Lectures
Special Pathophysiology
3. year Medical faculty
General medicine
Dentistry medicine

Pathophysiology of nervous system
Motor disorders

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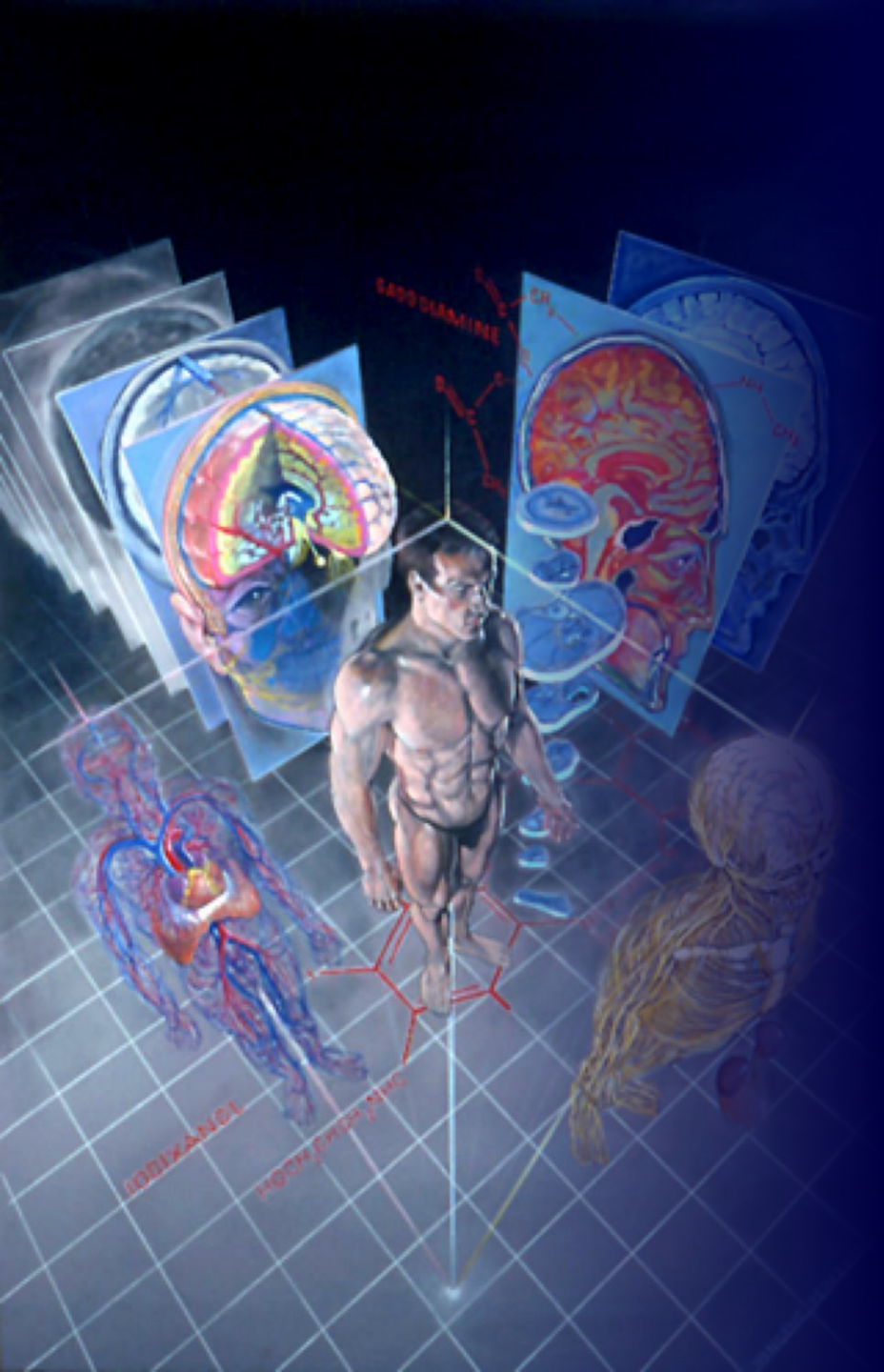

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Review of physiology

ORGANISATION OF MOTOR FUNCTION

Skeletomotor activities

What are skeletotomotor acitvities ?

- Movement of the limbs (waving, jumping), grimacing, smiling etc. – kinetic activities reciognized by anyone as motor performance
- Standing up, sitting, lying – motor behaviours with little apparent position change; just to fix up the skeleton
- Breathing – although known as vital function yet based on phasic motor behaviour of sceletal muscl. (diaphragm, intercostals, accesory m.)
- Old implicit „reflexes“ – sniffing, sneezing, swallowing, vomiting, balancing equilibrium, etc.

Motor processes always comprise 2 qualities:

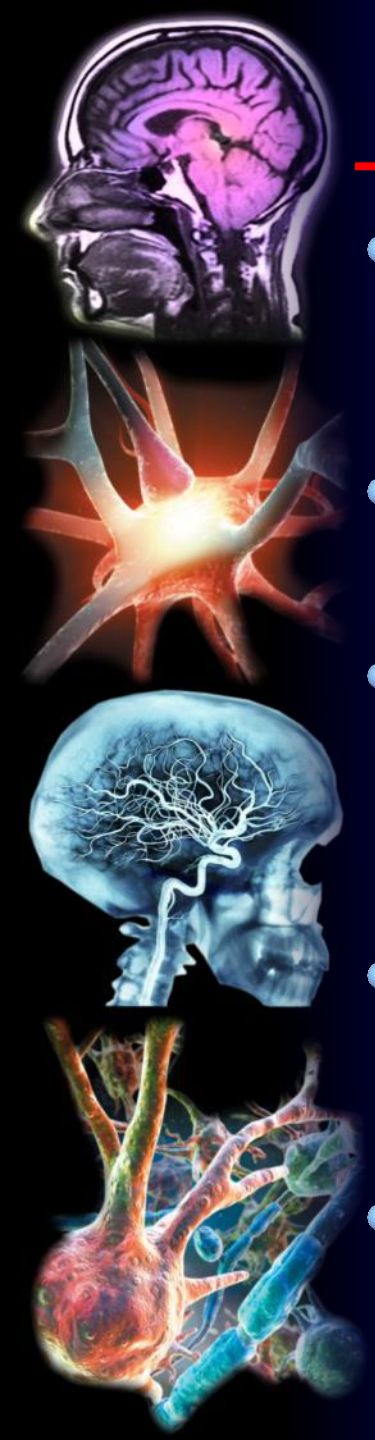
- Kinetic (Phasic) activity - episodic, contractions, spikes in EMG, prevail in kinetic muscles, mostly flexors, red muscle fibres, energy demanding, rapid fatigue,
- Tonic activity (muscle tone) - sustained, tightening of segments, muscle resistance & turgor, no evident EMG, prevail in postural muscles, mostly extensors, white muscle fibers, slower fatigue; one can feel this as tension, something as light tremor, effected by gravitation, anaesthesia, tirednes (postural tremor)

Skeletomotor activities

Sceletal muscles perform 2 forms of activites: Tonic activity – can not be precesly measured in EMG; Isometric – muscle length is kept stable; individual muscle fibres do not chage length; tonic activity is increased

- Movent is little (sometimes tremoric) or unvisible
- Typically involves antigravity musles (paravertebral bak muscles, extersors in extremities

- Isotonic – muscle tone is more or less stable; changes happen in the length of muscle;
  - Movement is visible
  - Typically involves flexors – kinetic muscles (biceps, hand and finger musclescalf muscles etc.)
- Combined izotomic and isometric -



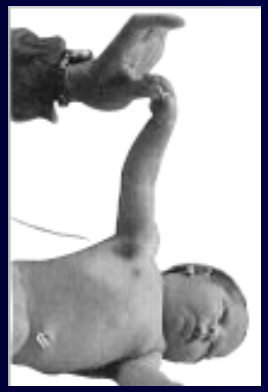
Principia

- We are born with minimum of skeletomotor capabilites → pritimive reflexes (blinking, grasping, stepping ,diving, rooting, sucking, tracking, startle - Moro reflex) →later rightening, protective and equilibrium reflexes are developing (up to 10 m) to allow movememnt on 4 extr. and later to climb up, to walk, to run
- All human related motor activities are learned and can be lost upon trauma, strokes and dementia; Motor patterns and programs are created and stored (motor memory – implicit memory) and can be lost after massive strokes
- Goal directed vs. voluntary movements - voluntuary is only intention, purpose, idea to do (we are not able to drive nor control all individual muscles involved in the movment; weare not aware of them --- robotics has proven how difficult is to program 3D movements. → cortex as iniciator (go – go), basal ganglia break and program; cerebellum as neuronal machine computing, checking and improving
- In man, as real bipedal organism, most motor functions were transferred into higher centra of CNS (process of cortcalisation) as we need more vestibular data, visual data and more advanced procesing to estimate direction of movment. Only a little is left in the spinal cord.
- Special and unique in man are fast phasic muscle activities as hand movment (writting, playing piano etc.), orofacial segment (face expressions) and larynx (vocal cords) → motor brain of man is unique.

„Primitive“ inborn reflexes

- Involuntary, brainstem-mediated automatic motor responses in infants that are assocted with immediate survival, contact with mother, feeding and protection and foster initial sensory-motor
- Later they are inhibited by higher brain centers by 4–6 months.
- Tests (grasp. Moro) are used to evaluatie psychomotor matuation;
- Asymmetric Tonic Neck Reflex (ATNR) (fencing - may help duting labor)

Blink reflex



Grasp reflex

Stepping reflex

Diving reflex

Fencing reflex

Rooting reflex

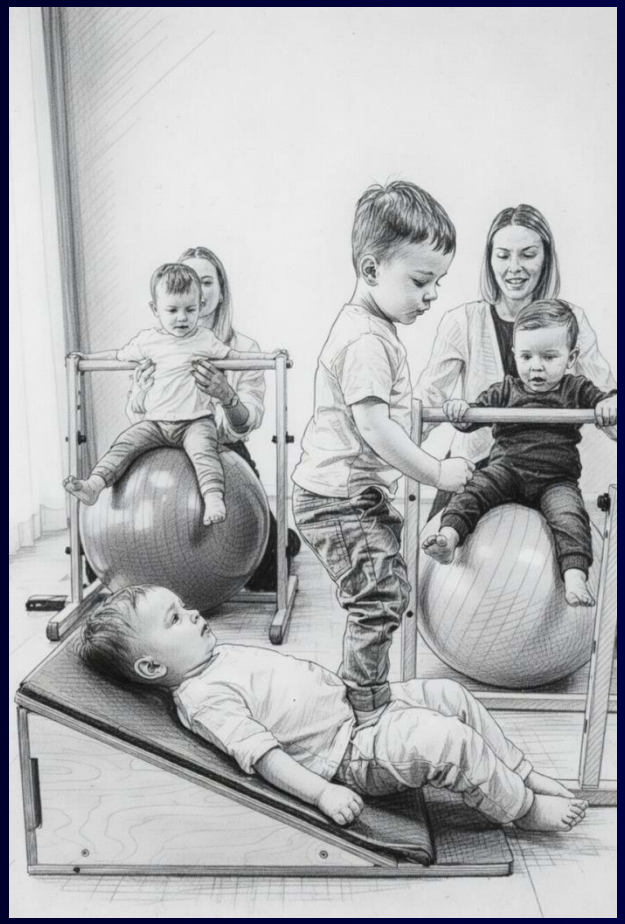
Sucking reflex

Tracking reflex

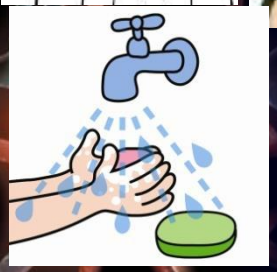
Startle reflex (Moro)

