CRANIAL NERVE TESTING IN ACUTE NEUROLOGY

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ANISOCORIA

Step 1: Examine the eyes both in the light and in the dark

Step 2:

THE PUPIL IN LIGHT VS. DARK	CAUSES
Difference greater in the dark = MIOSIS *There is a* **dilation lag** *meaning the smaller pupil is the abnormal one*	*Disruption to the sympathetic pathway* **First-order neurons**: Injury to brainstem and cervical spine, such as in Lateral Medullary Syndrome **Second-order neurons**: Pancoast tumor, chest pathology, brachial plexus pathology **Third-order neurons** (*no associated anhidrosis*): internal carotid artery dissection, neck surgery, cavernous sinus pathology
Difference greater in the light = MYDRIASIS *There is inability of the dilated pupil to constrict appropriately*	*Disruption to the parasympathetic pathway* **Cranial nerve III palsy**: posterior communicating artery aneurysm, tumor, temporal lobe uncal herniation **Cavernous sinus pathology** Remember that **anti-cholinergic drugs** can also result in mydriasis. In hospitalized patients, always look for a scopolamine patch or recent administration of nebulized ipratropium (in DuoNeb®) to assess whether the dilated pupil resulted from a medication effect

Step 3: Look for associated pathology.

☐ **If miosis**: look for a Horner's syndrome (anhidrosis and ptosis), for evidence of a lateral medullary syndrome (see "Brainstem Syndromes"), or for evidence of cavernous sinus pathology (testing extraocular eye movement and sensation in the V1/V2 distribution).

☐ **If mydriasis**: look for disorders of consciousness and consider a STAT scan; if awake, examine extraocular eye movements and facial sensation, consider CT angiogram to evaluate for expanding posterior communicating artery aneurysm.

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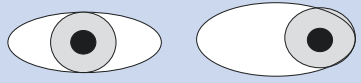
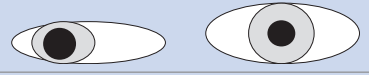
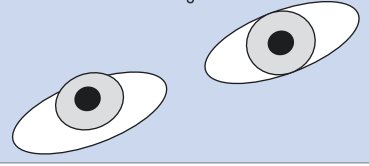
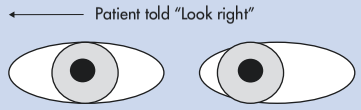
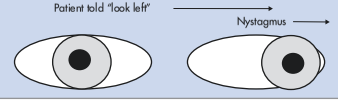
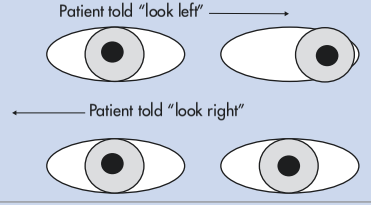
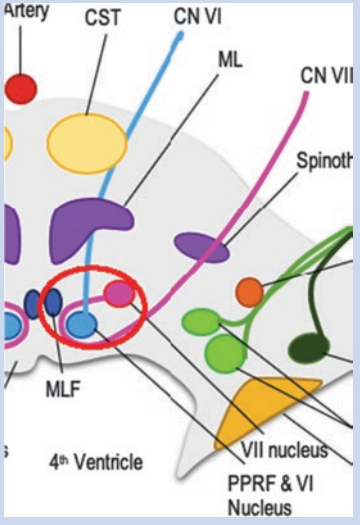
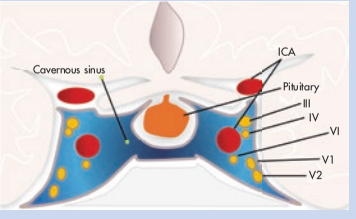
LOCALIZING EXTRAOCULAR EYE MOVEMENT ABNORMALITIES

LESION	PRESENTATION	COMMON AREAS OF INJURY	BEDSIDE TRICKS
CN III (Oculomotor) Nerve Palsy	Impairment in adduction (*medial rectus*), elevation (*superior rectus*), and depression (*inferior rectus*) + ptosis (*levator palpebrae*) and mydriasis (*parasympathetics*) Pupil-sparing/subtle right CN III: Patient told "look left" →  Full right CN III (looking straight): Full Right CN III: 	Midbrain nucleus, compression of the nerve by posterior communicating artery aneurysm, uncal herniation, pathology at the superior orbital fissure (SOF), cavernous sinus (CVS)	A compressive etiology leads first to pupil dilation prior to ophthalmoplegia
CNIV (Trochlear) Nerve Palsy	Impaired intorsion and depression in the adducted position (*superior oblique*) Right CN IV palsy: Patient told "tilt head to Right" 	Dorsal midbrain, pineal mass, pathology at the SOF/CVS	The diplopia worsens with head tilted toward the side of the lesion Patients may complain of diplopia when looking down (e.g., when going downstairs)

LESION	PRESENTATION	COMMON AREAS OF INJURY	BEDSIDE TRICKS
CN VI (Abducens) Nerve Palsy	Impaired abduction (*lateral rectus*) <u>Right CN VI palsy:</u> 	Increased intracranial pressure, cavernous sinus pathology, trauma	Diplopia is worse with far vision
Medial Longitudinal Fasciculus (MLF) Injury	Results in an *internuclear ophthalmoplegia (INO)*: impairs the coordination of ipsilateral CN III (impaired ipsilateral adduction) on contralateral gaze <u>Right INO:</u> On left gaze, the right eye does not adduct and the left eye usually displays nystagmus *Difference with CN III there is no ptosis and convergence is not impaired* 	Most commonly multiple sclerosis, or any stroke/lesion affecting the MLF	A right MLF lesion results in impaired right eye adduction when looking left Bilateral INO would result in inability to adduct either eye on horizontal gaze

