<table>
<thead>
<tr>
<th>VA far</th>
<th>VA near</th>
<th>Ishihara</th>
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<tr>
<th>Pupil diameter (mm)</th>
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Ductions

Nystagmus

Post head shake
Positional

RAPD

VF

Fundi

Light | Dark 5° | 20° | Conv | Palpe 

Lids (mm)

Fixation
Pursuit
Saccades
VOR can
Halmagyi
OKN
Vejence
### Nystagmus
- Post head shake
- Positional

### MMSE

<table>
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<tr>
<th>Cranial Nerves</th>
<th>V motor</th>
<th>V/sensory 1, 2, 3</th>
<th>touch, pain</th>
<th>VII upper</th>
<th>VII lower</th>
<th>palate tongue</th>
<th>corneal reflex</th>
<th>jaw jerk</th>
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<tr>
<td>Motor</td>
<td>UL Bulk</td>
<td>tone</td>
<td>power</td>
<td>reflexes</td>
<td>abn mov't</td>
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<tr>
<td>Sensory</td>
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<td>touch</td>
<td>vibration</td>
<td>JPS</td>
<td>graph/stereo/2pt</td>
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<tr>
<td>Coord</td>
<td>UL fine</td>
<td>Romberg</td>
<td>sharp</td>
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### Assessment

### Investigations / Management
Normal Pupil

• The normal pupil is 2 mm to 6 mm in diameter.
• In ordinary ambient light the pupils are usually 3 mm to 4 mm in diameter.
Normal Pupil

• The pupils are small and poorly reactive at birth and in early infancy, becoming normal size around ages 7 to 8.
• They are normally larger in adolescents and young adults, about 4 mm in diameter and perfectly round.
• In middle age, they are typically 3.5 mm in diameter and regular, and in old age 3 mm or less and often slightly irregular.
Miotic pupil

- Pupils less than 2 mm in diameter are miotic.
- Common causes of acquired miosis include old age, hyperopia, alcohol abuse, and drug effects.
Miotic pupil

• Neurologically significant causes of miosis include
  – neurosyphilis,
  – diabetes,
  – levodopa therapy
  – Horner's syndrome.
Miotic pupil

• Acute, severe brainstem lesions, such as pontine hematoma, may cause bilaterally tiny, “pinpoint” pupils that still react.
• Due to bilateral sympathetic pathway lesions
• Normal reaction is confirmed using magnifying lenses
Mydriatic Pupil

- Pupils more than 6 mm in diameter are dilated.
- Common causes of bilateral mydriasis include anxiety, fear, pain, myopia, and drug effects—especially anticholinergics.
- Large pupils were once considered a sign of youth and beauty, and the anticholinergic belladonna (Ital. “fair lady”) alkaloids were named for their ability to produce this effect.
Mydriatic Pupil

• Persons with light irises have larger pupils than those with dark irises.
• Only severe, bilateral lesions of the retina or anterior visual pathways, enough to cause near blindness, will affect the resting pupil size.
Mydriatic Pupil

- Neurologically significant bilateral mydriasis occurs in
  - midbrain lesions
  - comatose patients following cardiac arrest
  - cerebral anoxia
The Pupils

- Size, shape and symmetry
- Direct and indirect responses to bright light
- Accommodation reaction should be tested

- Ptosis with small pupil = Horner’s Syndrome
- Ptosis with large pupil = 3rd N Palsy

- Dilated pupils due to Atropine instillation
- Size changes with age

- Hippus phenomenon

- Size is controlled by PNS and SNS
IS ANISOCORIA GREATER IN LIGHT OR DARK?

