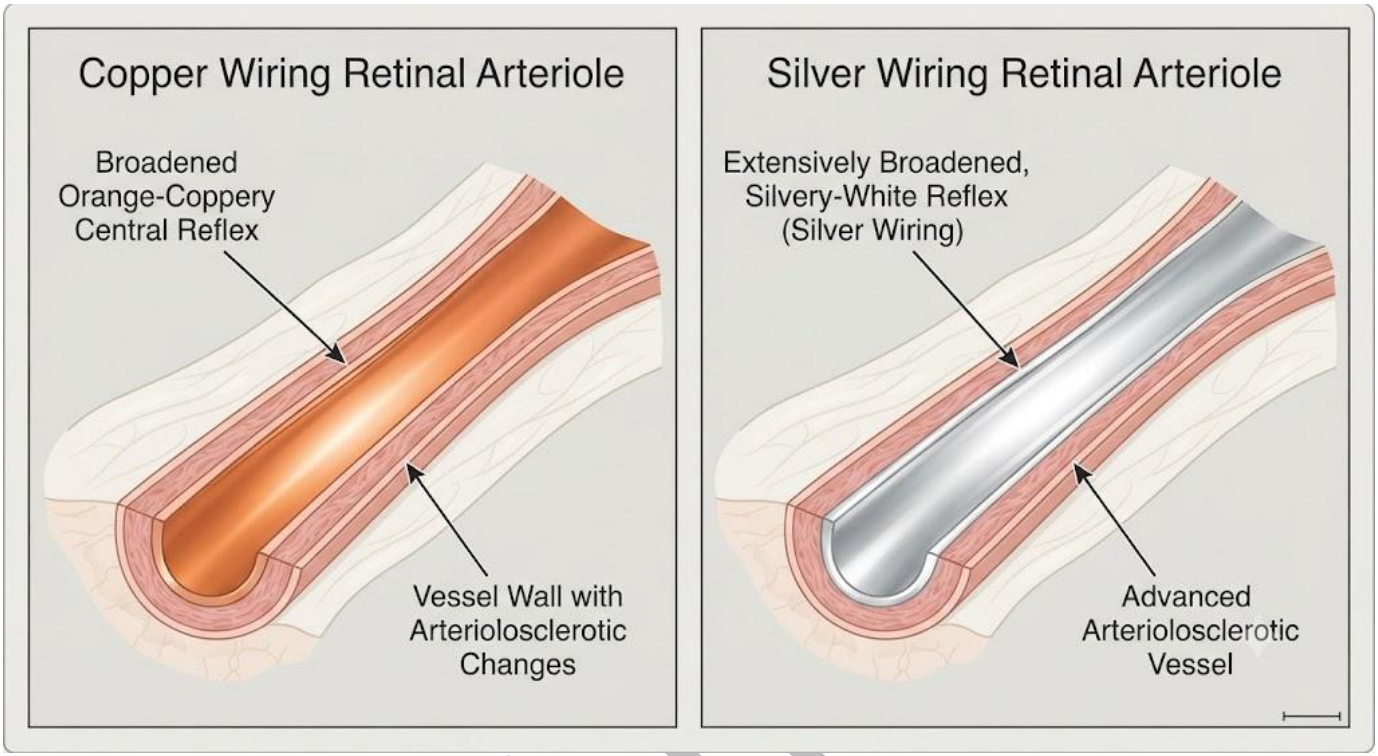

Figure 2.1: A diagram illustrating copper wiring and silver wiring, comparing progressive arteriolar wall thickening obscuring the visible blood column.

Pilot questions 2.1

- Describe arteriolar narrowing as an early hypertensive retinopathy sign. (Linked to SLO 2.1)
- Define arteriovenous nicking. (Linked to SLO 2.1)
- Distinguish copper wiring from silver wiring. (Linked to SLO 2.2)
- Describe the haemorrhages and cotton wool spots of hypertensive retinopathy. (Linked to SLO 2.2)
- Describe the macular star finding of malignant hypertension. (Linked to SLO 2.2)

2.2 Diabetic Retinopathy

2.2.1 microaneurysms and dot-blot haemorrhages

The earliest recognisable signs of diabetic microvascular damage

Microaneurysms, small, discrete, round red dots representing focal outpouching of weakened capillary walls, generally represent the earliest recognisable fundus sign of diabetic retinopathy, often first appearing in the temporal macular region.

Dot and blot haemorrhages, deeper retinal haemorrhages with a rounded rather than flame-shaped configuration, reflecting their location within the compact middle retinal layers rather than the superficial nerve fibre layer, generally accompany microaneurysms as diabetic retinopathy progresses beyond its earliest, mildest stage.

Fill in the blank: Dot and blot haemorrhages have a rounded rather than flame-shaped configuration, reflecting their location within the compact middle retinal layers rather than the superficial nerve fibre _____.

2.2.2 hard exudates and cotton wool spots

Lipid deposition and localised nerve fibre ischaemia

Hard exudates, yellow-white, sharply defined lipid deposits, result from chronic vascular leakage, frequently arranged in a circinate or ring pattern surrounding a leaking microaneurysm, and carry particular significance when approaching the fovea, given the genuine threat to central vision this proximity represents.

Cotton wool spots, fluffy, ill-defined white lesions representing localised retinal nerve fibre layer infarction from focal capillary occlusion, generally indicate progression toward the more ischaemic, pre-proliferative stage of diabetic retinopathy.

Fill in the blank: Hard exudates carry particular significance when approaching the fovea, given the genuine threat to central _____ this proximity represents.

2.2.3 proliferative diabetic retinopathy

Neovascularisation, the hallmark of advanced disease

Neovascularisation, new, abnormal, fragile vessel growth, discussed here as the defining feature of proliferative diabetic retinopathy, arises in response to widespread retinal ischaemia, generally appearing at the optic disc or elsewhere across the retinal surface, and carries substantial risk of vitreous haemorrhage or tractional retinal detachment.

Recognising proliferative disease at the earliest possible stage carries direct clinical urgency, since prompt treatment, generally panretinal laser photocoagulation or anti-vascular endothelial

growth factor therapy, can meaningfully reduce the risk of these genuinely sight-threatening complications.

Fill in the blank: Neovascularisation arises in response to widespread retinal ischaemia and carries substantial risk of vitreous haemorrhage or tractional retinal _____.

Table 2.2: Comparing key features of hypertensive and diabetic retinopathy.

Feature	Hypertensive retinopathy	Diabetic retinopathy
Earliest sign	Arteriolar narrowing	Microaneurysms
Vessel wall change	Copper and silver wiring	Not characteristic
Advanced sign	Disc oedema, macular star	Neovascularisation (proliferative disease)

Pilot questions 2.2

1. Define microaneurysm and describe its typical location. (Linked to SLO 2.3)
2. Distinguish dot-blot haemorrhages from flame-shaped haemorrhages. (Linked to SLO 2.3)
3. Describe hard exudates and their significance near the fovea. (Linked to SLO 2.4)
4. Describe cotton wool spots and their significance in diabetic retinopathy. (Linked to SLO 2.4)
5. Define neovascularisation and its clinical urgency. (Linked to SLO 2.4)

2.3 Disc Oedema and Integrating Fundus Findings

2.3.1 recognising disc oedema

A specific pattern of disc change distinct from normal appearance

Disc oedema produces blurred, elevated disc margins, loss of the normal physiological cup, and, frequently, venous engorgement and peripapillary haemorrhage, findings that should be actively sought during every fundus examination given their genuine diagnostic significance.

Distinguishing genuine disc oedema from a normal variant, such as a small, crowded, or tilted disc that may superficially appear somewhat elevated, requires careful attention to the specific

pattern of margin blurring and, where genuine doubt remains, comparison against any available prior fundus photograph or examination record.

Fill in the blank: Disc oedema produces blurred, elevated disc margins, loss of the normal physiological cup, and, frequently, venous engorgement and peripapillary _____.

2.3.2 causes of disc oedema relevant to systemic disease

Bilateral and unilateral patterns pointing toward different causes

Bilateral disc oedema in the context of malignant hypertension, discussed earlier in this Series, reflects genuinely severe, acutely elevated blood pressure, while bilateral disc oedema from raised intracranial pressure, papilloedema, reflects a genuinely different underlying mechanism entirely unrelated to local retinal vascular disease.

Unilateral disc oedema, by contrast, more typically reflects a localised process, such as optic neuritis or anterior ischaemic optic neuropathy, meaning the specific laterality pattern observed carries direct value in narrowing the relevant differential diagnosis.

Fill in the blank: Unilateral disc oedema more typically reflects a localised process, meaning the specific laterality pattern observed carries direct value in narrowing the relevant differential _____.

2.3.3 integrating fundus findings in systemic disease assessment

Combining individual signs into a coherent clinical picture

No single fundus finding examined throughout this Series should be interpreted in genuine isolation, since the overall combination and specific pattern of findings, arteriolar changes, haemorrhage type, exudate distribution, and disc appearance together, generally provides considerably more diagnostic confidence than any one sign alone.

This integrated approach to fundus interpretation carries direct relevance to the broader systemic disease context this course addresses, since the fundus frequently provides genuinely valuable, non-invasive evidence of a patient's overall vascular and metabolic health, complementing rather than replacing systemic clinical assessment.

Fill in the blank: An integrated approach to fundus interpretation provides genuinely valuable, non-invasive evidence of a patient's overall vascular and metabolic health, complementing rather than replacing systemic clinical _____.

Table 2.3: Comparing patterns of disc oedema by laterality and likely cause.

Laterality	Likely underlying process	Example cause
Bilateral	Systemic or intracranial process	Malignant hypertension, papilloedema
Unilateral	Localised optic nerve process	Optic neuritis, ischaemic optic neuropathy

Pilot questions 2.3

1. Describe the characteristic ophthalmoscopic findings of disc oedema. (Linked to SLO 2.5)
2. Explain how genuine disc oedema is distinguished from a normal variant appearance. (Linked to SLO 2.5)
3. Contrast bilateral and unilateral disc oedema in terms of likely underlying cause. (Linked to SLO 2.6)
4. Explain why no single fundus finding should be interpreted in isolation. (Linked to SLO 2.6)
5. Describe the value of an integrated approach to fundus interpretation in systemic disease. (Linked to SLO 2.6)

Series Summary

This Series worked through the fundus signs of hypertensive retinopathy, arteriolar narrowing, arteriovenous nicking, copper and silver wiring, and haemorrhage and exudate patterns, followed by diabetic retinopathy, microaneurysms, dot-blot haemorrhages, hard exudates, cotton wool spots, and the sight-threatening neovascularisation of proliferative disease, before concluding with disc oedema and the value of integrating rather than isolating individual fundus signs.