Reflexes – movement, ballance

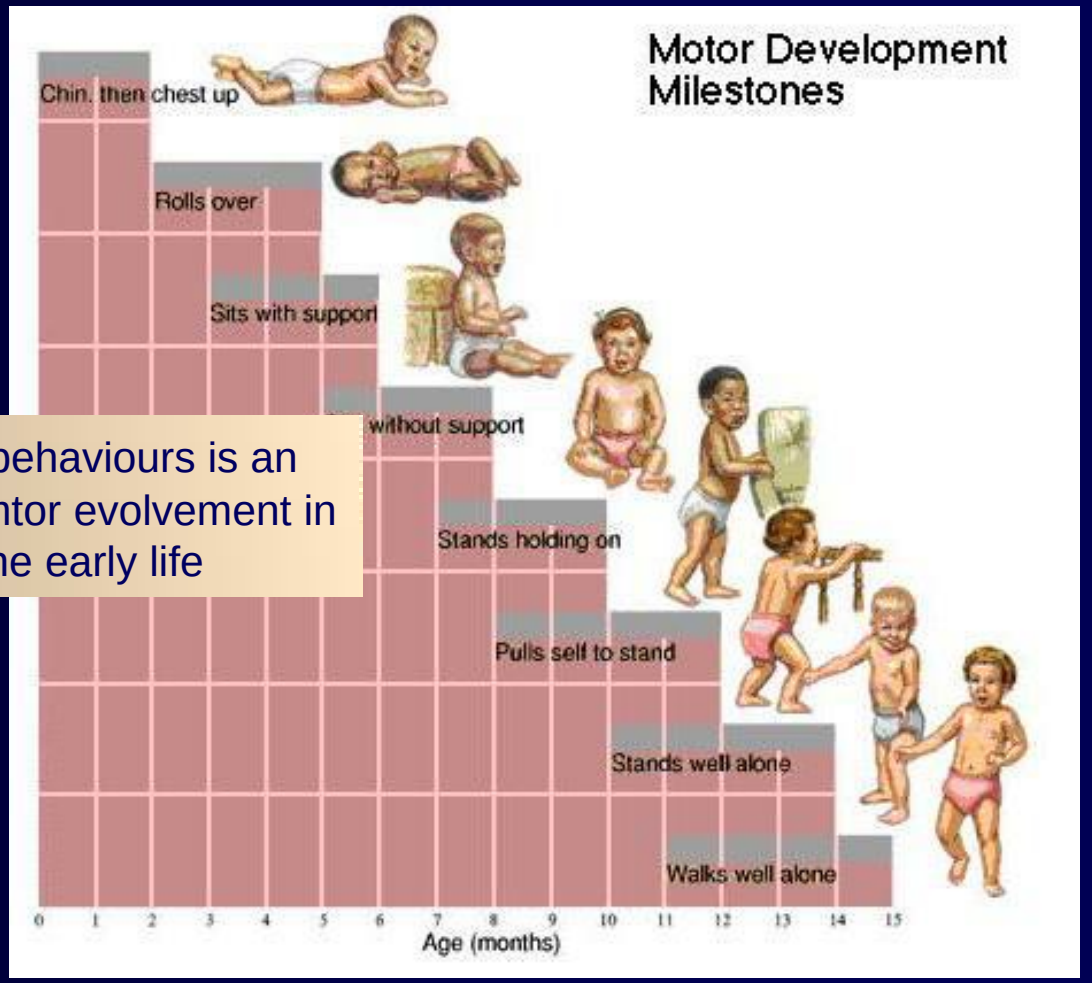
- Rightening reflex reactions – head – body arrangement
- Ballancing reflex reactions – to distribute the mass to keep the ballance
- Protective reflex reactions (parachute reflexes)



Motor developemental milestones



Richness of motor behaviours is an indicator of psychomtor evolvement in specific periods of the early life

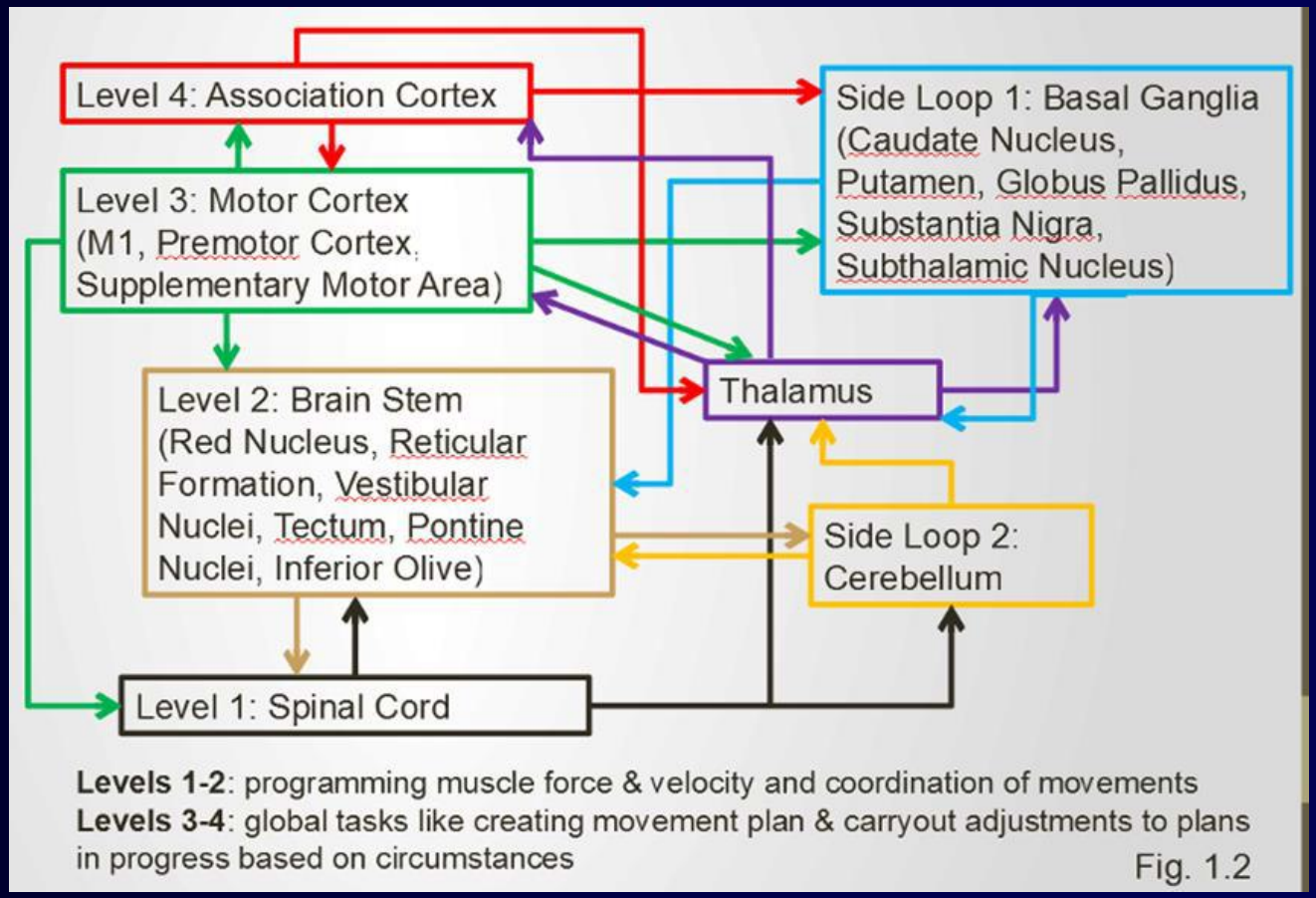


- Motor skills are built over the life-time (implicit motor memory)
- The enormous motor development is achieved within the first 2 years
- Skills in motor execution of complex tasks (language, drawing, writing etc) reach maximal development in first 10 years

Hierarchical organisation of motor activities

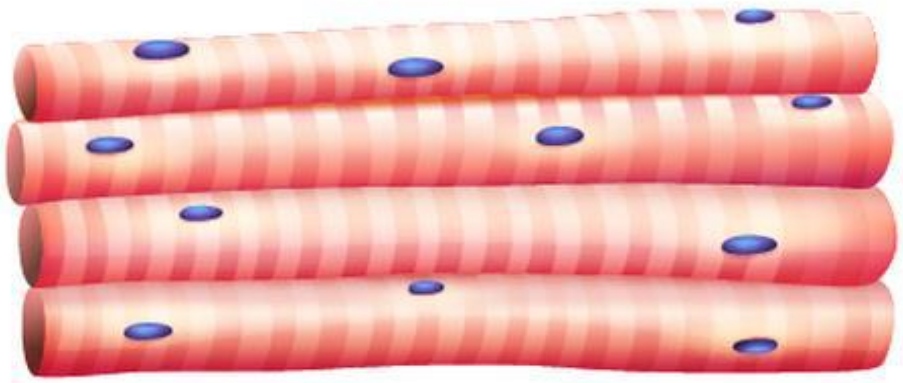
1. Motoneuronal orhganization (lower motoneurons = motor pattern generators
   - monosegmental, polysegmental reflexes of the spinal cord
   - reflexes of cranial nerves (masseter)
2. Brainstem motor organization (relexes, semireflexes)
   - orientation reflexes = searching (mhead rotation), myotonic
   - corneal, pupillary, vestibule-oculocephalic, oculocephalic,
   - branchiogenic = sniffing, sneezing, gagging, vomiting, cough, swallowing
   - more advanced activites = swimming, stepping, support to floor, axial reflexes, extensor
3. Basal gangia = algorithms, programms for movmemnt, learning (walkingm jumping, running downhill
4. Cerebellum = neuronal machine; control of all muscles whn to start what to do when to fininsh; control
5. Motor cortex = excitatory inputs down; fast kinetic movements (direct iniciation to spinal cord)
6. Association cortex = frontal + partietal (planing of activities accordng to the ideative plans)

Hierarchy

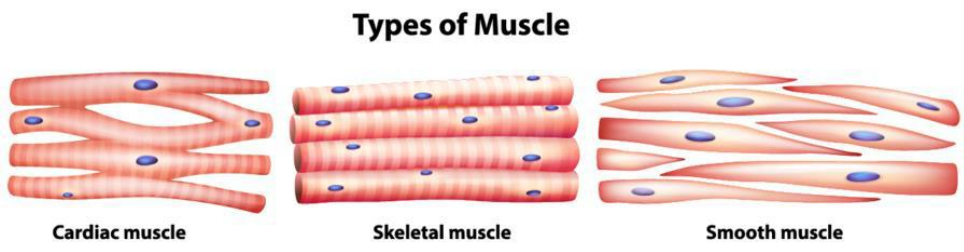


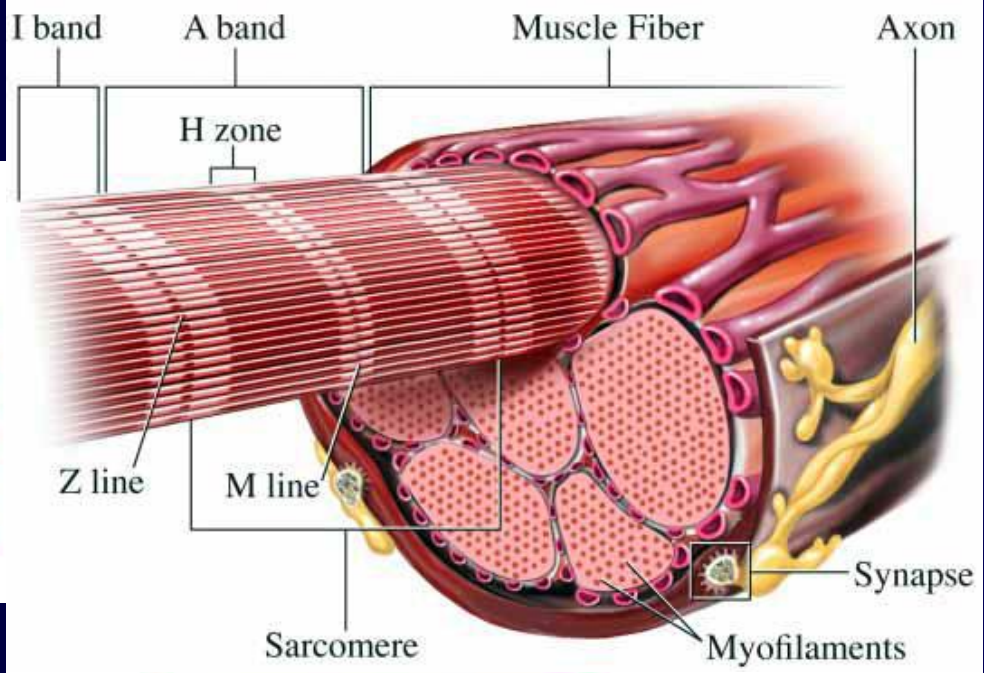
- Relatively little motor programming and commanding skeletomotor capabilities were left in the spinal cord in man ← spinal cord dammage.

