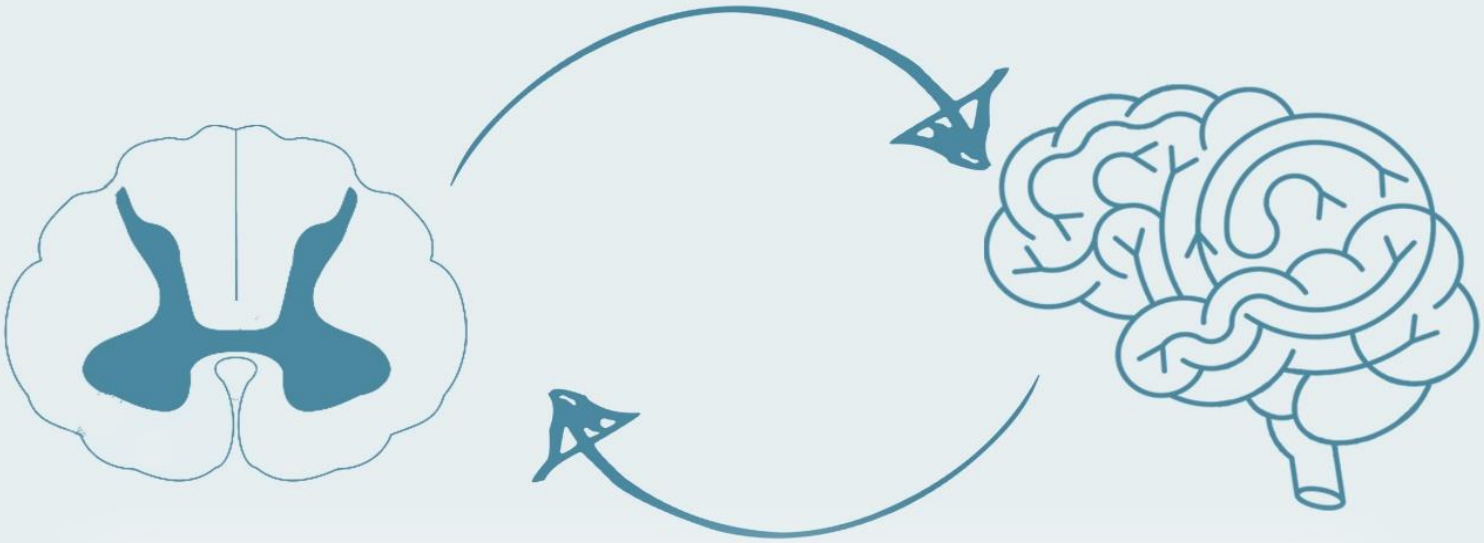

CNS

physiology

slides | sheets

number	12
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Introduction

In this sheet, we continue with the topic of pupillary light reflex and its lesions. We also discuss anisocoria and the topic of corticospinal and corticonuclear tracts.

Pupillary Light Reflex*; Tracts and Lesions*

In the previous lecture, we discussed the general aspects of this important reflex. We also mentioned the consequences of lesions along its pathway. For example, if there is a lesion in the right optic tract, then exposing either eye to light results in constriction in both eyes.

Optic Nerve and Optic Chiasm

We said previously that the nasal fibres of optic nerve cross in the optic chiasm; while the temporal fibres do not. The crossing:non-crossing ratio is not equal to 1:1; crossing fibres make 60% of total fibres, whereas 40% of fibres are non-crossing, and continue ipsilaterally.

According to that, when the right eye is exposed to light in normal settings, the right pretectal nucleus will receive 40% of the right eye fibres from the right optic tract, while the left pretectal nucleus will receive the remaining 60% from the left optic tract. After that, each Edinger–Westphal nucleus receives 100% of the activation (since each Edinger-westphal nucleus receives fibres from the two olivary pretectal nuclei). Consequently, both eyes constrict by 100% of expected.

To put that in an example; if there is a defect in the left optic tract, the following happens:

1. Light on right eye: both eyes' pupils constrict, but they only constrict by 40%; since only 40% of the impulses reached Edinger–Westphal nuclei on both sides.
2. Light on left eye: constriction of both eyes' pupils by 60% occurs (more consriction).

