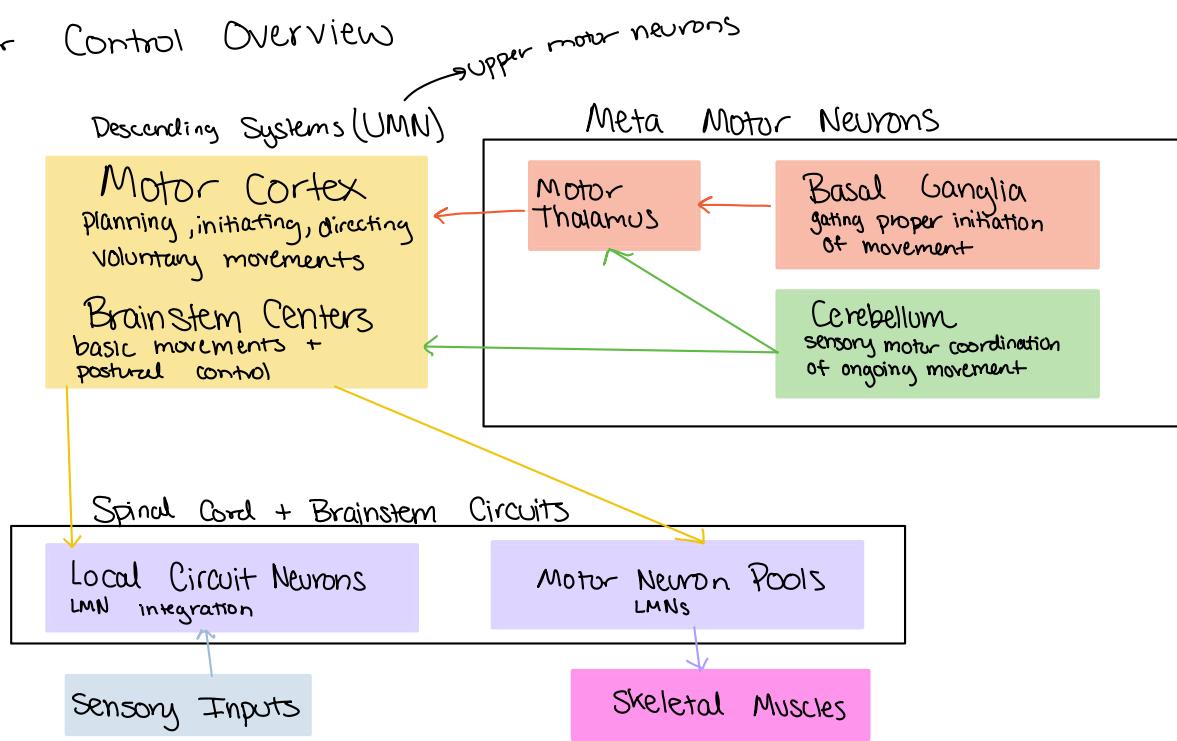
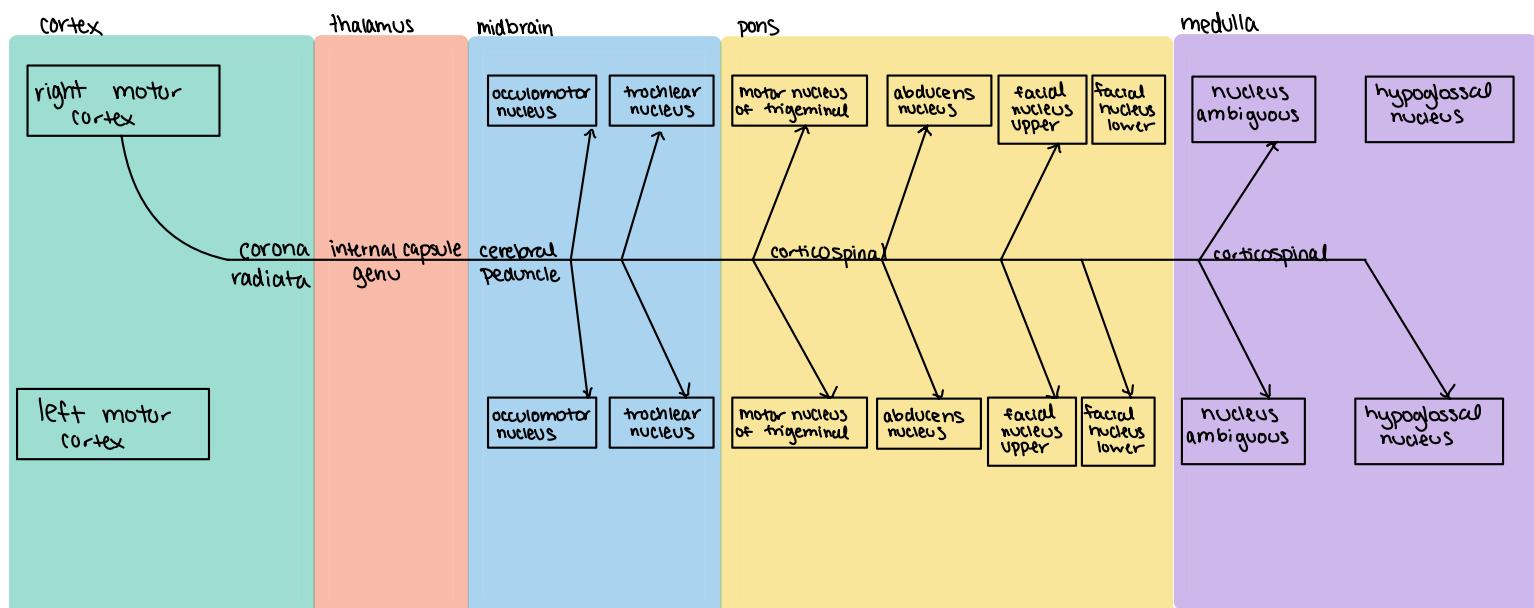