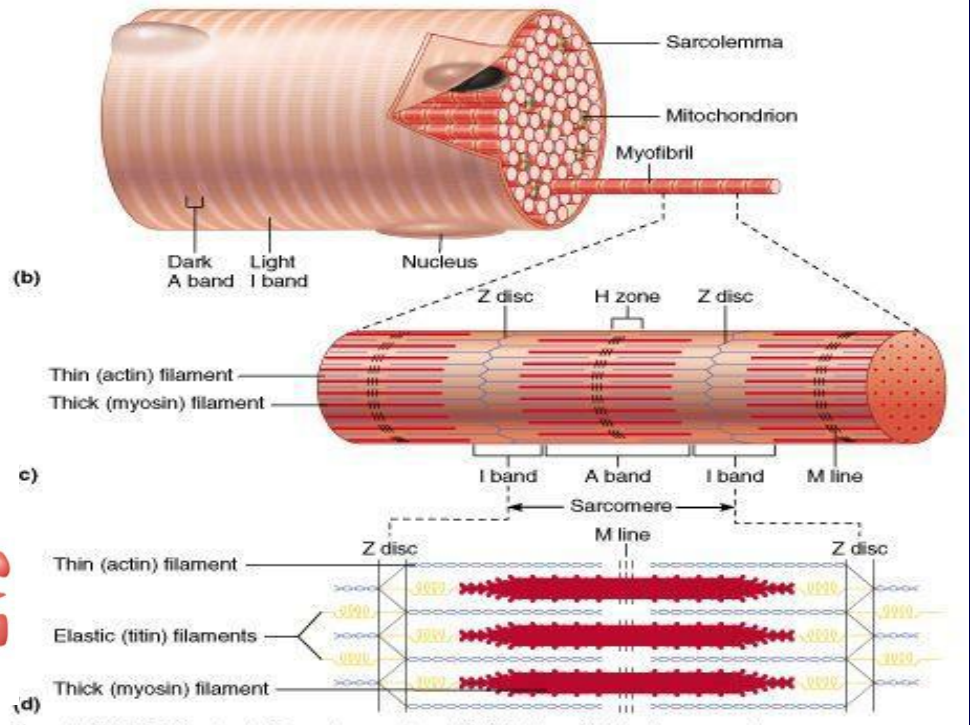
Skeletal muscle



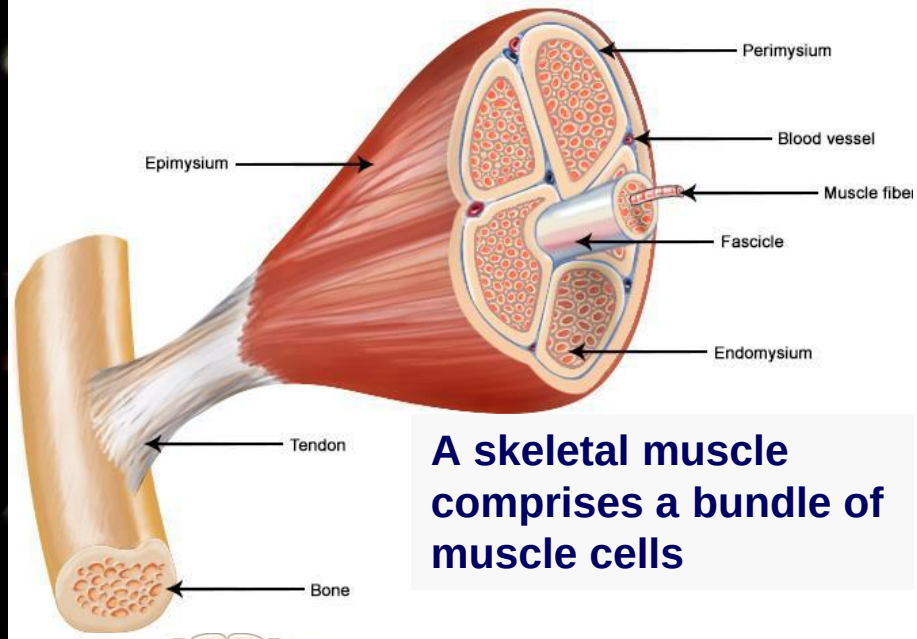
- - Muscle cell = myofilament (10-50x1000-40000 um; Salami shape, striation transparent sarcolema; mutinuclear sarcoplasmic retic.
- Cells do not convey the electical signal to each other; each cell receive nerve connection = synapse .= muscular plate.
- Motor unite = muscle cells inervated from 1 motoneuron







Skeletal muscles



A skeletal muscle comprises a bundle of muscle cells

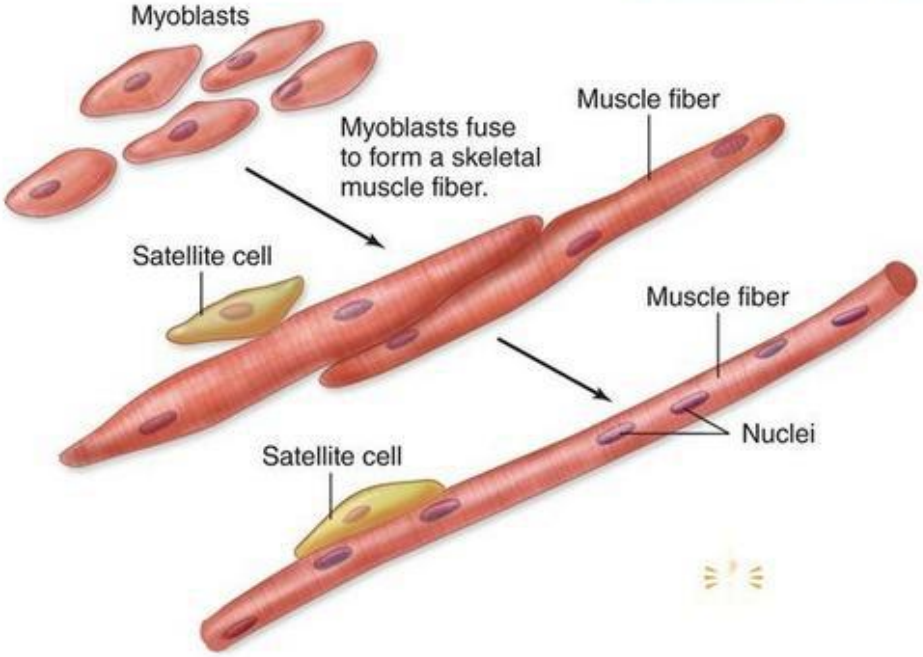
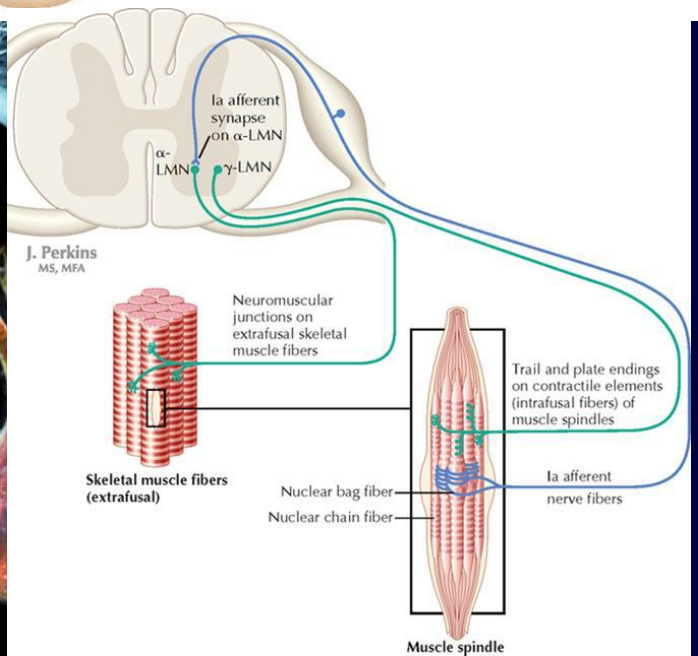


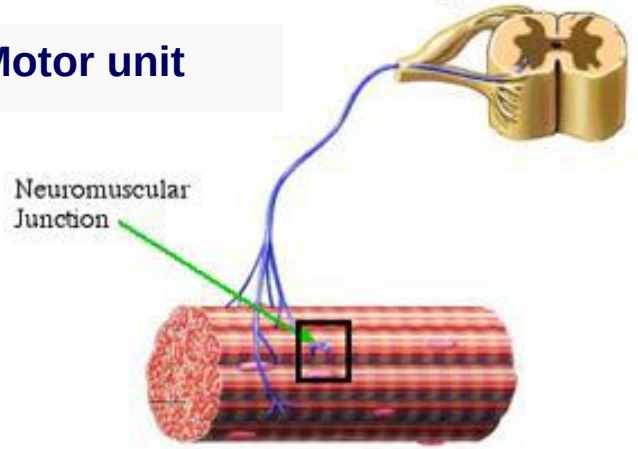
Figure 10.2

Development of Skeletal Muscle.

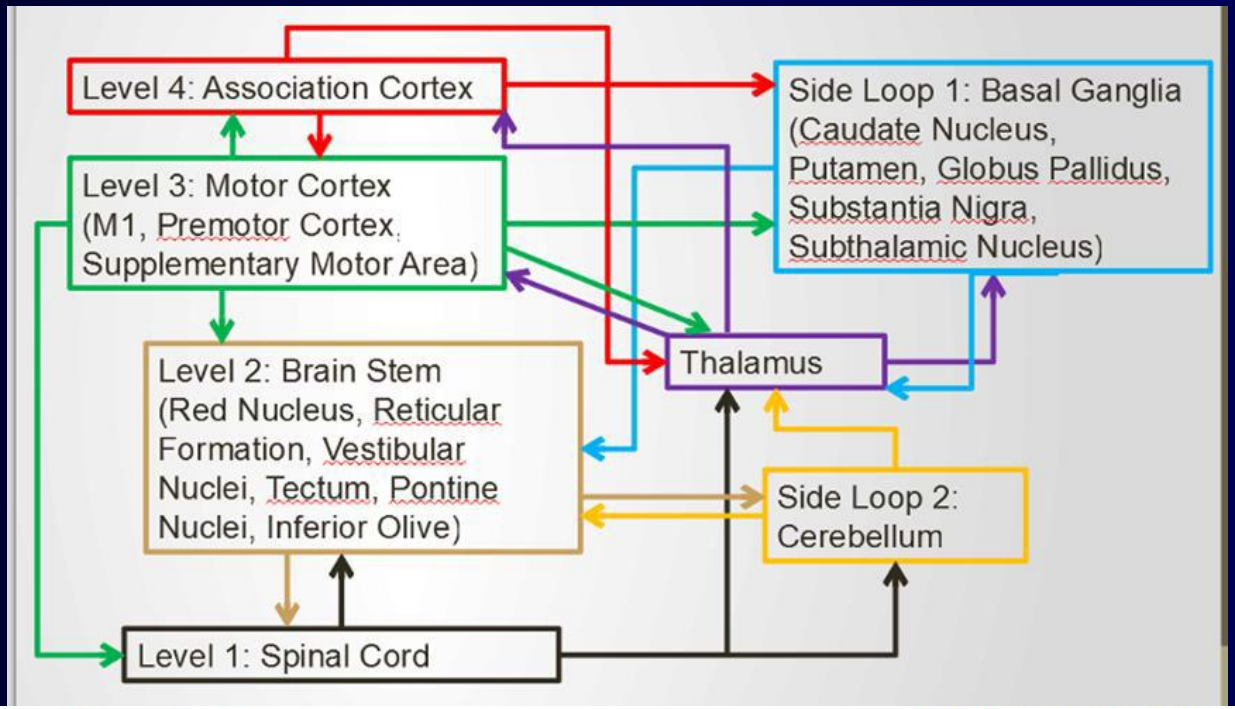
Embryonic muscle cells called myoblasts fuse to form a single skeletal muscle fiber. After development, both muscle fibers and satellite cells are present. Satellite cells are myoblasts that do not go on to form the skeletal muscle fiber. Instead, satellite cells remain with postnatal skeletal muscle tissue and assist in repair of muscles.