This vascular and metabolic disease foundation carries forward directly into visual field testing, hemianopia, and amaurosis fugax, examined in the next Series of this course.

Glossary of Terms

1. **Arteriolar narrowing:** reduction in the normal arteriole-to-venule diameter ratio, an early hypertensive sign.
2. **Arteriovenous nicking:** focal venous compression at an arteriovenous crossing point.
3. **Copper wiring:** a broadened, coppery central arteriolar light reflex from wall thickening.
4. **Silver wiring:** a pronounced, silvery arteriolar reflex from severe wall thickening.
5. **Macular star:** a stellate pattern of hard exudate around the fovea, seen in malignant hypertension.
6. **Microaneurysm:** a small, round red dot from focal capillary wall outpouching.
7. **Dot and blot haemorrhage:** a rounded, deep retinal haemorrhage characteristic of diabetic retinopathy.
8. **Hard exudate:** a yellow-white lipid deposit from chronic vascular leakage.
9. **Cotton wool spot:** a fluffy white lesion representing localised nerve fibre layer infarction.
10. **Neovascularisation:** abnormal new vessel growth marking proliferative diabetic retinopathy.
11. **Disc oedema:** swelling of the optic disc with blurred, elevated margins.
12. **Papilloedema:** bilateral disc oedema resulting from raised intracranial pressure.

Concept Check Questions [Series 2]

Objective questions

1. Arteriovenous nicking reflects thickening of the arteriolar wall compressing the shared adventitial sheath at the point where an arteriole crosses a _____.
2. Silver wiring reflects such severe arteriolar wall thickening that the blood column is almost entirely _____.
3. Dot and blot haemorrhages have a rounded rather than flame-shaped configuration, reflecting their location within the compact middle retinal layers rather than the superficial nerve fibre _____.
4. Neovascularisation arises in response to widespread retinal ischaemia and carries substantial risk of vitreous haemorrhage or tractional retinal _____.
5. Unilateral disc oedema more typically reflects a localised process, meaning the specific laterality pattern observed carries direct value in narrowing the relevant differential _____.

Short-answer questions

- Distinguish copper wiring from silver wiring.
- Describe the macular star finding of malignant hypertension.
- Define microaneurysm and describe its typical location.
- Describe cotton wool spots and their significance in diabetic retinopathy.
- Contrast bilateral and unilateral disc oedema in terms of likely underlying cause.

Long-answer questions

1. Discuss arteriolar narrowing, arteriovenous nicking, copper wiring, and silver wiring in hypertensive retinopathy. (Linked to SLO 2.1, SLO 2.2)
2. Discuss haemorrhages and exudates in hypertensive retinopathy. (Linked to SLO 2.2)
3. Discuss microaneurysms, dot-blot haemorrhages, hard exudates, and cotton wool spots in diabetic retinopathy. (Linked to SLO 2.3, SLO 2.4)
4. Discuss proliferative diabetic retinopathy. (Linked to SLO 2.4)
5. Discuss recognising disc oedema, its causes, and integrating fundus findings in systemic disease assessment. (Linked to SLO 2.5, SLO 2.6)

Answers to Concept Check Questions [Series 2]

Objective questions

6. Venule.
7. Obscured.
8. Layer.
9. Detachment.
10. Diagnosis.

Short-answer questions

11. Copper wiring shows a broadened coppery reflex from moderate wall thickening; silver wiring shows a near-complete silvery reflex from severe thickening.
12. A stellate pattern of hard exudate deposition around the fovea, reflecting genuinely dangerous, acutely elevated blood pressure.
13. A small, discrete, round red dot from focal capillary wall outpouching, often first appearing in the temporal macular region.
14. Fluffy, ill-defined white lesions from localised nerve fibre layer infarction, indicating progression toward pre-proliferative disease.

15. Bilateral disc oedema suggests a systemic or intracranial process such as malignant hypertension or papilloedema; unilateral suggests a localised optic nerve process.

Long-answer questions

16. **Basic response:** Hypertensive retinopathy begins with arteriolar narrowing and arteriovenous nicking, progressing through copper and, in more severe cases, silver wiring.

Value-added response: These vessel wall changes reflect the cumulative effect of chronic, sustained blood pressure elevation on arteriolar structure.

17. **Basic response:** Flame-shaped haemorrhages and cotton wool spots indicate more severe hypertensive retinopathy.

Value-added response: In malignant hypertension, disc oedema and a macular star reflect genuinely dangerous, acutely elevated blood pressure requiring urgent management.

18. **Basic response:** Diabetic retinopathy begins with microaneurysms and dot-blot haemorrhages, progressing to hard exudates and cotton wool spots as ischaemia develops.

Value-added response: Hard exudates threaten central vision when approaching the fovea, while cotton wool spots signal progression toward pre-proliferative disease.

19. **Basic response:** Proliferative diabetic retinopathy is defined by neovascularisation, abnormal vessel growth in response to widespread ischaemia.

Value-added response: Prompt recognition and treatment, generally laser or anti-VEGF therapy, meaningfully reduces the risk of vitreous haemorrhage or tractional detachment.

20. **Basic response:** Disc oedema shows blurred, elevated margins and loss of the physiological cup, distinguished from normal variants by careful pattern assessment.

Value-added response: Bilateral and unilateral patterns point toward different causes, and combining disc findings with other fundus signs supports the most confident overall systemic disease assessment.

Answers to Pilot Questions [Series 2]

Part 2.1

11. A reduction in the normal arteriole-to-venule diameter ratio, generally the earliest recognisable hypertensive fundus sign.
12. Focal compression or displacement of a venule at the point where an arteriole crosses over it.
13. Copper wiring is a broadened coppery reflex from moderate thickening; silver wiring is a near-complete silvery reflex from severe thickening.
14. Flame-shaped haemorrhages from superficial nerve fibre layer bleeding, and cotton wool spots from localised infarction.
15. A stellate pattern of hard exudate around the fovea, reflecting genuinely dangerous, acutely elevated blood pressure.

Part 2.2

1. A small, round red dot from focal capillary wall outpouching, often first appearing in the temporal macular region.
2. Dot-blot haemorrhages are rounded and deep; flame-shaped haemorrhages are superficial and follow the nerve fibre layer.
3. Yellow-white, sharply defined lipid deposits; near the fovea, they carry genuine threat to central vision.
4. Fluffy white lesions from localised nerve fibre layer infarction, indicating progression toward pre-proliferative disease.
5. Abnormal new vessel growth from widespread retinal ischaemia; urgent because it risks vitreous haemorrhage or tractional detachment.

Part 2.3

6. Blurred, elevated disc margins, loss of the physiological cup, venous engorgement, and peripapillary haemorrhage.
7. By careful attention to margin blurring pattern and comparison against any available prior fundus record.
8. Bilateral oedema suggests a systemic or intracranial cause; unilateral oedema suggests a localised optic nerve process.
9. Because the overall combination and pattern of findings provides considerably more diagnostic confidence than any single sign alone.
10. It provides valuable, non-invasive evidence of overall vascular and metabolic health, complementing systemic clinical assessment.

References and Attributions [Series 2]

11. Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
12. American Academy of Ophthalmology (2021). Retina and Vitreous, Basic and Clinical Science Course Section 12.
13. Wong, T. Y., & Mitchell, P. (2007). The Eye in Hypertension. The Lancet, 369(9559), 425-435.
14. Wilkinson, C. P., et al. (2003). Proposed international clinical diabetic retinopathy severity scales. Ophthalmology, 110(9), 1677-1682.

Figures, Plates, Tables, and Boxes

1. Figure 2.1: A diagram illustrating copper wiring and silver wiring, comparing progressive arteriolar wall thickening obscuring the visible blood column. AI-generated using the prompt: "A single clean, accurate two-panel schematic diagram of a retinal arteriole segment, one panel labelled copper wiring showing a broadened orange-coppery central reflex, the other labelled silver wiring showing an almost fully silvery-white vessel, textbook ophthalmology diagram style, neutral background, no photographic realism, no other panels."
2. Table 2.2: Comparing key features of hypertensive and diabetic retinopathy. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Key Features of Hypertensive and Diabetic Retinopathy, listing feature, hypertensive retinopathy, and diabetic retinopathy. Ophthalmology reference table style with outside border, no photographic elements."
3. Table 2.3: Comparing patterns of disc oedema by laterality and likely cause. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Patterns of Disc Oedema by Laterality and Likely Cause, listing laterality, likely underlying process, and example cause. Ophthalmology reference table style with outside border, no photographic elements."

Course code: OPT 611

Course title: Ocular Manifestations of Systemic Diseases

Course credit load: 1 Unit

Series 3: Visual Field Testing, Hemianopia, and Amaurosis Fugax

- **Part 3.1: Confrontation Visual Field Testing**
  - Sub Part 3.1.1: Technique of confrontation visual field testing
  - Sub Part 3.1.2: Four-quadrant assessment
  - Sub Part 3.1.3: Limitations of confrontation testing
- **Part 3.2: Recognition of Hemianopias**
  - Sub Part 3.2.1: Homonymous hemianopia
  - Sub Part 3.2.2: Bitemporal hemianopia
  - Sub Part 3.2.3: Localising hemianopic patterns
- **Part 3.3: Amaurosis Fugax**
  - Sub Part 3.3.1: Defining amaurosis fugax
  - Sub Part 3.3.2: Underlying causes and systemic significance
  - Sub Part 3.3.3: Clinical evaluation and urgency of amaurosis fugax

Introduction

Not every ocular manifestation of systemic disease shows up on the fundus itself. A genuinely large proportion of important visual complaints instead reflect problems elsewhere along the visual pathway or its blood supply, problems a simple bedside visual field test can reveal in minutes. This Series covers **confrontation visual field testing** as a practical skill, the **hemianopic** patterns this testing may uncover, and **amaurosis fugax**, transient visual loss that frequently represents a warning sign of genuine systemic vascular disease.

The aim of this Series is to equip you with a thorough, practical understanding of confrontation visual field testing, recognition of hemianopias, and the definition and significance of amaurosis fugax.

This Series begins with **confrontation visual field testing**, before turning to **recognition of hemianopias**. It concludes with **amaurosis fugax**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 3.1:** demonstrate confrontation visual field testing technique
- **SLO 3.2:** perform four-quadrant confrontation testing and recognise its limitations
- **SLO 3.3:** recognise homonymous hemianopia
- **SLO 3.4:** recognise bitemporal hemianopia and localise hemianopic patterns
- **SLO 3.5:** define amaurosis fugax and describe its underlying causes and systemic significance, and
- **SLO 3.6:** describe the clinical evaluation and urgency of amaurosis fugax.

