

Facial Nerve

Branchial Functions

Nucleus - facial motor nucleus (caudal pons)

Exits - exits brainstem via pontomedullary junction

- exits cranium via internal auditory meatus (w/ CN8)
- LMNs exit via stylomastoid foramen

Function: innervate muscles of facial expression, stapedius muscle in inner ear (dampens vibrations of oval window), and orbicularis oculi (closes eyelid)

Damage:

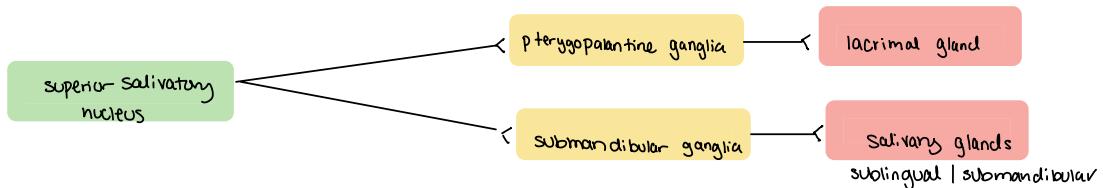
- to nucleus/tract → ipsilateral LMN paralysis (atrophy, flaccidity, and decreased reflexes) + hyperacusis
- Upper face muscles: forehead + orbicularis oculi (functions to close eye)
- lower face muscles: muscles involved in smiling
- blockage of stylomastoid foramen → facial nerve paralysis + hyperacusis
- blockage of internal auditory meatus → facial paralysis + loss of hearing b/c CN8 travels through internal auditory meatus, so hearing lost completely
- temporary paralysis (Bell's Palsy) due to virus /autoimmune disease
- damage to UMN, only get paralysis of lower face → primary motor cortex doesn't project to upper face

Visceral Motor Functions

① fibers originate in superior salivatory nucleus

② preganglionic fibers project to

- a) pterygopalantine ganglia → which projects to lacrimal glands
- b) submandibular ganglia → projects to sublingual (under tongue) and submandibular (saliva production) glands



Trigeminal Nerve

Classification: somatic sensory, branchial motor

Branchial Functions

Nucleus: trigeminal motor nucleus (middle pons)

Exits: branchial motor component exits with V_3 (mandibular) via foramen ovale

Innervates: Muscles of mastication (temporalis, masseter, pterygooids) and tensor tympani (muscle that dampens vibration of tympanic membrane)

Damage: damage to nerve or nucleus results in ipsilateral paralysis of muscles of mastication and hyperacusis (tensor tympani muscle paralysis results in hyper hearing)

Sensory Components

Function: provide sensory innervation to the face (general somatic sensing)

- contains 3 major branches/functional components → considered functional areas, not dermatomes, because not derived from somites, derived from branchial arches
 - ophthalmic (V_1) → superior orbital fissure
 - maxillary (V_2) → foramen rotundum
 - mandibular (V_3) → foramen ovale

Primary Afferents:

mesencephalic nucleus → proprioception

chief trigeminal sensory nucleus → fine touch, dental pressure

spinal trigeminal nucleus → crude touch pain, temperature

Glossopharyngeal + Vagus Nerve Branchial Functions

Nucleus: nucleus ambiguus (middle medulla) → rostral exit via glossopharyngeal, caudal exit via vagus

Exit: exit medulla → post olfactory sulcus exit skull → jugular foramen (with CN 9,10,11, jugular vein)

Functions:

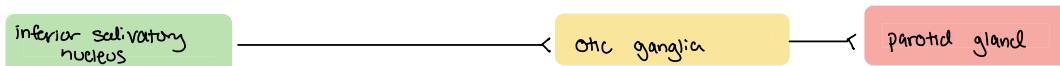
- pharynx (swallowing muscles) → CN 9 innervates stylopharyngeus, CN 10 innervates the rest (39/40 muscles)
- larynx (speech muscles) → innervated by vagus (CN 10)

Damage: Unilateral LMN syndrome

- Damage to CN 9 → can still swallow
- Damage to CN 10 → hoarse quiet voice (unilateral lesion) + difficulty swallowing