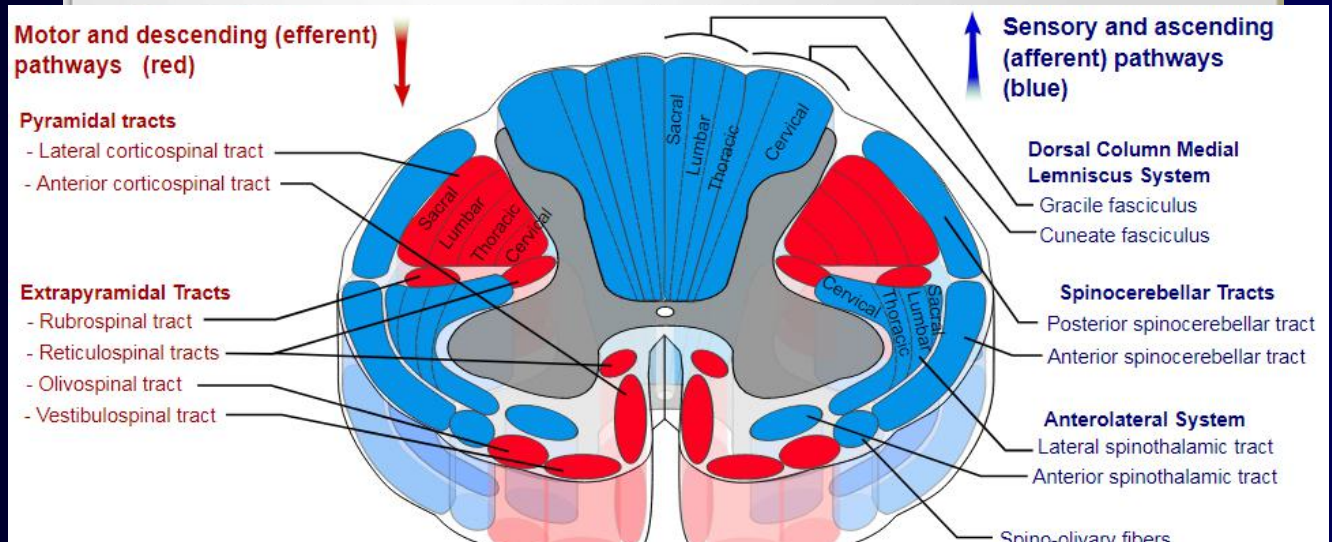


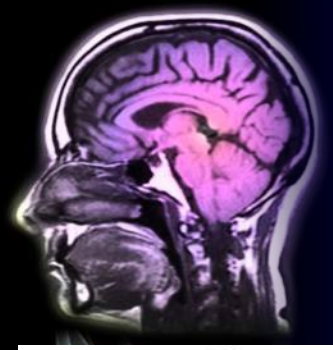
Motor unit



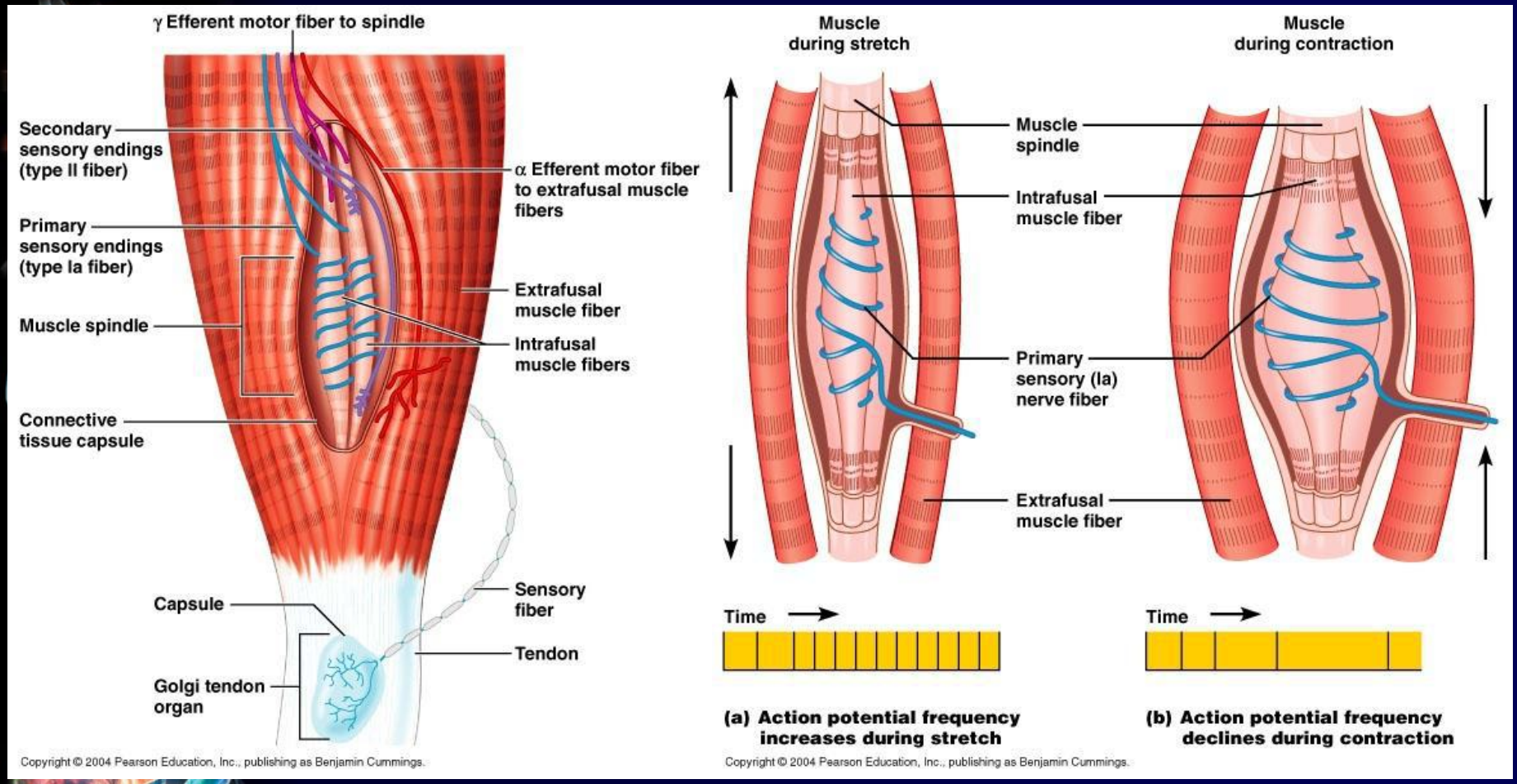
Hierarchy –Motoneuronal level



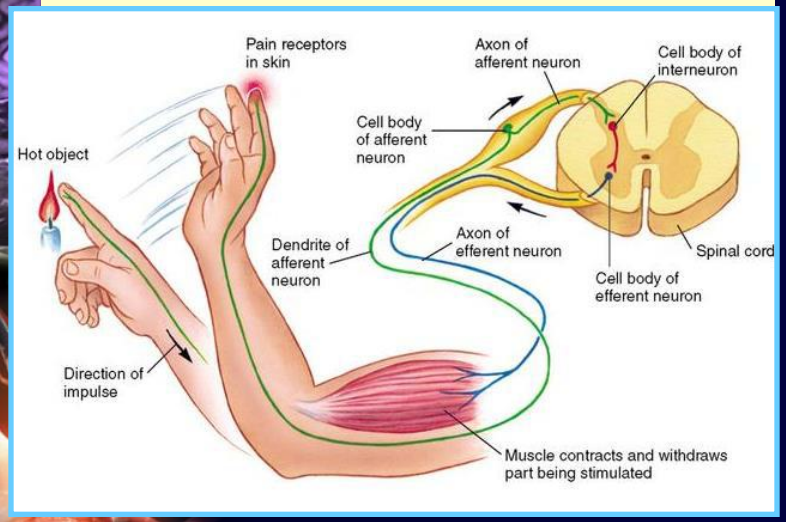


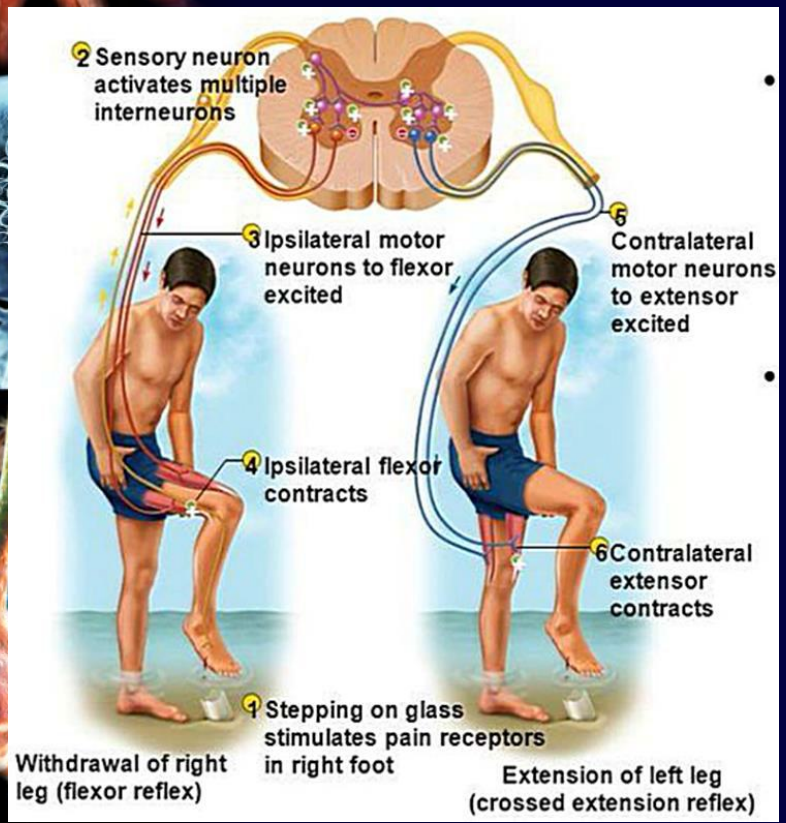


Proprioreceptors



Sensory - motor withdrawal reflexes





Reflexes

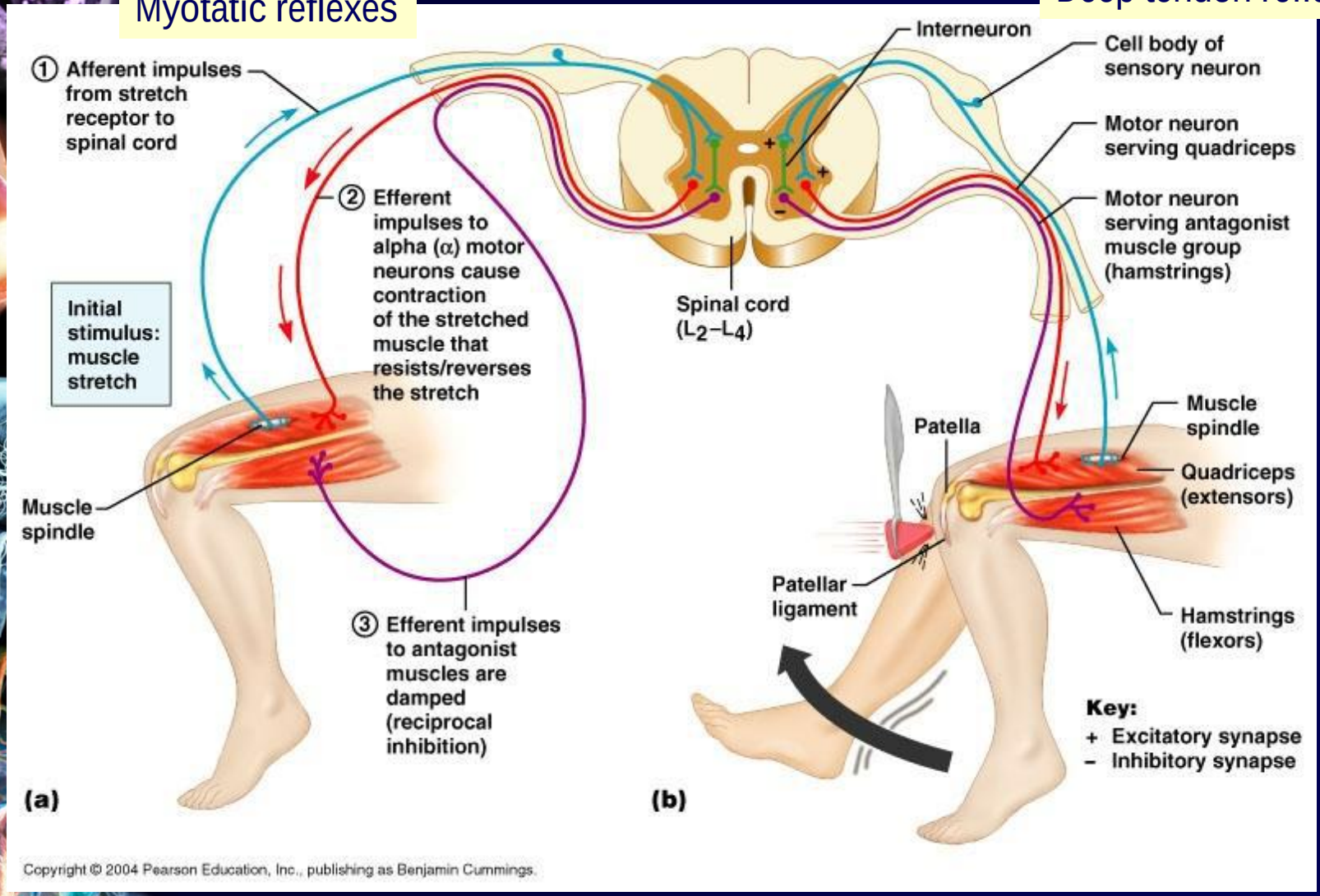
Spinal reflexes

- type of afferents
  - somatic spinal reflexes
  - visceral spinal reflexes
- type of somatosensor
  - proprioceptive reflexes
  - exteroceptive reflexes
- number of involved spinal segments
  - monosegmental spinal reflexes
  - polysegmental spinal reflexes
- number of synapses
  - monosynaptic reflexes
  - disynaptic reflexes
  - polysynaptic reflexes