More anisocoria in dim light or Good light reaction in both eyes

- Look for dilation lag
- Consider cocaine 4—10% test

- Absent dilation lag of smaller pupil
- Dilation occurs with cocaine test

More anisocoria in bright light or Poor light reaction in one eye

- Examine iris at slit lamp
- Check for constriction at near
- Consider pilocarpine 0.125% test

- Torn pupillary border
- Sluggish near constriction

Traumatic Mydriasis

- Round pupil
- Sector iris palsy
- Tonic near
- Constriction seen with pilocarpine 0.125%

Horner's Syndrome

- Present dilation lag of smaller pupil
- Poor or no dilation occurs with cocaine test

Hydroxyamphetamine 1% test

- Dilation occurs

Pilocarpine 1—2% test

- No dilation occurs

Constriction occurs

No constriction occurs

- Oculomotor Palsy
- Pharmacologic Mydriasis

Physiologic Anisocoria

- Preganglionic or Central Horner's Syndrome

- Postganglionic Horner's Syndrome

- Adie's Tonic Pupil
Normal Pupillary phenomena

• Physiologic Anisocoria
  – 20% of general population
  – Less than 0.5mm anisocoria
  – Degree of anisocoria can vary from day to day and even switch sides

• Pupillary Unrest (Hippus)
  – During distance fixation with constant, ambient illumination
  – Bilateral symmetrical, nonrhythmical unrest or variation in size (usu < 1mm)
Normal Pupillary phenomena

• Near Synkinesis
  – Light miosis is greater than near miosis

• Psychosensory reflex
  – During light or near stimuli, the patient is subjected to a loud noise or pain
  – The pupils will dilate
  – Due to active sympathetic discharges
  – Inhibition of EW nucleus
Pupillary Testing

- Direct Light Reflex
- Consensual Reflex
- Swinging flashlight test
  - Bright hand light in a darkened room
  - Patient should fix on a distant object
  - Light should cross from one eye to the other fairly rapidly and remain 3 to 5 seconds on each eye to allow pupillary stabilization
Light Reflex

direct  consensual
Parasympathetic Pathway
Sphincter Muscle of the Iris

• Innervated by Parasympathetic fibers that originate in the Edinger-Westphal nucleus.

4 Neuron Pathway

• Afferent neurons from retinal ganglion cells to pretectal area
• Interneurons from pretectal complex to EW nuclei
• Parasympathetic outflow with 3rd nerve to ciliary ganglion
• From ciliary ganglion to Iris sphincter
Accommodation

2.1 Correct technique for testing accommodation.
Near Synkinesis

- Origin at peristriate cortex
- Near synkinesis triad
  - Convergence of eyes
  - Accommodation of lenses
  - Miosis of pupils
- The pathway is more ventrally located than the pretectal afferent limb of the light reflex
- Anatomical location is basis for light near dissociation in eg Argyll Robertson pupils
- Final pathway is 3rd N, Ciliary ganglion and short ciliary nerves
Abnormalities of Light Reflex and Parasympathetic Pathway

- Marcus Gunn Pupil - Relative Afferent Pupillary defect
- Parinaud Syndrome
- Argyll Robertson Pupil
- 3rd Nerve palsy
- Holmes Adie Pupil
- Trauma
- Diphtheria
Parasympathetic Pathway

Aqueduct (note the pathways relaying around in the periaqueductal area)

Superior colliculus

Lateral geniculate body

Etlinger-Westphal nuclei

Convergence centre

Blood vessels on pia mater (supply surface of the nerve including pupillary fibres)

Filaments to pupil (lie dorsal and peripheral)

Plat sheath of nerve

Vasa nervorum

Vasa nervorum supplies central part of nerve

Third nerve

Short ciliary nerves (18-20 in all)

Optic chiasm

Ciliary ganglion (on branch to the inferior oblique muscle)

Sphincter pupillae

Parasympathetic pathways.
Relative afferent Pupillary Defect (Marcus Gunn Pupil)

- Amount of light transmitted from one eye is less than the other eye. During swinging flashlight, the fol may be noted from normal eye to defective eye

1+ Initial constriction. Greater escape to larger intermediate size

2+ No change in pupil size initially. Followed by dilatation of pupil

3+ Immediate dilation of pupil, instead of normal initial constriction

4+ Immediate dilation of pupil, NPL VA
Relative Afferent Pupillary Defect (Marcus Gunn Pupil)

