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OPT 607

NEURO- OPHTHALMOLOGY



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Course code: OPT 607

Course title: Neuro-Ophthalmology

Course credit load: 1 Unit

Series 1: Neuro-Ophthalmic Pathways and Optic Nerve Disease

- **Part 1.1: Afferent and Pupillary Pathways**
 - Sub Part 1.1.1: The afferent visual pathway
 - Sub Part 1.1.2: The pupillary pathway
 - Sub Part 1.1.3: The ocular autonomic pathway
- **Part 1.2: Ocular Motor Pathway and Cranial Nerve Palsies**
 - Sub Part 1.2.1: The ocular motor pathway
 - Sub Part 1.2.2: Third nerve palsy
 - Sub Part 1.2.3: Fourth and sixth nerve palsy
- **Part 1.3: Optic Nerve Disease**
 - Sub Part 1.3.1: Papilloedema
 - Sub Part 1.3.2: Optic atrophy
 - Sub Part 1.3.3: Papillitis

Introduction

Confident neuro-ophthalmic practice begins with a clear understanding of the essential **neural pathways** governing vision, pupillary function, ocular autonomic control, and ocular motility, together with the most important **optic nerve diseases** encountered in everyday practice. This opening Series establishes this essential anatomical and clinical foundation.

The aim of this Series is to equip you with a thorough understanding of the basic anatomy of the afferent visual pathway, ocular motor pathway, ocular autonomic pathway, and pupillary pathway, optic nerve diseases including papilloedema, optic atrophy, and papillitis, and acute onset third, fourth, and sixth cranial nerve palsy.



Series Learning Outcomes

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

- **SLO 1.1:** describe the afferent visual pathway
- **SLO 1.2:** describe the pupillary pathway and the ocular autonomic pathway
- **SLO 1.3:** describe the ocular motor pathway
- **SLO 1.4:** describe third, fourth, and sixth cranial nerve palsy
- **SLO 1.5:** describe papilloedema and optic atrophy, and
- **SLO 1.6:** describe papillitis.

1.1 Afferent and Pupillary Pathways

1.1.1 the afferent visual pathway

The route conveying visual information from retina to cortex

The **afferent visual pathway**, discussed in relation to phototransduction in an earlier related course, begins at the **retinal photoreceptors**, discussed in an earlier related course, with signal relayed through the retinal bipolar and ganglion cell layers before converging to form the **optic nerve**.

The optic nerve, discussed earlier in this Part, continues to the **optic chiasm**, where nasal retinal fibres decussate, discussed in relation to chiasmal anatomy in an earlier related course, before continuing via the optic tract, lateral geniculate body, optic radiations, and finally the **visual cortex**, completing the pathway's route from retina to conscious visual **perception**.

Fill in the blank: The optic nerve continues to the optic chiasm, where nasal retinal fibres decussate, before continuing via the optic tract, lateral geniculate body, optic radiations, and finally the visual _____.



1.1.2 the pupillary pathway

The **pupillary light reflex pathway**, discussed in relation to pupillary examination in an earlier related course, shares its initial afferent route with the visual pathway, discussed earlier in this Part, but diverges before the lateral geniculate body, instead projecting to the **pretectal nucleus** within the midbrain.

From the pretectal nucleus, discussed earlier in this sub-Part, signals project bilaterally to the **Edinger-Westphal nuclei**, providing the anatomical basis for the consensual light reflex, discussed in an earlier related course, before efferent parasympathetic fibres travel via the **oculomotor nerve** to produce pupillary **constriction**.

Fill in the blank: From the pretectal nucleus, signals project bilaterally to the Edinger-Westphal nuclei, before efferent parasympathetic fibres travel via the oculomotor nerve to produce pupillary _____.

1.1.3 the ocular autonomic pathway

The **ocular sympathetic pathway**, discussed in relation to Müller's muscle innervation in an earlier related course, follows a notably long, three-neuron route, originating in the hypothalamus, descending through the brainstem and cervical spinal cord, before synapsing at the **superior cervical ganglion**.

From the superior cervical ganglion, discussed earlier in this sub-Part, postganglionic sympathetic fibres travel along the internal carotid artery to reach the orbit, providing sympathetic innervation to the pupil dilator muscle and **Müller's muscle**, discussed in an earlier related course, with disruption anywhere along this extended pathway capable of producing **Horner's syndrome**.

Fill in the blank: Disruption anywhere along the extended ocular sympathetic pathway is capable of producing Horner's _____.



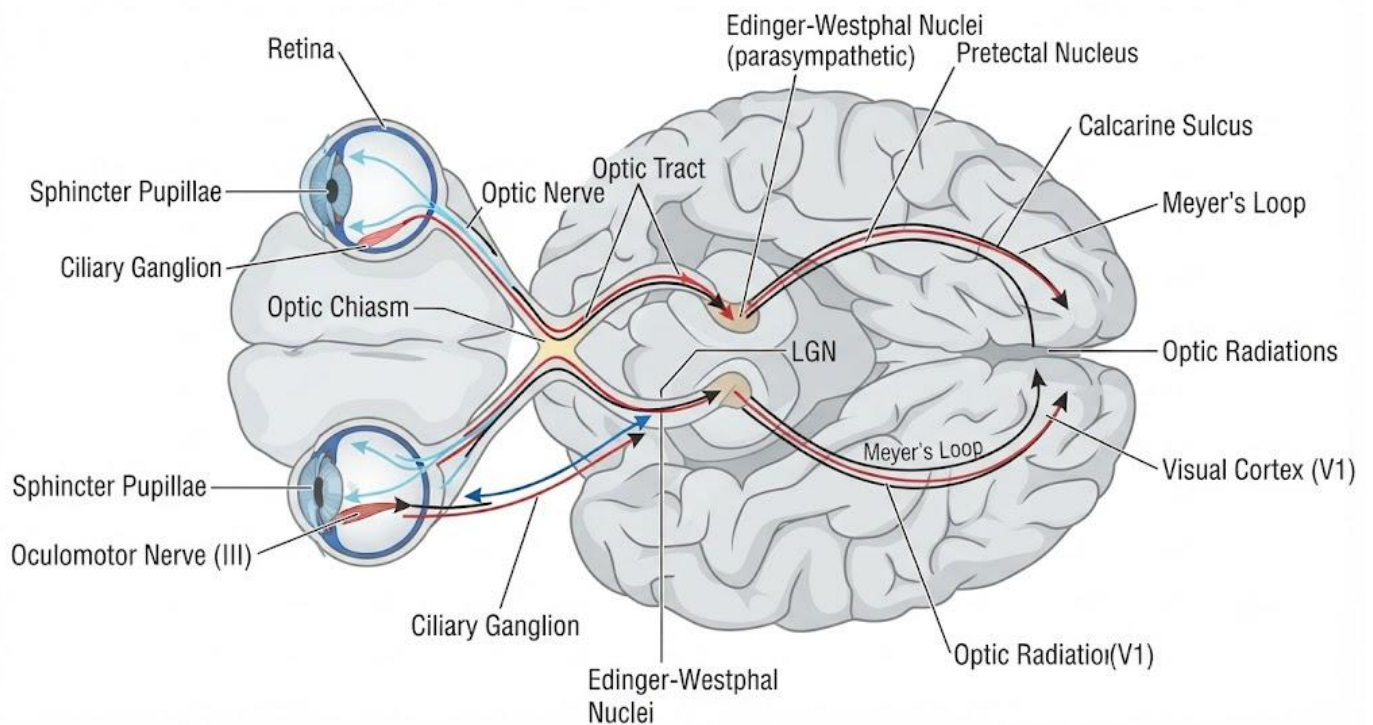


Figure 1.1: Diagram illustrating the afferent visual pathway from retina through the optic chiasm to the visual cortex, and the pupillary pathway diverging at the pretectal nucleus.

Pilot questions 1.1

1. Trace the afferent visual pathway from retina to visual cortex. (Linked to SLO 1.1)
2. Describe where the pupillary pathway diverges from the visual pathway. (Linked to SLO 1.2)
3. Describe the route of the pupillary light reflex pathway from the pretectal nucleus. (Linked to SLO 1.2)
4. Describe the three-neuron route of the ocular sympathetic pathway. (Linked to SLO 1.2)
5. Explain the relevance of the ocular sympathetic pathway to Horner's syndrome. (Linked to SLO 1.2)



1.2 Ocular Motor Pathway and Cranial Nerve Palsies

1.2.1 the ocular motor pathway

Three cranial nerves coordinating eye movement

The **ocular motor pathway**, discussed in relation to extraocular muscle anatomy in an earlier related course, comprises the **oculomotor**, **trochlear**, and **abducens** cranial nerves, originating from their respective brainstem nuclei before travelling through the orbital apex to reach their target extraocular muscles.

Coordinated function of these three cranial nerves, discussed earlier in this Part, produces smooth, conjugate eye movement, discussed in relation to ocular motility examination in an earlier related course, with disruption of any single nerve producing a characteristic, recognisable pattern of restricted movement and **diplopia**, discussed further later in this Series.

Fill in the blank: Coordinated function of the oculomotor, trochlear, and abducens nerves produces smooth, conjugate eye movement, with disruption of any single nerve producing a characteristic, recognisable pattern of restricted movement and _____.

1.2.2 third nerve palsy

Third (oculomotor) nerve palsy, discussed earlier in this Part, typically produces **ptosis**, discussed in relation to levator palpebrae innervation in an earlier related course, an eye positioned "**down and out**" given unopposed lateral rectus and superior oblique action, and, where the pupillary fibres are affected, a fixed, **dilated pupil**.

The specific presence or absence of pupillary involvement, discussed earlier in this sub-Part, carries direct clinical significance, since a **pupil-sparing** third nerve palsy more commonly reflects a microvascular cause, while a **pupil-involving** palsy raises greater concern for **compressive** pathology, such as a posterior communicating artery aneurysm, requiring urgent further investigation.

Fill in the blank: A pupil-sparing third nerve palsy more commonly reflects a microvascular cause, while a pupil-involving palsy raises greater concern for compressive pathology requiring urgent further _____.

1.2.3 fourth and sixth nerve palsy

Fourth (trochlear) nerve palsy, discussed earlier in this Part, produces weakness of the superior oblique muscle, characteristically producing **vertical diplopia**, worse on downgaze and on tilting



the head toward the affected side, with patients frequently adopting a compensatory head tilt away from the affected eye.

Sixth (abducens) nerve palsy, discussed earlier in this Part, produces weakness of the lateral rectus muscle, characteristically producing **horizontal diplopia** worse on gaze toward the affected side, with the affected eye showing an esotropic, or inward, resting position given unopposed medial rectus **action**.

Fill in the blank: Sixth nerve palsy produces weakness of the lateral rectus muscle, with the affected eye showing an esotropic, or inward, resting position given unopposed medial rectus _____.

Table 1.2: Comparing third, fourth, and sixth cranial nerve palsy.

Nerve	Muscle affected	Characteristic finding
Third (oculomotor)	Multiple, including levator and most recti	Ptosis, down-and-out eye, possible dilated pupil
Fourth (trochlear)	Superior oblique	Vertical diplopia worse on downgaze and head tilt
Sixth (abducens)	Lateral rectus	Horizontal diplopia, esotropic resting position

Pilot questions 1.2

1. List the three cranial nerves comprising the ocular motor pathway. (Linked to SLO 1.3)
2. Describe the characteristic clinical features of third nerve palsy. (Linked to SLO 1.4)
3. Explain the clinical significance of pupil involvement in third nerve palsy. (Linked to SLO 1.4)
4. Describe the characteristic features of fourth nerve palsy. (Linked to SLO 1.4)
5. Describe the characteristic features of sixth nerve palsy. (Linked to SLO 1.4)

1.3 Optic Nerve Disease

1.3.1 papilloedema

Optic disc swelling from raised intracranial pressure

Papilloedema, discussed in relation to normal optic disc appearance in an earlier related course, refers specifically to optic disc swelling resulting from **raised intracranial pressure**, generally



producing bilateral disc swelling given the shared cerebrospinal fluid space surrounding both optic nerves.

Characteristic ophthalmoscopic findings include blurred, elevated disc margins, discussed earlier in this sub-Part, loss of the normal physiological cup, and venous engorgement, with vision, in early papilloedema, generally relatively **preserved** despite these striking disc changes, an important distinguishing feature from papillitis discussed further later in this Series.

Fill in the blank: In early papilloedema, vision is generally relatively preserved despite striking disc changes, an important distinguishing feature from _____.

1.3.2 optic atrophy

Optic atrophy, discussed in relation to optic nerve fibre loss, refers to degeneration of optic nerve axons, generally resulting from a wide range of underlying causes including prior optic neuritis, discussed further later in this Series, compressive lesions, or ischaemic optic nerve damage.

Characteristic ophthalmoscopic findings include a **pale**, discussed in relation to normal disc colour in an earlier related course, well defined optic disc, generally accompanied by reduced visual acuity, colour vision impairment, and a visual field **defect**, discussed further in a later Series of this course, reflecting the specific underlying pattern of axonal **loss**.

Fill in the blank: Optic atrophy is characterised by a pale, well defined optic disc, generally accompanied by reduced visual acuity, colour vision impairment, and a visual field _____.

1.3.3 papillitis

Papillitis, discussed in relation to papilloedema earlier in this Series, refers to optic disc swelling resulting from inflammation of the optic nerve head itself, generally representing the anterior form of **optic neuritis**, typically presenting with **unilateral**, painful vision loss, in distinct contrast to the generally bilateral, painless presentation of papilloedema.

Unlike papilloedema, discussed earlier in this Part, papillitis characteristically produces significant, early **visual impairment**, together with reduced colour vision and a relative afferent pupillary defect, discussed in an earlier related course, reflecting genuine optic nerve dysfunction rather than the disc swelling alone characteristic of papilloedema.

Fill in the blank: Unlike papilloedema, papillitis characteristically produces significant, early visual impairment, together with reduced colour vision and a relative afferent pupillary

_____.



Table 1.3: Comparing papilloedema, optic atrophy, and papillitis.