3.1 Confrontation Visual Field Testing

3.1.1 technique of confrontation visual field testing

A rapid bedside comparison against the examiner's own field

Confrontation testing compares the patient's visual field, one eye tested at a time with the fellow eye occluded, against the examiner's own presumed normal field, generally performed with the examiner positioned directly opposite the patient at a comfortable arm's length distance while the patient maintains steady central fixation.

The examiner's own field serves as the practical reference standard throughout testing, making this approach genuinely efficient as a bedside screening tool despite requiring no specialised equipment beyond the examiner's own hands or a simple target.

Fill in the blank: Confrontation testing compares the patient's visual field, one eye tested at a time, against the examiner's own presumed normal _____.

3.1.2 four-quadrant assessment

A systematic sequence covering every part of the field

Each of the four quadrants, superotemporal, superonasal, inferotemporal, and inferonasal, is tested by presenting a moving finger or small target at the midpoint between examiner and patient, asking the patient to indicate when it first becomes visible while maintaining central fixation throughout.

Where a more sensitive assessment is needed, comparing the relative clarity of fingers held simultaneously in the patient's two hemifields can help detect subtler asymmetries that simple detection testing alone might otherwise miss, and both eyes should always be tested through the full quadrant sequence in turn.

Fill in the blank: Where a more sensitive assessment is needed, comparing the relative clarity of fingers held simultaneously in the patient's two hemifields can help detect subtler _____.

3.1.3 limitations of confrontation testing

A screening tool, not a definitive measurement

Confrontation testing can reliably detect only relatively dense field defects, meaning genuinely subtle or early field loss may pass entirely unnoticed despite careful, technically correct testing, and poor patient fixation during testing represents a further common, avoidable source of unreliable results.

Any identified or suspected abnormality on confrontation testing should therefore prompt referral for formal, automated perimetry, which provides a considerably more precise, quantified characterisation of field defect pattern and severity than bedside testing alone can ever achieve.

Fill in the blank: Any identified or suspected abnormality on confrontation testing should prompt referral for formal, automated _____.

Figure 3.1: A diagram illustrating confrontation visual field testing, with the examiner presenting a target at the midpoint between examiner and patient.

Pilot questions 3.1

- Describe the basic principle of confrontation visual field testing. (Linked to SLO 3.1)
- Describe how each of the four quadrants is tested. (Linked to SLO 3.2)
- Describe a technique for more sensitive detection of subtle field asymmetry. (Linked to SLO 3.2)
- List the recognised limitations of confrontation visual field testing. (Linked to SLO 3.2)
- Explain when referral for formal automated perimetry is appropriate. (Linked to SLO 3.2)

3.2 Recognition of Hemianopias

3.2.1 homonymous hemianopia

Loss of corresponding fields on the same side in both eyes

Homonymous hemianopia describes loss of the same, corresponding half of the visual field, either right or left, in both eyes, resulting from a lesion affecting the optic tract, optic radiations,

or visual cortex, all of which lie posterior to the point where the visual pathways from each eye have already crossed and combined.

The degree of congruity, how similar in shape and size the defect is between the two eyes, carries genuine additional localising value, with more congruous defects generally reflecting more posterior lesions closer to the visual cortex, and less congruous defects pointing toward a more anterior site closer to the optic tract.

Fill in the blank: Homonymous hemianopia results from a lesion affecting the optic tract, optic radiations, or visual cortex, all of which lie posterior to the point where the visual pathways from each eye have already _____.

3.2.2 bitemporal hemianopia

A pattern classically localising to the chiasm

Bitemporal hemianopia describes loss of the outer, temporal half of the visual field in each eye, resulting from a lesion at the optic chiasm itself, where the decussating nasal retinal fibres, carrying information from the temporal visual field, are specifically compressed or damaged.

This pattern is classically associated with a pituitary tumour, given the chiasm's close anatomical relationship to the pituitary gland immediately below it, meaning this specific field defect pattern can provide a genuinely valuable clinical clue toward the underlying diagnosis before any imaging has even been obtained.

Fill in the blank: Bitemporal hemianopia is classically associated with a pituitary tumour, given the chiasm's close anatomical relationship to the pituitary gland immediately _____.

3.2.3 localising hemianopic patterns

Applying visual pathway anatomy to the bedside finding

Distinguishing homonymous from bitemporal hemianopia rests on a genuinely simple observation, whether the defect involves the same side in both eyes, homonymous, or the outer field in each eye, bitemporal, with this distinction directly reflecting whether the underlying lesion sits before, at, or beyond the chiasm.

Careful confrontation testing, combined with attention to this specific pattern, therefore allows the examiner to generate a genuinely confident anatomical localisation at the bedside, directly guiding the choice of further neuro-ophthalmic or neuroimaging investigation.

Fill in the blank: Careful confrontation testing, combined with attention to the specific defect pattern, allows the examiner to generate a genuinely confident anatomical _____ at the bedside.

Table 3.2: Comparing homonymous and bitemporal hemianopia.

Feature	Homonymous hemianopia	Bitemporal hemianopia
Field affected	Same side in both eyes	Outer, temporal field in each eye
Lesion location	Posterior to the optic chiasm	At the optic chiasm itself
Classic association	Optic tract, radiation, or cortical lesion	Pituitary tumour

Pilot questions 3.2

1. Define homonymous hemianopia. (Linked to SLO 3.3)
2. Explain how congruity assists localisation of a homonymous defect. (Linked to SLO 3.3)
3. Define bitemporal hemianopia and its classic underlying cause. (Linked to SLO 3.4)
4. Explain why bitemporal hemianopia results specifically from a chiasmal lesion. (Linked to SLO 3.4)
5. Distinguish homonymous from bitemporal hemianopia. (Linked to SLO 3.4)

3.3 Amaurosis Fugax

3.3.1 defining amaurosis fugax

Transient, painless monocular vision loss

Amaurosis fugax refers to transient, generally painless monocular visual loss, characteristically described by patients as a curtain or shade descending or ascending across the visual field, with vision typically recovering completely within minutes.

This transient nature genuinely distinguishes amaurosis fugax from established vascular occlusion, discussed elsewhere in this course, since amaurosis fugax reflects a temporary interruption of retinal blood flow rather than a permanent, established vascular occlusion.

Fill in the blank: Amaurosis fugax refers to transient, generally painless monocular visual loss, with vision typically recovering completely within _____.

3.3.2 underlying causes and systemic significance

A warning sign frequently reflecting embolic disease

The most common recognised cause of amaurosis fugax is a small embolus, generally originating from atherosclerotic plaque within the ipsilateral carotid artery, temporarily lodging within the retinal circulation before dislodging and restoring normal blood flow.

Given this embolic mechanism, amaurosis fugax carries substantial systemic significance as a warning sign of underlying carotid or, less commonly, cardiac disease, meaning any patient presenting with this symptom warrants systemic evaluation directly addressing their broader stroke risk, not merely reassurance regarding the eye alone.

Fill in the blank: Amaurosis fugax carries substantial systemic significance as a warning sign of underlying carotid or, less commonly, cardiac _____.

3.3.3 clinical evaluation and urgency of amaurosis fugax

A genuine transient ischaemic attack equivalent

Clinical evaluation of amaurosis fugax generally includes carotid imaging, discussed in relation to embolic source assessment, together with cardiac evaluation where indicated, and appropriate assessment of broader vascular risk factors, given the recognised association with underlying systemic vascular disease.

Amaurosis fugax is best regarded clinically as the ocular equivalent of a transient ischaemic attack, meaning it warrants the same genuine urgency of evaluation, since it represents a recognised, meaningful warning sign for future stroke that should never be dismissed as a purely benign or self-limiting event.

Fill in the blank: Amaurosis fugax is best regarded clinically as the ocular equivalent of a transient ischaemic attack, warranting the same genuine urgency of clinical _____.

Table 3.3: Key features of amaurosis fugax.

Feature	Description	Clinical implication
Presentation	Transient, painless monocular vision loss lasting minutes	Distinguishes it from established vascular occlusion
Common cause	Embolus from carotid atherosclerotic plaque	Suggests need for carotid evaluation

Clinical significance	Ocular equivalent of a transient ischaemic attack	Warrants urgent systemic vascular assessment

Pilot questions 3.3

1. Define amaurosis fugax. (Linked to SLO 3.5)
2. Describe the typical patient description of amaurosis fugax. (Linked to SLO 3.5)
3. Describe the most common underlying cause of amaurosis fugax. (Linked to SLO 3.5)
4. Explain the systemic significance of amaurosis fugax. (Linked to SLO 3.6)
5. Describe the appropriate clinical evaluation and urgency of amaurosis fugax. (Linked to SLO 3.6)

Series Summary

This Series covered confrontation visual field testing, its technique, four-quadrant sequence, and recognised limitations, followed by the recognition and localisation of homonymous and bitemporal hemianopia, before concluding with amaurosis fugax, its definition, embolic cause, and genuine urgency as a transient ischaemic attack equivalent.

This foundation carries directly into sickle cell disease, thyroid orbitopathy, and myasthenia gravis, examined in the next Series of this course.

Glossary of Terms

1. **Confrontation visual field testing:** a bedside screening technique comparing the patient's field with the examiner's own.
2. **Homonymous hemianopia:** loss of the same, corresponding half of the visual field in both eyes.
3. **Bitemporal hemianopia:** loss of the outer, temporal half of the visual field in each eye, classically from a chiasmal lesion.
4. **Congruity:** the degree of similarity in shape and size between field defects in each eye.
5. **Optic chiasm:** the point where nasal retinal fibres decussate, the classic site of bitemporal hemianopia.
6. **Pituitary tumour:** a mass classically associated with bitemporal hemianopia given its proximity to the chiasm.
7. **Amaurosis fugax:** transient, generally painless monocular visual loss lasting minutes.

8. **Embolus:** a fragment of material, often atherosclerotic plaque, that travels within the bloodstream and lodges elsewhere.
9. **Carotid atherosclerotic plaque:** the most common source of the embolus causing amaurosis fugax.
10. **Transient ischaemic attack:** a temporary episode of neurological dysfunction from transiently interrupted blood flow, to which amaurosis fugax is the ocular equivalent.
11. **Carotid imaging:** an investigation used to assess for embolic source in amaurosis fugax.
12. **Vascular risk factors:** conditions and behaviours predisposing to embolic and vascular disease, relevant to amaurosis fugax evaluation.

Concept Check Questions [Series 3]

Objective questions

1. Confrontation testing compares the patient's visual field, one eye tested at a time, against the examiner's own presumed normal _____.
2. Any identified or suspected abnormality on confrontation testing should prompt referral for formal, automated _____.
3. Homonymous hemianopia results from a lesion affecting the optic tract, radiations, or cortex, all of which lie posterior to the point where the visual pathways have already _____.
4. Bitemporal hemianopia is classically associated with a pituitary tumour, given the chiasm's close anatomical relationship to the pituitary gland immediately _____.
5. Amaurosis fugax is best regarded clinically as the ocular equivalent of a transient ischaemic attack, warranting the same genuine urgency of clinical _____.