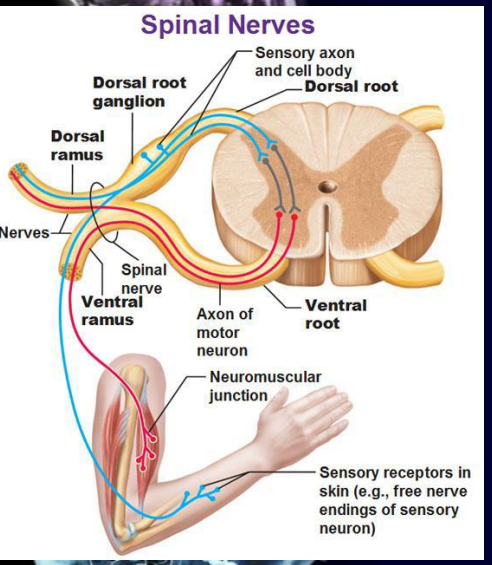
Basic monosegmental reflexes

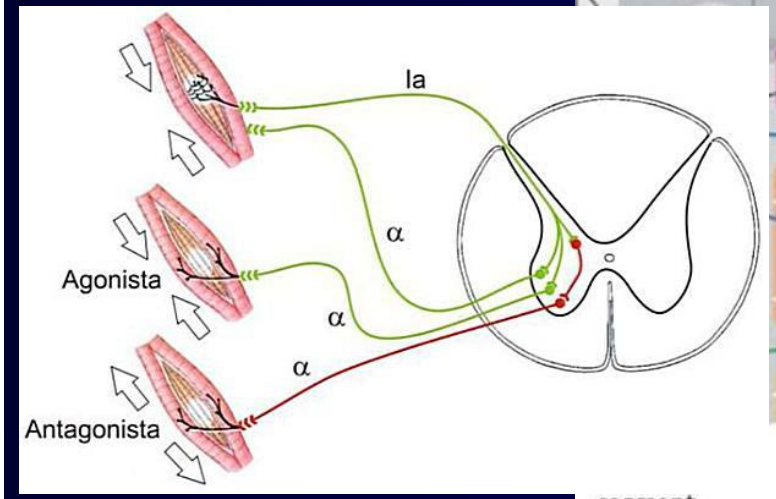
Myotatic reflexes

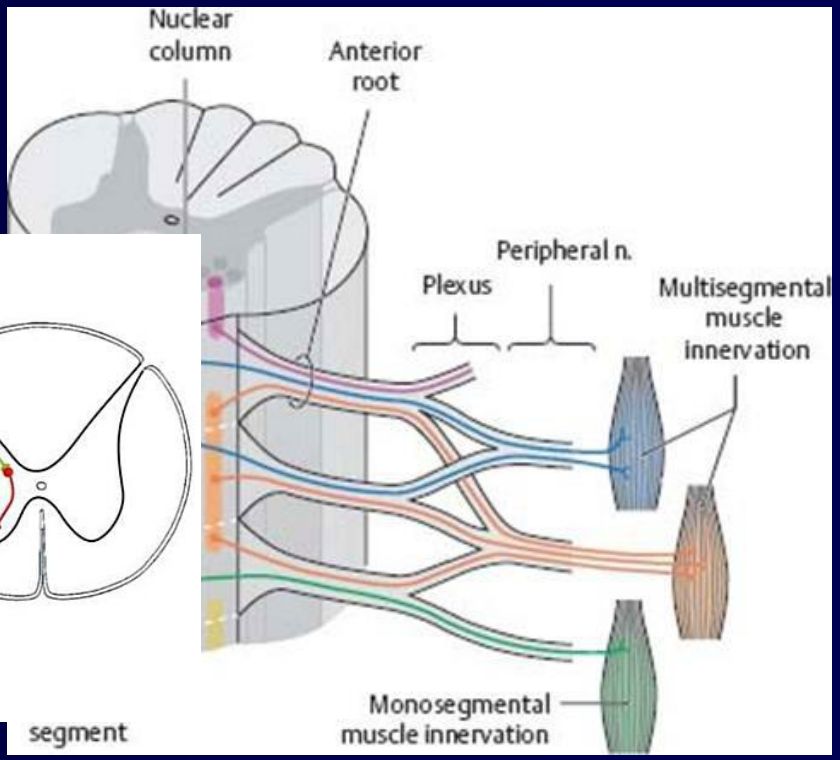
Deep tendon reflexeses

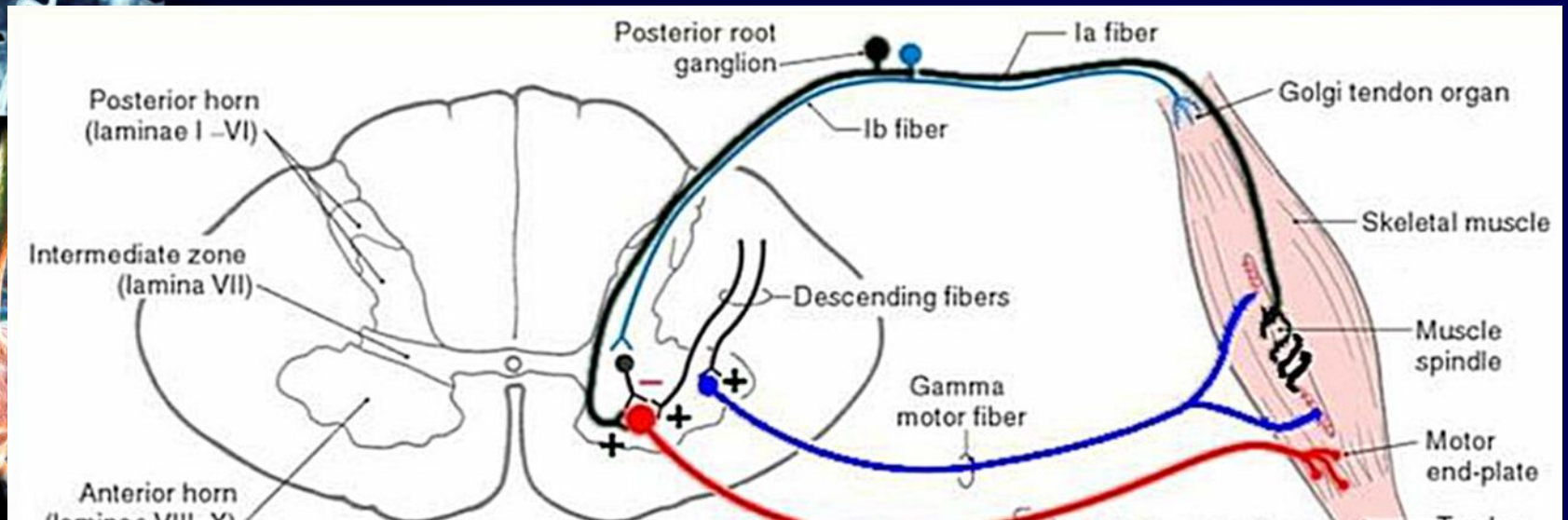


Motoneronal organisation





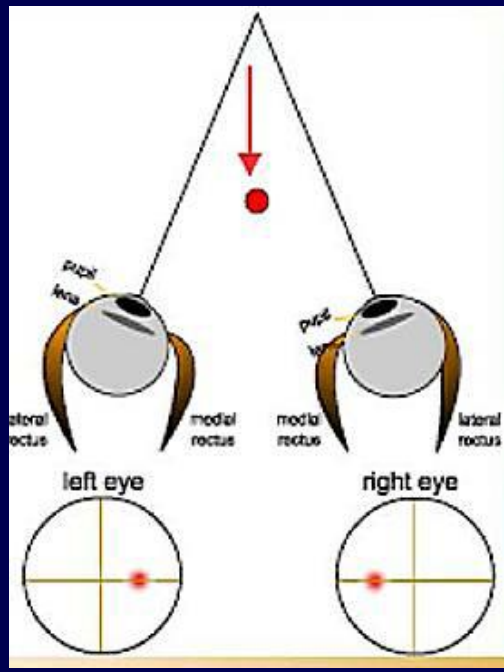




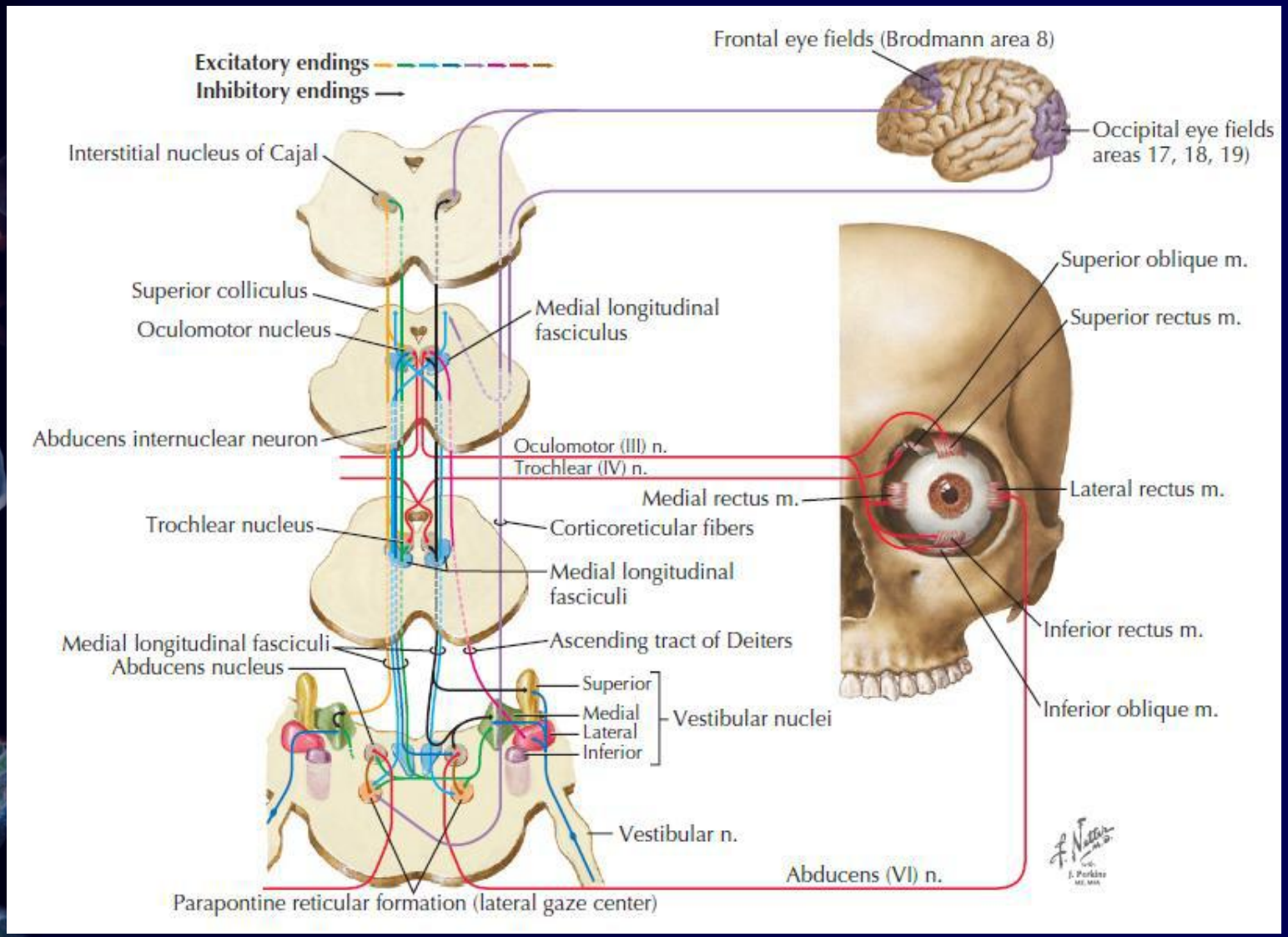
Eye movements

- Four basic types of eye movements
  - Vergence movements (convergemnt, divergent, consensual)