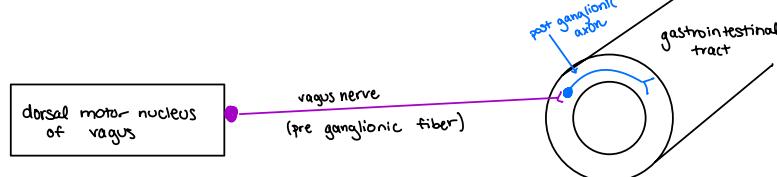
Glossopharyngeal Visceral Motor

- ① cell bodies located in inferior salivatory nucleus
- ② pre-ganglionic fibers project to otic motor ganglia
- ③ post-ganglionic fibers project to parotid gland (salivation)



Vagus Nerve - Visceral Motor (part of enteric nervous system)

- Nucleus - dorsal motor nucleus of vagus (in medulla)
- motor ganglia - thoracic and abdominal parasympathetic activation
- Auerbach's plexus (myenteric) - Organization of neurons in gut that controls motor aspects of gut function such as peristalsis
- Meissner's plexus (submucous) - located beneath mucous membrane of the gut, involved in chemical monitoring / gland secretion



Segmental Motor Control

Local Circuit Neurons → segmental motor control

- sensory inputs can make LMNs fire producing reflexive contraction (unconscious reflex control)
- primary somatosensory fibers connect to LMNs
 - monosynaptic reflex - direct synapse of somatosensory fiber to LMN
 - polysynaptic reflex - somatosensory fiber → interneuron → LMN (inhibitory interneurons are usually GABAergic)
- reflexes are found at all levels of spinal cord

Withdrawal Reflex

- cutaneous reflex → multisynaptic

- ① step on tack
- ② nociceptors activated
- ③ primary afferents (A delta or C fibers) travel to lower lumbar or upper sacral levels and release substance P
- ④ Ipsilateral

- flex the knee (lower lumbar) → cutaneous afferent fiber synapses onto excitatory interneuron (releasing acetylcholine) which activates flexor muscle (hamstring)
- inhibit knee extensor (lower lumbar) → primary afferent synapses on inhibitory interneuron, which then inhibits LMN activation of extensor muscle (quad)
- flex hip (upper lumbar levels) → fibers ascend via
 - dorsolateral fasciculus (Lissauer's tract) → 1/2 levels
 - propriospinal fasciculus → 3/4 levels

 } pain fibers that don't leave
 the spinal cord

Contralateral

- extend leg/hip for support → inhibit flexor muscle and activate extensor muscle (quads)
- fibers cross to the contralateral side via
 - ventral white commissure → same level
 - anterolateral system → other level

Deep Tendon Reflex (stretch reflex)

Muscle Fiber Types

- ⓐ extrinsic muscle fibers (myofibrils) → innervated by alpha motor neurons (LMNs) → damage results in LMN syndrome
- ⓑ intrinsic muscle fibers (muscle spindles)

• Two components:

- ① sensory mechanoreceptor — made up of bag and chain muscle fibers that detect stretch and change in muscle length via type 1a (bag) and type 2 (chain) primary afferents
- ② contractile fibers (myofibrils) — innervated by gamma motor neurons → damage doesn't result in LMN paralysis

Deep Tendon Reflex (stretch reflex) → monosynaptic reflex

• sensory receptor that activates reflex → muscle spindle

① triceps stretched by tap

② muscle spindle detects stretch → 1a fibers fire

③ 1a sensory fibers synapse on:

- LMN of the triceps → excitatory synapse so muscle contracts
- inhibitory interneuron → inhibits contraction of biceps (flexor)
- proprioceptive information travels via fasciculus cuneatus

Examples: spinal nerve → knee, elbow, ankle, wrist

Why does this exist? → example. Hold cup of coffee

• Brain sends feedforward command → hold cup level

• segmental feedback control to LMNs

- load changes (aka more coffee is added to cup) resulting in passive stretch
- LMNs correct by activating muscles to provide resistance