Condition	Ophthalmoscopic appearance	Typical presentation
Papilloedema	Bilateral swollen disc with blurred margins	Generally preserved vision, painless
Optic atrophy	Pale, well defined disc	Reduced acuity, colour vision, and field defect
Papillitis	Unilateral swollen disc	Painful, early significant vision loss

Pilot questions 1.3

1. Define papilloedema and describe its characteristic ophthalmoscopic findings. (Linked to SLO 1.5)
2. Explain why vision is generally preserved in early papilloedema. (Linked to SLO 1.5)
3. Define optic atrophy and describe its characteristic ophthalmoscopic findings. (Linked to SLO 1.5)
4. Define papillitis and describe how it differs from papilloedema. (Linked to SLO 1.6)
5. List the clinical features distinguishing papillitis from papilloedema. (Linked to SLO 1.6)

Series Summary

This opening Series established neuro-ophthalmic pathways and optic nerve disease, beginning with the **afferent and pupillary pathways**, the visual pathway, pupillary pathway, and ocular autonomic pathway, before examining the **ocular motor pathway and cranial nerve palsies**, third, fourth, and sixth nerve palsy.

We concluded with **optic nerve disease**, papilloedema, optic atrophy, and papillitis. Having worked through this Series, you should now be equipped to understand pupillary reactions and ocular motility assessment examined in the next Series of this course.



Glossary of Terms

- **Optic chiasm:** the point where nasal retinal nerve fibres decussate within the visual pathway.
- **Pretectal nucleus:** a midbrain structure receiving afferent pupillary pathway input before projecting to the Edinger-Westphal nuclei.
- **Edinger-Westphal nucleus:** the source of parasympathetic pupilloconstrictor fibres travelling via the oculomotor nerve.
- **Horner's syndrome:** a clinical syndrome resulting from disruption of the ocular sympathetic pathway.
- **Third (oculomotor) nerve palsy:** a palsy typically producing ptosis, a down-and-out eye, and, if pupil-involving, a dilated pupil.
- **Fourth (trochlear) nerve palsy:** a palsy producing vertical diplopia worse on downgaze and head tilt.
- **Sixth (abducens) nerve palsy:** a palsy producing horizontal diplopia and an esotropic resting eye position.
- **Papilloedema:** optic disc swelling resulting from raised intracranial pressure, generally bilateral.
- **Optic atrophy:** degeneration of optic nerve axons, producing a pale, well defined disc.
- **Papillitis:** optic disc swelling from inflammation of the optic nerve head, the anterior form of optic neuritis.
- **Optic neuritis:** inflammation of the optic nerve, presenting as papillitis when anterior.
- **Pupil-sparing palsy:** a nerve palsy in which pupillary function is unaffected, generally suggesting a microvascular cause.

Concept Check Questions [Series 1]

Objective questions

1. The optic nerve continues to the optic chiasm, where nasal retinal fibres decussate, before continuing to the visual _____.
2. Disruption anywhere along the extended ocular sympathetic pathway is capable of producing Horner's _____.
3. A pupil-sparing third nerve palsy more commonly reflects a microvascular cause, while a pupil-involving palsy raises greater concern for compressive pathology requiring urgent further _____.
4. In early papilloedema, vision is generally relatively preserved despite striking disc changes, an important distinguishing feature from _____.



5. Papillitis characteristically produces significant, early visual impairment, together with reduced colour vision and a relative afferent pupillary _____.

Short-answer questions

6. Trace the afferent visual pathway from retina to visual cortex.
7. Describe the three-neuron route of the ocular sympathetic pathway.
8. Describe the characteristic clinical features of third nerve palsy.
9. Describe the characteristic features of fourth and sixth nerve palsy.
10. Define papilloedema and describe its characteristic ophthalmoscopic findings.

Long-answer questions

11. Discuss the afferent visual pathway and the pupillary pathway. (Linked to SLO 1.1, SLO 1.2)
12. Discuss the ocular autonomic pathway and its relevance to Horner's syndrome. (Linked to SLO 1.2)
13. Discuss the ocular motor pathway and third nerve palsy. (Linked to SLO 1.3, SLO 1.4)
14. Discuss fourth and sixth nerve palsy. (Linked to SLO 1.4)
15. Discuss papilloedema, optic atrophy, and papillitis. (Linked to SLO 1.5, SLO 1.6)

Answers to Concept Check Questions [Series 1]

Objective questions

1. Cortex.
2. Syndrome.
3. Investigation.
4. Papillitis.
5. Defect.

Short-answer questions

6. Retina, optic nerve, optic chiasm, optic tract, lateral geniculate body, optic radiations, and visual cortex.
7. Originating in the hypothalamus, descending through the brainstem and cervical cord, synapsing at the superior cervical ganglion, then travelling to the orbit along the internal carotid artery.
8. Ptosis, a down-and-out eye position, and, if pupil-involving, a fixed, dilated pupil.



9. Fourth nerve palsy produces vertical diplopia worse on downgaze and head tilt; sixth nerve palsy produces horizontal diplopia with an esotropic eye position.
10. Optic disc swelling from raised intracranial pressure; findings include blurred, elevated margins, loss of the physiological cup, and venous engorgement.

Long-answer questions

11. **Basic response:** The afferent visual pathway carries visual signal from retina through the optic nerve, chiasm, tract, and radiations to the visual cortex.

Value-added response: The pupillary pathway shares this initial route but diverges before the lateral geniculate body toward the pretectal nucleus and Edinger-Westphal nuclei.

12. **Basic response:** The ocular sympathetic pathway follows a long, three-neuron route from the hypothalamus through the cervical cord to the superior cervical ganglion and along the carotid artery.

Value-added response: Disruption anywhere along this extended pathway can produce Horner's syndrome, reflecting the pathway's vulnerability at multiple anatomical levels.

13. **Basic response:** The ocular motor pathway comprises the oculomotor, trochlear, and abducens nerves coordinating eye movement.

Value-added response: Third nerve palsy produces ptosis, a down-and-out eye, and, if pupil-involving, a dilated pupil, with pupil involvement carrying important diagnostic significance.

14. **Basic response:** Fourth nerve palsy produces vertical diplopia worse on downgaze and head tilt, while sixth nerve palsy produces horizontal diplopia with an esotropic eye.

Value-added response: Each palsy produces a characteristic, recognisable pattern reflecting the specific muscle weakened.

15. **Basic response:** Papilloedema is bilateral disc swelling from raised intracranial pressure with generally preserved vision, while optic atrophy shows a pale disc with reduced function.

Value-added response: Papillitis is unilateral disc swelling from optic nerve inflammation with early, significant painful vision loss, distinguishing it from papilloedema.



Part 1.1

1. Retina, optic nerve, optic chiasm, optic tract, lateral geniculate body, optic radiations, and visual cortex.
2. Before the lateral geniculate body, instead projecting to the pretectal nucleus.
3. Bilateral projection to the Edinger-Westphal nuclei, then efferent parasympathetic fibres via the oculomotor nerve.
4. Hypothalamus, descending through brainstem and cervical cord to the superior cervical ganglion, then along the internal carotid artery to the orbit.
5. Disruption anywhere along this extended pathway can produce Horner's syndrome.

Part 1.2

1. Oculomotor, trochlear, and abducens nerves.
2. Ptosis, a down-and-out eye position, and, if pupil-involving, a fixed, dilated pupil.
3. Pupil-sparing suggests a microvascular cause; pupil-involving raises concern for compressive pathology requiring urgent investigation.
4. Vertical diplopia worse on downgaze and head tilt toward the affected side, with compensatory head tilt away from it.
5. Horizontal diplopia worse on gaze toward the affected side, with an esotropic resting eye position.

Part 1.3

1. Optic disc swelling from raised intracranial pressure; findings include blurred, elevated margins, loss of the cup, and venous engorgement.
2. Because the disc changes reflect raised pressure rather than direct optic nerve dysfunction, at least initially.
3. Degeneration of optic nerve axons; findings include a pale, well defined disc with reduced acuity, colour vision, and field defect.
4. Optic disc swelling from optic nerve head inflammation; it is generally unilateral and painful with early significant vision loss, unlike bilateral, painless papilloedema.
5. Unilateral involvement, pain, early significant vision loss, reduced colour vision, and a relative afferent pupillary defect.



References and Attributions [Series 1]

- Kanski, J. J., & Bowling, B. (2015). *Clinical Ophthalmology: A Systematic Approach* (8th ed.). Elsevier.
- Liu, G. T., Volpe, N. J., & Galetta, S. L. (2019). *Liu, Volpe, and Galetta's Neuro-Ophthalmology* (3rd ed.). Elsevier.
- Riordan-Eva, P., & Augsburger, J. J. (Eds.). (2017). *Vaughan and Asbury's General Ophthalmology* (19th ed.). McGraw-Hill.
- Kline, L. B., & Bajandas, F. J. (2008). *Neuro-Ophthalmology Review Manual* (6th ed.). SLACK Incorporated.

Figures, Plates, Tables, and Boxes

- Figure 1.1: Diagram illustrating the afferent visual pathway from retina through the optic chiasm to the visual cortex, and the pupillary pathway diverging at the pretectal nucleus. AI-generated using the prompt: "A single clean, accurate schematic diagram of the brain and orbits from above, tracing the afferent visual pathway from the retina through the optic nerve, chiasm, tract, and radiations to the visual cortex, with a separate labelled branch showing the pupillary pathway diverging to the pretectal nucleus and Edinger-Westphal nuclei, textbook neuroanatomy diagram style, neutral background, no photographic realism, no other panels."
- Table 1.2: Comparing third, fourth, and sixth cranial nerve palsy. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Third, Fourth, and Sixth Cranial Nerve Palsy, listing nerve, muscle affected, and characteristic finding. Ophthalmology reference table style with outside border, no photographic elements."
- Table 1.3: Comparing papilloedema, optic atrophy, and papillitis. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Papilloedema, Optic Atrophy, and Papillitis, listing condition, ophthalmoscopic appearance, and typical presentation. Ophthalmology reference table style with outside border, no photographic elements."



Course code: OPT 607

Course title: Neuro-Ophthalmology

Course credit load: 1 Unit

Series 2: Pupillary Reactions and Ocular Motility Assessment

- **Part 2.1: Pupillary Examination Technique**
 - Sub Part 2.1.1: Examining pupil size, shape, and symmetry
 - Sub Part 2.1.2: Testing direct and consensual light reflex with a penlight
 - Sub Part 2.1.3: The near reflex and its testing
- **Part 2.2: Relative Afferent Pupillary Defect**
 - Sub Part 2.2.1: The swinging flashlight test technique
 - Sub Part 2.2.2: Pathophysiology of relative afferent pupillary defect
 - Sub Part 2.2.3: Grading and clinical significance of RAPD
- **Part 2.3: Ductions and Versions**
 - Sub Part 2.3.1: Defining ductions and versions
 - Sub Part 2.3.2: Technique for testing ductions
 - Sub Part 2.3.3: Technique for testing versions and recognising restriction

Introduction

In the last Series, we established the neuro-ophthalmic pathways underlying vision, pupillary function, and ocular motility, together with the important optic nerve diseases and cranial nerve palsies these pathways help explain. We now apply that anatomical foundation directly to the bedside, translating the afferent, pupillary, and ocular motor pathways discussed in the previous Series into the practical, hands-on clinical skills of **pupillary examination**, the **swinging flashlight test** for relative afferent pupillary defect, and **ductions and versions testing**, together forming the core neuro-ophthalmic bedside assessment applied to every patient with a suspected neurological or ocular motor complaint.



The aim of this Series is to equip you with a thorough, practical understanding of how to test pupillary reactions with a penlight, recognise relative afferent pupillary defect, and perform ductions and versions testing, together with the underlying rationale for each of these essential clinical techniques and what their specific findings mean for the patient in front of you.

This Series begins with **pupillary examination technique**, before turning to **relative afferent pupillary defect**, its testing, pathophysiology, and clinical significance. It concludes with **ductions and versions**, the practical assessment of monocular and binocular eye movement.

Series Learning Outcomes

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

- **SLO 2.1:** examine pupil size, shape, and symmetry, and test the direct and consensual light reflex with a penlight
- **SLO 2.2:** describe and test the near reflex
- **SLO 2.3:** perform the swinging flashlight test
- **SLO 2.4:** describe the pathophysiology of relative afferent pupillary defect
- **SLO 2.5:** grade relative afferent pupillary defect and explain its clinical significance, and
- **SLO 2.6:** define, and perform testing of, ductions and versions.

2.1 Pupillary Examination Technique

2.1.1 examining pupil size, shape, and symmetry

A careful baseline inspection before any dynamic testing

Pupillary examination, discussed in relation to iris anatomy in an earlier related course, should begin with careful inspection under **dim ambient lighting**, generally using a penlight held obliquely rather than directed straight into the eye, allowing assessment of baseline pupil size, shape, and, critically, **symmetry** between the two eyes before any dynamic light testing is attempted. This baseline assessment should specifically note whether the pupil margin is regular and round or irregular, discussed further in relation to iris pathology in a later Series of this course, since an irregular pupil margin carries its own distinct diagnostic significance quite separate from any reflex abnormality.



A degree of **physiological anisocoria**, a small, generally symmetric difference in pupil size between the two eyes of up to around one millimetre, is a recognised normal finding present in a meaningful proportion of the healthy population, and should not automatically be assumed to reflect underlying pathology. Distinguishing this benign physiological anisocoria from **pathological anisocoria** generally requires assessment of whether the size difference changes under differing lighting conditions, discussed further in the next sub-Part, since a difference that remains genuinely constant in both bright and dim light is considerably more reassuring than one that varies.

Fill in the blank: A degree of physiological anisocoria, a small, generally symmetric difference in pupil size between the two eyes, is a recognised normal finding present in a meaningful proportion of the healthy _____.

2.1.2 testing direct and consensual light reflex with a penlight

Assessing the bilateral pupillary response to unilateral stimulation

The **direct light reflex**, discussed in relation to the pupillary pathway in the previous Series of this course, is tested by directing a penlight beam into one eye from an oblique angle and observing constriction of that same, directly illuminated pupil, while the **consensual light reflex** is assessed simultaneously by observing the contralateral, unstimulated pupil for an equivalent, synchronous degree of constriction. Both reflexes should be tested and recorded for each eye in turn, since a genuinely complete pupillary examination requires four separate observations, the direct and consensual response elicited from stimulating the right eye, and the direct and consensual response elicited from stimulating the left.