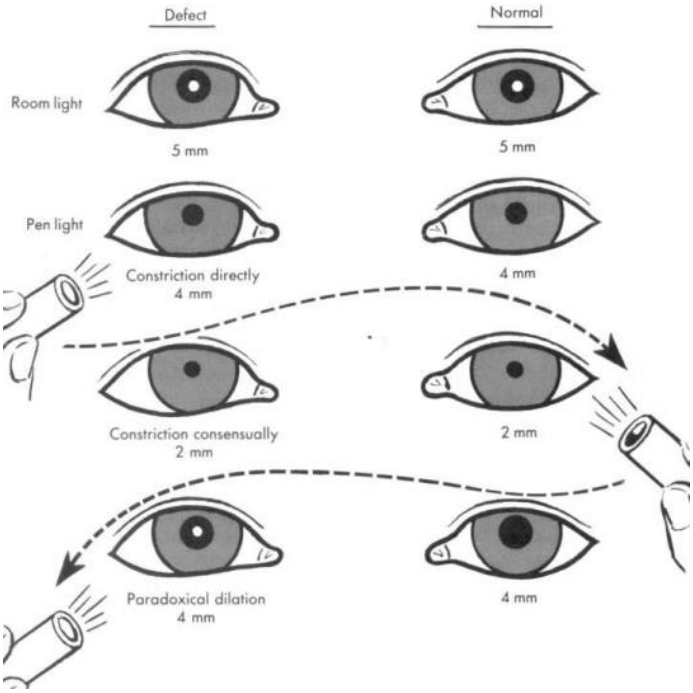
Clinically, it is hard to notice this difference in constriction in these two cases. So, we use the following test to diagnose such lesions.

Swinging-Flashlight Test

In this test, we do the following:

1. Shedding light on right eye, then on left eye.
2. Swing the light from one eye to the other and return back to the starting position.

Normally, constriction does not change while swinging the flashlight. However, in case of optic tract lesion for example, pupils expand when light is on the eye contralateral to the tract defected. So, in the previous example, the damaged tract is the one in the left side, and pupils dilate when light is being swung from the left eye to the right eye (the affected eye). In this case, we say that there is an afferent pupillary defect in the right eye (the one that its pupil dilated more in the test), and because this defect is not complete, we call it Relative Afferent Pupillary Defect (RAPD).



00:00 – 10:00

Relative Afferent Pupillary Defect (RAPD)

The swinging-flashlight test is used to test for the presence of RAPD. Although optic tract lesions can result with RAPD, it can be caused by other lesions and diseases that affect the afferent limb of the pupillary light reflex unilaterally. Etiology includes:

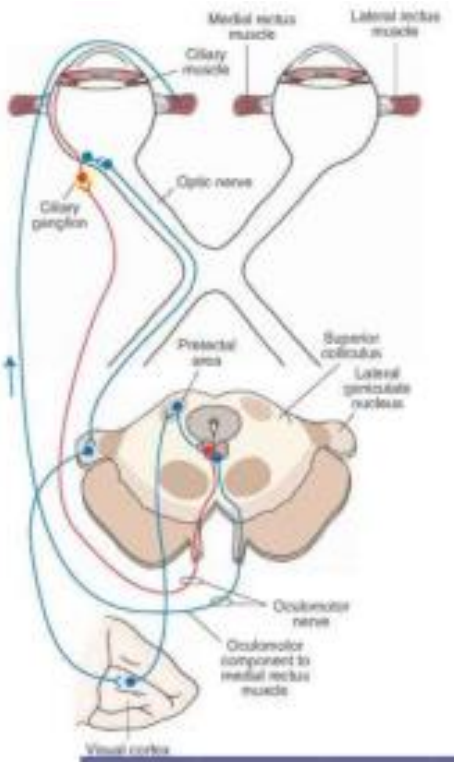
- Usually a before-chiasm problem, such as: glaucoma, retinal detachment, ischemic retina, optic nerve lesions (ischemia, compression neuritis, recovered neuritis … etc.), diabetic retinopathy and demyelination (Multiple Sclerosis). (Note that these problem in this case will not result in complete loss of the afferent limb)
- Unilateral Optic track lesion
- Unilateral midbrain lesion

Example: glaucoma in the right eye can result in retinal degeneration. This decreases the impulses generated from the eye when light hits it, and so less impulses reach the midbrain. So, in swinging-flashlight test, pupils dilate when light is on the right eye (the affected eye).