Golgi Tendon Organ

• somatosensory receptor (mechanoreceptor)

• 1b fibers form meshwork in connective tissue of most muscle

• activates in response to tension on tendon → important in active stretch

GTO vs. Muscle Spindles

Passive stretch → increase in load

- activity of muscle spindles increases to produce deep tendon reflex (muscle spindles in parallel w/ extrinsic fibers)
- activity of GTO increases slightly, but don't produce DTR (GTO in series w/ extrinsic fibers)

Active Contraction

• activity of muscle spindle decreases → don't provide proprioception during active contraction

• GTO activity increases and provides proprioception

Autogenic Inhibition — GTO

• GTO provides negative feedback during active stretch | provides proprioception to help control muscles

- ① signal of increased tension travels via Ib afferent fibers, activating inhibitory interneurons
- ② inhibitory interneurons inhibit stretched muscle's LMN and other interneurons activate antagonist muscle
 - may contribute to "clasp knife" reflex → abnormal reflex displayed in patients with higher than normal tone
 - when doing range of motion testing, tension increases and increases, until it suddenly releases

Muscle Tone

normal muscle tone → resistance to active stretch

two mechanism of tone:

- ① active tone - due to spontaneous activity of LMNs
- ② passive tone - tense sarcomeres from actin/mysosin crossbridging and connective tissue stiffness

Pathological active muscle tone:

- LMN disorders → very low tone b/c loss of muscle innervation
- UMN disorders → very high tone. Results in spasticity and rigidity due to loss of descending inhibition of gamma motor neurons

Gamma Activation of Tone + Reflexes

Gamma Loop

- ① gamma motor neurons are tonically active
 - ② muscle spindles contract
 - ③ increased activity in Ia fibers
 - ④ activate alpha motor neurons
 - ⑤ activate extrinsic muscle to raise tone
- descending motor pathway (upper motor neurons) inhibit gamma motor neurons, inhibiting gamma loop
 - Damage to UMNs reduces descending inhibition resulting in spastic paralysis (increased tone + reflexes)
 - pathological tone limits functional movements
 - to reduce tone, cut dorsal roots (Ia fiber) at affected levels (dorsal rhizotomy), which lowers tone
 - also means you lose touch, pain, temp, but decreased tone
 - now possible to cut specific rootlets to reduce specifically muscle spindle sensation w/o other senses

Reflexes

Jaw Jerk Reflex → stretch reflex (trigeminal in, trigeminal out)

- ① tap hammer on base of mandible → stretches muscles of mastication
- ② proprioceptive primary afferents (cell bodies in mesencephalic nucleus) synapse in chief sensory nucleus
 - or motor nucleus of trigeminal nerve

③ neurons in trigeminal motor nucleus are LMNs, which project to muscles of mastication (temporalis, masseter, pterygoid) that function to close the jaw

Damage to sensory or motor part of trigeminal nerve blocks this reflex

Corneal Reflex → withdrawal reflex (trigeminal in, facial out)

① poke cornea

② trigeminal carries sensory info and synapses in spinal trigeminal nucleus

③ interneuron projects — from spinal trigeminal nucleus to facial motor nucleus

④ LMNs of facial nerve project to orbicularis oculi to close eyelid

→ multisynaptic reflex

Gag Reflex

① noxious stimuli in uvula transmitted by glossopharyngeal synapses in spinal trigeminal nucleus

② interneurons project bilaterally to nucleus ambiguus

③ LMNs in nucleus ambiguus project to muscles in pharynx (via CN9 + 10)

④ muscles in pharynx move upwards on both sides → unilateral stimulation, bilateral activation

Conscious Gagging Sensation

- spinal trigeminal nucleus

- crosses midline

- travels to VPM via trigeminotthalamic tract

- VPM to somatosensory cortex (cingulate cortex)

Somatosensory Systems

Mechanical Sensory Transduction

- ① mechanical force activates somatosensory receptors
- ② cutaneous touch receptors have mechanically gated ion channels that produce local potentials (depolarizing) when stretched
- ③ if depolarization > threshold, amplitude of local potential is embedded as action potential frequency in sensory nerves