Motor Control Overview



Corticobulbar Tract



- cell bodies in motor cortex
- axons leave motor cortex via corona radiata
- enter internal capsule genu in thalamus
- midbrain → enter cerebral peduncle and some fibers exit bilaterally via CN3/CN4
- pons → fibers become corticospinals
 - synapse in motor nucleus of trigeminal in middle pons bilaterally
 - synapse in abducens nucleus in caudal pons bilaterally

- synapse in facial motor nucleus controlling upper facial muscles bilaterally
- synapse in facial motor nucleus controlling lower facial muscles on contralateral side
- CN 9, 10, 11 synapse bilaterally
- CN 12 synapse unilaterally

Damage to Corticobulbar

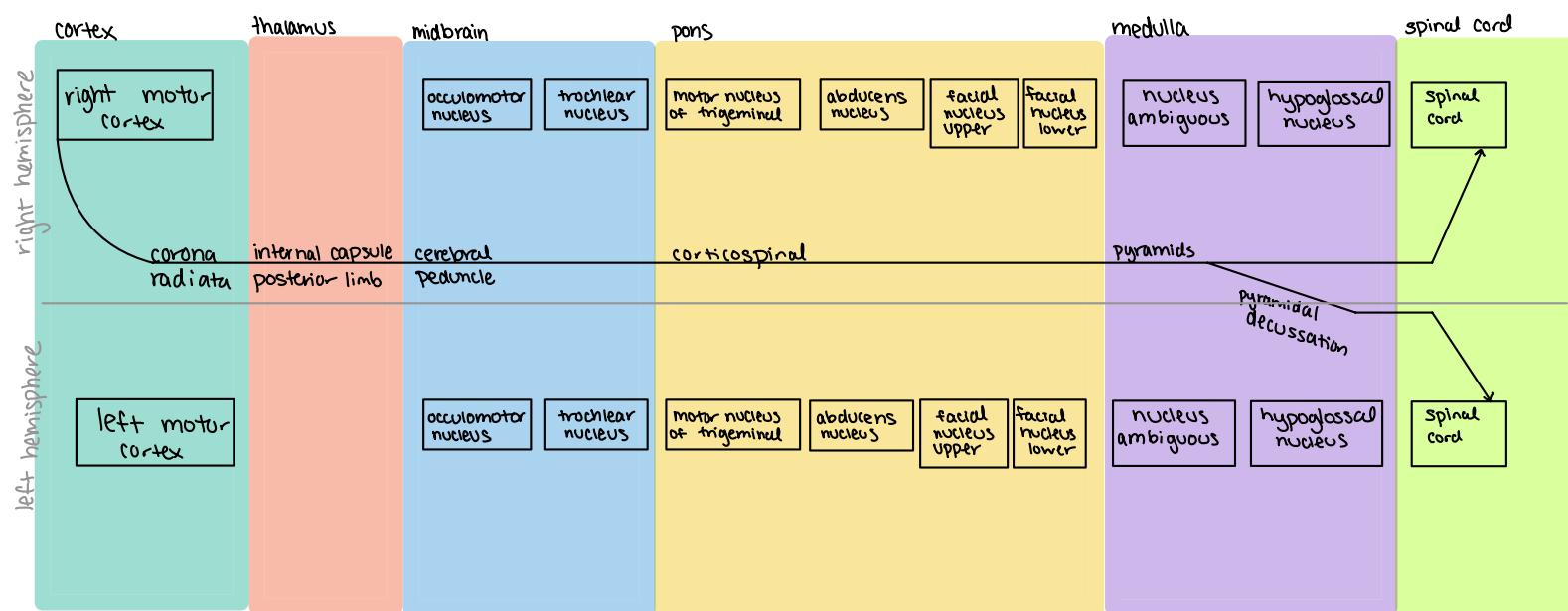
Hypoglossal nuclei UMN paralysis: fibers of corticobulbar cross at level of hypoglossal nuclei in the medulla