Note that in RAPD, the eye that results with dilation of pupils when light hits it is said to be the affected eye, or the eye with afferent pupillary defect.

The Pupil Near Reflex

Light is not the only thing that causes pupils constriction. When seeing near objects, pupils constrict, via the pupil near reflex. From physical perspective, the function of this reflex is to aid in optic focusing and to increase resolution by increasing the focal depth of retina and lens. Note that this reflex works even at dim light.



<u>The Neural Pathway</u>

Just like what we observed in the selective amplification pathway in hearing, the cortex is the center that realizes the need to initiate this reflex. So,

the pathway starts from the cortex (ipsilaterally), and then heads to pre-tectum (other area than olives).

From pre-tectum, fibres reach Edinger–Westphal nuclei to cause pupils constriction by the action of oculomotor nerves.

Pupillary Light Reflex Versus Near Reflex

You may have noticed that although they have the same target (pupillary constrictor muscles), these two reflexes differ in their afferent limb (cortex-pre-tectum for near reflex, and optic nerve-pre-tectum for light reflex).

Light-Near Dissociation (Adie's tonic pupil)

If one of these reflexes is damaged but the other is kept intact, constriction occurs with light exposure but not by near objects, or vice versa. This is called light-near dissociation, and is a result of conditions that damage the posterior part of the midbrain, which damages one of these two pathways without affecting the other. (That is because each one of these two pathways has its area in the midbrain.)

The following points are examples of such conditions (causing dorsal midbrain syndrome (Parinaud's Syndrome)):

- Stroke
- Meningitis
- Tumor
- Neurosyphilis
- Diabetic neuropathy: DM affects big and long axons more than short axons.
- Demyelination (Multiple Sclerosis)

Note: lesions in the visual pathway before the midbrain (optic tracts lesions, for example) do not cause light-near dissociation.

Anisocoria

Anisocoria is a condition characterized by an unequal size of the eyes' pupils. 20 to 30% of normal people have physiological anisocoria (not severe). That is because the afferent sensory limb and the efferent motor limb of the pupillary light reflex do not divide equally between the two eyes.

Causes of Anisocoria

Causes include any problem that affect the sensory limb or the motor limb of the pupillary light reflex unilaterally (one eye is affected). Most cases are caused by defects in the motor limb of the reflex. Of the causes are one-sided dorsal midbrain syndrome and Horner syndrome.

Horner syndrome is the most famous syndrome that can causes anisocoria. Horner syndrome results from an interruption of the sympathetic nerve supply to the eye and is characterized by miosis (constricted pupil; parasympathetic effect), partial ptosis, and loss of hemifacial sweating (anhidrosis).

one-sided dorsal midbrain syndrome is an example of a defect that results with anisocoria by affecting the sensory limb of the pupillary light reflex (pre-tectum is damaged).

10:00 – 20:00

(Other details on this topic are not required)

Corticospinal and Corticonuclear Projections

Corticospinal (pyramidal) and corticonuclear tracts are motor descending corticofugal pathways. In these motor systems, motor orders originate in the cortex, and descend to the brainstem and the spinal cord. The target of