  - Saccades (fast but small muvements to track the most from the objects or e.g.) impostant to get asmuch info per short time
  - Smooth pursuit movements (tracking the object o finterest)
  - Vestibulo - ocular movements

Vergence movements

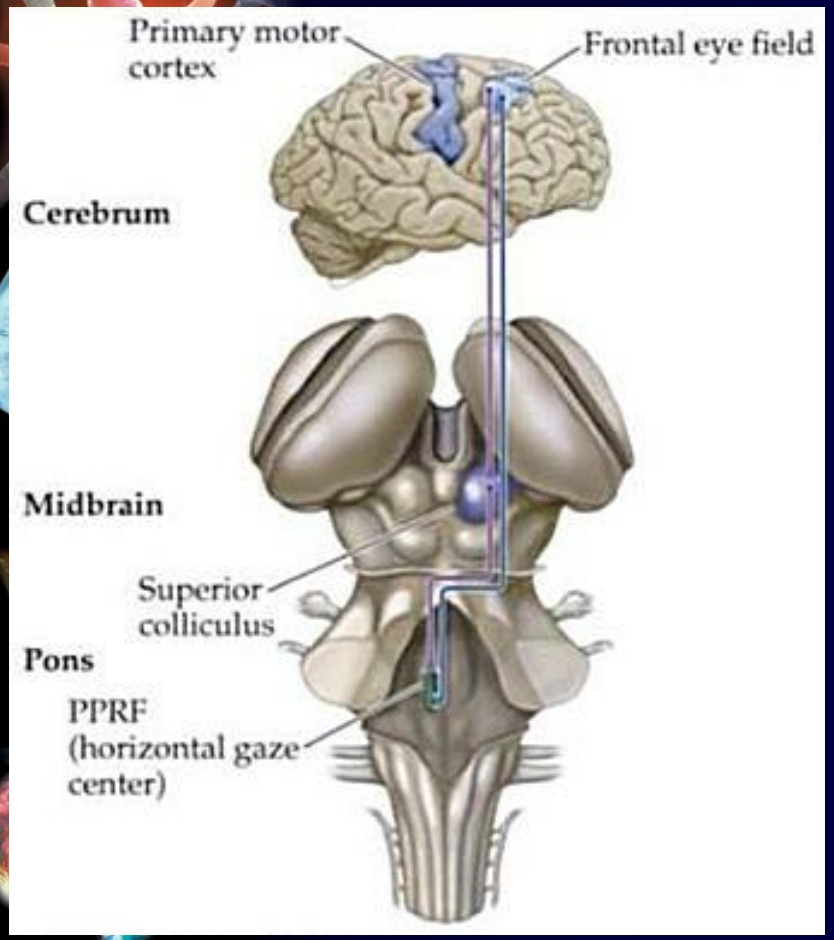


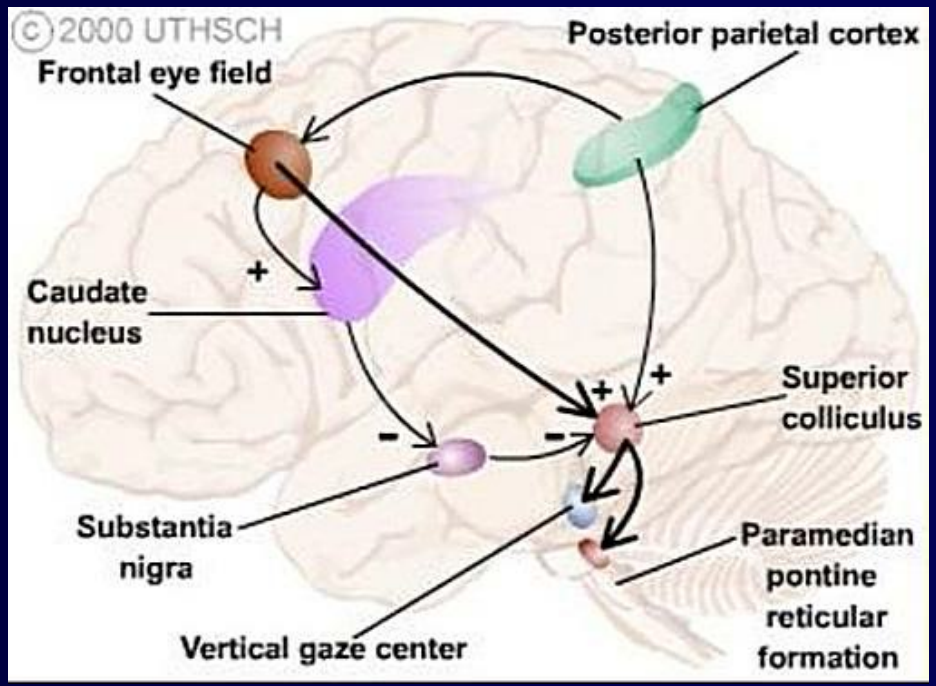
Central control of eye movement



Saccadic eye movements

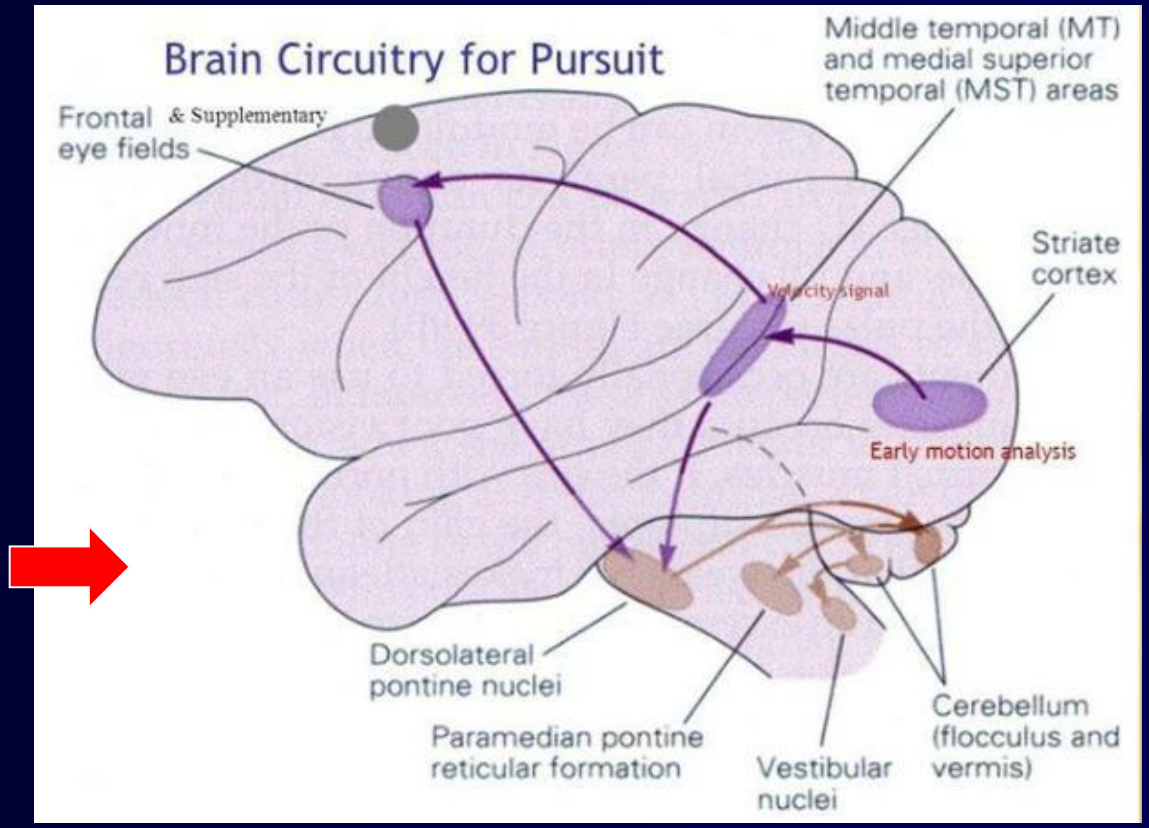
- horizontal gaze center - PPRF
- vertical gaze center - RF of the midbrain
- superior colliculi - information from FEF, retina, auditory,
- and tactile i.





