COURSE 660      OHP 1
EVALUATION OF THE PUPILS
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OVERVIEW OF LECTURE
• General why/when of pupil testing
• Neuro quick review
  – The anatomic basis of our strategy and clinically significant anatomy
• Strategy of pupil evaluation
  – Why are we doing what we are doing
• Techniques of pupil evaluation
  – How to evaluate pupils
• Common pupil anomalies

WHY EVALUATE PUPILS?
• Test of neurological integrity - integrity of afferent and efferent pupil pathways.
• Pupil reflexes are important in the differential diagnosis of the cause of vision loss
• Pupil signs are important signs in ocular disease especially anterior segment disease.
  Iritis → small pupils & significant photophobia
  Acute angle closure glaucoma → mid-dilated, fixed pupil (pupil stuck in the mid-dilated position)

WHEN TO EVALUATE PUPILS
• All comprehensive exams
• All cases of vision loss or visual field loss
• Always prior to pupillary dilation
• Prior to contact lens fitting
• All tests of neurovisual function and/or neurological status

WHAT IS EVALUATED IN A PUPIL EXAM?
• Neurological integrity/neurovisual function
  Afferent pathway –
  From eye to midbrain for pupillary light reflex.
  From eye to cortex for pupillary near reflex
  Efferent pathways – brain to the pupils (CN III to pupillary sphincter and oculosympathetic pathway to the pupillary dilator)
• Gross external ocular health

PUPIL NEURO
Quick Review
**THE PUPIL PATHWAYS!!**

- **Afferent pathway**
  - From eye to brain
  - Pre-geniculate visual pathways (optic nerve + anterior portion of optic tracts) + projections to midbrain
- **Efferent pathways**
  - From brain to eye
  - Third cranial nerve to pupillary sphincter
  - Oculosympathetic pathway to pupillary dilator

**AFFERENT PUPIL PATHWAY**

- **Retina**
  - Retinal receptors
  - Intra-retinal connections
  - Retinal ganglion cells (RGCs)
  - Ganglion cell axons (RNFL)
- **Optic nerve**
  - Optic nervehead (RGC axons)
  - Optic nerve (RGC axons)

**AFFERENT PUPIL PATHWAY (cont’d)**

- Optic chiasm (RGC axons)
- Optic tract (RGC axons)
- (Brachium of superior colliculus)
- (Pretectal nuclei)
- (Posterior commissure)

**Afferent Pupillary Light Pathway**

**CROSSOVER AT THE CHIASM**

- The fibers from nasal retina (53-55% of all) from each eye cross to the opposite side in the chiasm
- The fibers from the temporal retina (only 45-47% of all from an eye) stay on the same side

So...in the right optic tract 53-55% of fibers are from the **LEFT eye**!
CONSENSUAL PUPILLARY LIGHT REFLEX
ANATOMIC BASIS

- Optic chiasm
- Posterior commissure

A LESION IN THE AFFERENT PATHWAY PRODUCES

- Impaired pupillary light reflex
  - But *not* the near reflex
- Light - near dissociation
  - Near reflex is stronger, quicker than light reflex
- APD (*if* unilateral or asymmetric damage)

DAMAGE TO AFFERENT PATHWAY DOES **NOT** CAUSE

- Anisocoria
  - Difference in pupil size
- Change in near reflexes
  - Near reflex not changed if tested as described in this lecture

PUPILLARY LIGHT REFLEX IS A TWO-LIMBED REFLEX ARC

- Afferent pathway
- Efferent pathway (CN III)

EFFERENT PUPIL PATHWAYS

- Third cranial nerve
  (parasympathetic to pupillary sphincter - CN III)
- Oculosympathetic pathway
  (sympathetics to pupillary dilator)

EFFERENT PATHWAY DAMAGE CAUSES

- Change in pupil size
  - If asymmetric or unilateral → anisocoria
- Reduced pupil function
  - If CN III damage → reduced response to light and near
    or
  - If oculosympathetic pathway damage → reduced or slower dilation in dark
The Oculosympathetic Pathway