• Optic Neuropathy must be unilateral or markedly asymmetric

• Ocular media opacities (corneal scar, cataract, vitreous hrr) will not cause MG pupil

• Maculopathy, or amblyopic ‘lazy eye’ will not cause MG pupil. Unless VA < 20/200, then 1+ MG phenomenon
Afferent Pupillary Defect (Marcus Gunn Pupil)

• Extensive retinal damage will cause a significant MG phenomenon

• Amaurotic pupil: maximum MG pupil imaginable. ‘blind eye’

• No such thing as bilateral MG pupils
Afferent Pupillary Defect (Marcus Gunn Pupil)

- Isolated, unilateral optic neuropathy does not cause the ipsilateral pupil to be larger. Due to consensual light reflex.

Amaurotic Mydriasis does not exist

- Detection of APD requires only one ‘working’ pupil
Equal pupil size

Normal direct and Consensual light reflexes

Left afferent pupil defect

Marcus Gunn Pupil

normal
Normal Pupil

Left afferent pupil defect
Right afferent and efferent pupil defect
Normal left pupil
Dilated Right pupil-efferent pupil defect
Left RAPD – afferent pupil defect
Argyll Robertson Pupil

• Miotic irregular pupils

• Light-near dissociation

• Poor dilation in dark and in response to mydriatic agents

• Usually bilateral, often asymmetric

• Cause: Neurosyphilis. Other reported causes, Diabetes mellitus, chronic alcoholism, multiple sclerosis, sarcoid

• Site of lesion; most likely region of Sylvian Aqueduct in rostral midbrain
Argyll Robertson Pupil

Note: Ptosis, pupil is small, irregular, does not react to light but does to accommodation.

With 1% atropine solution in both eyes

2.3 Argyll Robertson pupil. (For illustrative purposes, patient’s left pupil is shown as normal. AR pupils are usually bilateral, although they can be asymmetrical.)
Argyll robertson pupils
Adie’s Tonic Pupil

• Idiopathic, benign cause of internal ophthalmoplegia

• 80% initially unilateral

• Become bilateral at rate of 4% per year

• Female predilection (70% vs 30%)

• Young adults (20 to 40 yrs)
Adie’s Tonic Pupil

- Dilated pupil with poor to absent light reaction
- Slow constriction to prolonged near effort and slow redilation
- Initial accommodative paresis. Resolves over months
- Segmental palsy of iris sphincter muscle
Adie’s Tonic Pupil

- Cholinergic supersensitivity to weak Pilocarpine solutions (0.125% or 0.1%)

- Aetiology unknown. Lesion in ciliary ganglion or short posterior ciliary nerves

- Aberrant regeneration of more numerous fibers innervating the ciliary muscle (97%) than iris sphincter (3%)

- Adie’s syndrome: pupillary abnormalities with diminished tendon reflexes
Holmes Adie Pupil

Note: No ptosis, pupil is large, regular, does not react to light (other than prolonged exposure) but may show some response to accommodation

2.5% methacholine (mecholyl) in both eyes
The sensitized H-A pupil promptly constricts No effect on normal eye

Holmes-Adie pupils are usually unilateral; another feature to distinguish them from the AR pupil
1.8% pilocarpine drops in affected eye
Pupil constricts

2.4 Holmes-Adie pupil.
.125% Pilocarpine

Pharmacologic
Segmental palsy of sphincter muscles

Right dilated pupil

R pupil no light reflex

Adies pupil

Pupils react to accommodation
Sympathetic Pathway

Pathway starts in hypothalamus

possibly damaged by ischaemia
a carotid artery thrombosis
migraine spasm

superior cervical ganglion
peripheral synapse

Lesions of cervical sympathetic chain: Thyroid carcinoma
Thyroid surgery
Neoplastic lesions
Local trauma
Surgical extirpation

Lesions of spinal root at T1: Apical carcinoma of the lung
Cervical ribs
Aortic aneurysm
Avulsion of the lower plexus