Cutaneous Receptors

^{vibration}
Encapsulated (touch) - contain capsule formed by connective tissue to make receptors more sensitive → all innervated by A_B fibers

- Meissner's corpuscles → light pressure, most superficial encapsulated receptor
- Merkel's Disks - light touch, located in crypts of epidermis
- Ruffini Ending - slow adapting mechanoreceptor to low frequency vibration
- Pacinian Corpuscle - responds to vibration
- Hair follicle receptors - nerve ending that wraps around base of hair, so when hair moves, receptor stretches

Unencapsulated (Pain + Temp)

- free nerve endings

Somatosensory Fiber Types

Sensory Function	receptor type	afferent axon type	axon diameter	axon velocity
proprioception	muscle spindle	A _A , type II	13-20 μm	80-120 m/s
touch	merkel, meissner, pacinian, ruffini	A _B , type II	6-12 μm	35-30 m/s
pain temp	free nerve endings	A _F ^{delta} , type III	1-5 μm	5-30 m/s
pain temp itch	free nerve endings	C, type III (no myelin)	.2-1.5 μm	0.5-2 m/s

Somatosensory Pathway Crossings

Pathway	function	crossing
DCL	fine touch, proprioception	caudal medulla (sensory decussation)
Anterolateral system	pain + temp	spinal cord
trigeminothalamic	fine touch, conscious proprioception, pain, temp from face	multiple places
dorsal, ventral spinocerebellar, cuneocerebellar	unconscious proprioception	multiple places

Dorsal Column Medical Lemniscus

Function: fine touch, vibration, conscious proprioception

Primary Afferents:

- cell bodies in dorsal root ganglia
- primary afferents receive input from mitscher's discs, etc.
- no synapse or crossing in spinal cord
- travel via
 - fasciculus gracilis - primary afferents from lower body T6 and below and synapses in nucleus gracilis
 - fasciculus cuneatus - primary afferents from upper body above T6 and synapse in nucleus cuneatus

Secondary Afferents

- cell bodies in nucleus gracilis/cuneatus
- secondary afferents cross in internal arcuate fibers then form medial lemniscus in closed medulla
- Once crossing fiber reaches the midline, it becomes medial lemniscus

Tertiary Afferents (Thalamus)

- secondary afferents synapse in VPL of thalamus → sensation of body
- tertiary afferents project from thalamus to cortex via internal capsule and corona radiata
- synapse in postcentral gyrus of cortex in Brodmann's Areas 1,2,3

LESIONS

peripheral nerve damage → ipsilateral loss fine touch + proprioception distal to damage

spinal cord → ipsilateral loss fine touch + proprioception at and below level

lower medulla (below internal arcuate fibers) → ipsilateral loss fine touch + proprioception at and below level (entire body)

upper medulla and above (contralateral) loss of fine touch + proprioception

Somatotopic Organization of DCML

spinal cord/caudal medulla → sensation from feet medial, arms lateral

rostro medulla → in medial lemniscus arms dorsal feet ventral (standing on pyramids)

pons → arms medial, feet lateral

midbrain + thalamus → feet dorsal, arms ventral

cortex → feet medial, arms lateral (face is most lateral)

Anterolateral System — (Classic) Lateral Pain Pathway

Primary Afferents

- type C (IV) or A delta fibers (III) w/ cells in dorsal root ganglia

• synapse at dorsal horn of spinal level in posteromedial nucleus/substantia gelatinosa and lamina B + C

and release substance P

• Or travel via Lissauer's Tract to synapse at dorsal horn at another level

- Lissauer's tract → ascending or descending primary afferents prior to synapse

- damage to dorsal horn reduces pain/temp at level and multi-segmental sensory loss or reflexes

- loss of pain isn't as significant above/below level of damage

Secondary Afferents

- cell bodies in dorsal horn

- cross at level via ventral white commissure

- ascend spinal cord via ALF

- somatotopic organization: cervical medial, sacral lateral

- travel to VPL of thalamus (responsible for touch localization)