**Oculosympathetic Pathway**

**First Order Neuron**
- Hypothalamus
- Brainstem
- Upper spinal cord
- Ciliospinal center of Budge (C8, T1, T2)

**Second Order Neuron**
- White rami communicantes
- Stellate ganglion (no synapse)
- Over the apex of the lung
- Joins common carotid artery (CCA)
- Superior cervical ganglion (synapse)

**Third Order Neuron to Eye/Globe**
- Follows internal carotid artery
- Cavernous sinus
- Cranial nerve V, ophthalmic division, nasociliary
- Long ciliary nerves

**Third Order Neuron to Facial Sweating**
- External carotid artery
- Also goes to sudomotor (sweating) and vasomotor control in face
SYMPATHETIC INNERVATION IN HEAD/EYE & FUNCTIONS

- Pupillary dilator - pupillary dilation
- Mueller’s muscles - lid retraction
  - Upper & lower lid
- Facial sweating
- Lacrimal glands - lacrimation
- Ciliary muscle → accommodation (?)
- Vessels of conjunctiva - vasoconstriction
- Iris melanin development in early life

Oculosympathetic dysfunction (Horners’ syndrome)

- From www.thyroid-eyes.com/ptosis
- From: www.wikidoc.org/index.php/Horner%27s_syndrome

Based on this gross external view what is the most probable cause of this patient’s problem?

LID RETRACTORS

- Mueller’s muscle (sympathetic)
- Levator palpebrae superioris (CN III)

Lid Retractors

NEUROGENIC PTOSIS

- Third nerve palsy - large ptosis
- Oculosympathetic paresis - small ptosis + inverse ptosis
The Parasympathetic Pathway to the Pupil

**Parasympathetic Pathway to the Pupillary Sphincter**
- Edinger-Westphal nucleus
- CN III
  - Midbrain
  - Cavernous sinus
  - Superior orbital fissure
  - Orbit
- Inferior division of CN III
- Ciliary ganglion
- Short ciliary nerves

**CN II and CN III (Pupil light response)**

**The Path of CN III to the Orbit & Globe**

**Clinical Key Points**
- CN III Anatomy
  - In cavernous sinus pupil fibers are located dorsal and superficial in CN III → aneurysm of posterior communicating artery often ↓ CN III pupil function
  - ~97% of CN III parasympathetics → go to ciliary muscle; only 3% to sphincter
  - CN III parasympathetics to pupil are isolated from other CN III fibers in only one place → ciliary ganglion
Aneurysm of Posterior Communicating Artery and its Effect on CN III (Pupillomotor Fibers)

Damage to CN III Parasympathetics Causes

- ↓ light response and ↓ near response
- ↓ accommodation

Third Cranial Nerve Functions

- Inferior oblique - upgaze on adduction
- Ciliary muscle - accommodation
- Pupillary sphincter- pupil constriction
- Levator palpebrae superioris - superior lid retraction
- Superior rectus - upgaze in abduction
- Medial rectus - adduction
- Inferior rectus - downgaze in abduction

The Strategy for Clinical Evaluation of the Pupils

Why you do what you do in the pupil evaluation!

Three (or five) Steps in Pupil Evaluation

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect

Three (or five) Steps in Pupil Evaluation

- Evaluate for anisocoria—
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect
EVALUATION FOR ANISOCORIA
LIGHTING
• If no anisocoria seen or suspected → use room illumination
• If anisocoria seen or suspected → measure in bright and dim
• Possible reasons to suspect anisocoria
  – Patient complaint of anisocoria
  – Lung cancer
  – Carotid surgery e.g. carotid endarterectomy
  – Neck trauma
  – Other signs, symptoms or history suggestive of Horner’s or CN III palsy

ANISOCORIA
THREE CLINICAL QUESTIONS
• Is the anisocoria pathologic?
• Which pupil is abnormal?
• What is the cause of the abnormality?
  – Local iris damage
  – Neuromuscular disorder
  – Nerve/neurogenic
  – Central (brain)