A **brisk**, prompt pupillary constriction followed by a degree of sustained constriction, rather than an immediate return to baseline size, represents the expected normal finding, with a sluggish, delayed, or incomplete response, discussed further in relation to RAPD later in this Series, warranting careful further characterisation. The examiner should additionally allow adequate time between successive light stimulations, generally several seconds, to allow the pupil to genuinely return to its resting baseline size before the next test is performed, since testing too rapidly in succession risks producing a misleadingly reduced or fatigued response unrelated to any genuine underlying pathology.

Fill in the blank: The examiner should allow adequate time between successive light stimulations to allow the pupil to genuinely return to its resting baseline size, since testing too rapidly risks producing a misleadingly reduced or fatigued _____.



2.1.3 the near reflex and its testing

Confirming intact accommodative pupillary constriction

The **near reflex**, discussed in relation to the near triad and accommodation physiology in an earlier related course, is tested by asking the patient to shift fixation from a distant target to a near target, generally the examiner's own finger held a short distance in front of the patient's face, while observing for the expected pupillary constriction accompanying this shift in fixation distance. This test is particularly valuable in specific clinical circumstances where the light reflex itself is found to be reduced or absent, since certain recognised conditions can selectively spare the near response while impairing the light response, a dissociation carrying specific diagnostic relevance.

This pattern, termed **light-near dissociation**, occurs when the near reflex remains intact despite a genuinely poor or absent direct and consensual light reflex, reflecting the fact that the near reflex pathway, though anatomically related to the light reflex pathway discussed in the previous Series of this course, is not entirely identical to it and can therefore be selectively affected by certain specific lesions. Recognising this dissociation on careful bedside testing therefore adds genuine diagnostic value beyond simple light reflex assessment alone, and should be routinely tested wherever the light reflex itself appears abnormal.

Fill in the blank: Light-near dissociation occurs when the near reflex remains intact despite a genuinely poor or absent direct and consensual light _____.



Figure 2.1: A clinician performing a penlight pupillary examination, assessing baseline pupil size and symmetry under dim lighting.



Pilot questions 2.1

- Describe the correct technique for baseline pupillary inspection. (Linked to SLO 2.1)
- Define physiological anisocoria and how it is distinguished from pathological anisocoria. (Linked to SLO 2.1)
- Describe how the direct and consensual light reflex are tested. (Linked to SLO 2.1)
- Explain why adequate time should be allowed between successive light stimulations. (Linked to SLO 2.1)
- Define light-near dissociation and explain its diagnostic value. (Linked to SLO 2.2)

2.2 Relative Afferent Pupillary Defect

2.2.1 the swinging flashlight test technique

Alternately illuminating each eye to compare afferent input

The **swinging flashlight test**, discussed in relation to the afferent pupillary pathway in the previous Series of this course, is performed by directing a penlight beam onto one pupil for approximately one to three seconds, observing the resulting constriction, then **swinging** the light smoothly and promptly to the contralateral eye and observing its response, before repeating this alternating sequence several times to build a reliable clinical impression. The light must be moved briskly between the two eyes, avoiding any pause over the nasal bridge, since an unnecessarily slow transition allows both pupils time to partially redilate in darkness, which can obscure or mimic a genuine defect.

In a genuinely normal examination, each eye should show either a **sustained constriction** or, at most, an initial slight constriction followed by only mild dilation as the swinging light reaches it, reflecting essentially symmetric afferent input from both eyes to the shared bilateral pupillary constrictor pathway discussed in the previous Series of this course. Reliable performance of this test requires adequate ambient room lighting to be dimmed beforehand, a sufficiently bright, focused light source, and, ideally, several repeated swings rather than a single pass, since a single observation can occasionally be misleading or difficult to interpret with confidence.

Fill in the blank: The swinging flashlight test requires the light to be moved briskly between the two eyes, avoiding any pause over the nasal bridge, since an unnecessarily slow transition allows both pupils time to partially _____ in darkness.



2.2.2 pathophysiology of relative afferent pupillary defect

Asymmetric afferent signal producing paradoxical dilation

A **relative afferent pupillary defect**, or **RAPD**, discussed in relation to the afferent visual pathway in the previous Series of this course, reflects a genuine asymmetry in the strength of afferent visual signal reaching the brainstem from each eye, generally resulting from unilateral or markedly asymmetric optic nerve or extensive retinal pathology, discussed throughout the previous Series of this course, rather than any abnormality of the efferent, pupil-constricting pathway itself. Because the pupillary constriction response ultimately reflects the combined input from both eyes converging onto the shared bilateral Edinger-Westphal output, discussed in relation to the pupillary pathway earlier in this course, an eye with weaker afferent signal will produce less constriction, and therefore relatively more dilation, when directly stimulated compared with the response elicited when the healthier fellow eye was most recently stimulated.

This produces the characteristic finding of **paradoxical dilation** when the swinging light reaches the affected eye, discussed earlier in this Part, since the pupil appears to dilate in direct response to light being shone into it, a finding that would be genuinely counterintuitive without an understanding of the underlying comparative mechanism involved. It is essential to appreciate that RAPD is, by definition, a **relative**, comparative finding between the two eyes rather than an absolute measure of how poorly either individual eye's own pupil reacts to light, meaning RAPD cannot occur in the setting of genuinely symmetric bilateral optic nerve or retinal disease, even where both eyes are significantly and equally affected.

Fill in the blank: RAPD is, by definition, a relative, comparative finding between the two eyes, meaning it cannot occur in the setting of genuinely symmetric bilateral optic nerve or _____ disease.

2.2.3 grading and clinical significance of rapd

Quantifying severity and interpreting the finding

RAPD is generally graded on a clinical scale from **trace or mild** through to **severe**, or, in more formal neuro-ophthalmic assessment, quantified using **neutral density filters** of increasing strength placed in front of the healthier eye until the paradoxical dilation in the affected eye is neutralised, providing an objective, reproducible numerical estimate of defect severity discussed further in a later Series of this course. This graded, quantified approach supports genuinely reliable tracking of change over time in a given patient, which can carry direct relevance to monitoring disease progression or, conversely, treatment response, in conditions such as optic neuritis discussed in the previous Series of this course.



The presence of a RAPD carries substantial clinical significance as an **objective sign** of genuine optic nerve or extensive retinal pathology, independent of the patient's own subjective visual acuity report, and its absence, conversely, argues meaningfully against significant unilateral or markedly asymmetric optic nerve disease, though it does not entirely exclude bilateral, symmetric pathology as discussed earlier in this Part. Careful documentation of RAPD, whether present or absent, together with its specific graded severity where relevant, should therefore form a routine, essential component of every neuro-ophthalmic and, indeed, general ophthalmic clinical assessment.

Fill in the blank: The presence of a RAPD carries substantial clinical significance as an objective sign of genuine optic nerve or extensive retinal pathology, independent of the patient's own subjective visual acuity _____.

Table 2.2: Key principles of the swinging flashlight test and RAPD.

Principle	Description	Clinical implication
Brisk light transition	Light moved quickly between eyes, avoiding pauses	Prevents redilation artefact
Relative, comparative sign	RAPD compares afferent input between the two eyes	Cannot occur with symmetric bilateral disease
Neutral density grading	Filters of increasing strength neutralise the defect	Provides objective, reproducible severity measurement

Pilot questions 2.2

1. Describe the correct technique for performing the swinging flashlight test. (Linked to SLO 2.3)
2. Explain why the light must be moved briskly between the two eyes during the swinging flashlight test. (Linked to SLO 2.3)
3. Describe the underlying pathophysiology of relative afferent pupillary defect. (Linked to SLO 2.4)
4. Explain why RAPD cannot occur with genuinely symmetric bilateral optic nerve disease. (Linked to SLO 2.4)
5. Describe how RAPD is graded and explain its clinical significance. (Linked to SLO 2.5)

2.3 Ductions and Versions

2.3.1 defining ductions and versions



Monocular versus binocular conjugate eye movement

Ductions, discussed in relation to extraocular muscle anatomy and the ocular motor pathway in the previous Series of this course, refer to monocular eye movements, meaning the movement of a single eye tested independently with the fellow eye occluded, generally assessed in each of the six cardinal directions, adduction, abduction, elevation, depression, and the intermediate oblique directions, to isolate the function of individual extraocular muscles. This monocular testing approach is particularly valuable where a binocular abnormality has already been identified, since it helps determine whether the underlying restriction genuinely reflects weakness of a specific muscle in one eye alone, rather than a more complex binocular coordination problem.

Versions, by contrast, refer to **binocular**, conjugate eye movements, in which both eyes move together in the same direction simultaneously, generally assessed by asking the patient to follow a moving target through the same cardinal positions of gaze discussed earlier in this sub-Part, with both eyes open and viewing together. Versions testing reflects the coordinated, yoked action of specific muscle pairs across both eyes, discussed further in the next sub-Part, and represents the more commonly performed routine clinical test in everyday ophthalmic and neuro-ophthalmic practice, since most patients present with a binocular visual complaint such as diplopia rather than isolated monocular symptoms.

Fill in the blank: Versions refer to binocular, conjugate eye movements, in which both eyes move together in the same direction simultaneously, reflecting the coordinated, yoked action of specific _____ pairs across both eyes.

2.3.2 technique for testing ductions

Isolating single-eye movement with the fellow eye occluded

Ductions are tested by occluding the fellow, non-tested eye, discussed earlier in this Part, and asking the patient to follow a target, generally the examiner's finger or a small fixation object, as it is moved smoothly through each of the six cardinal directions in turn, with the examiner carefully observing the full range and quality of movement achieved in each specific direction. Any restriction identified should be graded, generally on a simple qualitative scale from full, normal range through mild, moderate, and severe restriction, since consistent, reproducible grading supports reliable comparison between the two eyes and reliable tracking of any change over subsequent examinations.

Careful ductions testing, discussed earlier in this sub-Part, is particularly valuable for distinguishing a genuine, isolated muscle **paresis**, weakness, from **mechanical restriction**, in which the eye is physically prevented from moving fully regardless of how strong the relevant muscle's contraction may genuinely be, such as with orbital trauma or thyroid eye disease



discussed in an earlier related course. This distinction carries direct relevance to further investigation and management planning, since paretic and restrictive causes of limited eye movement require genuinely different diagnostic and therapeutic approaches despite potentially appearing superficially similar on cursory observation alone.

Fill in the blank: Careful ductions testing is particularly valuable for distinguishing a genuine, isolated muscle paresis from mechanical restriction, in which the eye is physically prevented from moving fully regardless of the relevant muscle's _____.

2.3.3 technique for testing versions and recognising restriction

Assessing coordinated binocular movement for asymmetry

Versions are tested with both eyes open, discussed earlier in this Part, asking the patient to follow a single target smoothly through each cardinal direction of gaze while the examiner observes both eyes **simultaneously**, specifically comparing the relative position and range of movement achieved by each eye at every point throughout the test sequence, since asymmetry between the two eyes during versions testing represents the key finding of genuine clinical interest. A lag or restriction of one eye relative to the other during a specific direction of gaze generally indicates weakness or restriction of the muscle primarily responsible for movement in that particular direction within the affected eye.

Where versions testing identifies an area of restriction, discussed earlier in this sub-Part, follow-up ductions testing of each eye individually, discussed earlier in this Series, should generally be performed to help localise the specific underlying muscle or nerve involved, since the pattern of restriction observed on versions, correlated with the specific ductions findings in each eye separately, together support confident clinical localisation consistent with the cranial nerve palsy patterns discussed in the previous Series of this course. This combined ductions-and-versions approach represents the genuinely standard, comprehensive clinical method for evaluating any patient presenting with suspected ocular motor dysfunction or diplopia.

Fill in the blank: Where versions testing identifies an area of restriction, follow-up ductions testing of each eye individually should generally be performed to help _____ the specific underlying muscle or nerve involved.

Table 2.3: Comparing ductions and versions testing.

Feature	Ductions	Versions
Eyes tested	One eye at a time, fellow eye occluded	Both eyes together, simultaneously



Primary purpose	Isolate individual muscle function in one eye	Assess coordinated, conjugate binocular movement
Key finding sought	Restricted range in a specific direction	Asymmetry or lag between the two eyes

Pilot questions 2.3

1. Distinguish ductions from versions. (Linked to SLO 2.6)
2. Describe the correct technique for testing ductions. (Linked to SLO 2.6)
3. Explain the value of ductions testing in distinguishing paresis from mechanical restriction. (Linked to SLO 2.6)
4. Describe the correct technique for testing versions. (Linked to SLO 2.6)
5. Explain why ductions testing generally follows identification of restriction on versions testing. (Linked to SLO 2.6)

Series Summary

This Series examined **pupillary reactions and ocular motility assessment**, beginning with **pupillary examination technique**, baseline inspection, the direct and consensual light reflex, and the near reflex, before turning to **relative afferent pupillary defect**, the swinging flashlight test, its underlying pathophysiology, and grading and clinical significance.

We concluded with **ductions and versions**, distinguishing monocular from binocular eye movement testing and the practical technique for each. Having worked through this Series, you should now be equipped to understand visual field testing and visual field defects examined in the next Series of this course.

Glossary of Terms

1. **Physiological anisocoria:** a small, generally symmetric, normal difference in pupil size between the two eyes.
2. **Direct light reflex:** pupillary constriction in the eye directly stimulated by light.
3. **Consensual light reflex:** simultaneous pupillary constriction in the contralateral, unstimulated eye.



4. **Near reflex:** pupillary constriction accompanying a shift in fixation from a distant to a near target.
5. **Light-near dissociation:** an intact near reflex despite a poor or absent light reflex.
6. **Swinging flashlight test:** a test alternately illuminating each eye to detect afferent pathway asymmetry.
7. **Relative afferent pupillary defect (RAPD):** paradoxical pupillary dilation on swinging light toward an eye with afferent pathway disease.
8. **Neutral density filter:** a filter used to objectively quantify RAPD severity.
9. **Ductions:** monocular eye movements tested with the fellow eye occluded.
10. **Versions:** binocular, conjugate eye movements tested with both eyes open.
11. **Paresis:** weakness of a muscle, in this context an extraocular muscle.
12. **Mechanical restriction:** physical limitation of eye movement independent of muscle strength.