Short-answer questions

- Describe how each of the four quadrants is tested in confrontation testing.
- Define homonymous hemianopia.
- Define bitemporal hemianopia and its classic underlying cause.
- Define amaurosis fugax.
- Describe the most common underlying cause of amaurosis fugax.

Long-answer questions

1. Discuss the technique, four-quadrant assessment, and limitations of confrontation visual field testing. (Linked to SLO 3.1, SLO 3.2)
2. Discuss homonymous hemianopia. (Linked to SLO 3.3)

3. Discuss bitemporal hemianopia and how hemianopic patterns are localised. (Linked to SLO 3.4)
4. Discuss the definition, causes, and systemic significance of amaurosis fugax. (Linked to SLO 3.5)
5. Discuss the clinical evaluation and urgency of amaurosis fugax. (Linked to SLO 3.6)

Answers to Concept Check Questions [Series 3]

Objective questions

6. Field.
7. Perimetry.
8. Crossed.
9. Below.
10. Evaluation.

Short-answer questions

11. By presenting a moving finger or target at the midpoint between examiner and patient in each of the four quadrants while the patient maintains central fixation.
12. Loss of the same, corresponding half of the visual field in both eyes.
13. Loss of the outer, temporal half of the visual field in each eye; classically caused by a pituitary tumour.
14. Transient, generally painless monocular visual loss, typically recovering completely within minutes.
15. A small embolus, generally originating from atherosclerotic plaque within the ipsilateral carotid artery.

Long-answer questions

16. **Basic response:** Confrontation testing compares the patient's field with the examiner's own, testing each eye independently in all four quadrants.

Value-added response: It can only reliably detect relatively dense defects and remains vulnerable to poor patient fixation, meaning suspected abnormalities warrant formal perimetry.

17. **Basic response:** Homonymous hemianopia affects the same side in both eyes, resulting from a postchiasmal lesion.

Value-added response: Congruity of the defect provides further localising value, with more congruous defects reflecting more posterior lesions.

18. **Basic response:** Bitemporal hemianopia affects the outer field in each eye, resulting from a chiasmal lesion, classically a pituitary tumour.

Value-added response: Distinguishing homonymous from bitemporal patterns directly reflects whether a lesion sits before, at, or beyond the chiasm.

19. **Basic response:** Amaurosis fugax is transient, painless monocular visual loss, most commonly from a carotid embolus.

Value-added response: This embolic mechanism gives it substantial systemic significance as a warning sign of underlying vascular disease.

20. **Basic response:** Evaluation includes carotid imaging, cardiac assessment, and broader vascular risk factor review.

Value-added response: Amaurosis fugax should be treated with the same urgency as a transient ischaemic attack, never dismissed as benign.

Answers to Pilot Questions [Series 3]

Part 3.1

11. It compares the patient's own visual field, one eye at a time, against the examiner's own presumed normal field.
12. By presenting a target at the midpoint between examiner and patient in each quadrant, asking the patient to indicate when it becomes visible.
13. Comparing the relative clarity of the examiner's fingers held simultaneously in the two hemifields.
14. It can only reliably detect relatively dense defects, and is vulnerable to poor patient fixation.
15. Whenever confrontation testing identifies or suggests an abnormality warranting more precise characterisation.

Part 3.2

1. Loss of the same, corresponding half of the visual field in both eyes, from a postchiasmal lesion.
2. More congruous defects generally reflect more posterior lesions; less congruous defects reflect more anterior lesions.
3. Loss of the outer, temporal half of the field in each eye; classically caused by a pituitary tumour.
4. Because the decussating nasal retinal fibres carrying temporal field information are specifically affected at the chiasm.
5. Homonymous affects the same side in both eyes from a postchiasmal lesion; bitemporal affects the outer field in each eye from a chiasmal lesion.

Part 3.3

6. Transient, generally painless monocular visual loss, typically recovering completely within minutes.
7. A curtain or shade descending or ascending across the visual field.
8. A small embolus, generally originating from atherosclerotic plaque within the ipsilateral carotid artery.
9. It carries substantial significance as a warning sign of underlying carotid or cardiac disease.
10. Carotid imaging, cardiac evaluation, and vascular risk factor assessment, treated with the same urgency as a transient ischaemic attack.

References and Attributions [Series 3]

11. Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
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13. Biousse, V., & Newman, N. J. (2015). Ischemic Optic Neuropathies. New England Journal of Medicine, 372(25), 2428-2436.
14. Bickley, L. S. (2020). Bates' Guide to Physical Examination and History Taking (13th ed.). Wolters Kluwer.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: A diagram illustrating confrontation visual field testing, with the examiner presenting a target at the midpoint between examiner and patient. AI-generated using the prompt: "A single clean, realistic clinical illustration of an examiner seated opposite a patient, holding a finger target at arm's length midway between them, both facing each other, calm clinical setting, no text overlays, no other panels, respectful depiction, no identifying facial features."
2. Table 3.2: Comparing homonymous and bitemporal hemianopia. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Homonymous and Bitemporal Hemianopia, listing feature, homonymous hemianopia, and bitemporal hemianopia. Ophthalmology reference table style with outside border, no photographic elements."
3. Table 3.3: Key features of amaurosis fugax. AI-generated using the prompt: "Create a clean clinical reference table titled Key Features of Amaurosis Fugax, listing feature, description, and clinical implication. Ophthalmology reference table style with outside border, no photographic elements."

Course code: OPT 611

Course title: Ocular Manifestations of Systemic Diseases

Course credit load: 1 Unit

Series 4: Sickle Cell Disease, Thyroid Orbitopathy, and Myasthenia Gravis

- **Part 4.1: Sickle Cell Disease and the Eye**
  - Sub Part 4.1.1: Ocular manifestations of sickle cell disease
  - Sub Part 4.1.2: Traumatic hyphaema in sickle cell disease
  - Sub Part 4.1.3: Management implications of hyphaema in sickle cell disease
- **Part 4.2: Thyroid (Graves) Orbitopathy**
  - Sub Part 4.2.1: Clinical features of thyroid orbitopathy
  - Sub Part 4.2.2: The Werner classification
  - Sub Part 4.2.3: Treatment of thyroid orbitopathy
- **Part 4.3: Myasthenia Gravis**
  - Sub Part 4.3.1: Pathophysiology of myasthenia gravis
  - Sub Part 4.3.2: Ocular features of myasthenia gravis
  - Sub Part 4.3.3: Diagnosis and management of ocular myasthenia gravis

Introduction

Three genuinely different disease processes share this Series, a haematological disorder, an autoimmune thyroid condition, and a neuromuscular junction disorder, yet each produces ocular signs distinctive and important enough that recognising them can meaningfully change a patient's management. This Series works through **sickle cell disease**, with particular attention to the genuine emergency of traumatic hyphaema in this population, **thyroid orbitopathy**, and **myasthenia gravis**.

The aim of this Series is to equip you with a thorough understanding of the importance of recognising traumatic hyphaema as a risk factor for acute visual loss in sickle cell anaemia, and the clinical features, Werner classification, and treatment of thyroid orbitopathy.

This Series begins with **sickle cell disease and the eye**, before turning to **thyroid (Graves) orbitopathy**. It concludes with **myasthenia gravis**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 4.1:** describe the ocular manifestations of sickle cell disease
- **SLO 4.2:** explain the importance of recognising traumatic hyphaema as a risk factor for acute visual loss in sickle cell anaemia
- **SLO 4.3:** describe the clinical features of thyroid orbitopathy
- **SLO 4.4:** discuss the Werner classification and treatment of thyroid orbitopathy
- **SLO 4.5:** describe the pathophysiology and ocular features of myasthenia gravis, and
- **SLO 4.6:** describe the diagnosis and management of ocular myasthenia gravis.

4.1 Sickle Cell Disease and the Eye

4.1.1 ocular manifestations of sickle cell disease

A retinal vasculopathy of variable severity

Sickle cell disease produces a spectrum of ocular manifestations, ranging from asymptomatic peripheral retinal vascular changes, comma-shaped conjunctival vessels, and salmon patch haemorrhages to genuinely sight-threatening **proliferative sickle retinopathy**, characterised by peripheral retinal neovascularisation.

This retinal vasculopathy reflects repeated microvascular occlusion by sickled erythrocytes within the peripheral retinal circulation, a process that generally develops insidiously over years and may remain entirely asymptomatic until relatively advanced, underscoring the value of periodic dilated fundus screening in this population.

Fill in the blank: Sickle cell retinopathy reflects repeated microvascular occlusion by sickled erythrocytes within the peripheral retinal _____.

4.1.2 traumatic hyphaema in sickle cell disease

A genuinely dangerous combination

Hyphaema, blood within the anterior chamber following blunt or penetrating ocular trauma, poses a substantially greater risk in patients with sickle cell disease or trait than in the general population, since sickled red cells within the anterior chamber obstruct the trabecular meshwork considerably more readily than normal erythrocytes.

This obstruction produces a genuinely dangerous, disproportionate rise in intraocular pressure compared with hyphaema in patients without sickle haemoglobin, meaning even a relatively small hyphaema in a sickle cell patient carries a meaningfully higher risk of acute, rapid visual loss than the equivalent finding would carry in an unaffected patient.

Fill in the blank: Sickled red cells within the anterior chamber obstruct the trabecular meshwork considerably more readily than normal erythrocytes, producing a genuinely dangerous, disproportionate rise in intraocular _____.

4.1.3 management implications of hyphaema in sickle cell disease

Modified management given the elevated risk

Any patient presenting with traumatic hyphaema should be specifically asked about, or tested for, sickle cell status, since this single piece of information directly changes the urgency and intensity of subsequent monitoring, given the disproportionate pressure risk discussed in the previous sub-Part.

Certain medications used more routinely in hyphaema management, including some carbonic anhydrase inhibitors, require caution or avoidance in sickle cell patients given their potential to further promote red cell sickling, meaning genuinely individualised, sickle-status-aware management represents an essential safety principle rather than an optional refinement.

Fill in the blank: Certain medications used in hyphaema management require caution or avoidance in sickle cell patients given their potential to further promote red cell _____.

Table 4.1: Comparing hyphaema management considerations by sickle cell status.