Anisocoria – Diagnostic Value of Bright and Dim Illumination Testing
• Anisocoria ↑ or appears only in bright illumination → suggests CN III lesion
• Anisocoria ↑ or appears only in dark illumination → suggests oculosympathetic lesion
• Anisocoria same in dark and in bright → suggests physiologic anisocoria

ANISOCORIA INCREASES WITH LUMINANCE IF DUE TO A PARASYMPATHETIC LESION AND DECREASES WITH LUMINANCE IF DUE TO A SYMPATHETIC LESION

THREE (or FIVE) STEPS IN PUPIL EVALUATION
• Evaluate for anisocoria
• Evaluate lid position ←
  – If anisocoria was found in prior step
• Evaluate responses to light
• Evaluate responses to near
  – If ↓ light response
• Evaluate for afferent pupil defect

When to check lid position?
• If anisocoria is found
WHY CHECK LID POSITION?

If anisocoria is present and is pathological it may be due to damage to either CNIII or to oculosympathetic pathway.

Superior Lid Position Neurological Control

Superior lid position controlled by:
1. Levator
   - CN III innervation
   - Primary retractor of the upper lid
2. Superior tarsal muscle (Mueller's muscle)
   - Sympathetic innervation
   - Secondary lid (both upper and lower lid) retractor

NEUROGENIC PTOSIS

- Third nerve palsy - large (usually ≥ 5mm) ptosis possible
- Oculosympathetic paresis (Horner’s syndrome) - small (1 to 2 mm) ptosis of superior lid usually + inverse ptosis (inferior lid up slightly)

NORMAL LID POSITION

- Patient should look in primary gaze and judge lid position relative to the limbus
- Upper lid crosses limbus at 2:00 and 10:00
- Upper lid covers 1 to 2 mm of superior cornea
- Lower lid just grazes inferior limbal region
- Do NOT judge lid position relative to the pupil margin
- Watch out for frontalis contraction

Normal lid position?
WARNING!!

Do not measure/evaluate lid position relative to the pupil margin. Use the distance from limbus to lid margin.

LID - PUPIL RELATIONS

- CN III - superior lid down (a lot usually), pupil larger
- Horner’s - superior lid down a little; pupil smaller
  - May not detect ptosis if lid margin evaluated/measured from pupil margin!! Use the limbus as the landmark.

SIGNIFICANCE OF LID POSITION EVALUATION

- Helps to confirm presence of efferent pathway lesion especially Horner’s (88% of Horner’s have ptosis)
- Helps to localize a lesion within pathway especially CN III
- Helps to differentiate type of lesion

THREE (or FIVE) STEPS IN PUPIL EVALUATION

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light →
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect
**PUPILLARY LIGHT REFLEX IS A TWO-LIMBED REFLEX ARC**

- Afferent pathway (CN II)
- Efferent pathway (CN III)

**PUPILLARY LIGHT REFLEX CHECKS BOTH SIDES OF THE TWO-LIMBED REFLEX ARC**

- Afferent pathway
  - Important to pupillary light reflex
- Efferent pathway
  - Important to pupillary function
  - Damage causes poor light response, near response or poor dilation in dark (oculo-sympathetic path)
  - Important to pupil size
  - Damage can cause an anisocoria

**POOR DIRECT LIGHT REFLEX**

Differential Diagnosis

- Afferent pathway lesion
- Efferent pathway (CN III) lesion
- Pharmacologic block of sphincter
- Mechanical damage/restriction of sphincter i.e. trauma, posterior synechiae
- Adies pupil

**CONSENSUAL LIGHT REFLEX USE IN DECISION-MAKING EXAMPLE**

- 4mm OU
- No direct response in OD; no consensual response in OS
  - What is the ddx at this point?
- Normal direct response in OS with normal consensual response in OD
  - Which of the differentials are ruled out by this finding?

What is the most probable cause?

**THREE (or FIVE) STEPS IN PUPIL EVALUATION**

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect

**NEAR PUPIL REFLEX**

- Response of pupils to a near stimulus
- Normally slightly slower and slightly weaker than light reflexes
- No need to evaluate near responses if normal light responses
THE NEAR PUPIL REFLEX

Why check it?