→ damage to uppermotor neurons results in contralateral tongue not able to tongue, so tongue deviates away from the lesion

Facial Nerve UMN Paralysis: primary motor cortex doesn't innervate upper facial muscles

- innervation to upper face muscles from motor part of cingulate cortex (bilateral innervation)
- damage to UMN results in contralateral lower face muscle paralysis

Corticospinal Tract



- corticospinal tract originates in cortex, where it travels via corona radiata
- in thalamus, it becomes the internal capsule of the posterior limb
- in midbrain → part of cerebral peduncle
- in pons → corticospinal
- in medulla → pyramids (crossing occurs at pyramidal decussation)

Lateral Corticospinal tract

Function: controls distal limb muscles and fine motor control

Cross: all cross in pyramidal decussation

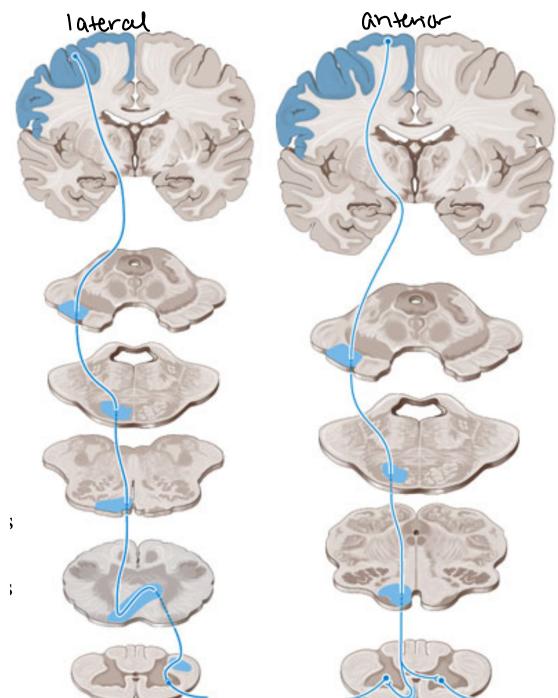
Innervate: travel all throughout spinal cord

Anterior Corticospinal tract

Function: axial/proximal muscles and postural motor control (bilateral)

Cross: crosses at spinal levels or in the pyramids

Innervates: mostly innervates cervical (but some in all levels), so it has ;
bilateral innervation

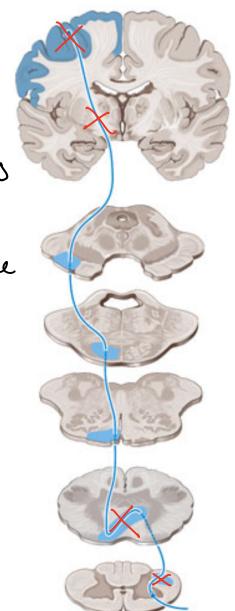


Lesions

- damage to upper motor neurons or axons in the corticospinal tract results in spastic paralysis (lateral tract)
 - can't make limb move
 - no atrophy, fasciculations, or fibrillations
 - increases tone/reflexes
- } Upper Motor Neuron Syndrome