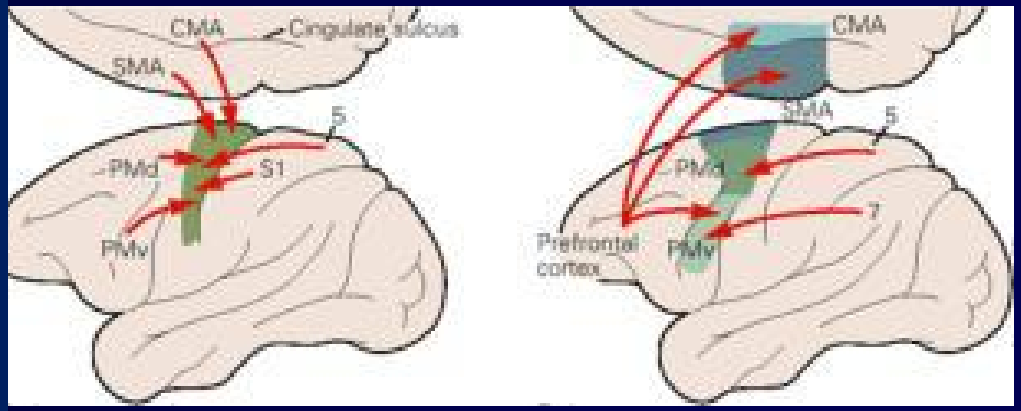
Smooth pursuit movements

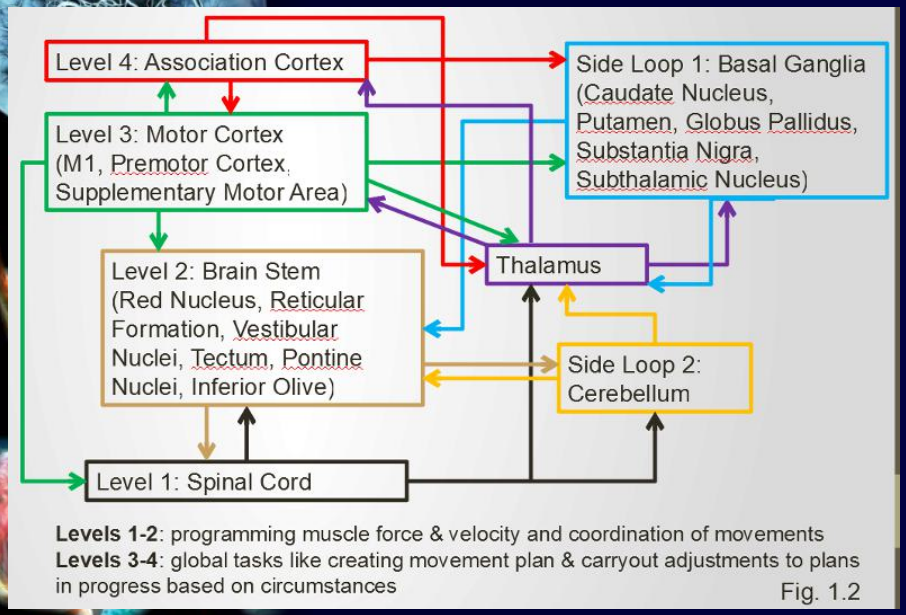
- correction movements elicited by targeting an object (e.g. hunting pray in animals, wathich a game, movie
- voluntarily or under direction

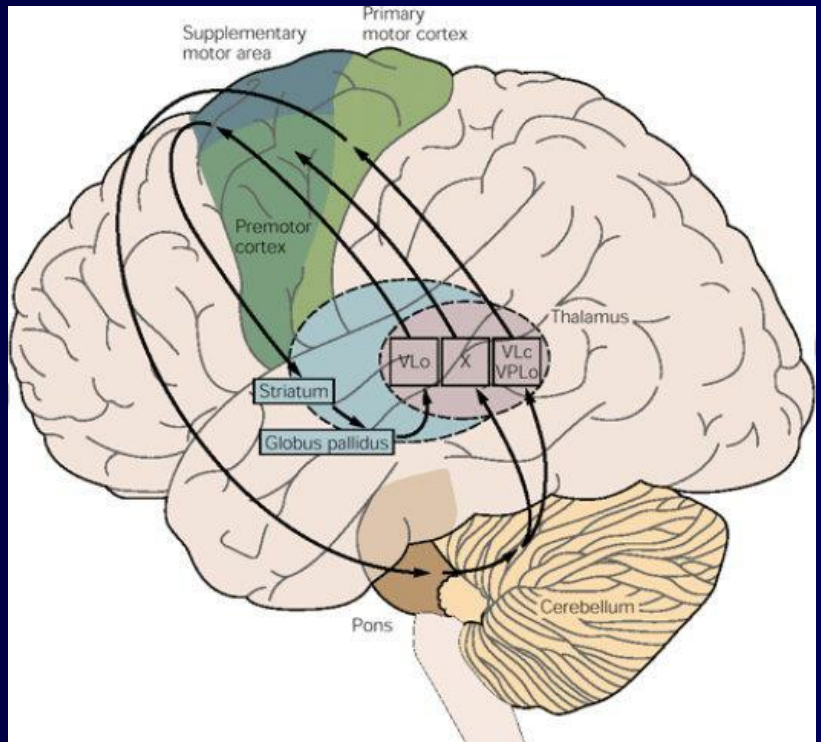


Hierarchy - cortical motor areas

- Primary motor cortex (BA4) – best topological org. (number of neurons) → fast kinetic activity ; plans from the PM and SMA + proprio feedback from somatosensory cor. (S1, BA3,2,1, BA5)..
- Suplementary motor cortex (SMA) (BA6,8) – caudal (motor), rostral cognitive; self –initiated actions, motor planning; bimanual coordination
- Premotor motor cortex (PM) (BA 6)



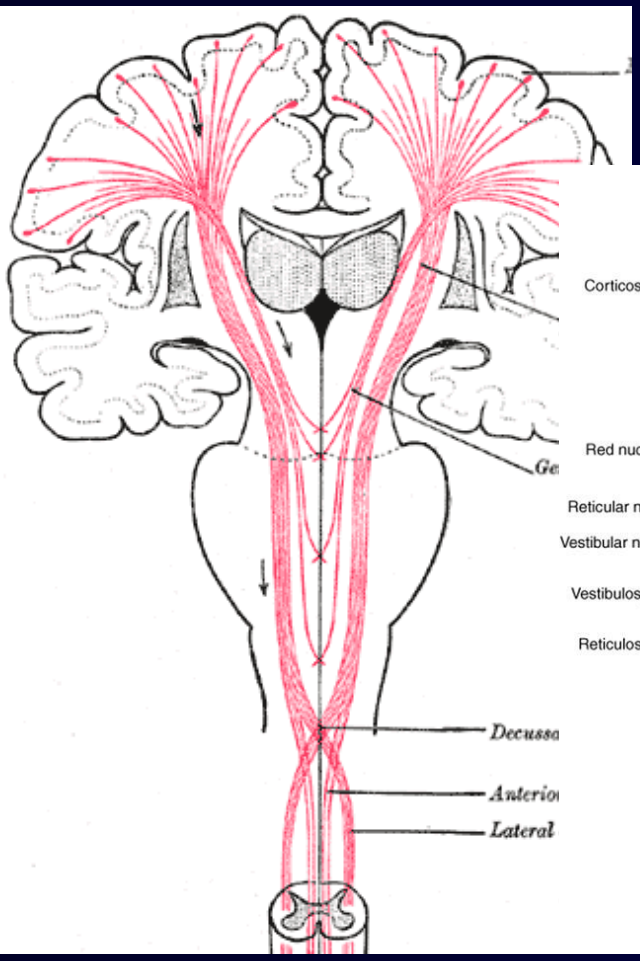


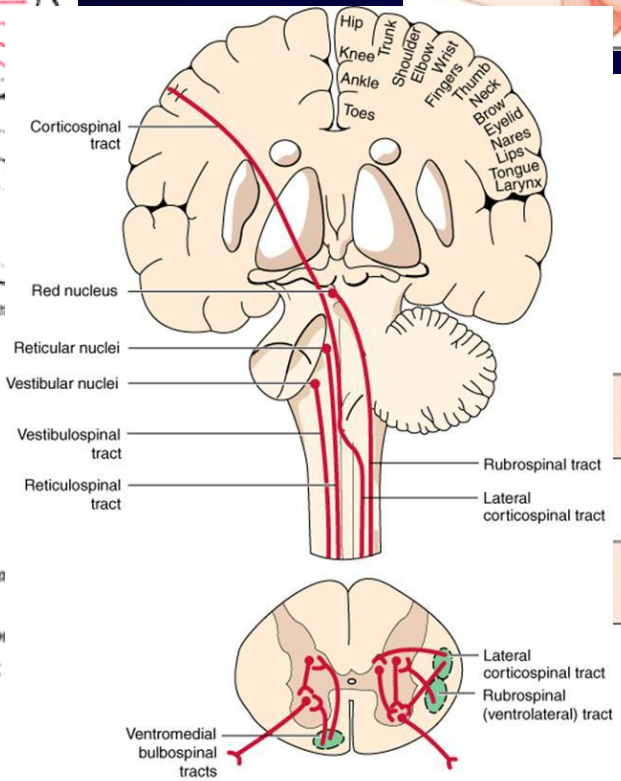


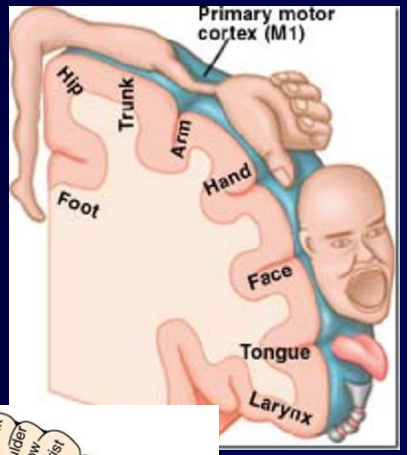
Main skeletomotor areas

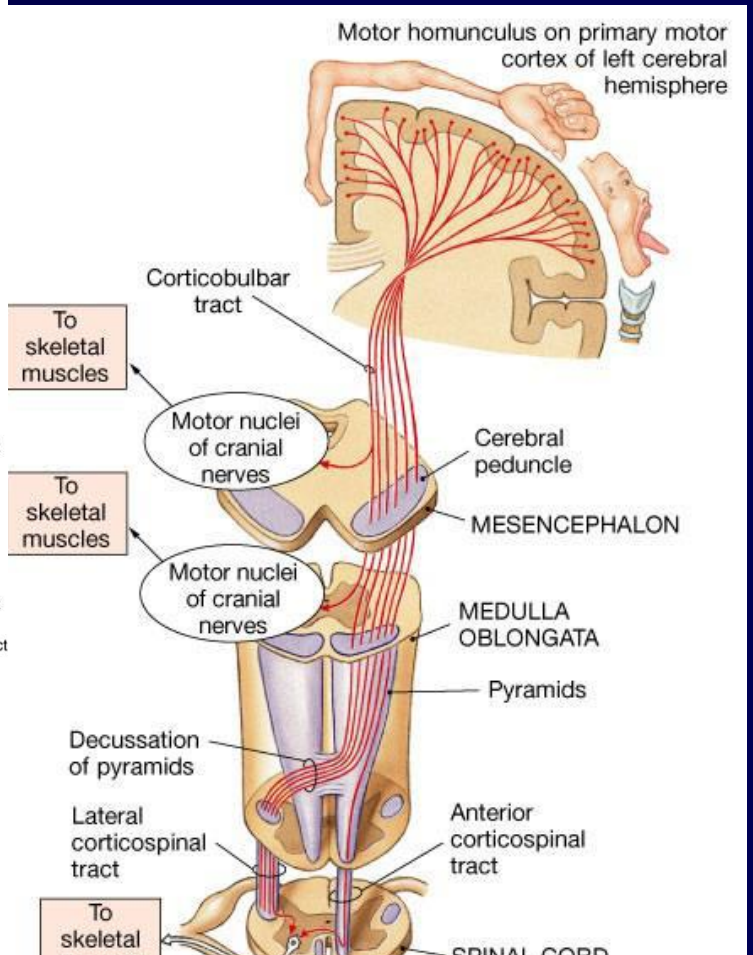
Location	Role	Effects of Unilateral Damage
Primary Motor Cortex (M1) BA4 Precentral gyrus	**Execution** (common pathway) **Ultra fast kinetic movement of contralateral body (motor homunculus)**	**Contralateral hemiparesis/hemiplegia** (worst in **face/hand)**
Supplementary Motor Area (SMA) BA 6 (medial, caudal) Medial surface – superior frontal gyrus (above cingulate sulcus)	**Planning** (internal/self-initiated) **Motor sequencing,** bimanual coordination, **initiation** of complex actions, **motor learning**	**Transient akinesia (ataxia) (**impaired movement initiation bimanual discoordination, alien-hand or grasping reflex)
Premotor Cortex (PM) BA 6 (lateral)Lateral frontal lobe, immediately anterior to M1	**Planning** (external/sensory-guided) **Preparation of goal-directed movements** based on visual/tactile cues; proximal & postural control	**Ideomotor apraxia** (cannot perform skilled movements on command despite intact strength), difficulty with tool use or sequenced actions
Frontal Eye Field (FEF) BA 8 Posterior part of middle frontal gyrus (junction of BA 6/9)	**Planning + Execution** (oculomotor) Voluntary saccadic eye movements, gaze shifting, visual spatial attention	**Contralateral saccadic gaze palsy, (**impaired visual scanning,, difficulty to focuse eyes)
Broca's Area 44 & 45 Inferior frontal gyrus (pars opercularis = 44, pars triangularis = 45); dominant (usually left) hemisphere	**Planning + Execution** (speech motor) Articulatory planning, grammatical encoding, speech production & orofacial motor sequences	**Broca's (non-fluent/expressive) aphasia** (effortful, agrammatic speech with intact comprehension; apraxia of speech)