• To bypass the afferent pathway in order to prove that the efferent (CN III) pathway is normal
• An afferent path lesion affects the light response but not the near response (if proprioception is used to drive the near response)

LIGHT-NEAR DISSOCIATION

Near reflex is stronger/faster than light reflex

DDx of LIGHT-NEAR DISSOCIATION

• Afferent pathway lesion
• Adies pupil
• Aberrant regeneration of CN III
• Diabetes
• Argyll-Robertson pupils
• Midbrain lesion

NEAR REFLEX USE IN DECISION-MAKING - EXAMPLE

• 4mm OU
• No direct response in OD
• Normal direct response in OS
• Normal near response OU

What does the normal near response in OD indicate?
What is the most probable cause of this?

THREE (or FIVE) STEPS IN PUPIL EVALUATION

• Evaluate for anisocoria
• Evaluate lid position
  — If anisocoria
• Evaluate responses to light
• Evaluate responses to near
  — If ↓ light response
• Evaluate for afferent pupillary defect —

How to check for an afferent pupillary defect?

Compare the direct pupil response in one eye to the direct pupil response in the other eye
Compare speed, magnitude and presence of “escape”
What is an afferent pupil defect (APD)?

The light response in one eye is less than the light response in the other eye due to an afferent pathway lesion (not some other cause). An APD is due to unilateral or asymmetric afferent pathway damage.

EVALUATE FOR AFFERENT PUPIL DEFECT

• How? Compare direct light response in OD to that in OS by checking the light responses in rapid succession.
• Why? To detect very subtle difference between the pupillary light reflex of OD compared to OS.

AFFERENT PUPIL DEFECT

Other Names

• APD or +APD
• Relative afferent pupil defect (RAPD)
• Marcus-Gunn pupil
• + Swinging flashlight test (+ SFLT)
• Pupillary escape

Differential Diagnoses

• Optic nerve disease
• Macular disease
  Very gross macular lesion
• Retinal detachment
  Large RD
• Chiasmal disease
• Optic tract lesion
  Very extensive or complete tract lesion produces only a mild (1+ or 2+) APD

Most common causes:

• Optic nerve disease
  − Only a little ON damage, especially inflammation of ON (optic neuritis), causes large APD.
  − May or may not (retrobulbar damage) be able to visualize the ON damage on ophthalmoscopy → depends on where the damage is and how soon you look after the damage has occurred.
• Extensive retinal damage
  − Large macular/retinal lesion causes little APD
  − You should easily see the fundus lesion.

CLINICAL TIP

If a large APD is present with macular/retinal disease or amblyopia → rule out ON disease!
OTHER CAUSES OF APD

- Chiasmal disease
- Contralateral extensive optic tract lesion
- Gross macular disease
- Large, extensive retinal lesion
  - RD
  - BRAO/CRAO
  - Ischemic CRVO

Case Example

30 y/o male
CVC: ↓ vision and side vision OU for over 1 year, getting worse
Hab VAs: 20/50, 20/50
CF confrontations: Very dim in superior temporal quadrant OU
Pupils: 5 mm OU, 2+ direct OU, 4+ near OU, 1+ APD OS
D.O. see slides
Case Example
Will this patient have a pupil anomaly?

Bilateral APDs???
Is it possible?

Bilateral APDs do not exist!!!
Bilateral L-N dissociation does.
An APD only occurs when the light response is worse in one eye than the other eye.

CLINICAL TIP
There will be ophthalmoscopic evidence of visual pathway damage for most lesions causing APD.
Look for: ON pallor/atrophy, RNFL dropout, macular lesion, RD etc.

CLINICAL TIP
Cataracts rarely produce an afferent pupillary defect.
(A dense cataract can produce an APD in the other eye!!)
CLINICAL TIP
If an APD is present on the same side as a cataract rule out afferent pathway damage especially ON disease

CLINICAL TIP
If a patient has both anisocoria and an APD → there are two different causes/lesions.