Spastic Paralysis

- Measure Stretch reflex → score 0-5
 - normal = 2
 - UMN syndrome > 2
 - LMN syndrome < 2
- Measure Withdrawal Reflex (plantar-babinski reflex)
 - normally toes curl down
 - corticospinal tract damage → toes flare down
 - infants exhibit toes flaring b/c corticospinal tract is not yet myelinated



Damage

- damage at cerebral cortex: contralateral decorticate UMN syndrome at damaged levels of homunculus
- internal capsule: contralateral UMN syndrome (decorticate — flexion of upper limbs)
- damage in brainstem above decussation: contralateral UMN syndrome (decerbrate rigidity — increase in tone and upper/lower limbs are extended)
- damage at decussation: bilateral paralysis on both sides at all levels at all levels (decerebrate)
- damage at spinal cord: ipsilateral paralysis at and below lesion b/c its after crossing (decerebrate)
- If lesioned just pyramids + nothing else, you get paralysis w/ no change in tone + reflexes

Acute spinal Cord Damage → damage to ventral horn + corticospinal tract

- if you damage T4 level, you get LMN syndrome at T4 level w/ flaccid paralysis of thoracic muscles with fasciculations, then atrophy in a week on ipsilateral side
- below lesion level → no atrophy and decreased tone + reflexes → tone returns in a week then UMN syndrome appears due to loss of inhibition of gamma motor neurons
due to spinal shock

Reticular Nuclei of the Brainstem

- reticulum of neurons with long dendrites and axons
- integrate many sensory and motor functions in the brainstem
- invertebrates and humans have reticular nuclei
- important reticular nuclei
 - Periaqueductal gray (PAG), locus caeruleus, Ventral tegmental area, raphe nuclei
- functions:

- ascending projections to the cortex (alertness, sleep)
- descending projections to spinal cord (carry motor info) → reticulospinal projection to ventral horn neurons

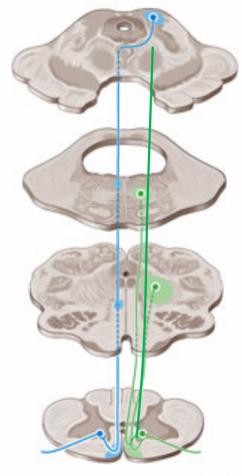
Reticulospinal tract

- travels from reticular nuclei to spinal cord → specifically to the medial pool in the ventral horn
- reticular nuclei from midbrain, pons, and medulla all contribute to reticulospinal tract
- has ipsilateral projection to spinal cord (in anterior fasciculus)

Functions:

- descending pain control
- gait control → pattern generators
- inhibits extensor tone by inhibiting the gamma loop

Damage to reticular nuclei → increase of extensor tone and reflexes on the contralateral side (unclear where crossing occurs)



Vestibulospinal Tracts

Medial Vestibulospinal tract:

Originates: medial vestibular nucleus

Terminates: bilaterally in medial ventral horn in cervical levels

Function: stabilizes head + gaze

Travels through medial vestibulospinal tract (or also called descending part of MLF by Purvis) → anterior funiculus

Lateral Vestibulospinal Tract

Originates: lateral vestibular nucleus in pons

Terminates: ipsilateral innervation of medial pools in ventral horn throughout entire spinal cord

Function: balance + posture

Medial Longitudinal Fasiculus

Originates: all vestibular nuclei

Terminates: trochlear nucleus, oculomotor nucleus, abducens nucleus

Function: stabilizing eyes + gaze → gives rise to vestibulo-ocular reflex (rotate head right, eyes stay fixed)

Tectospinal tract

• Originates: tectum (tectum is made up of inferior/superior colliculi)

• Terminates: in cervical levels of spinal cord

Crosses: crosses immediately (in midbrain)

Function: controls head + eye movements

- important in orienting to visual and auditory stimuli
- optokinetic reflex (eyes follow moving object) and startle reflex (jump when hear loud noise)

Rubrospinal Tract

Originates: red nucleus (midbrain) and travels in lateral funiculus in spinal cord

Crosses: in the midbrain

Terminates: lateral regions of ventral horn

Function: plays a role in movements of upper limbs by projecting to local circuit neurons

Theory of Red Nucleus

- damage to corticospinal tract above red nucleus results in decorticate rigidity → elbow flexion
- damage to CST below/at red nucleus → decerebrate rigidity (elbow extension)
- indicates red nucleus produces upper limb flexion tone. Motor cortex inhibits red nucleus. Damage to corticorubro fibers disinhibits red nucleus

→ rubrospinal tract might not exist in humans