Concept Check Questions [Series 2]

Objective questions

1. A degree of physiological anisocoria is a recognised normal finding present in a meaningful proportion of the healthy _____.
2. Light-near dissociation occurs when the near reflex remains intact despite a genuinely poor or absent direct and consensual light _____.
3. The swinging flashlight test requires the light to be moved briskly between the two eyes, since an unnecessarily slow transition allows both pupils time to partially _____ in darkness.
4. RAPD is, by definition, a relative, comparative finding between the two eyes, meaning it cannot occur in the setting of genuinely symmetric bilateral optic nerve or _____ disease.
5. Versions refer to binocular, conjugate eye movements, reflecting the coordinated, yoked action of specific muscle _____ across both eyes.

Short-answer questions

- Describe how the direct and consensual light reflex are tested.
- Describe the correct technique for performing the swinging flashlight test.
- Describe the underlying pathophysiology of relative afferent pupillary defect.
- Distinguish ductions from versions.
- Explain the value of ductions testing in distinguishing paresis from mechanical restriction.



Long-answer questions

1. Discuss pupillary examination technique, including baseline inspection, the light reflex, and the near reflex. (Linked to SLO 2.1, SLO 2.2)
2. Discuss the swinging flashlight test technique and the pathophysiology of relative afferent pupillary defect. (Linked to SLO 2.3, SLO 2.4)
3. Discuss the grading and clinical significance of RAPD. (Linked to SLO 2.5)
4. Discuss ductions and versions and how they are defined and distinguished. (Linked to SLO 2.6)
5. Discuss the practical technique for testing ductions and versions and how findings are correlated. (Linked to SLO 2.6)

Answers to Concept Check Questions [Series 2]

Objective questions

6. Population.
7. Reflex.
8. Redilate.
9. Retinal.
10. Pairs.

Short-answer questions

11. The direct reflex is tested by observing constriction of the directly illuminated eye; the consensual reflex is assessed by observing the contralateral eye simultaneously.
12. Alternately illuminating each eye for one to three seconds, swinging the light briskly between them, and repeating the sequence several times.
13. It reflects asymmetric afferent visual signal reaching the brainstem, producing paradoxical relative dilation in the more affected eye when the light swings toward it.
14. Ductions are monocular movements tested with the fellow eye occluded; versions are binocular, conjugate movements tested with both eyes open.
15. It helps distinguish weakness of a specific muscle in one eye from physical restriction preventing movement regardless of muscle strength.

Long-answer questions

16. **Basic response:** Pupillary examination begins with baseline inspection of size, shape, and symmetry, followed by testing of the direct and consensual light reflex.



Value-added response: The near reflex should additionally be tested, particularly where light-near dissociation is suspected, since it adds genuine diagnostic value beyond light reflex assessment alone.

17. **Basic response:** The swinging flashlight test alternately illuminates each eye to compare afferent input, requiring brisk transitions to avoid redilation artefact.

Value-added response: RAPD reflects asymmetric afferent signal strength between the two eyes, producing characteristic paradoxical dilation in the more affected eye.

18. **Basic response:** RAPD is graded from trace to severe, or quantified using neutral density filters for objective, reproducible measurement.

Value-added response: Its presence provides objective evidence of optic nerve or retinal pathology independent of subjective visual acuity, making it an essential routine examination component.

19. **Basic response:** Ductions test monocular movement with the fellow eye occluded, isolating individual muscle function; versions test binocular, conjugate movement with both eyes open.

Value-added response: This distinction supports different diagnostic purposes, with versions representing the more commonly performed routine clinical test.

20. **Basic response:** Ductions and versions are tested through the cardinal positions of gaze, observing range of movement and any restriction or asymmetry.

Value-added response: Correlating versions findings with subsequent ductions testing in each eye separately supports confident localisation of the underlying muscle or nerve involved.

Answers to Pilot Questions [Series 2]

Part 2.1

11. Under dim ambient lighting, using a penlight held obliquely, assessing baseline size, shape, and symmetry before dynamic testing.
12. Physiological anisocoria is a small, symmetric, normal difference of up to about one millimetre; it is distinguished from pathological anisocoria by whether the difference changes with lighting conditions.



13. By directing light into one eye and observing constriction of that eye (direct) while simultaneously observing the contralateral eye (consensual).
14. Because testing too rapidly risks producing a misleadingly reduced or fatigued response unrelated to genuine pathology.
15. An intact near reflex despite a poor or absent light reflex; it adds diagnostic value since certain lesions selectively spare the near pathway.

Part 2.2

1. Alternately illuminating each eye for one to three seconds, swinging the light briskly between them without pausing, repeated several times.
2. Because an unnecessarily slow transition allows both pupils time to partially redilate in darkness, which can obscure or mimic a genuine defect.
3. It reflects asymmetric afferent visual signal strength reaching the shared bilateral pupillary constrictor pathway from each eye.
4. Because RAPD is a relative, comparative finding between the two eyes, so equally affected eyes produce no detectable asymmetry.
5. Graded from trace to severe, or quantified with neutral density filters; its presence is an objective sign of significant unilateral or asymmetric optic nerve or retinal pathology.

Part 2.3

6. Ductions are monocular movements tested with the fellow eye occluded; versions are binocular, conjugate movements tested with both eyes open.
7. Occluding the fellow eye and following a target through each cardinal direction, grading any restriction observed.
8. It helps determine whether restriction reflects weakness of a specific muscle in one eye or physical restriction independent of muscle strength.
9. With both eyes open, following a single target through cardinal gaze positions while comparing the relative position and range of each eye.
10. Because correlating versions findings with individual ductions testing supports confident localisation of the specific underlying muscle or nerve involved.

References and Attributions [Series 2]

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13. Kline, L. B., & Bajandas, F. J. (2008). Neuro-Ophthalmology Review Manual (6th ed.). SLACK Incorporated.
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Figures, Plates, Tables, and Boxes

1. Figure 2.1: A clinician performing a penlight pupillary examination, assessing baseline pupil size and symmetry under dim lighting. AI-generated using the prompt: "A single clean, realistic clinical illustration of a clinician holding a penlight at an oblique angle toward a seated patient's eyes in a dimly lit room, both attentive, calm clinical setting, no text overlays, no other panels, respectful depiction, no identifying facial features."
2. Table 2.2: Key principles of the swinging flashlight test and RAPD. AI-generated using the prompt: "Create a clean clinical reference table titled Key Principles of the Swinging Flashlight Test and RAPD, listing principle, description, and clinical implication. Ophthalmology reference table style with outside border, no photographic elements."
3. Table 2.3: Comparing ductions and versions testing. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Ductions and Versions Testing, listing feature, ductions, and versions. Ophthalmology reference table style with outside border, no photographic elements."



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Series 3: Visual Field Testing and Visual Field Defects

- **Part 3.1: Confrontation Visual Field Testing Technique**
 - Sub Part 3.1.1: Principles of confrontation visual field testing
 - Sub Part 3.1.2: Testing technique in four quadrants
 - Sub Part 3.1.3: Sources of error and limitations
- **Part 3.2: Scotoma and Localised Visual Field Defects**
 - Sub Part 3.2.1: Defining scotoma
 - Sub Part 3.2.2: Types of scotoma
 - Sub Part 3.2.3: Localising visual field defects to the pathway
- **Part 3.3: Hemianopic Visual Field Defects**
 - Sub Part 3.3.1: Homonymous hemianopia
 - Sub Part 3.3.2: Bitemporal hemianopia
 - Sub Part 3.3.3: Comparing hemianopic patterns and their localisation

Introduction

In the last Series, we established pupillary examination and ocular motility assessment. We now turn to **confrontation visual field testing**, the essential bedside technique for screening the visual field in each of the four quadrants, together with the **visual field defects** this testing may reveal, scotoma and the characteristic hemianopic patterns that carry direct localising value along the afferent visual pathway established in an earlier Series of this course.

The aim of this Series is to equip you with a thorough understanding of confrontation visual field testing in four quadrants for each eye, and visual field defects, including scotoma, hemianopia, homonymous hemianopia, and bitemporal hemianopia.

This Series begins with **confrontation visual field testing technique**, before examining **scotoma and localised visual field defects**. It concludes with **hemianopic visual field defects**.



Series Learning Outcomes

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

- **SLO 3.1:** describe the principles of confrontation visual field testing
- **SLO 3.2:** demonstrate confrontation visual field testing in four quadrants for each eye and recognise its limitations
- **SLO 3.3:** define scotoma and describe types of scotoma
- **SLO 3.4:** localise visual field defects to a specific point along the visual pathway
- **SLO 3.5:** describe homonymous hemianopia, and
- **SLO 3.6:** describe bitemporal hemianopia and compare hemianopic patterns.

3.1 Confrontation Visual Field Testing Technique

3.1.1 principles of confrontation visual field testing

A rapid bedside comparison against the examiner's own field

Confrontation visual field testing, discussed in relation to the visual pathway in an earlier Series of this course, provides a rapid, accessible bedside screening method comparing the patient's own visual field, one eye tested at a time, against the examiner's own, presumed normal, field. Each eye is tested **independently**, with the fellow eye occluded, while the patient maintains steady central fixation on the examiner's face throughout testing.

The examiner is generally positioned directly opposite the patient at a comfortable arm's length distance, with the examiner's own visual field serving as the practical reference standard against which the patient's responses are compared. This approach makes confrontation testing genuinely useful as an efficient screening tool, though it remains considerably less sensitive than formal automated perimetry for detecting subtle field defects.

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



3.1.2 testing technique in four quadrants

Systematic assessment of each visual field quadrant

Testing is generally performed by presenting a moving finger, or, for more precise assessment, a small target, in each of the four **quadrants**, superotemporal, superonasal, inferotemporal, and inferonasal, at the midpoint between the examiner and patient, asking the patient to indicate when the target first becomes visible while maintaining central fixation throughout. Each quadrant should be tested in both eyes separately, and gross confrontation defects can generally be identified through **finger counting** in each quadrant where more subtle detection testing proves unnecessary.

Where a more sensitive assessment is required, static finger counting or the specific **comparison technique**, asking the patient to compare the relative clarity of the examiner's fingers held simultaneously in the patient's two hemifields, can help detect more subtle field asymmetries that might otherwise be missed by simple detection testing alone. Testing all four quadrants of both eyes in this systematic sequence forms the essential minimum standard for any confrontation field examination.

Fill in the blank: Gross confrontation defects can generally be identified through finger counting in each quadrant, while a comparison technique can help detect more subtle field _____.

3.1.3 sources of error and limitations

Recognising the constraints of a screening technique

Confrontation testing, discussed throughout this Part, carries important recognised **limitations**, since it can reliably detect only relatively dense, substantial field defects, meaning genuinely subtle or early field loss may be entirely missed despite careful, technically correct testing. Poor patient **fixation** during testing represents a further common, avoidable source of unreliable results, since drifting fixation can falsely suggest either normal field or an apparent defect that does not genuinely exist.

Given these recognised limitations, confrontation testing should be understood as a genuine **screening** tool rather than a definitive diagnostic assessment, with any identified or suspected abnormality warranting referral for formal, automated perimetry, discussed further in a later Series of this course, to fully characterise the specific field defect pattern and its precise severity.

Fill in the blank: Given the recognised limitations of confrontation testing, any identified or suspected abnormality warrants referral for formal, automated _____.





Figure 3.1: Diagram illustrating confrontation visual field testing, with the examiner presenting a target in each of the four quadrants while the patient maintains central fixation.

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 the comparison technique used for more sensitive field assessment. (Linked to SLO 3.2)
- List the recognised limitations of confrontation visual field testing. (Linked to SLO 3.2)
- Explain why confrontation testing should be regarded as a screening tool rather than a definitive assessment. (Linked to SLO 3.2)

3.2 Scotoma and Localised Visual Field Defects

3.2.1 defining scotoma

An isolated area of visual loss within an otherwise normal field

A **scotoma**, discussed in relation to visual field terminology, refers to an area of reduced or absent visual sensitivity within the visual field, surrounded by an area of relatively preserved,



normal vision, discussed further later in this Part. This isolated pattern of visual loss distinguishes a scotoma from more generalised field constriction or the hemianopic patterns discussed further later in this Series.

A **physiological scotoma**, corresponding to the normal optic disc's lack of photoreceptors, discussed in relation to retinal anatomy in an earlier related course, is a genuinely normal finding present in every healthy visual field, located roughly fifteen degrees temporal to central fixation, and should not be mistaken for a pathological finding on careful field testing.

Fill in the blank: A scotoma refers to an area of reduced or absent visual sensitivity within the visual field, surrounded by an area of relatively preserved, normal _____.

3.2.2 types of scotoma

Distinguishing patterns of pathological visual loss

A **central scotoma**, discussed in relation to macular function in an earlier related course, involves the point of fixation itself, generally producing significant, disproportionate reduction in visual acuity given the central retina's role in high-resolution vision, and is classically associated with optic neuritis, discussed in an earlier Series of this course, or macular disease.

An **arcuate scotoma**, following the characteristic curved course of the retinal nerve fibre layer, discussed in relation to nerve fibre anatomy in an earlier related course, carries particular relevance to glaucomatous optic nerve damage, discussed further in a later Series of this course, while a **paracentral scotoma** lies close to, but does not directly involve, the point of fixation itself.

Fill in the blank: An arcuate scotoma follows the characteristic curved course of the retinal nerve fibre layer, carrying particular relevance to glaucomatous optic nerve _____.

3.2.3 localising visual field defects to the pathway

Applying pathway anatomy to interpret field patterns

The specific **pattern** of a visual field defect, discussed throughout this Part, carries direct localising value along the afferent visual pathway established in an earlier Series of this course, since lesions at different points along this pathway, from retina through optic nerve, chiasm, and optic radiations to the visual cortex, each produce genuinely characteristic, recognisable field defect patterns.

A defect confined to **one eye** generally localises to that eye's own retina or optic nerve, occurring before the optic chiasm, discussed in an earlier Series of this course, while defects affecting



corresponding portions of **both** visual fields generally indicate a lesion at or posterior to the chiasm, discussed further in the next Part of this Series.

Fill in the blank: A defect confined to one eye generally localises to that eye's own retina or optic nerve, occurring before the optic _____.

Table 3.2: Comparing types of scotoma.