Consideration	Non-sickle patient	Sickle cell patient
Intraocular pressure risk	Standard monitoring generally adequate	Disproportionately elevated risk, requiring closer monitoring

Certain medications	Standard agents generally appropriate	Some agents require caution given sickling risk
Overall urgency	Routine hyphaema management	Genuinely heightened clinical urgency

Pilot questions 4.1

- List ocular manifestations of sickle cell disease. (Linked to SLO 4.1)
- Define hyphaema. (Linked to SLO 4.2)
- Explain why hyphaema poses a disproportionate risk in sickle cell patients. (Linked to SLO 4.2)
- Explain why sickle status should be established in any patient with traumatic hyphaema. (Linked to SLO 4.2)
- Explain why certain medications require caution in hyphaema management for sickle cell patients. (Linked to SLO 4.2)

4.2 Thyroid (Graves) Orbitopathy

4.2.1 clinical features of thyroid orbitopathy

Orbital tissue expansion driven by autoimmune inflammation

Thyroid orbitopathy, an autoimmune condition generally associated with Graves disease, results from immune-mediated inflammation and subsequent fibrosis of the extraocular muscles and orbital fat, producing tissue expansion within the fixed volume of the bony orbit.

This tissue expansion produces the condition's characteristic clinical features, **proptosis**, forward displacement of the globe, **eyelid retraction**, restricted ocular motility from fibrotic muscle involvement, and, in more severe cases, exposure keratopathy and optic nerve compression from crowding at the orbital apex.

Fill in the blank: Thyroid orbitopathy results from immune-mediated inflammation and subsequent fibrosis of the extraocular muscles and orbital fat, producing tissue expansion within the fixed volume of the bony _____.

4.2.2 the werner classification

A structured mnemonic for documenting disease features

The **Werner classification**, often remembered through the mnemonic **"NO SPECS"**, provides a structured framework for documenting thyroid orbitopathy severity, spanning from Class 0, No signs or symptoms, through progressively more severe categories including lid retraction, soft tissue involvement, proptosis, extraocular muscle involvement, corneal involvement, and, finally, sight loss from optic nerve compression.

While this classification carries genuine historical and educational value, its clinical use has been increasingly supplemented by activity and severity scoring systems that more directly guide treatment decisions, since the Werner classification alone does not reliably distinguish active, inflammatory disease from stable, burnt-out disease.

Fill in the blank: The Werner classification provides a structured framework spanning from no signs or symptoms through progressively more severe categories, finally reaching sight loss from optic nerve _____.

4.2.3 treatment of thyroid orbitopathy

Addressing both systemic thyroid status and local orbital disease

Treatment of thyroid orbitopathy requires attention to both the underlying **systemic thyroid status**, discussed in relation to endocrine management, since restoring euthyroidism represents an essential foundation of care, and the **local orbital disease** itself, with mild cases generally managed conservatively using lubrication and protective measures.

More severe or actively inflammatory disease may require **systemic corticosteroids** or other immunosuppressive therapy, with **orbital decompression surgery** reserved for cases with sight-threatening optic nerve compression or genuinely disfiguring proptosis unresponsive to medical management alone.

Fill in the blank: More severe or actively inflammatory thyroid orbitopathy may require systemic corticosteroids, with orbital decompression surgery reserved for sight-threatening optic nerve _____.

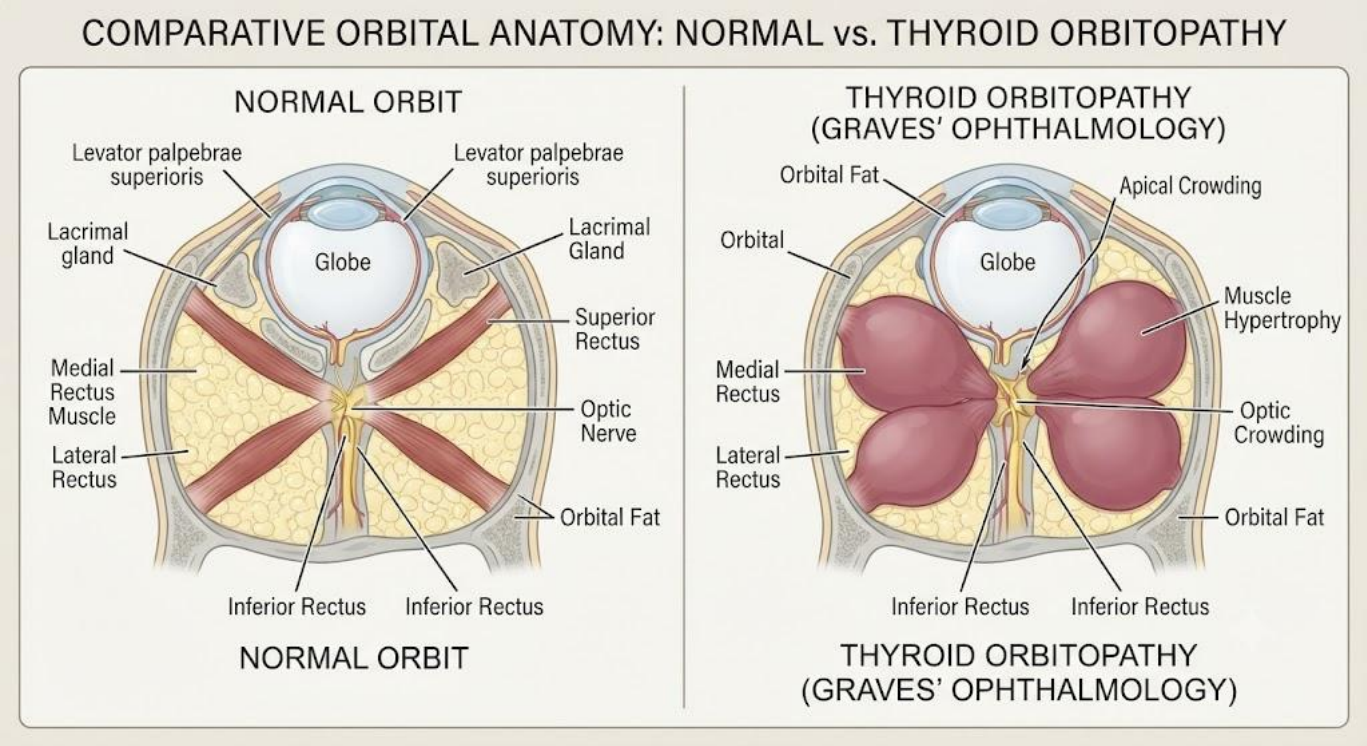

Figure 4.2: A diagram illustrating orbital tissue expansion in thyroid orbitopathy, showing enlarged extraocular muscles crowding the orbital apex.

Pilot questions 4.2

1. Describe the pathophysiology of thyroid orbitopathy. (Linked to SLO 4.3)
2. List the characteristic clinical features of thyroid orbitopathy. (Linked to SLO 4.3)
3. Describe the Werner classification and its NO SPECS mnemonic. (Linked to SLO 4.4)
4. Explain a limitation of the Werner classification. (Linked to SLO 4.4)
5. Describe the treatment approach for thyroid orbitopathy, from mild to severe disease. (Linked to SLO 4.4)

4.3 Myasthenia Gravis

4.3.1 pathophysiology of myasthenia gravis

Autoimmune destruction of the neuromuscular junction

Myasthenia gravis is an autoimmune disorder in which antibodies target the **acetylcholine receptor**, or, less commonly, related proteins, at the postsynaptic neuromuscular junction,

progressively reducing the number of functional receptors available to receive the neurotransmitter signal.

This reduced receptor availability produces the condition's characteristic **fatigability**, muscle weakness that worsens with repeated or sustained use and improves with rest, distinguishing myasthenia gravis from most other causes of muscle weakness, which generally remain relatively constant regardless of activity.

Fill in the blank: Myasthenia gravis is an autoimmune disorder in which antibodies target the acetylcholine receptor at the postsynaptic neuromuscular _____.

4.3.2 ocular features of myasthenia gravis

The eyes as a frequent, often earliest, site of involvement

Ocular involvement, present in the substantial majority of myasthenia gravis patients at some point during their illness, characteristically produces **fluctuating ptosis** and **variable diplopia**, reflecting fatigable weakness of the levator palpebrae and extraocular muscles respectively.

This fluctuating pattern, worsening as the day progresses or with sustained upward gaze, and the **absence of pupillary involvement**, since the pupil sphincter is smooth rather than skeletal muscle and therefore unaffected by this specific disease process, together provide genuinely useful clues distinguishing myasthenic ptosis and diplopia from other causes of similar presentation.

Fill in the blank: Ocular myasthenia gravis characteristically produces fluctuating ptosis and variable diplopia, with an absence of pupillary involvement since the pupil sphincter is smooth rather than skeletal _____.

4.3.3 diagnosis and management of ocular myasthenia gravis

Bedside signs supporting a confident clinical diagnosis

Bedside diagnostic techniques include the **ice pack test**, applying cold to a ptotic eyelid and observing genuine, temporary improvement, given cold's effect on acetylcholinesterase activity, and observing fatigability directly, worsening ptosis with sustained upgaze, together supporting a confident clinical diagnosis before formal confirmatory testing.

Management generally begins with **acetylcholinesterase inhibitor** therapy, prolonging acetylcholine availability at the neuromuscular junction, with immunosuppressive therapy reserved for cases with inadequate response, and referral for broader systemic evaluation

essential given the recognised risk of progression to generalised, potentially life-threatening myasthenia affecting respiratory and bulbar muscles.

Fill in the blank: Management of ocular myasthenia gravis generally begins with acetylcholinesterase inhibitor therapy, prolonging acetylcholine availability at the neuromuscular _____.

Table 4.3: Distinguishing myasthenic ptosis and diplopia from other causes.

Feature	Myasthenia gravis	Other causes (e.g. third nerve palsy)
Pattern over time	Fluctuating, worse with fatigue	Generally constant
Pupil involvement	Absent	Often present
Bedside test	Improves with ice pack test	Not applicable

Pilot questions 4.3

1. Describe the pathophysiology of myasthenia gravis. (Linked to SLO 4.5)
2. Describe the characteristic ocular features of myasthenia gravis. (Linked to SLO 4.5)
3. Explain why pupillary involvement is absent in myasthenia gravis. (Linked to SLO 4.5)
4. Describe the ice pack test and its diagnostic value. (Linked to SLO 4.6)
5. Describe the general management approach for ocular myasthenia gravis. (Linked to SLO 4.6)

Series Summary

This Series worked through sickle cell disease and its ocular manifestations, with particular attention to the disproportionate, genuinely dangerous risk hyphaema poses in this population, thyroid orbitopathy, its clinical features, Werner classification, and treatment, and myasthenia gravis, its pathophysiology, characteristic fluctuating ocular features, and diagnosis and management.

This Series completes the systemic disease foundation carried forward into HIV and tropical ophthalmology, leprosy, trachoma, and onchocerciasis, examined in the final Series of this course.