ANISOCORIA IS VERY RARELY RELATED VIA A SINGLE DISEASE OR LESION TO AN APD

APD GRADING SYSTEM
4+ little, if any, light response in affected eye → big difference in light reflexes between the two eyes
3+ some light response (near normal) but quicker-than-normal pupil escape
2+ slight response to light in one eye
1+ very slight difference between the two eyes light responses. Affected eye has near normal light response but quicker escape.

TECHNIQUE OF CLINICAL EVALUATION OF THE PUPILS
How to test pupils!
THREE (or FIVE) STEPS IN PUPIL EVALUATION

• Evaluate for anisocoria ←
• Evaluate lid position
  – If anisocoria
• Evaluate responses to light
• Evaluate responses to near
  – If ↓ light response
• Evaluate for afferent pupil defect

EVALUATION FOR ANISOCORIA

Technique – Room Lighting

• Use dim or semi-dark room to look for anisocoria
• If no anisocoria then guessitmate (no need to measure) the pupil sizes
• If anisocoria seen or suspected then measure pupil sizes in bright and dark

EVALUATION FOR ANISOCORIA

Technique

• Gross external evaluation to rule out evidence of trauma or external disease
• Guessitmate pupil size; critically evaluate for anisocoria
• Measure pupil size in bright and dark if anisocoria seen or suspected
• Fixation at distance
  – Watch out for miosis as you approach eye to measure size
• No drops prior to pupil testing
• Burton lamp excellent for dim

CLINICAL TIP

Always rule out anisocoria before checking the light reflexes and before evaluating for APD

WHY?
THREE (or FIVE) STEPS IN PUPIL EVALUATION

- Evaluate for anisocoria
- Evaluate lid position →
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect

EVALUATION OF LID POSITION

Technique

- Distance fixation
- Gross external exam
- Note lid positions relative to limbus
  - Don’t judge relative to pupil margin

Normal lid position??

Measure lid position/ptosis relative to the limbus

THREE (or FIVE) STEPS IN PUPIL EVALUATION

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light →
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect

PUPILLARY LIGHT REFLEX EVALUATION

Technique

- No prior drops or tonometry
- Identify any anisocoria FIRST
- Subdued room illumination
- Patient fixates a distant target
- Use bright, cool light source
- Direct source into eye from just below the visual axis
- Evaluate direct response (2-3 trials)
- Grade response from 4+ (strong, brisk) to 0 (no response)
- View from side, not on patient’s line of sight
NO DIRECT LIGHT REFLEX
(Or no light perception claimed)
Strategy to Detect Minimal Light Response

- Transilluminator
- BIO on maximum
- Slit lamp on maximum

THE NORMAL DIRECT LIGHT REFLEX

1. Pupil constricts briskly
2. Slow dilation to intermediate size
3. Pupillary unrest (Hippus)

POOR OR NO DIRECT LIGHT RESPONSE

Potential causes:
- Afferent pathway lesion
- CN III lesion
- Pharmacologic block of sphincter
- Local mechanical iris/sphincter damage
- Adies pupil

THREE (or FIVE) STEPS IN PUPIL EVALUATION

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect

EVALUATION OF THE NEAR REFLEX
(Evaluation for Light-Near Dissociation)

- Distance fixation
- Patient holds finger on nose & looks at it
  - Strong proprioceptive stimulus is best→ patient’s finger on patient’s nose
- Evaluate for near miosis
- Direct fixation to distance
- Evaluate for redilation on distance fixation
  - Redilation may be much easier to see
In testing the near reflex, the redilation on viewing distance (after near viewing) is very often much easier to detect than the near miosis.