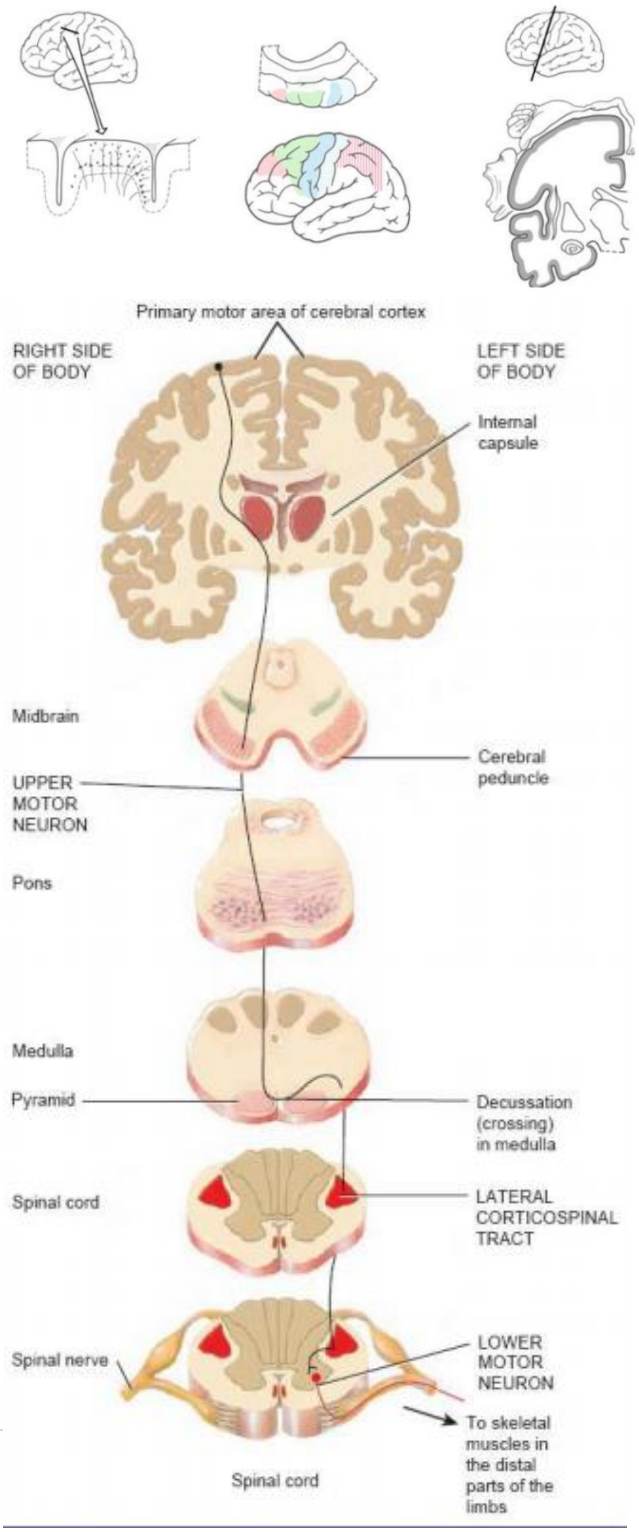
corticospinal tract is the cell bodies of lower motor neurons in the spinal cord, whereas the target of corticonuclear tract is cell bodies of the lower motor neurons in the motor nuclei of the cranial nerves in the brainstem.

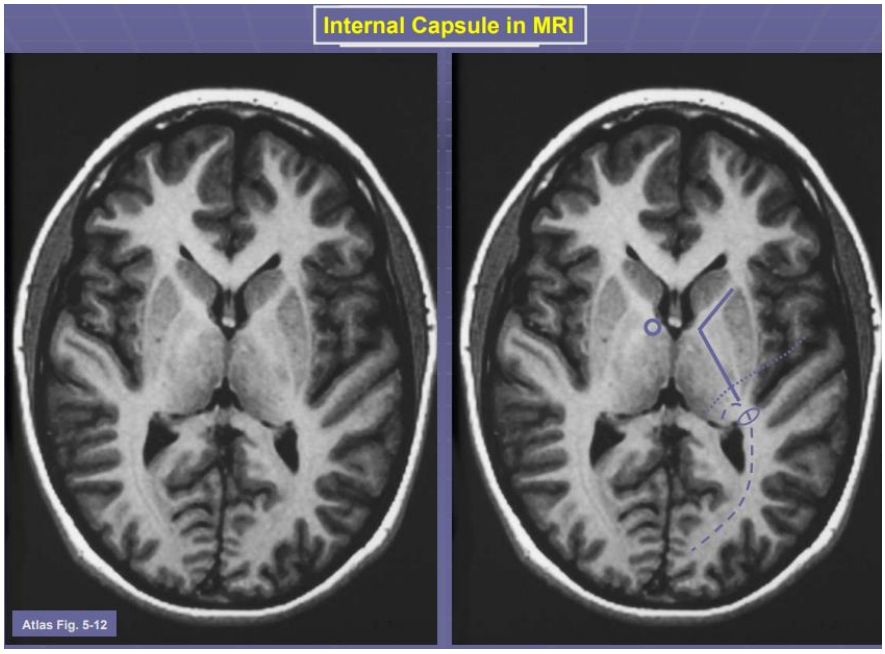
The Neural Pathways; Somatotopic Organizations