Glossary of Terms

1. **Proliferative sickle retinopathy:** sight-threatening peripheral retinal neovascularisation from sickle cell disease.
2. **Hyphaema:** blood within the anterior chamber, generally resulting from ocular trauma.
3. **Trabecular meshwork obstruction:** blockage of aqueous outflow, disproportionately severe when sickled cells are involved.
4. **Thyroid orbitopathy:** autoimmune orbital tissue expansion associated with Graves disease.
5. **Proptosis:** abnormal forward protrusion of the globe.
6. **Werner classification:** a structured mnemonic (NO SPECS) for documenting thyroid orbitopathy severity.
7. **Orbital decompression surgery:** surgery relieving orbital crowding in severe thyroid orbitopathy.
8. **Myasthenia gravis:** an autoimmune disorder targeting the acetylcholine receptor at the neuromuscular junction.
9. **Fatigability:** weakness worsening with repeated or sustained use, characteristic of myasthenia gravis.
10. **Ice pack test:** a bedside test showing temporary ptosis improvement with cold application in myasthenia gravis.
11. **Acetylcholinesterase inhibitor:** a medication prolonging acetylcholine availability at the neuromuscular junction.
12. **Generalised myasthenia:** progression of myasthenia gravis to affect respiratory and bulbar muscles.

Concept Check Questions [Series 4]

Objective questions

1. Sickle cell retinopathy reflects repeated microvascular occlusion by sickled erythrocytes within the peripheral retinal _____.
2. Sickled red cells within the anterior chamber obstruct the trabecular meshwork considerably more readily than normal erythrocytes, producing a disproportionate rise in intraocular _____.
3. Thyroid orbitopathy results from immune-mediated inflammation and fibrosis of the extraocular muscles and orbital fat, producing tissue expansion within the fixed volume of the bony _____.
4. Myasthenia gravis is an autoimmune disorder in which antibodies target the acetylcholine receptor at the postsynaptic neuromuscular _____.

5. Ocular myasthenia gravis characteristically produces fluctuating ptosis and diplopia, with an absence of pupillary involvement since the pupil sphincter is smooth rather than skeletal _____.

Short-answer questions

- List ocular manifestations of sickle cell disease.
- Explain why hyphaema poses a disproportionate risk in sickle cell patients.
- List the characteristic clinical features of thyroid orbitopathy.
- Describe the Werner classification and its NO SPECS mnemonic.
- Describe the ice pack test and its diagnostic value.

Long-answer questions

1. Discuss the ocular manifestations of sickle cell disease and the importance of traumatic hyphaema. (Linked to SLO 4.1, SLO 4.2)
2. Discuss the clinical features of thyroid orbitopathy. (Linked to SLO 4.3)
3. Discuss the Werner classification and treatment of thyroid orbitopathy. (Linked to SLO 4.4)
4. Discuss the pathophysiology and ocular features of myasthenia gravis. (Linked to SLO 4.5)
5. Discuss the diagnosis and management of ocular myasthenia gravis. (Linked to SLO 4.6)

Answers to Concept Check Questions [Series 4]

Objective questions

6. Circulation.
7. Pressure.
8. Orbit.
9. Junction.
10. Muscle.

Short-answer questions

11. Comma-shaped conjunctival vessels, salmon patch haemorrhages, and proliferative sickle retinopathy.
12. Because sickled cells obstruct the trabecular meshwork considerably more readily than normal erythrocytes, producing a disproportionate pressure rise.

13. Proptosis, eyelid retraction, restricted ocular motility, and, in severe cases, exposure keratopathy and optic nerve compression.
14. A staged classification from no signs through lid retraction, soft tissue involvement, proptosis, muscle involvement, corneal involvement, to sight loss.
15. Applying cold to a ptotic eyelid and observing genuine, temporary improvement; it supports a confident bedside diagnosis.

Long-answer questions

16. **Basic response:** Sickle cell disease produces peripheral retinal vascular changes ranging from asymptomatic findings to sight-threatening proliferative retinopathy.

Value-added response: Traumatic hyphaema carries disproportionate risk in this population, since sickled cells obstruct the trabecular meshwork more readily, requiring sickle-status-aware urgent management.

17. **Basic response:** Thyroid orbitopathy results from autoimmune orbital tissue expansion, producing proptosis, lid retraction, and restricted motility.

Value-added response: Severe cases risk exposure keratopathy and optic nerve compression from crowding at the orbital apex.

18. **Basic response:** The Werner classification (NO SPECS) provides a structured staging system for documenting disease severity.

Value-added response: Treatment addresses both systemic thyroid status and local orbital disease, ranging from conservative measures to corticosteroids and, in severe cases, decompression surgery.

19. **Basic response:** Myasthenia gravis results from antibody-mediated acetylcholine receptor loss, producing characteristic fatigable weakness.

Value-added response: Ocular involvement produces fluctuating ptosis and diplopia without pupillary involvement, distinguishing it from other causes of similar presentation.

20. **Basic response:** Diagnosis relies on bedside signs including the ice pack test and observed fatigability with sustained gaze.

Value-added response: Management begins with acetylcholinesterase inhibitors, with attention to the risk of progression to generalised, potentially life-threatening disease.

Answers to Pilot Questions [Series 4]

Part 4.1

11. Comma-shaped conjunctival vessels, salmon patch haemorrhages, and proliferative sickle retinopathy.
12. Blood within the anterior chamber, generally resulting from ocular trauma.
13. Because sickled cells obstruct the trabecular meshwork more readily than normal erythrocytes, causing a disproportionate pressure rise.
14. Because sickle status directly changes the urgency and intensity of subsequent monitoring given the elevated pressure risk.
15. Because some agents can further promote red cell sickling, worsening the underlying risk.

Part 4.2

1. Immune-mediated inflammation and fibrosis of the extraocular muscles and orbital fat, producing tissue expansion within the fixed orbit.
2. Proptosis, eyelid retraction, restricted ocular motility, and, in severe cases, exposure keratopathy and optic nerve compression.
3. A staged classification (NO SPECS) from no signs through lid retraction, soft tissue involvement, proptosis, muscle involvement, corneal involvement, to sight loss.
4. It does not reliably distinguish active, inflammatory disease from stable, burnt-out disease.
5. Conservative measures for mild disease, systemic corticosteroids for more severe or active disease, and decompression surgery for sight-threatening or disfiguring cases.

Part 4.3

6. Antibodies target the acetylcholine receptor at the postsynaptic neuromuscular junction, reducing functional receptor availability.
7. Fluctuating ptosis and variable diplopia, worsening with fatigue or sustained gaze.
8. Because the pupil sphincter is smooth rather than skeletal muscle, unaffected by this specific disease process.
9. Applying cold to a ptotic eyelid and observing genuine, temporary improvement; it supports a confident bedside diagnosis.
10. Beginning with acetylcholinesterase inhibitor therapy, with immunosuppression for inadequate response and referral given generalised disease risk.

References and Attributions [Series 4]

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12. Downes, S. M. (2016). Ocular associations of sickle cell disease. Eye, 30(7), 924-931.
13. Bahn, R. S. (2010). Graves' Ophthalmopathy. New England Journal of Medicine, 362(8), 726-738.
14. Kerty, E., et al. (2014). EFNS/ENS Guidelines for the treatment of ocular myasthenia. European Journal of Neurology, 21(5), 687-693.

Figures, Plates, Tables, and Boxes

1. Table 4.1: Comparing hyphaema management considerations by sickle cell status. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Hyphaema Management Considerations by Sickle Cell Status, listing consideration, non-sickle patient, and sickle cell patient. Ophthalmology reference table style with outside border, no photographic elements."
2. Figure 4.2: A diagram illustrating orbital tissue expansion in thyroid orbitopathy, showing enlarged extraocular muscles crowding the orbital apex. AI-generated using the prompt: "A single clean, accurate cross-sectional schematic diagram of the orbit, one panel labelled normal showing slender extraocular muscles around the optic nerve, the other labelled thyroid orbitopathy showing markedly enlarged muscles crowding the nerve at the orbital apex, textbook ophthalmology diagram style, neutral background, no photographic realism, no other panels."
3. Table 4.3: Distinguishing myasthenic ptosis and diplopia from other causes. AI-generated using the prompt: "Create a clean clinical reference table titled Distinguishing Myasthenic Ptosis and Diplopia from Other Causes, listing feature, myasthenia gravis, and other causes. Ophthalmology reference table style with outside border, no photographic elements."

Course code: OPT 611

Course title: Ocular Manifestations of Systemic Diseases

Course credit load: 1 Unit

Series 5: HIV and Tropical Ophthalmology: Leprosy, Trachoma, and Onchocerciasis

- **Part 5.1: Ocular Manifestations of HIV/AIDS**
  - Sub Part 5.1.1: Anterior segment manifestations of HIV/AIDS
  - Sub Part 5.1.2: HIV retinopathy and CMV retinitis
  - Sub Part 5.1.3: Other opportunistic and neoplastic ocular manifestations
- **Part 5.2: Ocular Leprosy and Trachoma**
  - Sub Part 5.2.1: Ocular manifestations of leprosy
  - Sub Part 5.2.2: Trachoma: pathogenesis and staging
  - Sub Part 5.2.3: Complications and prevention of trachoma
- **Part 5.3: Onchocerciasis**
  - Sub Part 5.3.1: Pathogenesis of ocular onchocerciasis
  - Sub Part 5.3.2: Anterior and posterior segment features of onchocerciasis
  - Sub Part 5.3.3: Prevention and control of onchocerciasis

Introduction

Few areas of ophthalmology illustrate the connection between eye disease and broader public health as clearly as the conditions closing this course. **HIV/AIDS** produces a recognisable spectrum of ocular findings directly tracking a patient's degree of immunosuppression, while **leprosy**, **trachoma**, and **onchocerciasis** remain among the world's most important preventable causes of blindness, each with a genuinely distinct pathogenesis and each still carrying real relevance in many practice settings.

The aim of this Series is to equip you with a thorough understanding of the ocular manifestations of HIV/AIDS, leprosy, trachoma, and onchocerciasis.

This Series begins with **ocular manifestations of HIV/AIDS**, before turning to **ocular leprosy and trachoma**. It concludes with **onchocerciasis**.

Series Learning Outcomes

At the end of this Series, you should be able to:

- **SLO 5.1:** list anterior segment ocular manifestations of HIV/AIDS
- **SLO 5.2:** describe HIV retinopathy and CMV retinitis and other opportunistic or neoplastic ocular manifestations of HIV/AIDS
- **SLO 5.3:** describe ocular manifestations of leprosy
- **SLO 5.4:** describe the pathogenesis and staging of trachoma
- **SLO 5.5:** describe the complications and prevention of trachoma, and
- **SLO 5.6:** describe the pathogenesis, clinical features, and prevention of ocular onchocerciasis.