ALF contains

tract vs. fasciculus

- spinothalamic tract

- tract → fibers have same origin/termination

- spinoreticular tract

- fasciculus → can contain multiple tracts

- spinoencephalic tract

- propriospinal tract

LESIONS

Spinal nerves → ipsilateral loss of pain distal to lesion

Spinal cord:

- dorsal root → ipsilateral loss at level of lesion

- dorsal horn → ipsilateral loss at level of lesion

- Lissauer's tract → ipsilateral loss near level of lesion (up/down a level)

- ALF → contralateral loss at and below level of lesion

Thalamus (VPL) → contralateral entire body (not face)

Somatosensory Cortex → pain persists, but don't know where

Discriminative Pain (fast, "epicritic", neospinothalamic pain)

- pain localization/intensity

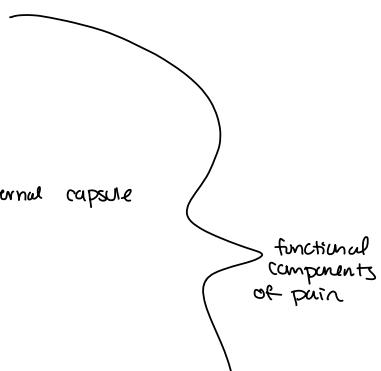
- A delta fibers

- lateral pain pathway to VPL and SI of somatosensory cortex via internal capsule

Affective Pain (protopathic, slow pain, paleospinothalamic pain)

- unpleasantness and emotional aspects of pain

- C-fibers



- medial pain pathway of cingulate cortex + insula



Medial Pain Pathway

- primary afferents same as lateral pathway
- secondary afferents travel to thalamus in two ways
 1. spinothalamic → direct to thalamus
 2. spinoreticular → synapse in reticular nuclei in brainstem and then reticulothalamic
- synapse in midline/intralaminar thalamic nuclei
- project through internal capsule to cingulate cortex + insula

Discriminative VS. Affective Pain

- damage to somatosensory cortex (S1, S2) → have pain, don't know where
- damage to VPL → have pain, don't know where
- damage to entire thalamus → no pain sensation
- damage to anterior cingulate cortex (bilaterally) → you have pain, know where/how much, but don't care

Somatosensing Pathways of the Face Pain + Temperature

- ① pain detected by nociceptors in face, teeth, tongue, cornea, dura, external ear (CN 5, 7, 9, 10)
- ② primary afferent in trigeminal (semilunar, gasserian) ganglia located outside dura at base of skull (in cavernous sinus)
- ③ primary afferent passes through middle cerebellar peduncle and descends via spinal trigeminal tract to synapse in spinal trigeminal nucleus

Touch + Some Proprioception

- ① touch receptors (Merkel's discs, Meissner's discs, pacinian corpuscles) in face, external ear, tongue, back of throat (9 pharynx)
- ② travels to trigeminal sensory ganglia
- ③ synapses in chief sensory trigeminal nucleus in middle pons

Proprioception Pathway

- ① proprioception receptors in muscles of mastication, tongue, eye muscles (muscle spindles)
- ② cell bodies located in mesencephalic nucleus or trigeminal ganglia
 - mesencephalic "nucleus" → these neurons are derived from neural crest cells
- ③ synapse in chief sensory nucleus

Discriminative Pathways of the Face

- ① primary afferents synapse in either chief sensory nucleus or spinal trigeminal nucleus
 - ② secondary afferents then cross immediately at whatever level they are at
 - ③ once they cross, secondary afferents ascend via trigeminotthalamic tract
 - ④ synapse in ventral posterior medial nucleus (VPM)
 - ⑤ VPM cells project through posterior limb of internal capsule, through corona radiata to lateral somatosensory cortex
- discriminative pathway for fine touch and localized facial pain (lateral pain pathway)

Medial Pain Pathway of the Face

- ① primary afferents synapse in chief sensory and spinal trigeminal nucleus
- ② crosses and ascends via trigeminal lemniscus
- ③ synapses in midline + intralaminar thalamic nuclei
- ④ synapses in cingulate gyrus