Type	Location	Typical association
Central scotoma	Involves the point of fixation	Optic neuritis, macular disease
Arcuate scotoma	Follows the retinal nerve fibre layer course	Glaucomatous optic neuropathy
Paracentral scotoma	Close to, but not involving, fixation	Early localised nerve fibre damage

Pilot questions 3.2

1. Define scotoma. (Linked to SLO 3.3)
2. Define physiological scotoma and its normal location. (Linked to SLO 3.3)
3. Describe central and arcuate scotoma and their typical associations. (Linked to SLO 3.3)
4. Explain how a unilateral field defect is generally localised along the visual pathway. (Linked to SLO 3.4)
5. Explain how a bilateral, corresponding field defect is generally localised. (Linked to SLO 3.4)

3.3 Hemianopic Visual Field Defects

3.3.1 homonymous hemianopia

Loss of corresponding fields on the same side in both eyes

Homonymous hemianopia, discussed in relation to visual pathway anatomy in an earlier Series of this course, describes loss of the same, corresponding half of the visual field in both eyes, either the right or left hemifield, resulting from a lesion affecting the optic tract, optic radiations, or visual cortex, discussed further in this sub-Part, posterior to the optic chiasm.

The degree of **congruity**, or similarity in shape and size, between the field defects in each eye carries further localising value, with more congruous defects generally reflecting more posterior lesions closer to the visual cortex, and less congruous defects generally reflecting more anterior lesions closer to the optic tract itself.



Fill in the blank: Homonymous hemianopia describes loss of the same, corresponding half of the visual field in both eyes, resulting from a lesion posterior to the optic _____.

3.3.2 bitemporal hemianopia

A defect classically localising to the chiasm

Bitemporal hemianopia, discussed in relation to chiasmal anatomy in an earlier Series of this course, 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 affected.

This pattern is classically associated with a **pituitary tumour**, discussed in relation to chiasmal compression from below, given the chiasm's close anatomical relationship to the pituitary gland, with the specific pattern of field loss providing a genuinely important clinical clue toward this underlying diagnosis even before further imaging is obtained.

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

3.3.3 comparing hemianopic patterns and their localisation

Applying field pattern to confidently localise the lesion

Distinguishing homonymous from bitemporal hemianopia, discussed throughout this Part, rests on whether the field defect involves the **same** side in both eyes, homonymous, or the **outer**, temporal field in each eye, bitemporal, with this distinction directly reflecting the specific anatomical location of the underlying lesion relative to the optic chiasm.

Careful confrontation testing, discussed earlier in this Series, combined with careful attention to this specific pattern of field loss, therefore allows the examiner to generate a genuinely confident anatomical localisation at the bedside, directly guiding appropriate further neuro-ophthalmic and neuroimaging **investigation**.

Fill in the blank: Careful confrontation testing combined with attention to the specific pattern of field loss allows the examiner to generate a genuinely confident anatomical localisation at the bedside, directly guiding appropriate further _____.



Table 3.3: 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 3.3

1. Define homonymous hemianopia. (Linked to SLO 3.5)
2. Explain how congruity of a homonymous defect assists localisation. (Linked to SLO 3.5)
3. Define bitemporal hemianopia and its classic underlying cause. (Linked to SLO 3.6)
4. Explain why bitemporal hemianopia results specifically from a chiasmal lesion. (Linked to SLO 3.6)
5. Distinguish homonymous from bitemporal hemianopia. (Linked to SLO 3.6)

Series Summary

This Series examined **visual field testing and visual field defects**, beginning with **confrontation visual field testing technique**, its principles, four-quadrant testing method, and recognised limitations, before examining **scotoma and localised visual field defects**, definitions, types, and their localising value.

We concluded with **hemianopic visual field defects**, homonymous and bitemporal hemianopia and their comparison. Having worked through this Series, you should now be equipped to understand the anterior chamber angle, aqueous humour, and intraocular pressure 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. **Scotoma:** an area of reduced or absent visual sensitivity surrounded by relatively preserved vision.
3. **Physiological scotoma:** the normal blind spot corresponding to the optic disc.
4. **Central scotoma:** a scotoma involving the point of fixation, associated with optic neuritis or macular disease.
5. **Arcuate scotoma:** a scotoma following the curved course of the retinal nerve fibre layer, relevant to glaucoma.
6. **Homonymous hemianopia:** loss of the same, corresponding half of the visual field in both eyes.
7. **Bitemporal hemianopia:** loss of the outer, temporal half of the visual field in each eye, classically from a chiasmal lesion.
8. **Congruity:** the degree of similarity in shape and size between field defects in each eye.
9. **Optic chiasm:** the point where nasal retinal fibres decussate, the classic site of bitemporal hemianopia.
10. **Optic tract:** the pathway continuing from the chiasm toward the lateral geniculate body.
11. **Optic radiations:** fibres continuing from the lateral geniculate body toward the visual cortex.
12. **Pituitary tumour:** a mass classically associated with bitemporal hemianopia given its proximity to the chiasm.

Concept Check Questions [Series 3]

Objective questions

1. Confrontation visual field testing compares the patient's own visual field against the examiner's own, presumed normal, _____.
2. Given the recognised limitations of confrontation testing, any identified or suspected abnormality warrants referral for formal, automated _____.
3. An arcuate scotoma follows the characteristic curved course of the retinal nerve fibre layer, carrying particular relevance to glaucomatous optic nerve _____.
4. Homonymous hemianopia describes loss of the same, corresponding half of the visual field in both eyes, resulting from a lesion posterior to the optic _____.
5. Bitemporal hemianopia is classically associated with a pituitary tumour, given the chiasm's close anatomical relationship to the pituitary _____.

Short-answer questions

- Describe how each of the four quadrants is tested in confrontation testing.



- Define scotoma and physiological scotoma.
- Describe central and arcuate scotoma and their typical associations.
- Define homonymous hemianopia.
- Define bitemporal hemianopia and its classic underlying cause.

Long-answer questions

1. Discuss the principles and technique of confrontation visual field testing in four quadrants. (Linked to SLO 3.1, SLO 3.2)
2. Discuss the limitations of confrontation visual field testing. (Linked to SLO 3.2)
3. Discuss scotoma, its definition, and types. (Linked to SLO 3.3)
4. Discuss how visual field defects are localised along the visual pathway. (Linked to SLO 3.4)
5. Discuss homonymous and bitemporal hemianopia and how they are compared and localised. (Linked to SLO 3.5, SLO 3.6)

Answers to Concept Check Questions [Series 3]

Objective questions

6. Field.
7. Perimetry.
8. Damage.
9. Chiasm.
10. Gland.

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. Scotoma is an area of reduced or absent visual sensitivity surrounded by preserved vision; physiological scotoma is the normal blind spot corresponding to the optic disc.
13. Central scotoma involves fixation, associated with optic neuritis or macular disease; arcuate scotoma follows the nerve fibre layer course, associated with glaucoma.
14. Loss of the same, corresponding half of the visual field in both eyes.
15. Loss of the outer, temporal half of the visual field in each eye; classically caused by a pituitary tumour.



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 using a moving target or finger counting.

Value-added response: More sensitive comparison techniques can detect subtler asymmetries, though the overall approach remains a screening rather than definitive assessment.

17. **Basic response:** Confrontation testing can only reliably detect relatively dense defects and is vulnerable to poor patient fixation.

Value-added response: Any identified or suspected abnormality should prompt referral for formal, automated perimetry to fully characterise the defect.

18. **Basic response:** A scotoma is an isolated area of visual loss surrounded by normal vision, distinguished from generalised constriction or hemianopic patterns.

Value-added response: Central, arcuate, and paracentral scotomas each carry characteristic associations, with arcuate scotoma particularly relevant to glaucoma.

19. **Basic response:** A unilateral field defect generally localises before the chiasm, to the retina or optic nerve of that eye alone.

Value-added response: A bilateral, corresponding defect generally indicates a lesion at or posterior to the chiasm, reflecting the specific point of pathway crossing.

20. **Basic response:** Homonymous hemianopia affects the same side in both eyes from a postchiasmal lesion, while bitemporal hemianopia affects the outer field in each eye from a chiasmal lesion.

Value-added response: Congruity assists further localisation of homonymous defects, while the bitemporal pattern classically points toward a pituitary tumour.



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 while maintaining central fixation.
13. Asking the patient to compare 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. Because it detects only dense defects and remains considerably less sensitive than formal automated perimetry.

Part 3.2

1. An area of reduced or absent visual sensitivity surrounded by relatively preserved, normal vision.
2. The normal blind spot corresponding to the optic disc, located roughly fifteen degrees temporal to fixation.
3. Central scotoma involves fixation, associated with optic neuritis or macular disease; arcuate scotoma follows the nerve fibre layer, associated with glaucoma.
4. It generally localises to that eye's own retina or optic nerve, before the optic chiasm.
5. It generally indicates a lesion at or posterior to the optic chiasm.

Part 3.3

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



References and Attributions [Series 3]

11. Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
12. Liu, G. T., Volpe, N. J., & Galetta, S. L. (2019). Liu, Volpe, and Galetta's Neuro-Ophthalmology (3rd ed.). Elsevier.
13. Bickley, L. S. (2020). Bates' Guide to Physical Examination and History Taking (13th ed.). Wolters Kluwer.
14. Kline, L. B., & Bajandas, F. J. (2008). Neuro-Ophthalmology Review Manual (6th ed.). SLACK Incorporated.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: Diagram illustrating confrontation visual field testing, with the examiner presenting a target in each of the four quadrants while the patient maintains central fixation. 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 at an upper quadrant position, both facing each other, calm clinical setting, no text overlays, no other panels, respectful depiction, no identifying facial features."
2. Table 3.2: Comparing types of scotoma. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Types of Scotoma, listing type, location, and typical association. Ophthalmology reference table style with outside border, no photographic elements."
3. Table 3.3: 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."



Course code: OPT 607

Course title: Neuro-Ophthalmology

Course credit load: 1 Unit

Series 4: Anterior Chamber Angle, Aqueous Humour, and Intraocular Pressure

- **Part 4.1: Anatomy of the Anterior Chamber Angle**
 - Sub Part 4.1.1: Structures forming the anterior chamber angle
 - Sub Part 4.1.2: The trabecular meshwork
 - Sub Part 4.1.3: Anterior chamber depth estimation with a pen torch
- **Part 4.2: Aqueous Humour Production and Circulation**
 - Sub Part 4.2.1: Aqueous humour production
 - Sub Part 4.2.2: Circulation of aqueous humour
 - Sub Part 4.2.3: Outflow pathways
- **Part 4.3: Intraocular Pressure and Tonometry**
 - Sub Part 4.3.1: Normal intraocular pressure and diurnal variation
 - Sub Part 4.3.2: Types of tonometry
 - Sub Part 4.3.3: Clinical relevance of accurate IOP measurement

Introduction

In the last Series, we established confrontation visual field testing and visual field defects. We now turn to the **anterior chamber angle**, its underlying anatomy and clinical assessment, **aqueous humour** production and circulation, and **intraocular pressure**, together with the practical tonometry techniques used to measure it, forming the essential foundation for understanding glaucoma examined in the final Series of this course.

The aim of this Series is to equip you with a thorough understanding of the basic anatomy of the anterior chamber angle, aqueous humour production and circulation, the normal value range and diurnal variation of intraocular pressure, and types of tonometry.



This Series begins with the **anatomy of the anterior chamber angle**, before examining **aqueous humour production and circulation**. It concludes with **intraocular pressure and tonometry**.

Series Learning Outcomes

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

- **SLO 4.1:** describe the structures forming the anterior chamber angle and the trabecular meshwork
- **SLO 4.2:** demonstrate anterior chamber depth estimation with a pen torch
- **SLO 4.3:** describe aqueous humour production
- **SLO 4.4:** describe the circulation and outflow pathways of aqueous humour
- **SLO 4.5:** enumerate the normal value range and diurnal variation of intraocular pressure, and
- **SLO 4.6:** describe types of tonometry and the clinical relevance of accurate IOP measurement.

4.1 Anatomy of the Anterior Chamber Angle

4.1.1 structures forming the anterior chamber angle

The junction between the cornea and iris root

The **anterior chamber angle**, discussed in relation to anterior chamber anatomy in an earlier related course, is formed at the junction between the peripheral cornea and the iris root, bordered by the **corneoscleral junction**, the **trabecular meshwork**, discussed further in the next sub-Part, and the **ciliary body band**.

The overall **width** of this angle, whether open or narrowed, directly determines the ease with which aqueous humour, discussed further later in this Series, can access the trabecular meshwork for drainage, meaning angle anatomy carries direct relevance to glaucoma risk discussed further in a later Series of this course.

Fill in the blank: The overall width of the anterior chamber angle directly determines the ease with which aqueous humour can access the trabecular meshwork for _____.



4.1.2 the trabecular meshwork

A sponge-like structure providing the principal drainage route

The **trabecular meshwork**, discussed earlier in this Part, is a sponge-like, porous structure located within the anterior chamber angle, providing the principal pathway through which aqueous humour drains from the anterior chamber into **Schlemm's canal**, discussed further later in this Series.

The trabecular meshwork's resistance to aqueous outflow, discussed earlier in this sub-Part, represents the primary physiological determinant of intraocular pressure, discussed further later in this Series, meaning structural or functional changes within this specific tissue carry direct relevance to **glaucoma** pathogenesis.

Fill in the blank: The trabecular meshwork's resistance to aqueous outflow represents the primary physiological determinant of intraocular _____.

4.1.3 anterior chamber depth estimation with a pen torch

A simple bedside screening technique for angle narrowing

Anterior chamber depth estimation, discussed in relation to angle anatomy earlier in this Part, is performed by directing a penlight beam from the temporal side across the eye, observing the extent to which the nasal iris is illuminated relative to the temporal iris.

In a **deep**, open angle, the entire iris surface is generally illuminated relatively evenly, while in a **shallow**, narrow angle, the nasal iris remains in shadow given its bowed, anteriorly displaced configuration, a simple, genuinely useful bedside screening finding relevant to acute angle-closure glaucoma risk discussed further in a later Series of this course.

Fill in the blank: In a shallow, narrow angle, the nasal iris remains in shadow given its bowed, anteriorly displaced _____.