Corticofugal tracts come from different areas in the cortex; including primary motor area (area 4), less from area 6 and further less come from areas posterior to the central sulcus (areas 1, 2, 3, 5 and 7). (Details about such tracts will be discussed in later lectures.)

These tracts flow through the cortex, telencephalon, diencephalon and then to the brainstem, where the corticonuclear tracts reach the motor nuclei. The somatotopic arrangement of the motor cortex is similar to that of the sensory area; the lower limb is medial, and then come the trunk and the upper limb, and the face is the most laterally located (from which originate the corticonuclear tract).

When the fibres reach the diencephalon, they flip; face fibres are the most medial, and then come the fibres of the upper limb and the trunk, and the fibres of the lower limb are the most lateral. When the fibres pass through the internal capsule, corticonuclear tract goes through the genu of the internal capsule, whereas the corticospinal tract goes through the posterior limb of the internal capsule.





After that, fibres continue as that through the brainstem. In the midbrain, the fibres are found in the crus cerebri (in the same arrangement). During passing the brainstem, corticonuclear fibres (face fibres) exit to the motor nuclei of the cranial nerves.

When fibres of the corticospinal tract reach the pyramids, the arrangement does not change; the upper limb fibres are the most medial, and the lower limb fibres are the most lateral. In the pyramids, fibres undergo crossing, but they continue in the same arrangement. In the spinal cord, fibres to the upper limb (most medially located) leave in the cervical segments.

<u>Lesions in Relation to The Somatotopic Organization</u>

- Lesion in the spinal cord:
  Since the fibres of the upper limb are medial to those of the lower limb, an extradural (extramedullay) tumour would result with weakness in the lower limb first, and then in the upper limb, both ipsilaterally.
  (Note that tumours anterior to the dentate ligament would affect the ALS tract; however, tumours posterior to the dentate ligament would affect the corticospinal tract.)

- Remember the differences between upper motor neuron lesions and lower motor neuron lesions.

TABLE 16.1
Signs and Symptoms of Upper and Lower Motor Neuron Lesions

Upper Motor Neuron Syndrome	*Lower Motor Neuron Syndrome*
Weakness	Weakness or paralysis
Spasticity	Decreased superficial reflexes
Increased tone	Hypoactive deep reflexes
Hyperactive deep reflexes	Decreased tone
Clonus	Fasciculations and fibrillations
Babinski's sign	Severe muscle atrophy
Loss of fine voluntary movements	

- Pyramidotomy, leading to damage to the corticospinal tract:

  Experiments show that pyramidotomy does not result with complete paralysis. It rather leads to weakness and loss of ability to perform fine movements.
  In an experiment, a monkey underwent pyramidotomy. The following points were noticed:
  1. In less than a week, the monkey could stand, move and climb cages. The presence of multisynapse pathways in the extrapyramidal tracts, in addition to the presence of other tracts for motor function can explain such results.
  2. The monkey lost the ability to perform fine movements; for example, instead of holding things in its fingers, it used the palms of its hands.

Corticonuclear Projections*; Motor Nuclei and Lesions*

As we said, corticonuclear fibres start from the cortex, and then pass in the genu of the internal capsule. They proceed to reach the motor nuclei of cranial nerves in the brainstem. These nuclei are the accessory, facial, trigeminal motor, hypoglossal and ambiguus.