**CLINICAL TIP**

**THREE (or FIVE) STEPS IN PUPIL EVALUATION**

- Evaluate for anisocoria
- Evaluate lid position
  - If anisocoria
- Evaluate responses to light
- Evaluate responses to near
  - If ↓ light response
- Evaluate for afferent pupil defect —

**SWINGING FLASHLIGHT TEST TECHNIQUE**

- Identify any anisocoria first
- Room illumination: subdued to dark
- Patient fixation: distant object
- Light source: transilluminator
- Direct beam into OD from just below visual axis
- Observe direct light reflex (in OD) until maximal miosis
- At maximal miosis swing to OS
- Observe direct light response in OS
- Swing at moment of maximal miosis
- Watch carefully for “escape”

**CLINICAL TIP**

**Common causes of pseudo-APD**

- Anisocoria
- Penlight (poor light source)
EXAMPLE OF APD
MILD (1-2+) APD OD

- Light into OD → constriction followed more quickly than normal by dilation (pupillary escape)
- Light into OS → constriction followed by dilation

EXAMPLE OF APD
SEVERE APD OD

- Light into OD → minimal or no constriction
- Light into OS → strong, rapid constriction

SWINGING FLASHLIGHT TEST

Comparison of direct response in OD to that of OS to detect very subtle difference.

Go to UC Davis home page and search "eye simulator"

UC Davis Eye Simulator
Watch demo, test pupils yourself or take quiz!!!
Bilateral APDs???
Is it possible?

Bilateral APDs do not exist!!!
(Bilateral L-N dissociation does.)
An APD occurs when the light response is worse in one eye than the other eye.

CLINICAL TIP
Reverse SFLT

Only one working pupil is needed to check for APD.
So use the reverse swinging flashlight test anytime only one pupil is functional or visible or if you don’t trust one!

REVERSE SWINGING FLASHLIGHT TEST

Technique
Direct light into normal pupil, note its response
Upon maximal pupillary constriction quickly direct light into abnormal pupil while noting the response (consensual response) in the normal pupil
Again direct light into normal pupil, note its response

Indications:
• Abnormal or possibly abnormal pupillary light reflex
• Corneal scar
• Pharmacological block of the sphincter
• Traumatic sphincter damage
• Posterior synechiae
• Old CN III, old Adies
• You don’t “trust” the pupil

SUBJECTIVE MARCUS GUNN TEST

• Subjective, unilateral reduction in perceived brightness due to afferent pathway lesion
• Relative to the other eye
• Not as reliable as regular swinging flashlight test

Technique:
• Present the light source before each eye in sequence
• Direct patient fixation toward the light source
• Ask for the patient to note any apparent interocular difference in perceived brightness
• Try to quantify - “If this is 100% bright (light source before eye with greater perceived brightness) what would this be worth (light source before other eye)?”
CLINICAL TIP

Biggest problem with the subjective Marcus Gunn test → its subjective!!

PUPIL EVALUATION

Recording the results

Minimum necessary on a normal pupil

- Pupil size (guessitmate)
- Light reflexes (graded)
- Presence or absence of afferent defect

PUPIL EVALUATION - RECORDING

Additional recording as needed:

- Anisocoria
  - Record pupil size in dark and in bright
- Abnormal lid position
  - Describe position relative to the normal position
- Abnormal light reflex
  - Perform and record near reflex
- Regular SFLT not possible
  - Record reverse SFLT

What is the most probable cause of this?

MINIMUM RECORDING - NORMAL PUPILS

P 5 RRL 4+ -APD

In Examwriter:
5 mm OU, 4+ OU, -APD
ANISOCORIA
THREE CLINICAL QUESTIONS

• Is the anisocoria pathologic?
• Which pupil is abnormal?
• What is the cause of the abnormality?
  – Local
  – Neuromuscular
  – Nerve
  – Central (brain)

P^4_{R} RRL^{2+} A^{4+} 2+ APD (OS) (reverse)

P^{8\rightarrow8}_{5\rightarrow2} RRL^{0+} A^{0+} - Rev APD
(Rt ptosis 3-4 mm)

P^{8\rightarrow8}_{5\rightarrow2} RRL^{0+} A^{0+} 0+ APD (rev)
(No ptosis, normal CN III)

NORMAL PUPIL FINDINGS

Size: 3 to 7 mm, reducing with age
Shape: Round
Equality: 80% of normals - equal
~20% of normals have physiologic anisocoria

PHYSIOLOGIC ANISOCORIA

• Anisocoria - always rule out efferent pathway lesion
• No lesion in efferent pathways
• Mimics more significant causes of anisocoria i.e., Horner’s
  – Horner’s can be life-threatening!
PHYSIOLOGIC ANISOCORIA
- 20% of population under age 17
- 30% of population over age 60
- 4% of general population
  anisocoria > 1mm