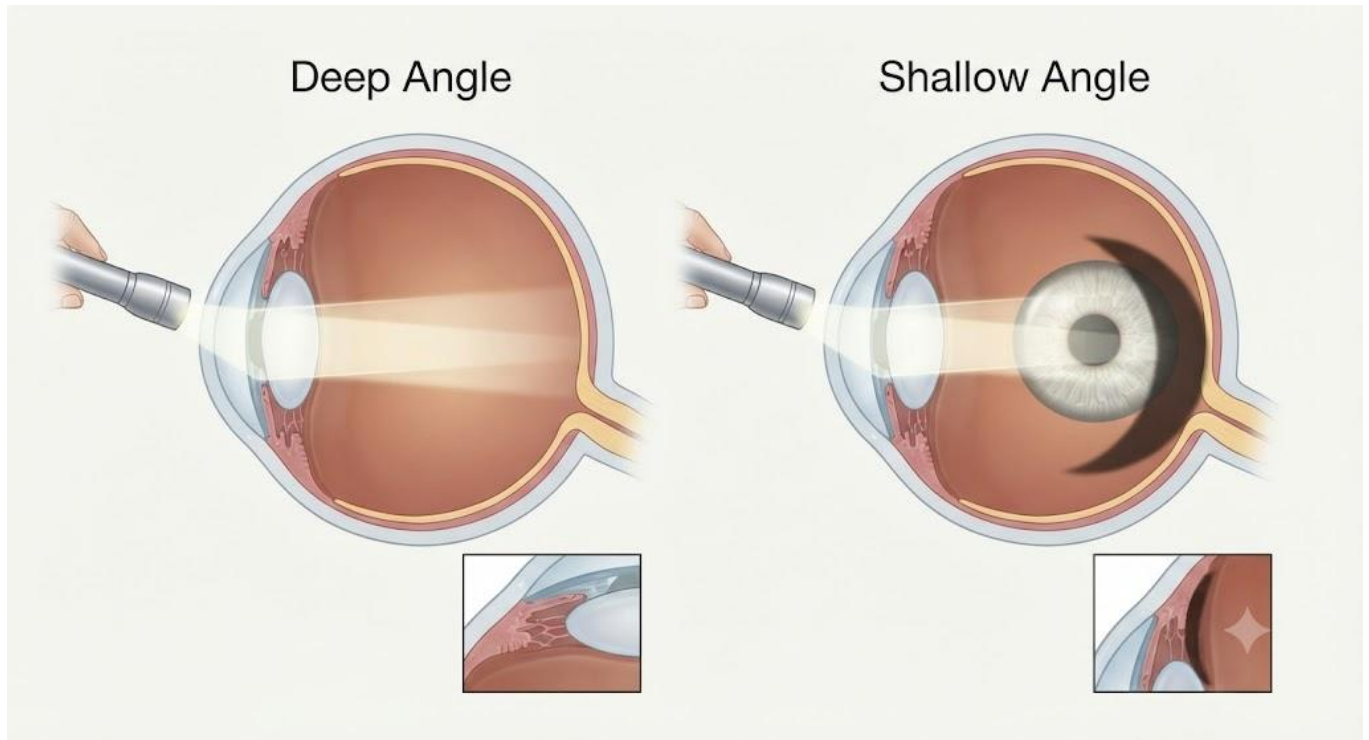


Figure 4.1: Diagram illustrating the pen torch anterior chamber depth estimation technique, comparing an evenly illuminated deep angle with a shadowed nasal iris in a shallow angle.

Pilot questions 4.1

- Describe the structures forming the anterior chamber angle. (Linked to SLO 4.1)
- Explain the clinical relevance of anterior chamber angle width. (Linked to SLO 4.1)
- Describe the trabecular meshwork and its function. (Linked to SLO 4.1)
- Describe the technique for pen torch anterior chamber depth estimation. (Linked to SLO 4.2)
- Distinguish the pen torch appearance of a deep angle from a shallow angle. (Linked to SLO 4.2)

4.2 Aqueous Humour Production and Circulation

4.2.1 aqueous humour production

Active secretion by the ciliary processes

Aqueous humour, discussed in relation to ciliary body anatomy in an earlier related course, is continuously produced by the **ciliary processes**, generally through a combination of active



secretion, ultrafiltration, and simple diffusion, with active secretion representing the predominant **contributing** mechanism.

This continuous production, discussed earlier in this sub-Part, replaces aqueous humour lost through outflow, discussed further later in this Series, maintaining a stable overall aqueous volume and, consequently, a relatively stable intraocular **pressure** under normal physiological circumstances.

Fill in the blank: Aqueous humour is continuously produced by the ciliary processes, replacing aqueous lost through outflow and maintaining a relatively stable intraocular _____.

4.2.2 circulation of aqueous humour

A defined pathway from posterior to anterior chamber

Following production by the ciliary processes, discussed earlier in this Part, aqueous humour flows first into the **posterior chamber**, the space between the iris and lens, before passing through the **pupil** into the **anterior chamber**, discussed in relation to anterior segment anatomy in an earlier related course.

This circulatory route, discussed earlier in this sub-Part, additionally provides essential nutritional support to the avascular lens and cornea, discussed in an earlier related course, before the aqueous ultimately reaches the anterior chamber angle for **drainage**, discussed further in the next sub-Part.

Fill in the blank: The aqueous circulatory route additionally provides essential nutritional support to the avascular lens and cornea before ultimately reaching the anterior chamber angle for _____.

4.2.3 outflow pathways

Conventional and uveoscleral drainage routes

The **conventional**, or trabecular, outflow pathway, discussed earlier in this Series, accounts for the majority of aqueous drainage, passing through the trabecular meshwork into **Schlemm's canal** and subsequently into the episcleral venous system.

The **uveoscleral**, or unconventional, outflow pathway accounts for a smaller proportion of overall drainage, with aqueous instead passing directly through the ciliary muscle and sclera, representing a further recognised route of genuine relevance to certain glaucoma medications discussed further in a later Series of this course.



Fill in the blank: The uveoscleral, or unconventional, outflow pathway accounts for a smaller proportion of overall drainage, with aqueous instead passing directly through the ciliary muscle and _____.

Table 4.2: Comparing conventional and uveoscleral aqueous outflow pathways.

Pathway	Route	Proportion of outflow
Conventional (trabecular)	Trabecular meshwork to Schlemm's canal to episcleral veins	Majority of outflow
Uveoscleral (unconventional)	Directly through ciliary muscle and sclera	Smaller proportion of outflow

Pilot questions 4.2

1. Describe how aqueous humour is produced. (Linked to SLO 4.3)
2. Trace the circulatory pathway of aqueous humour from production to the anterior chamber. (Linked to SLO 4.4)
3. Explain the nutritional role of aqueous humour circulation. (Linked to SLO 4.4)
4. Describe the conventional outflow pathway. (Linked to SLO 4.4)
5. Describe the uveoscleral outflow pathway. (Linked to SLO 4.4)

4.3 Intraocular Pressure and Tonometry

4.3.1 normal intraocular pressure and diurnal variation

A physiological range with recognised daily fluctuation

Intraocular pressure, discussed throughout this Series, normally ranges from approximately **10 to 21 mmHg**, reflecting the balance between aqueous production and outflow discussed earlier in this Series, with values outside this range warranting further clinical evaluation.

Intraocular pressure additionally demonstrates a recognised **diurnal variation**, generally peaking in the early morning hours and gradually declining throughout the day, meaning a single isolated measurement may not fully represent a patient's genuine overall pressure profile, particularly relevant to glaucoma **monitoring** discussed further in a later Series of this course.



Fill in the blank: Intraocular pressure additionally demonstrates a recognised diurnal variation, generally peaking in the early morning hours and gradually declining throughout the _____.

4.3.2 types of tonometry

Methods used to measure intraocular pressure

Goldmann applanation tonometry, discussed in relation to slit lamp examination, represents the current clinical gold standard, measuring the force required to flatten a standardised area of cornea, while **non-contact tonometry**, using a calibrated air pulse, provides a useful, widely available screening alternative.

Further recognised tonometry methods include **Perkins tonometry**, a portable, handheld applanation device particularly useful where the patient cannot be positioned at a standard slit lamp, and **Tono-Pen** tonometry, a further portable device providing rapid, though somewhat less precise, intraocular pressure **measurement**.

Fill in the blank: Perkins tonometry is a portable, handheld applanation device particularly useful where the patient cannot be positioned at a standard slit _____.

4.3.3 clinical relevance of accurate iop measurement

A central component of glaucoma risk assessment

Accurate intraocular pressure measurement, discussed throughout this Part, represents an essential component of glaucoma risk assessment, discussed further in the final Series of this course, given elevated intraocular pressure's status as the single most important modifiable risk factor for glaucomatous optic nerve damage.

Careful attention to technique and recognised sources of **measurement error**, including corneal thickness variation, discussed in relation to corneal anatomy in an earlier related course, remains essential to obtaining genuinely reliable, clinically meaningful intraocular pressure readings across every tonometry method discussed earlier in this Part.

Fill in the blank: Careful attention to technique and recognised sources of measurement error, including corneal thickness variation, remains essential to obtaining genuinely reliable, clinically meaningful intraocular pressure _____.



Table 4.3: Comparing key types of tonometry.

Method	Principle	Typical use
Goldmann applanation	Force required to flatten a standard corneal area	Current clinical gold standard
Non-contact tonometry	Calibrated air pulse	Widely available screening tool
Perkins/Tono-Pen	Portable applanation or electronic measurement	Bedside or non-slit-lamp settings

Pilot questions 4.3

1. State the normal range of intraocular pressure. (Linked to SLO 4.5)
2. Describe the diurnal variation of intraocular pressure and its clinical relevance. (Linked to SLO 4.5)
3. Describe Goldmann applanation tonometry. (Linked to SLO 4.6)
4. Describe non-contact and portable tonometry methods. (Linked to SLO 4.6)
5. Explain the clinical relevance of accurate intraocular pressure measurement. (Linked to SLO 4.6)

Series Summary

This Series examined **anterior chamber angle, aqueous humour, and intraocular pressure**, beginning with the **anatomy of the anterior chamber angle**, its structures, the trabecular meshwork, and pen torch depth estimation, before examining **aqueous humour production and circulation**, production, circulation, and outflow pathways.

We concluded with **intraocular pressure and tonometry**, normal values and diurnal variation, types of tonometry, and the clinical relevance of accurate measurement. Having worked through this Series, you should now be equipped to understand glaucoma examined in the final Series of this course.

Glossary of Terms

1. **Anterior chamber angle:** the junction between the peripheral cornea and iris root.



2. **Trabecular meshwork:** a porous structure providing the principal drainage route for aqueous humour.
3. **Schlemm's canal:** the channel receiving aqueous drainage from the trabecular meshwork.
4. **Anterior chamber depth estimation:** a pen torch technique screening for angle narrowing.
5. **Ciliary processes:** the structures producing aqueous humour.
6. **Conventional outflow pathway:** the trabecular route accounting for the majority of aqueous drainage.
7. **Uveoscleral outflow pathway:** the unconventional route draining aqueous directly through the ciliary muscle and sclera.
8. **Diurnal variation (IOP):** the recognised daily fluctuation in intraocular pressure, generally peaking in the morning.
9. **Goldmann applanation tonometry:** the clinical gold standard method of measuring intraocular pressure.
10. **Non-contact tonometry:** a screening tonometry method using a calibrated air pulse.
11. **Perkins tonometry:** a portable, handheld applanation tonometry device.
12. **Corneal thickness:** a recognised source of tonometry measurement error.

Concept Check Questions [Series 4]

Objective questions

1. The overall width of the anterior chamber angle directly determines the ease with which aqueous humour can access the trabecular meshwork for _____.
2. The trabecular meshwork's resistance to aqueous outflow represents the primary physiological determinant of intraocular _____.
3. Aqueous humour is continuously produced by the ciliary processes, replacing aqueous lost through outflow and maintaining a relatively stable intraocular _____.
4. Intraocular pressure demonstrates a recognised diurnal variation, generally peaking in the early morning hours and gradually declining throughout the _____.
5. Careful attention to technique and recognised sources of measurement error remains essential to obtaining genuinely reliable, clinically meaningful intraocular pressure _____.

Short-answer questions

- Describe the structures forming the anterior chamber angle.



- Describe the technique for pen torch anterior chamber depth estimation.
- Trace the circulatory pathway of aqueous humour from production to the anterior chamber.
- Describe the conventional and uveoscleral outflow pathways.
- State the normal range of intraocular pressure and describe Goldmann applanation tonometry.

Long-answer questions

1. Discuss the anatomy of the anterior chamber angle and the trabecular meshwork. (Linked to SLO 4.1)
2. Discuss anterior chamber depth estimation with a pen torch. (Linked to SLO 4.2)
3. Discuss aqueous humour production and circulation. (Linked to SLO 4.3, SLO 4.4)
4. Discuss the conventional and uveoscleral aqueous outflow pathways. (Linked to SLO 4.4)
5. Discuss the normal value range and diurnal variation of intraocular pressure and types of tonometry. (Linked to SLO 4.5, SLO 4.6)

Answers to Concept Check Questions [Series 4]

Objective questions

6. Drainage.
7. Pressure.
8. Pressure.
9. Day.
10. Readings.

Short-answer questions

11. The corneoscleral junction, the trabecular meshwork, and the ciliary body band.
12. Directing a penlight beam from the temporal side, observing how much of the nasal iris is illuminated.
13. Produced by the ciliary processes into the posterior chamber, through the pupil into the anterior chamber.
14. Conventional: trabecular meshwork to Schlemm's canal to episcleral veins; uveoscleral: directly through the ciliary muscle and sclera.



15. Approximately 10 to 21 mmHg; Goldmann applanation tonometry measures the force required to flatten a standard corneal area, the clinical gold standard.

Long-answer questions

16. **Basic response:** The anterior chamber angle is formed at the corneoscleral junction, trabecular meshwork, and ciliary body band, with angle width relevant to glaucoma risk.

Value-added response: The trabecular meshwork provides the principal aqueous drainage route, with its resistance directly determining intraocular pressure.

17. **Basic response:** Pen torch anterior chamber depth estimation directs light from the temporal side, comparing nasal iris illumination between eyes.

Value-added response: A shallow angle shows a shadowed nasal iris, a useful bedside screening finding for angle-closure risk.