First synapse in intermediodorsal and intermediolateral cells, the caudal spinal centre at Bulb

Lesions of the pathway in prestomalateral brain stem:
Wallenberg's syndrome
Multiple sclerosis
Pontine glioma
Poliomyelitis

Lesion in cervical cord, usually caused by central lesions:
Syringomyelia
Ependymoma
Chiari

Palsy of the eyelid
Papillary constriction

Third nerve
Nasociliary branch of the ophthalmic nerve
Fibres to blood vessels traverse ganglion without synapse
Fibres to pupil as long ciliary nerves in the nasociliary branch of ophthalmic nerve

Ipsilateral palpebra superioris

lexor palpebrae superioris
Dilator Muscle of the Iris

- Innervated by Sympathetic fibers. 3 Neuron pathway
- 1\textsuperscript{st} order neuron; From Hypothalamus to C8-T2 level of spinal cord

- 2\textsuperscript{nd} order neuron (preganglionic); From the cord, to the paravertebral sympathetic chain and end at the superior cervical ganglion (SCG)

- 3\textsuperscript{rd} order neuron (postganglionic); From SCG, along the internal carotid artery, joins the ophthalmic nerve and enters the orbit through the sup orbital fissure. Terminate at the pupil and Muller’s muscle.

Sudomotor and Vasomotor fibers to face travel with external carotid artery
Horner Syndrome

• Small pupil

• Ptosis

• Conjunctival injection

• Anhidrosis

• Enophthalmos – apparent

• Congenital Horner’s
Causes of Horner Syndrome

- Hemispheric lesions
- Brainstem lesions
- Cervical cord lesions
- Root lesions at T1
- Sympathetic Chain
- Cavernous sinus and orbit
- Migraine and cluster Headache
Horner's Syndrome
Apraclonidine: weak $\alpha_1$ agonist, strong $\alpha_2$ agonist. Fol Horner syndrome, upregulation of $\alpha_1$ receptors. Denervation supersensitivity – Abn pupil dilates, lid elevates Normal pupil constricts due to $\alpha_2$ activity preventing release of NA
Congenital R Horner's syndrome
Unconscious Patient

- **Normal pupils** – metabolic encephalopathy (70%) DM, hypoglycaemia, uraemia, liver failure, drug overdose

- **Unequal pupils** – dilated pupil = herniated temporal lobe

- **Bilateral dilated pupils**
  - progressive tentorial herniation
  - irreversible brain damage

- **Bilateral pinpoint pupils**
  - massive pontine haemorrhage
  - opiates
  - pilocarpine drops
<table>
<thead>
<tr>
<th>Reaction</th>
<th>Small (miotic) pupils</th>
<th>Large (mydriatic) pupils</th>
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</thead>
<tbody>
<tr>
<td>Non-reactive to light</td>
<td>Argyll Robinson pupils (usually irregular)</td>
<td>Holmes–Adie (will slowly react to light)</td>
</tr>
<tr>
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<td>Pontine haemorrhage – may react with very bright light (observed by using magnifying lens)</td>
<td>Post-traumatic iridoplegia</td>
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<td>Opiates</td>
<td>Mydriatic drops</td>
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<td></td>
<td>Pilocarpine drops</td>
<td>Atropine drops</td>
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<td>Belladonna</td>
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<td>Datura (jimson weed)</td>
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<td>Brain death</td>
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<td>Reactive to light</td>
<td>Old age</td>
<td>Childhood</td>
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<td>Holmes–Adie pupil (in constricted phase)</td>
<td>Anxiety states</td>
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<td>Horner's syndrome</td>
<td>Physiological anisocoria</td>
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Physiologic Anisocoria

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- Dilation occurs

Horner’s Syndrome

Pilocarpine 1—2% test

- No dilation occurs

- Constriction occurs

- No constriction occurs

Adie’s Tonic Pupil

Preganglionic or Central Horner’s Syndrome

Postganglionic Horner’s Syndrome

Oculomotor Palsy

Pharmacologic Mydriasis