5.1 Ocular Manifestations of HIV/AIDS

5.1.1 anterior segment manifestations of hiv/aids

Findings across immunosuppression severity

Anterior segment manifestations of HIV/AIDS include a genuinely increased susceptibility to common infections such as **molluscum contagiosum**, herpes zoster ophthalmicus, and microbial keratitis, each occurring more frequently, and often with a more severe or atypical course, than in immunocompetent patients.

Kaposi sarcoma, a vascular neoplasm strongly associated with human herpesvirus 8, may present as a purplish conjunctival or eyelid lesion, occasionally the presenting sign leading to an initial HIV diagnosis, meaning careful examination of these structures carries genuine diagnostic value beyond simply the eye itself.

Fill in the blank: Kaposi sarcoma, a vascular neoplasm strongly associated with human herpesvirus 8, may present as a purplish conjunctival or eyelid _____.

5.1.2 HIV retinopathy and cmv retinitis

Findings that track the degree of immunosuppression

HIV retinopathy, the most common ocular manifestation of HIV/AIDS overall, produces cotton wool spots and occasional small retinal haemorrhages, generally reflecting direct microvascular effects of the virus rather than opportunistic infection, and is typically asymptomatic.

Cytomegalovirus retinitis, by contrast, represents a genuinely sight-threatening opportunistic infection, occurring specifically at very low CD4 counts, producing a characteristic full-thickness retinal necrosis with haemorrhage, often described as a "pizza pie" or "cheese and ketchup" fundus appearance, requiring urgent antiviral treatment to prevent progression to retinal detachment or bilateral blindness.

Fill in the blank: Cytomegalovirus retinitis occurs specifically at very low CD4 counts, producing a characteristic full-thickness retinal necrosis with _____.

5.1.3 other opportunistic and neoplastic ocular manifestations

A broader range of infective and malignant complications

Further recognised opportunistic ocular infections in advanced HIV/AIDS include ocular toxoplasmosis, presenting as focal retinochoroiditis, and, less commonly, progressive outer retinal necrosis, a rapidly destructive varicella-zoster-related retinitis carrying a genuinely poor visual prognosis even with prompt treatment.

Primary intraocular lymphoma represents a further important, though uncommon, neoplastic manifestation, generally presenting with vitreous cellular infiltration that can closely mimic uveitis, meaning genuine diagnostic vigilance is required in any HIV patient presenting with atypical or treatment-resistant intraocular inflammation.

Fill in the blank: Primary intraocular lymphoma generally presents with vitreous cellular infiltration that can closely mimic _____.

Table 5.1: Comparing HIV retinopathy and CMV retinitis.

Feature	HIV retinopathy	CMV retinitis
Mechanism	Direct microvascular effect	Opportunistic viral infection
CD4 relevance	Occurs across a range of counts	Occurs specifically at very low counts

Visual threat	Generally asymptomatic, benign	Sight-threatening, requires urgent treatment

Pilot questions 5.1

- List anterior segment manifestations of HIV/AIDS. (Linked to SLO 5.1)
- Describe Kaposi sarcoma as an ocular manifestation of HIV/AIDS. (Linked to SLO 5.1)
- Describe HIV retinopathy. (Linked to SLO 5.2)
- Describe CMV retinitis and its characteristic fundus appearance. (Linked to SLO 5.2)
- Describe primary intraocular lymphoma and why it can be diagnostically challenging. (Linked to SLO 5.2)

5.2 Ocular Leprosy and Trachoma

5.2.1 ocular manifestations of leprosy

Findings reflecting both direct infection and nerve damage

Leprosy, caused by *Mycobacterium leprae*, produces ocular manifestations through both direct bacillary invasion of ocular tissue and, indirectly, through facial and trigeminal nerve damage, with **lagophthalmos**, incomplete eyelid closure from facial nerve involvement, and **corneal anaesthesia**, from trigeminal nerve damage, together producing a genuinely dangerous combination for ocular surface integrity.

Direct ocular involvement includes **madarosis**, loss of eyelashes, and **iridocyclitis**, chronic anterior uveitis, each contributing to the substantial overall burden of blindness historically associated with untreated or inadequately treated leprosy.

Fill in the blank: Leprosy produces lagophthalmos from facial nerve involvement and corneal anaesthesia from trigeminal nerve damage, together producing a genuinely dangerous combination for ocular surface _____.

5.2.2 trachoma: pathogenesis and staging

A chronic conjunctival infection with defined disease stages

Trachoma, caused by *Chlamydia trachomatis*, produces a chronic, recurrent conjunctival infection generally acquired in early childhood, staged using the World Health Organization's

simplified grading system, spanning from follicular inflammation through conjunctival scarring to the disease's most sight-threatening late stage.

Repeated episodes of infection and inflammation, generally occurring across many years, progressively produce conjunctival scarring, which in turn distorts the eyelid, leading eventually to **trichiasis**, inward-turned eyelashes, discussed further in the next sub-Part, the final common pathway toward the disease's most severe complications.

Fill in the blank: Trachoma is staged using the World Health Organization's simplified grading system, spanning from follicular inflammation through conjunctival scarring to the disease's most sight-threatening late _____.

5.2.3 complications and prevention of trachoma

Trichiasis-driven corneal damage and public health prevention

Trichiasis, discussed in the previous sub-Part, produces ongoing mechanical corneal abrasion from the misdirected lashes, eventually leading to corneal scarring and, in advanced cases, genuine blindness, meaning surgical correction of trichiasis represents a critical, sight-preserving intervention wherever this stage is identified.

Prevention follows the World Health Organization's **SAFE strategy**, **S**urgery for trichiasis, **A**ntibiotics to treat active infection, **F**acial cleanliness, and **E**nvironmental improvement addressing water access and sanitation, reflecting the condition's fundamentally public health, rather than purely individual clinical, nature.

Fill in the blank: Trachoma prevention follows the World Health Organization's SAFE strategy: surgery, antibiotics, facial cleanliness, and environmental _____.

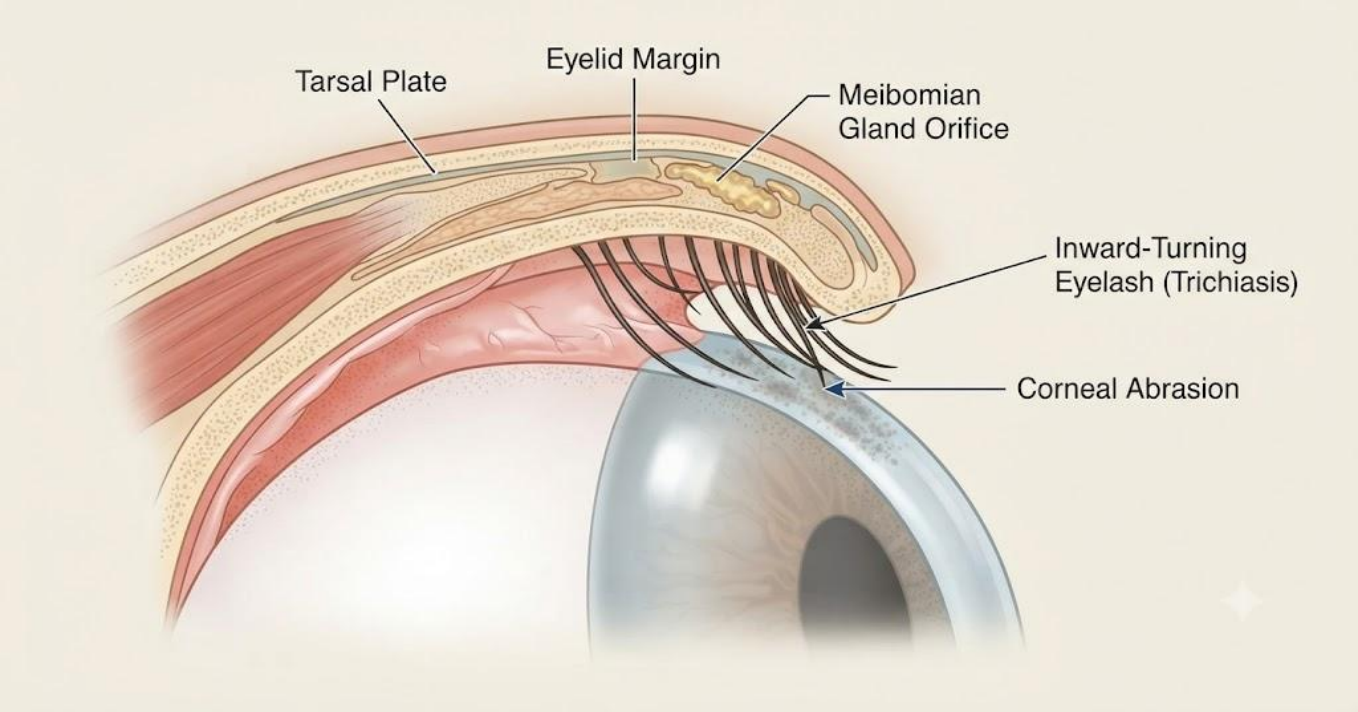

Figure 5.2: A diagram illustrating trichiasis, showing inward-turned eyelashes abrading the corneal surface as a complication of trachoma.

Pilot questions 5.2

1. Describe the ocular manifestations of leprosy relating to facial and trigeminal nerve involvement. (Linked to SLO 5.3)
2. Describe direct ocular manifestations of leprosy, including madarosis and iridocyclitis. (Linked to SLO 5.3)
3. Describe the causative organism and general staging approach of trachoma. (Linked to SLO 5.4)
4. Define trichiasis and describe how it develops in trachoma. (Linked to SLO 5.5)
5. State the components of the WHO SAFE strategy for trachoma prevention. (Linked to SLO 5.5)

5.3 Onchocerciasis

5.3.1 pathogenesis of ocular onchocerciasis

A parasitic infection transmitted by blackfly bite

Onchocerciasis, or river blindness, results from infection with the filarial parasite *Onchocerca volvulus*, transmitted through the bite of infected **Simulium** blackflies, with adult worms producing microfilariae that migrate throughout the skin and, critically, the eye.

Ocular damage results primarily from the host's own inflammatory response to dying microfilariae within ocular tissue, rather than from the parasite's direct mechanical presence alone, meaning the disease's genuinely destructive potential reflects an immune-mediated process rather than simple parasitic occupation.

Fill in the blank: Ocular damage in onchocerciasis results primarily from the host's own inflammatory response to dying microfilariae, rather than from the parasite's direct mechanical presence _____.

5.3.2 anterior and posterior segment features of onchocerciasis

Progressive damage across both segments of the eye

Anterior segment findings include **punctate keratitis**, small, discrete corneal opacities corresponding to dying microfilariae, and, with chronic disease, **sclerosing keratitis**, progressive corneal scarring beginning peripherally and advancing centrally, ultimately threatening vision directly.