18. **Basic response:** Aqueous humour is produced by the ciliary processes, flowing from the posterior chamber through the pupil to the anterior chamber.

Value-added response: This circulation provides nutritional support to the avascular lens and cornea before reaching the angle for drainage.

19. **Basic response:** The conventional pathway drains the majority of aqueous through the trabecular meshwork and Schlemm's canal.

Value-added response: The uveoscleral pathway drains a smaller proportion directly through the ciliary muscle and sclera, relevant to certain glaucoma medications.

20. **Basic response:** Normal intraocular pressure ranges from approximately 10 to 21 mmHg, with recognised diurnal variation peaking in the morning.

Value-added response: Goldmann applanation tonometry is the gold standard, with non-contact and portable methods providing further screening and bedside options.



Answers to Pilot Questions [Series 4]

Part 4.1

11. The corneoscleral junction, the trabecular meshwork, and the ciliary body band.
12. Angle width determines ease of aqueous access to the trabecular meshwork, directly relevant to glaucoma risk.
13. A porous structure providing the principal drainage route for aqueous humour into Schlemm's canal.
14. Directing a penlight from the temporal side and observing illumination of the nasal iris relative to the temporal iris.
15. A deep angle shows even illumination; a shallow angle shows a shadowed nasal iris.

Part 4.2

1. Through active secretion, ultrafiltration, and diffusion by the ciliary processes, with active secretion predominant.
2. From the ciliary processes into the posterior chamber, through the pupil, into the anterior chamber.
3. It provides essential nutritional support to the avascular lens and cornea.
4. Drainage through the trabecular meshwork into Schlemm's canal and the episcleral venous system, accounting for the majority of outflow.
5. Drainage directly through the ciliary muscle and sclera, accounting for a smaller proportion of outflow.

Part 4.3

6. Approximately 10 to 21 mmHg.
7. IOP peaks in the early morning and gradually declines through the day; a single measurement may not represent the overall pressure profile.
8. It measures the force required to flatten a standardised area of cornea; it is the current clinical gold standard.
9. Non-contact tonometry uses a calibrated air pulse; Perkins and Tono-Pen are portable devices for bedside or non-slit-lamp use.
10. It is essential to glaucoma risk assessment, since elevated pressure is the single most important modifiable risk factor for optic nerve damage.



References and Attributions [Series 4]

11. Kanski, J. J., & Bowling, B. (2015). Clinical Ophthalmology: A Systematic Approach (8th ed.). Elsevier.
12. American Academy of Ophthalmology (2021). Glaucoma, Basic and Clinical Science Course Section 10.
13. Riordan-Eva, P., & Augsburger, J. J. (Eds.). (2017). Vaughan and Asbury's General Ophthalmology (19th ed.). McGraw-Hill.
14. Khurana, A. K. (2019). Comprehensive Ophthalmology (7th ed.). Jaypee Brothers Medical Publishers.

Figures, Plates, Tables, and Boxes

1. Figure 4.1: Diagram illustrating the pen torch anterior chamber depth estimation technique, comparing an evenly illuminated deep angle with a shadowed nasal iris in a shallow angle. AI-generated using the prompt: "A single clean, clinically accurate two-panel illustration of an eye viewed from the side with a penlight directed from the temporal edge, one panel labelled deep angle showing the entire iris evenly lit, the other labelled shallow angle showing a dark crescent shadow on the nasal iris, medical textbook diagram style, neutral background, no photographic realism, no other panels."
2. Table 4.2: Comparing conventional and uveoscleral aqueous outflow pathways. AI-generated using the prompt: "Create a clean physiology reference table titled Comparing Conventional and Uveoscleral Aqueous Outflow Pathways, listing pathway, route, and proportion of outflow. Ophthalmology reference table style with outside border, no photographic elements."
3. Table 4.3: Comparing key types of tonometry. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Key Types of Tonometry, listing method, principle, and typical use. Ophthalmology reference table style with outside border, no photographic elements."



Course code: OPT 607

Course title: Neuro-Ophthalmology

Course credit load: 1 Unit

Series 5: Glaucoma

- **Part 5.1: Definition, Types, and Epidemiology of Glaucoma**
 - Sub Part 5.1.1: Defining glaucoma
 - Sub Part 5.1.2: Types of glaucoma
 - Sub Part 5.1.3: Epidemiology and risk factors
- **Part 5.2: Optic Disc Signs and Visual Field Changes in Glaucoma**
 - Sub Part 5.2.1: Glaucomatous cupping
 - Sub Part 5.2.2: Further optic disc signs of glaucomatous optic neuropathy
 - Sub Part 5.2.3: Visual field changes in glaucoma
- **Part 5.3: Acute Angle-Closure Glaucoma and Management**
 - Sub Part 5.3.1: Acute angle-closure glaucoma
 - Sub Part 5.3.2: Clinical features and management of open-angle glaucoma
 - Sub Part 5.3.3: Overview of glaucoma management approaches

Introduction

In the last Series, we established the anterior chamber angle, aqueous humour, and intraocular pressure. We now conclude this course with **glaucoma**, applying this foundation to its definition, types, epidemiology, and risk factors, the characteristic **optic disc signs** and **visual field changes** of glaucomatous optic neuropathy, and the genuine emergency of **acute angle-closure glaucoma**, together with an overview of glaucoma management.

The aim of this Series is to equip you with a thorough understanding of the definition, types, epidemiology, risk factors, clinical features, and management of glaucoma, the optic disc signs of glaucomatous optic neuropathy, and acute angle-closure glaucoma.



This Series begins with the **definition, types, and epidemiology of glaucoma**, before examining **optic disc signs and visual field changes in glaucoma**. It concludes with **acute angle-closure glaucoma and management**.

Series Learning Outcomes

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

- **SLO 5.1:** define glaucoma and describe its types
- **SLO 5.2:** describe the epidemiology and risk factors for glaucoma
- **SLO 5.3:** recognise glaucomatous cupping and further optic disc signs of glaucomatous optic neuropathy
- **SLO 5.4:** describe visual field changes in glaucoma
- **SLO 5.5:** describe the clinical features and management of acute angle-closure glaucoma, and
- **SLO 5.6:** describe the clinical features and management approaches for open-angle glaucoma.

5.1 Definition, Types, and Epidemiology of Glaucoma

5.1.1 defining glaucoma

A progressive optic neuropathy, not simply raised pressure

Glaucoma, discussed in relation to intraocular pressure and the trabecular meshwork in the previous Series of this course, is defined as a progressive **optic neuropathy** characterised by a specific, recognisable pattern of optic nerve damage, discussed further later in this Series, generally though not invariably associated with elevated intraocular pressure.

This definition, discussed earlier in this Part, deliberately emphasises the characteristic **optic nerve damage** and associated visual field loss, discussed further later in this Series, rather than elevated pressure alone, since glaucomatous damage can occur even at statistically normal pressure levels, discussed in relation to normal-tension glaucoma in the next sub-Part.

Fill in the blank: Glaucoma is defined as a progressive optic neuropathy characterised by a specific, recognisable pattern of optic nerve damage, generally though not invariably associated with elevated intraocular _____.



5.1.2 types of glaucoma

Open-angle, angle-closure, and secondary forms

Primary open-angle glaucoma, discussed in relation to trabecular meshwork function in the previous Series of this course, represents the most common glaucoma type, developing gradually with an anatomically open, normal-appearing anterior chamber angle despite increased trabecular outflow resistance.

Primary angle-closure glaucoma, discussed further later in this Series, results from physical obstruction of the trabecular meshwork by the peripheral iris, while **normal-tension glaucoma** produces characteristic glaucomatous damage despite statistically normal intraocular pressure, and **secondary glaucoma** develops as a consequence of another identifiable underlying ocular or systemic **condition**.

Fill in the blank: Secondary glaucoma develops as a consequence of another identifiable underlying ocular or systemic _____.

5.1.3 epidemiology and risk factors

A leading cause of irreversible blindness worldwide

Glaucoma, discussed throughout this Part, represents a leading global cause of **irreversible blindness**, with primary open-angle glaucoma representing the predominant type in most populations, though primary angle-closure glaucoma carries disproportionate representation among genuinely severe, advanced presentations.

Recognised risk factors include elevated intraocular pressure, discussed in the previous Series of this course, advancing **age**, family history, discussed in relation to genetic predisposition, and, for angle-closure glaucoma specifically, a shallow anterior chamber, discussed in relation to angle depth estimation in the previous Series of this course, and hyperopic **refractive** error.

Fill in the blank: Recognised risk factors for angle-closure glaucoma specifically include a shallow anterior chamber and hyperopic refractive _____.

Table 5.1: Comparing key types of glaucoma.

Type	Angle status	Key feature
Primary open-angle	Open, normal-appearing	Most common type; increased outflow resistance
Primary angle-closure	Obstructed by peripheral iris	Risk of acute presentation



Normal-tension	Open	Glaucomatous damage despite normal pressure
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Pilot questions 5.1

- Define glaucoma. (Linked to SLO 5.1)
- Explain why the definition of glaucoma emphasises optic nerve damage rather than pressure alone. (Linked to SLO 5.1)
- Describe primary open-angle glaucoma. (Linked to SLO 5.1)
- Distinguish primary angle-closure glaucoma from normal-tension glaucoma. (Linked to SLO 5.1)
- List risk factors for glaucoma. (Linked to SLO 5.2)

5.2 Optic Disc Signs and Visual Field Changes in Glaucoma

5.2.1 glaucomatous cupping

Progressive enlargement of the physiological cup

Glaucomatous cupping, discussed in relation to the normal physiological cup in an earlier related course, describes progressive enlargement of the optic disc's central cup, generally assessed using the **cup-to-disc ratio**, with a ratio exceeding **0.6**, or a significant asymmetry between the two eyes, warranting careful further evaluation.

This progressive cupping, discussed earlier in this sub-Part, reflects loss of the optic nerve's neuroretinal rim tissue, discussed further in the next sub-Part, as retinal ganglion cell axons are progressively lost to glaucomatous damage, meaning the cup-to-disc ratio serves as a genuinely useful, though imperfect, indirect **marker** of underlying axonal loss.

Fill in the blank: A cup-to-disc ratio exceeding 0.6, or a significant asymmetry between the two eyes, warrants careful further _____.

5.2.2 further optic disc signs of glaucomatous optic neuropathy

Rim thinning, notching, and disc haemorrhage

Further recognised optic disc signs, discussed earlier in this Part, include **neuroretinal rim thinning**, particularly at the inferior and superior poles given their relative vulnerability to



glaucomatous damage, and focal **rim notching**, a localised area of particularly pronounced rim tissue loss.

Disc haemorrhage, discussed in relation to optic disc vascular supply, a small, flame-shaped haemorrhage at the disc margin, represents a further important sign, generally indicating active, ongoing glaucomatous damage and carrying particular relevance to disease **progression** monitoring, discussed further later in this Series.

Fill in the blank: Disc haemorrhage, a small, flame-shaped haemorrhage at the disc margin, generally indicates active, ongoing glaucomatous damage and carries particular relevance to disease progression _____.

5.2.3 visual field changes in glaucoma

Characteristic patterns reflecting nerve fibre layer damage

Glaucomatous visual field defects, discussed in relation to arcuate scotoma in an earlier Series of this course, characteristically follow the curved course of the retinal nerve fibre layer, producing **arcuate scotomas** and, in more advanced disease, a **nasal step**, a defect respecting the horizontal meridian.

These field changes, discussed earlier in this sub-Part, generally correlate closely with the specific pattern of optic disc damage observed on ophthalmoscopy, discussed earlier in this Part, meaning combined structural and functional assessment, correlating disc appearance with field testing, supports genuinely confident glaucoma diagnosis and reliable ongoing disease **monitoring**.

Fill in the blank: Glaucomatous visual field changes generally correlate closely with the specific pattern of optic disc damage observed on ophthalmoscopy, supporting genuinely confident diagnosis and reliable ongoing disease _____.



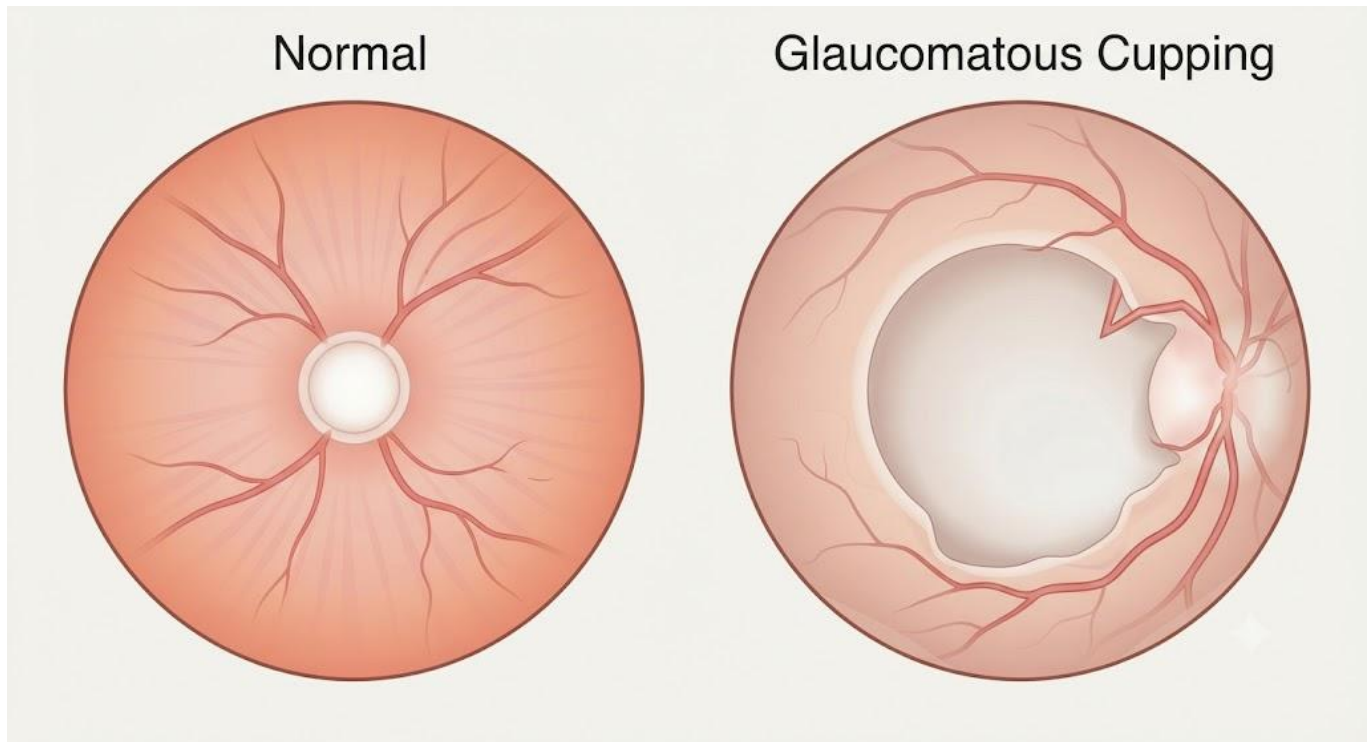


Figure 5.2: Diagram illustrating progressive glaucomatous cupping, comparing a normal cup-to-disc ratio with an enlarged, asymmetric cup showing rim thinning.