Upper Motor Neurons

Corticobulbar Tract

- Cell bodies in motor cortex
- leave motor cortex via corona radiata
- pass through internal capsule genu in thalamus
- midbrain → cerebral peduncle
- pons → corticospinals
- medulla → corticospinals

Synapse Bilaterally in:

- midbrain → oculomotor, trochlear
- pons → trigeminal motor, abducens, facial motor nucleus (upper face muscles)
- medulla → nucleus ambiguus

Synapse Contralaterally

- pons → facial motor nucleus controlling lower face muscles

Damage:

Hypoglossal nuclei UMN paralysis: fibers of corticobulbar cross at level of hypoglossal nuclei in the medulla
→ damage to UMN result in contralateral tongue not able to push, so tongue deviates away from lesion

Facial Nerve UMN paralysis: primary motor cortex doesn't innervate upper face muscles

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

Corticospinal Tract

Cortex	thalamus	midbrain	pons medulla spinal cord
corona radiata	internal capsule posterior limb	cerebral peduncle	corticospinal pyramids

Lateral Corticospinal Tract

Function: controls distal limb muscles + fine motor control

Cross: all cross in pyramidal decussation

Innervate: travel throughout entire spinal cord

Anterior Corticospinal Tract

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

Cross: crosses at spinal levels or in pyramids (some don't cross)

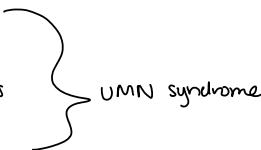
Innervates: mostly innervates cervical (but some in all levels) → has bilateral innervation

Damage

- cerebral cortex → contralateral decorticate UMN syndrome at damaged levels of homunculus
- internal capsule → contralateral UMN syndrome (decorticate → arms flexed over chest)
- brainstem (above decussation) → contralateral UMN syndrome (decerebrate rigidity → increase in tone, upper limbs are extended)
- damage at decussation → bilateral paralysis at all levels (decerebrate)
- damage at spinal cord → ipsilateral paralysis at and below lesion b/c its after crossing (decerebrate)
- lesion to just pyramids + nothing else, you get paraparesis w/ no change in tone + reflexes

UMN syndrome

- damage to lateral corticospinal tract results in spastic paralysis
 - can't make limbs move
 - no atrophy, fasciculations, or fibrillations
 - increased tone / reflexes



Measurement of Spastic Paralysis

- Measure stretch reflex → score 0-5
 - normal = 2
 - UMN syndrome > 2
 - LMN syndrome < 2

- Measure withdrawal reflex

- measure plantar (babinski) reflex
 - normally toes curl down
 - Corticospinal tract damage → toes flare up
 - Infants exhibit toes flaring b/c corticospinal tract is not yet myelinated

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

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

Reticular Nuclei

- reticular nuclei are formations of neurons w/ long axons + dendrites that form a meshwork
- integrate many sensory + motor functions in the brainstem
- invertebrates also have reticular nuclei

- important reticular nuclei
 - Periaqueductal grey (PAG), locus caeruleus, ventral tegmental area, raphe nuclei
- Functions
 - ascending projections to the cortex (alertness, sleep)
 - descending projections to spinal cord (carry motor information) → reticulospinal projection to ventral horn

Reticulospinal Tract

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

Functions:

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

Damage: Damage to reticular nuclei → increase of extensor tone / reflexes on contralateral side
 → unclear where crossing occurs

Vestibulospinal Tracts

Medial vestibulospinal tract

Originates: medial vestibular nucleus

Terminates: bilaterally in medial ventral horn (cervical levels)

Function: stabilize head + gaze

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 Fasciculus

Originates: all vestibular nuclei

Terminates: trochlear nucleus, oculomotor nucleus, abducens nucleus (this is ascending)

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

Tectospinal Tract

Originates: tectum (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 + auditory stimuli
- optokinetic reflex (eyes following moving object) and startle reflex (jump w/ loud noise)

Rubrospinal Tract

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

Crosses: in midbrain

Terminates: lateral region of ventral horn

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

Theory of Red Nucleus (might not exist in humans)

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