Posterior segment involvement includes chorioretinitis and optic atrophy, together contributing substantially to the overall global blindness burden historically attributed to this condition, particularly within heavily endemic regions of sub-Saharan Africa.

Fill in the blank: Anterior segment onchocerciasis findings include punctate keratitis, small, discrete corneal opacities corresponding to dying _____.

5.3.3 prevention and control of onchocerciasis

Mass drug administration as the primary control strategy

Ivermectin, an antiparasitic agent effective against microfilariae, forms the cornerstone of onchocerciasis control, generally delivered through **mass drug administration** programmes across endemic communities, reducing both individual microfilarial burden and, through repeated community-wide treatment, overall transmission.

Vector control measures targeting Simulium blackfly breeding sites, generally fast-flowing rivers and streams, provide a further important complementary control strategy, together with

ivermectin distribution reflecting decades of sustained public health effort that has meaningfully reduced this disease's overall global burden.

Fill in the blank: Ivermectin forms the cornerstone of onchocerciasis control, generally delivered through mass drug administration programmes reducing both individual microfilarial burden and overall _____.

Table 5.3: Comparing key features of trachoma and onchocerciasis.

Feature		**Trachoma**	**Oncho**
Causative organism		Chlamydia trachomatis	Onchoc
Mode of transmission		Direct and fomite spread, often in childhood	Blackfl
Primary prevention strategy		WHO SAFE strategy	Mass iv vector

Pilot questions 5.3

1. Describe the causative organism and transmission of onchocerciasis. (Linked to SLO 5.6)
2. Explain the mechanism by which onchocerciasis produces ocular damage. (Linked to SLO 5.6)
3. Describe punctate and sclerosing keratitis in onchocerciasis. (Linked to SLO 5.6)
4. Describe posterior segment involvement in onchocerciasis. (Linked to SLO 5.6)
5. Describe the primary strategies used to control onchocerciasis. (Linked to SLO 5.6)

Series Summary

This final Series covered the ocular manifestations of HIV/AIDS, spanning anterior segment findings, HIV retinopathy, CMV retinitis, and further opportunistic and neoplastic complications, ocular leprosy and trachoma, including trachoma's staging, trichiasis complications, and the WHO SAFE prevention strategy, and onchocerciasis, its immune-mediated pathogenesis, anterior and posterior segment features, and control through mass drug administration.

Across this entire course, you have now worked through retinal anatomy and dilated fundus examination, the fundus signs of hypertension and diabetes, visual field testing and amaurosis fugax, sickle cell disease, thyroid orbitopathy, and myasthenia gravis, and, finally, HIV and

tropical ophthalmology, together forming a genuinely comprehensive introduction to the ocular manifestations of systemic disease addressed throughout OPT 611.

Glossary of Terms

1. **Kaposi sarcoma:** a vascular neoplasm associated with human herpesvirus 8, occurring in HIV/AIDS.
2. **HIV retinopathy:** cotton wool spots and haemorrhage from direct HIV microvascular effect.
3. **CMV retinitis:** sight-threatening opportunistic retinal necrosis occurring at very low CD4 counts.
4. **Lagophthalmos:** incomplete eyelid closure, in leprosy resulting from facial nerve involvement.
5. **Madarosis:** loss of eyelashes, a direct ocular manifestation of leprosy.
6. **Trachoma:** a chronic conjunctival infection caused by Chlamydia trachomatis.
7. **Trichiasis:** inward-turned eyelashes causing corneal abrasion, a complication of trachoma.
8. **SAFE strategy:** the WHO trachoma prevention framework: Surgery, Antibiotics, Facial cleanliness, Environmental improvement.
9. **Onchocerciasis:** river blindness, caused by the filarial parasite Onchocerca volvulus.
10. **Microfilariae:** the larval stage of Onchocerca volvulus, whose death provokes ocular inflammation.
11. **Sclerosing keratitis:** progressive corneal scarring in chronic onchocerciasis.
12. **Ivermectin:** an antiparasitic agent forming the cornerstone of onchocerciasis control.

Concept Check Questions [Series 5]

Objective questions

1. Kaposi sarcoma, a vascular neoplasm strongly associated with human herpesvirus 8, may present as a purplish conjunctival or eyelid _____.
2. CMV retinitis occurs specifically at very low CD4 counts, producing a characteristic full-thickness retinal necrosis with _____.
3. Trachoma is staged using the World Health Organization's simplified grading system, spanning from follicular inflammation through conjunctival scarring to the disease's most sight-threatening late _____.

4. Ocular damage in onchocerciasis results primarily from the host's own inflammatory response to dying microfilariae, rather than from the parasite's direct mechanical presence _____.
5. Ivermectin forms the cornerstone of onchocerciasis control, generally delivered through mass drug administration programmes reducing both individual microfilarial burden and overall _____.

Short-answer questions

- Describe HIV retinopathy.
- Describe CMV retinitis and its characteristic fundus appearance.
- Describe the ocular manifestations of leprosy relating to facial and trigeminal nerve involvement.
- Define trichiasis and describe how it develops in trachoma.
- Describe the mechanism by which onchocerciasis produces ocular damage.

Long-answer questions

1. Discuss the anterior segment manifestations of HIV/AIDS. (Linked to SLO 5.1)
2. Discuss HIV retinopathy, CMV retinitis, and other opportunistic or neoplastic manifestations of HIV/AIDS. (Linked to SLO 5.2)
3. Discuss the ocular manifestations of leprosy and the pathogenesis and staging of trachoma. (Linked to SLO 5.3, SLO 5.4)
4. Discuss the complications and prevention of trachoma. (Linked to SLO 5.5)
5. Discuss the pathogenesis, clinical features, and prevention of ocular onchocerciasis. (Linked to SLO 5.6)

Answers to Concept Check Questions [Series 5]

Objective questions

6. Lesion.
7. Haemorrhage.
8. Stage.
9. Alone.
10. Transmission.

Short-answer questions

11. Cotton wool spots and occasional small haemorrhages from direct HIV microvascular effect, generally asymptomatic.
12. Sight-threatening retinal necrosis at very low CD4 counts, producing a pizza pie or cheese and ketchup fundus appearance.
13. Lagophthalmos from facial nerve involvement and corneal anaesthesia from trigeminal nerve damage.
14. Inward-turned eyelashes; it develops from conjunctival scarring caused by repeated trachoma infection distorting the eyelid.
15. The host's own inflammatory response to dying microfilariae within ocular tissue.

Long-answer questions

16. **Basic response:** HIV/AIDS produces increased susceptibility to common infections and the vascular neoplasm Kaposi sarcoma affecting the conjunctiva and eyelid.

Value-added response: These findings occasionally represent the presenting sign leading to an initial HIV diagnosis.

17. **Basic response:** HIV retinopathy is a common, generally asymptomatic microvascular finding, while CMV retinitis is a sight-threatening opportunistic infection at low CD4 counts.

Value-added response: Toxoplasmosis, progressive outer retinal necrosis, and primary intraocular lymphoma represent further recognised, more severe complications.

18. **Basic response:** Leprosy affects the eye directly and through facial/trigeminal nerve damage, while trachoma produces chronic conjunctival infection staged by WHO grading.

Value-added response: Both conditions reflect the interplay between direct infection and secondary structural or neurological damage over time.

19. **Basic response:** Trichiasis from conjunctival scarring produces ongoing corneal abrasion, eventually causing blindness if untreated.

Value-added response: The WHO SAFE strategy addresses both individual treatment and broader public health prevention.

20. **Basic response:** Onchocerciasis pathogenesis is immune-mediated, with corneal and posterior segment damage from the host response to dying microfilariae.

Value-added response: Control relies on mass ivermectin distribution and vector control, reflecting a sustained public health approach.

Answers to Pilot Questions [Series 5]

Part 5.1

11. Increased susceptibility to molluscum contagiosum, herpes zoster ophthalmicus, and microbial keratitis, among others such as Kaposi sarcoma.
12. A vascular neoplasm associated with human herpesvirus 8, presenting as a purplish conjunctival or eyelid lesion.
13. Cotton wool spots and occasional small haemorrhages from direct HIV microvascular effect, generally asymptomatic.
14. Sight-threatening retinal necrosis with haemorrhage occurring at very low CD4 counts, with a pizza pie or cheese and ketchup appearance.
15. A neoplastic manifestation with vitreous cellular infiltration that can closely mimic uveitis, requiring diagnostic vigilance.

Part 5.2

1. Lagophthalmos from facial nerve involvement and corneal anaesthesia from trigeminal nerve damage.
2. Madarosis, loss of eyelashes, and iridocyclitis, chronic anterior uveitis.
3. Chlamydia trachomatis, staged from follicular inflammation through conjunctival scarring to the most sight-threatening late stage.
4. Inward-turned eyelashes; develops from conjunctival scarring caused by repeated infection distorting the eyelid.
5. Surgery, Antibiotics, Facial cleanliness, and Environmental improvement.

Part 5.3

6. Onchocerca volvulus, transmitted through the bite of infected Simulium blackflies.
7. Through the host's own inflammatory response to dying microfilariae within ocular tissue.
8. Punctate keratitis is small, discrete corneal opacities from dying microfilariae; sclerosing keratitis is progressive scarring beginning peripherally.
9. Chorioretinitis and optic atrophy.
10. Mass ivermectin distribution and vector control targeting Simulium breeding sites.

References and Attributions [Series 5]

11. Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
12. Cunningham, E. T., & Margolis, T. P. (1998). Ocular manifestations of HIV infection. New England Journal of Medicine, 339(4), 236-244.
13. World Health Organization (2022). Trachoma: Key Facts and SAFE Strategy Overview.
14. World Health Organization (2022). Onchocerciasis (River Blindness): Key Facts.

Figures, Plates, Tables, and Boxes

1. Table 5.1: Comparing HIV retinopathy and CMV retinitis. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing HIV Retinopathy and CMV Retinitis, listing feature, HIV retinopathy, and CMV retinitis. Ophthalmology reference table style with outside border, no photographic elements."
2. Figure 5.2: A diagram illustrating trichiasis, showing inward-turned eyelashes abrading the corneal surface as a complication of trachoma. AI-generated using the prompt: "A single clean, clinically accurate illustration of an eyelid margin with several eyelashes turned inward, touching the corneal surface with a small labelled arrow indicating contact and abrasion, medical textbook diagram style, neutral background, no photographic realism, no other panels."
3. Table 5.3: Comparing key features of trachoma and onchocerciasis. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Key Features of Trachoma and Onchocerciasis, listing feature, trachoma, and onchocerciasis. Ophthalmology reference table style with outside border, no photographic elements."