Pilot questions 5.2

1. Define glaucomatous cupping and the cup-to-disc ratio. (Linked to SLO 5.3)
2. Describe neuroretinal rim thinning and notching. (Linked to SLO 5.3)
3. Describe disc haemorrhage and its clinical significance. (Linked to SLO 5.3)
4. Describe the characteristic visual field defects of glaucoma. (Linked to SLO 5.4)
5. Explain the value of correlating optic disc appearance with visual field testing. (Linked to SLO 5.4)

5.3 Acute Angle-Closure Glaucoma and Management

5.3.1 acute angle-closure glaucoma

A genuine ophthalmic emergency requiring urgent treatment

Acute angle-closure glaucoma, discussed in relation to angle anatomy and primary angle-closure glaucoma earlier in this Series, results from sudden obstruction of aqueous outflow,



producing rapid, severe intraocular pressure elevation, characteristically presenting with severe eye pain, headache, nausea, and vomiting.

Characteristic examination findings include a **fixed, mid-dilated pupil**, corneal oedema producing a hazy appearance, and a markedly elevated intraocular pressure on tonometry, discussed in the previous Series of this course, together representing a genuine ophthalmic emergency requiring **urgent** treatment to prevent permanent visual loss.

Fill in the blank: Acute angle-closure glaucoma characteristic examination findings include a fixed, mid-dilated pupil, corneal oedema, and a markedly elevated intraocular pressure, representing a genuine ophthalmic emergency requiring _____ treatment.

5.3.2 clinical features and management of open-angle glaucoma

A typically asymptomatic, slowly progressive condition

Primary open-angle glaucoma, discussed earlier in this Series, typically presents **asymptotically** in its early stages, given the slow, gradual nature of visual field loss and the tendency for peripheral field defects to remain unnoticed until relatively advanced, meaning routine screening carries genuine importance for early detection.

Management of open-angle glaucoma, discussed earlier in this sub-Part, generally begins with **topical medication** aimed at lowering intraocular pressure, discussed in relation to aqueous dynamics in the previous Series of this course, with **laser trabeculoplasty** or **surgical** intervention reserved for cases inadequately controlled by medical therapy alone.

Fill in the blank: Management of open-angle glaucoma generally begins with topical medication aimed at lowering intraocular pressure, with laser trabeculoplasty or surgical intervention reserved for cases inadequately controlled by _____ therapy alone.

5.3.3 overview of glaucoma management approaches

Lowering intraocular pressure remains the central strategy

Across every glaucoma type discussed throughout this Series, **lowering intraocular pressure** remains the single central management strategy, since it represents the only currently modifiable factor demonstrated to reliably slow or halt further glaucomatous optic nerve damage, discussed earlier in this Series, regardless of a patient's specific baseline pressure level.

In **acute angle-closure glaucoma**, discussed earlier in this Part, urgent pressure-lowering therapy is generally followed by a definitive **laser peripheral iridotomy**, creating a permanent alternative aqueous outflow route bypassing the obstructed angle, while chronic glaucoma



management, discussed earlier in this sub-Part, generally requires sustained, lifelong **monitoring** and treatment adjustment.

Fill in the blank: In acute angle-closure glaucoma, urgent pressure-lowering therapy is generally followed by a definitive laser peripheral iridotomy, creating a permanent alternative aqueous _____ route bypassing the obstructed angle.

Table 5.3: Comparing acute angle-closure and primary open-angle glaucoma.

Feature	Acute angle-closure glaucoma	Primary open-angle glaucoma
Onset	Sudden, acute	Gradual, chronic
Symptoms	Severe pain, headache, nausea	Typically asymptomatic until advanced
Definitive treatment	Laser peripheral iridotomy	Topical medication, laser, or surgery

Pilot questions 5.3

1. Describe the pathophysiology and presentation of acute angle-closure glaucoma. (Linked to SLO 5.5)
2. List the characteristic examination findings of acute angle-closure glaucoma. (Linked to SLO 5.5)
3. Explain why open-angle glaucoma is often diagnosed late. (Linked to SLO 5.6)
4. Describe the general management approach for open-angle glaucoma. (Linked to SLO 5.6)
5. Describe the definitive treatment for acute angle-closure glaucoma. (Linked to SLO 5.5)

Series Summary

This final Series examined **glaucoma**, beginning with its **definition, types, and epidemiology**, primary open-angle, angle-closure, normal-tension, and secondary glaucoma, and recognised risk factors, before examining **optic disc signs and visual field changes in glaucoma**, glaucomatous cupping, rim changes, and characteristic field defects.

We concluded with **acute angle-closure glaucoma and management**, its urgent presentation, and management approaches for both acute and chronic glaucoma types. Having worked through this Series, and indeed this entire course, you should now possess a comprehensive introductory



foundation in neuro-ophthalmology, from neuro-ophthalmic pathways and optic nerve disease through pupillary and ocular motility assessment, visual field testing, the anterior chamber angle and intraocular pressure, and, finally, glaucoma addressed throughout OPT 607.

Glossary of Terms

1. **Glaucoma:** a progressive optic neuropathy characterised by a specific pattern of optic nerve damage.
2. **Primary open-angle glaucoma:** the most common glaucoma type, with an anatomically open angle.
3. **Primary angle-closure glaucoma:** glaucoma resulting from physical obstruction of the trabecular meshwork by the peripheral iris.
4. **Normal-tension glaucoma:** glaucomatous damage occurring despite statistically normal intraocular pressure.
5. **Cup-to-disc ratio:** the proportion of the optic disc occupied by the physiological cup, used to assess glaucomatous cupping.
6. **Neuroretinal rim:** the tissue of the optic disc surrounding the cup, thinned by glaucomatous damage.
7. **Disc haemorrhage:** a flame-shaped haemorrhage at the disc margin indicating active glaucomatous damage.
8. **Nasal step:** a visual field defect respecting the horizontal meridian, characteristic of glaucoma.
9. **Acute angle-closure glaucoma:** sudden aqueous outflow obstruction producing rapid, severe intraocular pressure elevation.
10. **Laser peripheral iridotomy:** the definitive treatment for angle-closure glaucoma, creating an alternative aqueous outflow route.
11. **Laser trabeculoplasty:** a laser procedure used to lower intraocular pressure in open-angle glaucoma.
12. **Topical medication (glaucoma):** eye drop therapy representing the generally first-line treatment for open-angle glaucoma.

Concept Check Questions [Series 5]

Objective questions

1. Glaucoma is defined as a progressive optic neuropathy generally though not invariably associated with elevated intraocular _____.



2. Secondary glaucoma develops as a consequence of another identifiable underlying ocular or systemic _____.
3. A cup-to-disc ratio exceeding 0.6, or a significant asymmetry between the two eyes, warrants careful further _____.
4. Acute angle-closure glaucoma represents a genuine ophthalmic emergency requiring _____ treatment.
5. In acute angle-closure glaucoma, urgent pressure-lowering therapy is generally followed by a definitive laser peripheral iridotomy, creating a permanent alternative aqueous _____ route.

Short-answer questions

- Define glaucoma.
- List risk factors for glaucoma.
- Describe disc haemorrhage and its clinical significance.
- List the characteristic examination findings of acute angle-closure glaucoma.
- Describe the general management approach for open-angle glaucoma.

Long-answer questions

1. Discuss the definition and types of glaucoma. (Linked to SLO 5.1)
2. Discuss the epidemiology and risk factors for glaucoma. (Linked to SLO 5.2)
3. Discuss glaucomatous cupping and further optic disc signs of glaucomatous optic neuropathy. (Linked to SLO 5.3)
4. Discuss visual field changes in glaucoma. (Linked to SLO 5.4)
5. Discuss acute angle-closure glaucoma and the management of both acute and open-angle glaucoma. (Linked to SLO 5.5, SLO 5.6)

Answers to Concept Check Questions [Series 5]

Objective questions

6. Pressure.
7. Condition.
8. Evaluation.
9. Urgent.
10. Outflow.



Short-answer questions

11. A progressive optic neuropathy characterised by a specific pattern of optic nerve damage, generally associated with elevated intraocular pressure.
12. Elevated intraocular pressure, advancing age, family history, and, for angle-closure specifically, shallow anterior chamber and hyperopic refractive error.
13. A flame-shaped haemorrhage at the disc margin; it generally indicates active, ongoing glaucomatous damage.
14. Fixed, mid-dilated pupil, hazy corneal appearance from oedema, and markedly elevated intraocular pressure.
15. Beginning with topical medication to lower intraocular pressure, with laser trabeculoplasty or surgery reserved for inadequately controlled cases.

Long-answer questions

16. **Basic response:** Glaucoma is a progressive optic neuropathy with characteristic nerve damage, classified as open-angle, angle-closure, normal-tension, or secondary.

Value-added response: This classification directly informs the specific underlying mechanism and, consequently, the most appropriate management approach for a given patient.

17. **Basic response:** Glaucoma represents a leading global cause of irreversible blindness, with open-angle glaucoma predominant in most populations.

Value-added response: Risk factors include elevated pressure, age, family history, and, for angle-closure specifically, shallow anterior chamber and hyperopic refractive error.

18. **Basic response:** Glaucomatous cupping reflects progressive neuroretinal rim tissue loss, assessed via the cup-to-disc ratio, rim thinning, and notching.

Value-added response: Disc haemorrhage represents a further important sign of active, ongoing damage relevant to progression monitoring.

19. **Basic response:** Glaucomatous visual field defects characteristically follow the nerve fibre layer course, producing arcuate scotomas and, later, a nasal step.

Value-added response: These field changes correlate closely with optic disc appearance, supporting combined structural and functional diagnostic confidence.

20. **Basic response:** Acute angle-closure glaucoma is a genuine emergency requiring urgent treatment followed by laser peripheral iridotomy, while open-angle glaucoma is managed with topical medication progressing to laser or surgery as needed.



Value-added response: Lowering intraocular pressure remains the central management strategy across every glaucoma type, since it is the only currently modifiable factor demonstrated to slow disease progression.

Answers to Pilot Questions [Series 5]

Part 5.1

11. A progressive optic neuropathy characterised by a specific pattern of optic nerve damage.
12. Because glaucomatous damage can occur even at statistically normal pressure, as in normal-tension glaucoma.
13. The most common glaucoma type, developing gradually with an anatomically open, normal-appearing angle despite increased outflow resistance.
14. Primary angle-closure results from physical iris obstruction of the angle; normal-tension shows damage despite normal pressure with an open angle.
15. Elevated intraocular pressure, advancing age, family history, and, for angle-closure, shallow anterior chamber and hyperopic refractive error.

Part 5.2

1. Progressive enlargement of the optic disc's central cup; the cup-to-disc ratio quantifies the proportion of disc occupied by the cup.
2. Neuroretinal rim thinning, particularly at the inferior and superior poles, and focal rim notching, a localised area of pronounced rim loss.
3. A small, flame-shaped haemorrhage at the disc margin, indicating active, ongoing glaucomatous damage.
4. Arcuate scotomas following the nerve fibre layer course, and, in advanced disease, a nasal step respecting the horizontal meridian.
5. It supports genuinely confident glaucoma diagnosis and reliable ongoing disease monitoring by correlating structural and functional findings.

Part 5.3

6. Sudden obstruction of aqueous outflow producing rapid, severe pressure elevation, presenting with severe pain, headache, nausea, and vomiting.
7. Fixed, mid-dilated pupil, hazy corneal appearance, and markedly elevated intraocular pressure.



8. Because it is typically asymptomatic in early stages given slow, gradual visual field loss that remains unnoticed until advanced.
9. Topical medication first-line, with laser trabeculoplasty or surgery reserved for inadequately controlled cases.
10. Laser peripheral iridotomy, creating a permanent alternative aqueous outflow route bypassing the obstructed angle.

References and Attributions [Series 5]

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12. American Academy of Ophthalmology (2021). *Glaucoma, Basic and Clinical Science Course Section 10*.
13. Weinreb, R. N., Aung, T., & Medeiros, F. A. (2014). The Pathophysiology and Treatment of Glaucoma: A Review. *JAMA*, 311(18), 1901-1911.
14. Khurana, A. K. (2019). *Comprehensive Ophthalmology* (7th ed.). Jaypee Brothers Medical Publishers.

Figures, Plates, Tables, and Boxes

1. Table 5.1: Comparing key types of glaucoma. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Key Types of Glaucoma, listing type, angle status, and key feature. Ophthalmology reference table style with outside border, no photographic elements."
2. Figure 5.2: Diagram illustrating progressive glaucomatous cupping, comparing a normal cup-to-disc ratio with an enlarged, asymmetric cup showing rim thinning. AI-generated using the prompt: "A single clean, accurate two-panel schematic diagram of the optic disc on fundoscopy, one panel labelled normal showing a small, centred pale cup, the other labelled glaucomatous cupping showing an enlarged cup with thinned inferior rim tissue, textbook ophthalmology diagram style, neutral background, no photographic realism, no other panels."
3. Table 5.3: Comparing acute angle-closure and primary open-angle glaucoma. AI-generated using the prompt: "Create a clean clinical reference table titled Comparing Acute Angle-Closure and Primary Open-Angle Glaucoma, listing feature, acute angle-closure glaucoma, and primary open-angle glaucoma. Ophthalmology reference table style with outside border, no photographic elements."