(Motor pathways of eye movements are discussed in the next lectured)

20:00 – 30:00

1. Accessory nucleus:

This nucleus is located in the lower part of the medulla. It mainly receives uncrossed (ipsilateral) fibres. Accessory nerve innervates two muscles:

- Sternocleidomastoid: tilts the head towards the shoulder on the same side, Thereby, rotating the head to turn the face to the opposite direction
- Trapezius: elevates the shoulder

A lesion affecting the accessory nerve on one side results with loss of head rotation to the opposite side and loss of the ability to elevate the ipsilateral shoulder.

Example: damaging the right accessory nerve or nucleus or a lesion affecting the right cortex, all these conditions result with loss of head rotation to the left side and loss of the ability to elevate the right shoulder.

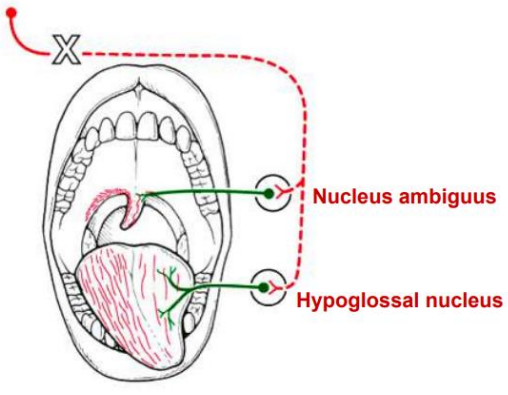
Note: if the patient presents with accessory nerve damage symptoms, we can predict the level and the side of the damage.

2. Nuclei that innervate genioglossus and uvula:

Nucleus ambiguus and nucleus of hypoglossal nerve receive mainly crossed fibres. The nucleus of hypoglossal nerve is present in the middle level of the medulla, whereas the nucleus ambiguus (the part that supplies the uvula) is present in the superior part of the medulla, just inferior to pons.

Functions:

- Inside uvula there is a muscle called musculus uvulae which has a pulling action (receives fibres from nucleus ambiguus through vagus nerve). Defects result with weakness of the muscle and tilting to the



side of the healthy muscle. A defect in the nerve or the nucleus results with tilting to the other side. However, a defect in the cortex or the corticonucleur tract will result with tilting to the same side.

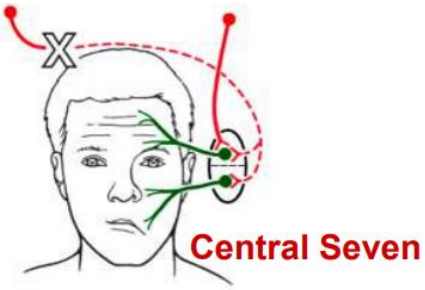
Example: a defect in the left internal capsule leads to weakness in the right muscle, resulting in tilting to the left. On the other hand, a defect in the right nucleus results with weakness in the right muscle and tilting to the left.

- Tongue:
  Genioglossus pushes the tongue outwards. A defect results with weakness of the muscle and deviation of the tongue to the side of the weakened muscle.

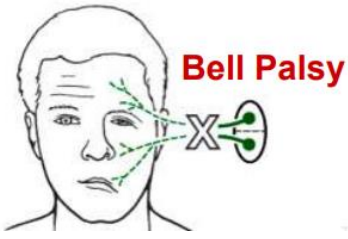
  Example: a lesion affecting the right nerve or the right nucleus results with weakened right muscle and deviation of the tongue to the right. However, damaging the left cortex or the left corticonuclear tract results with deviation of the tongue to the right.

3. Facial nucleus:

   This nucleus is located in pons. The part of the nucleus that supplies the upper part of the face is innervated equally bilaterally. However, the part of the nucleus that supplies the lower part of the face is mainly innervated contralaterally.



   Lesions in the nerve or the nucleus result with ipsilateral lesions to the face muscles. Defects in the cortex or the internal capsule do not cause upper face lesions; because the upper face is supplied bilaterally, however, they do result with lower part lesions, contralateral to the causative defect.



30:00 – 40:00

Note: nearly all the nuclei are supplied bilaterally, but the dominant side of supply is what we discuss here.