PHYSIOLOGICAL ANISOCORIA
- Often transient
- Often switches eyes
- No neurological signs
  - Normal pupil function, normal EOMs, no ptosis
- Anisocoria roughly equal in bright and dark
  - Rarely > 1 mm (~4% of population); generally < 1/2 mm

PHYSIOLOGICAL ANISOCORIA vs. HORNERS SYNDROME
- No dilation lag
- No ptosis, no inverse ptosis
- Watch out for dermatochalasis + physiological anisocoria – it will mimic the appearance of Horner’s

PUPIL SIZE
- BILATERAL INCREASED PUPILS
  - Normal: Young, blue-eyed/blond, myopes, anxiety
  - Other factors:
    - Hyperthyroidism, OTC meds (sympathomimetics or anticholinergics), recreational drugs i.e., cocaine

PUPIL SIZE
- BILATERAL PUPIL CONstriction
  - Infants
  - Elderly
  - Parasympathomimetics (miotics)
  - Iritis
  - Garden poisons (cholinesterase)
  - Diabetes
HORNER’S SYNDROME (Oculosympathetic paresis)

Disruption of the 1st, 2nd or 3rd order sympathetic neuron in the oculosympathetic pathway

- Miosis
- Dilation lag
  - Check within 5-6 seconds of turning lights out then again 10-15 seconds after
- Slight ptosis
- Inverse ptosis
- Apparent enophthalmos
- Facial anhydrosis (if pre-ganglionic)
- Heterochromia irides (if congenital)
- Conjunctival hyperemia
- Increased accommodative amp?

HORNER’S SYNDROME

DILATION LAG

- Check (measure) pupils in bright
- Turn off lights (dark)
- Remeasure pupils in dark (Burton lamp) within 5 seconds
- Remeasure pupils after 15 seconds
- Anisocoria increases (due to slow dilation) within 5 seconds then decreases at > 15 seconds

HORNER’S SYNDROME

CLINICAL SIGNIFICANCE

- Many preganglionic Horner’s are due to malignancies!!
- Most post-ganglionic (3rd order) Horner’s are benign
HORNER’S SYNDROME
COMMON CAUSES BY AGE OF ONSET
• Birth to 20 - congenital, trauma
• 20 to 50 - tumor (~ 50%)
• ≥ 50 - neoplasm is leading cause!

HORNER’S SYNDROME
DATING THE ONSET
• Congenital - heterochroma irides
• Old photos - look for anisocoria, enophthalmos or ptosis, heterochromia if child

HORNER’S SYNDROME
DDx of PRE- vs. POST-GANGLIONIC
• Lack of facial sweating → pre-ganglionic
• Symptoms/signs of apical lung carcinoma
  – Cough, arm pain, long term smoker
• Pharmacologic tests

ADIES TONIC PUPIL
• Disruption of CN III pupil/ accommodation fibers
• Probable site is ciliary ganglion behind globe

ADIES PUPIL
• Viral URI
• Sinusitis
• Trauma to orbit
• Tooth extraction
### ADIES PUPIL

#### Typical Patient
- Middle-aged
- Female
- Recent URI (viral)

#### ADIES PUPIL
- Anisocoria greatest in bright
  - Due to sphincter (CN III parasympathetics) paresis
- 80-90% are unilateral
- Acutely - poor or no light response
- Tonic (slow) near response
  - Light - near dissociation
- Partial accommodative paresis
- Segmental iris sphincter paresis → segmental miosis
- Possibly ↓ DTRs (Adies syndrome)

#### ADIES PUPIL

**DDX From CN III**
- Light response ↓ or absent but better near response
- Normal EOMs - SR, MR, IO, IR
- Normal levator (no ptosis)

#### ADIES PUPIL

**Long-term Changes**
- Usually small & sluggish to light
- Pupil may be normal in size (but not responsive to light)
- Near reflex improves
- Accommodation usually normal within 2 years
- DTRs ↓