LESION	PRESENTATION	COMMON AREAS OF INJURY	BEDSIDE TRICKS
One-and-a-half Syndrome	A lesion affects the crossed MLF + PPRF and/or CN VI There is no horizontal gaze to the affected side because of the CN VI/PPRF involvement Adduction of the ipsilateral eye on contralateral gaze is impaired due to MLF disruption between CN VI and CN III <u>Right one-and-a-half</u> (due to R-INO + R-CN VI/PPRF lesion): *Only the left eye can abduct* 	Lesion in the caudal pons 	This leaves only one horizontal movement: *abduction of the eye which is contralateral to the lesion* Often will cause an ipsilateral LMN pattern of facial weakness (by affecting CN VII, which is sometimes termed an "eight-and-a-half syndrome" (i.e., 7 + 1.5)
Cavernous Sinus (CS) Pathology	When severe, results in complete ophthalmoplegia on the effected side Pathology in the CS usually starts with a sixth nerve palsy	Cavernous sinus thrombosis, carotid-cavernous fistula, pituitary tumor, Tolosa-Hunt syndrome, pituitary apoplexy	
Skew Deviation	Usually caused by a central lesion resulting in vertical misalignment	Can be caused by lesion to brainstem, cerebellum or rarely CN VIII injury	Usually the eye on the side of the lesion is higher and intorted

CALORIC TESTING

Both warm and cold water can be used to activate the endolymph of the inner ear resulting in a current that activates the hair cells. This movement of the hair cells results in polarization (warm) or hyperpolarization (cold) of the ipsilateral vestibular verve and apparatus of the brainstem [1]. In the ICU, cold water is preferentially used.

Cold water irrigation of the external auditory canal results in movement of the endolymph in a way that causes hyperpolarization resulting in the inhibition of the vestibular nerve.

The normal response to cold water:

- A slow movement of the eyes *towards* from the stimulates, with the fast component of nystagmus beating *away* from the stimulated ear.

In coma:

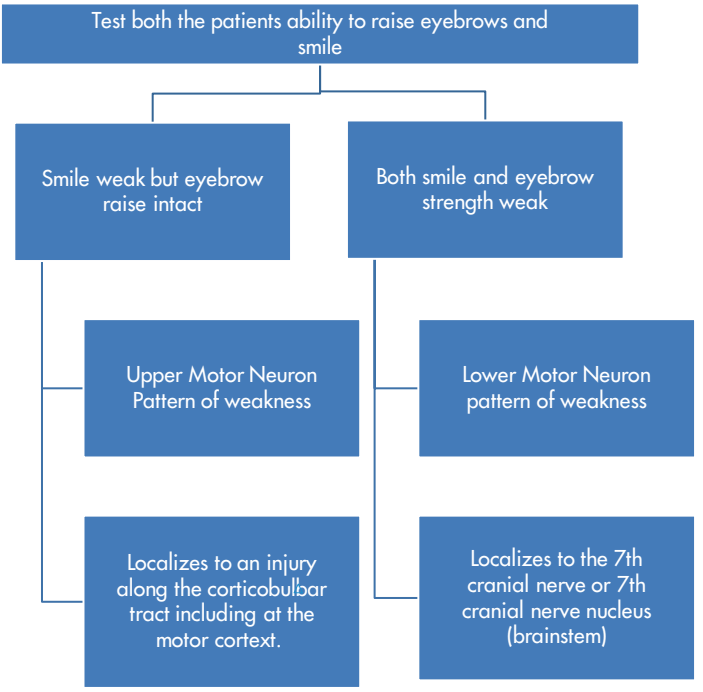
- There is no corrective saccade because the frontal eye fields are not activated (due to absent cortical function), thus the eyes will *only* have the slow movement *towards* the cold stimulus.

In brain death testing:

- There is *no* movement of the eye when the patient's ear canal is irrigated with cold water.

GENERAL PATTERNS OF FACIAL WEAKNESS

Note that testing facial weakness in less acute patients should involve assessing auditory function (for hyperacusis) and taste. Note that the facial nerve also receives projections from the extrapyramidal systems and frontal lobe which control emotional expression. Thus, patients with upper motor pattern of weakness may actually be able to activate their face involuntarily when associated with an emotional expression.



In comatose patients, the seventh and fifth cranial nerves are assessed by testing the corneal reflex:

The cornea is the clear layer of tissue over the iris. Touching the cornea transmits a signal to the brainstem via the Trigeminal Nerve (CN V) and the blink motor response is carried out due to innervation from the Facial Nerve (CN VII). If either of these are damaged (such as by a stroke or bleed effecting the brainstem) the patient will not blink to the gentle touch of the cornea with a cotton swab. Often tested at the bedside by dropping a drop of a saline flush into each eye, this is less sensitive but does not risk injury to the cornea. Take care to actually touch the cornea and not just the sclera (the white of the eye).

TESTING THE GAG REFLEX (PHARYNGEAL REFLEX)

Sensation mediated predominantly by the glossopharyngeal nerve (CN IX). Motor response mediated by the vagus nerve (CN X). In ICU patients, this is best tested by advancing a tongue depressor around the endotracheal tube and stimulating the oropharynx.

TESTING COUGH REFLEX

Mediated by the vagus nerve. In intubated patients, it is most easily tested by advancing in-line suction through the endotracheal tube which will stimulate the trachea. Be aware that coughing will typically trigger a temporary high airway pressure alarms on the ventilator.

REFERENCE

1. Gonçalves DU, Felipe L, Lima TM. Interpretation and use of caloric testing. Braz J Otorhinolaryngol. 2008;74(3):440–6.