Corticospinal pathway





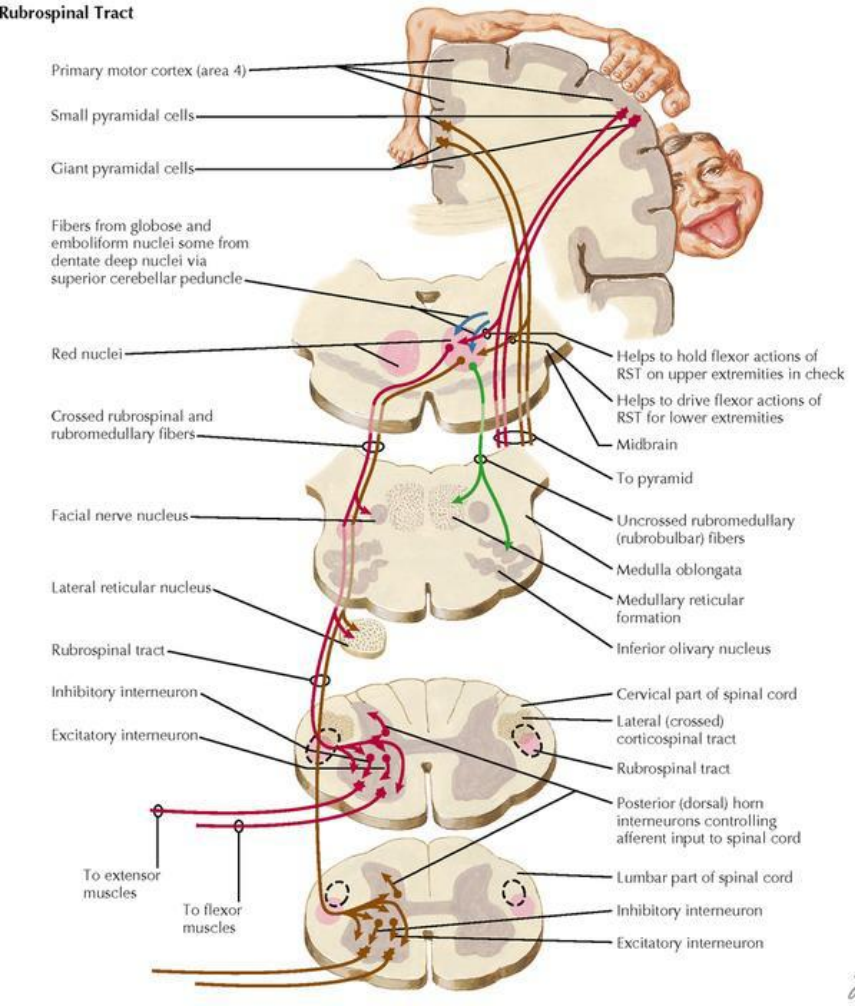


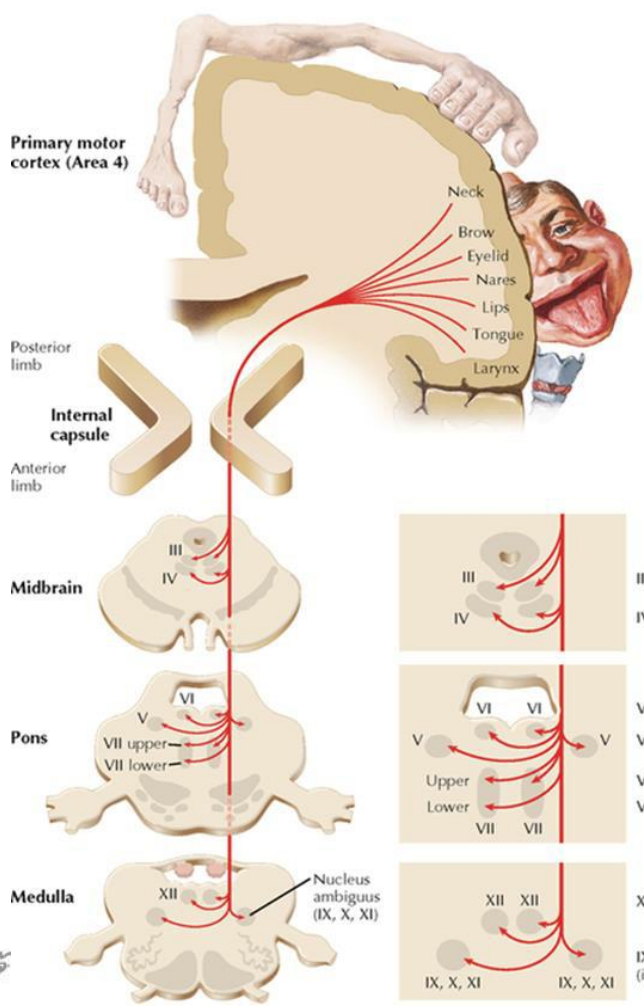


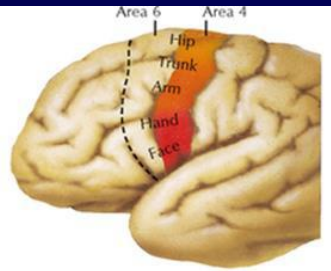
MI is well developed in humans and serves for rapid 2 synaptic motor drive into fast-responding muscles of the hand, face, mouth, pharynx, tongue. It is not essential but disables impairs activites typical for humans

Rubrospinal pathways

Corticobulbar and corticospinal pathways







Lateral aspect of cerebral cortex to show topographic projection of motor centers on precentral gyrus and premotor and supplemental motor cortex

III (ipsilateral and contralateral)

IV (ipsilateral and contralateral)

VI (ipsilateral and contralateral)

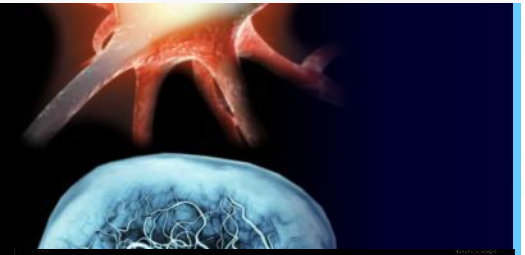
V (ipsilateral and contralateral)

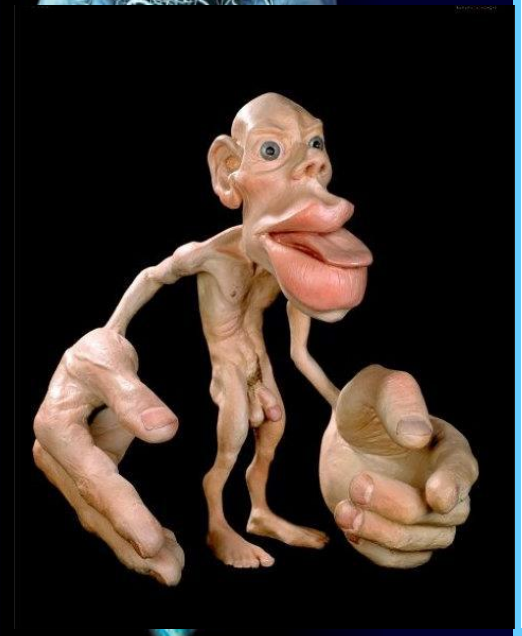
VII to upper face (ipsilateral and contralateral)

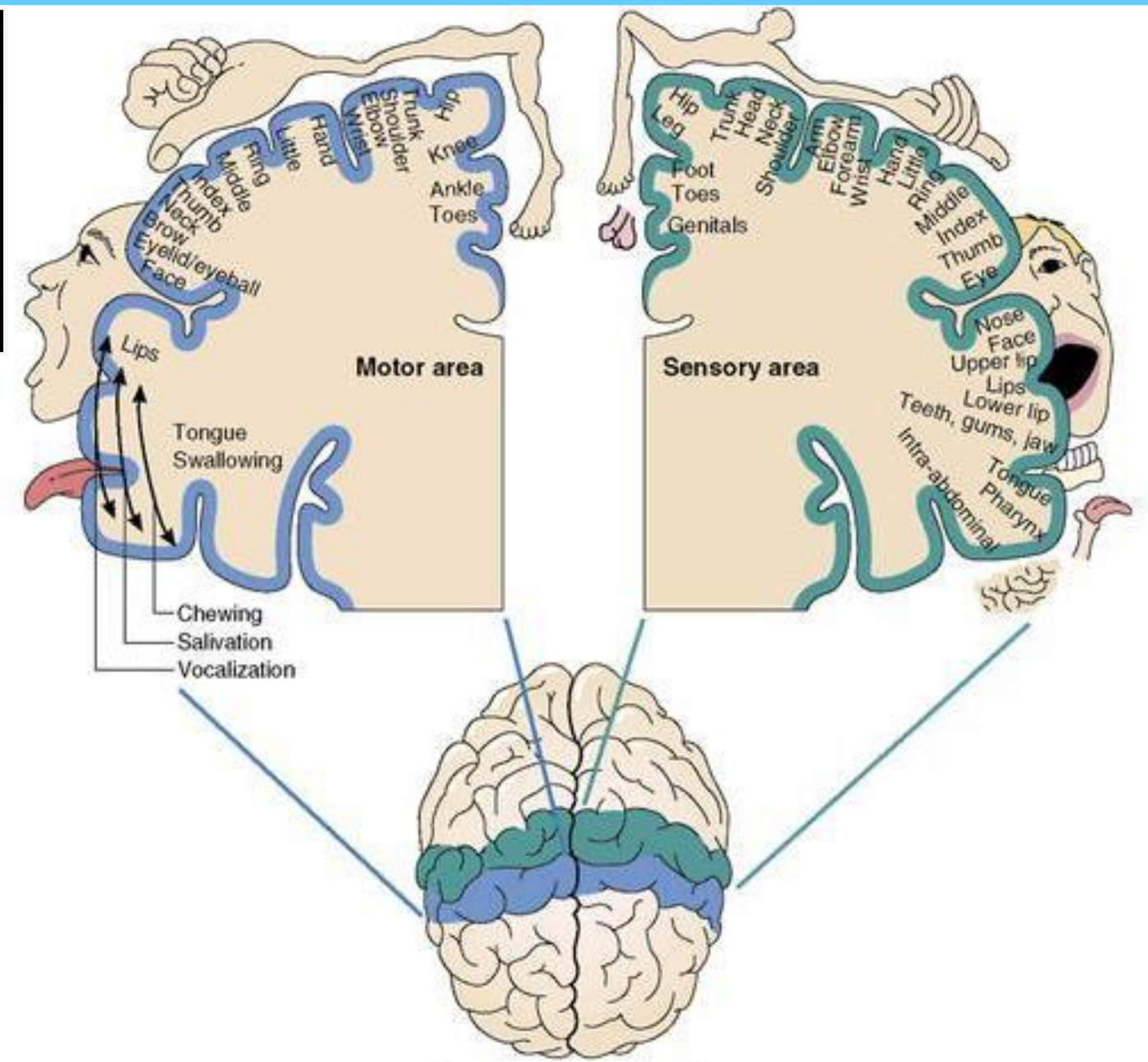
VII to lower face (contralateral only)

XII (ipsilateral and contralateral)

IX, X, and XI (ipsilateral and contralateral)

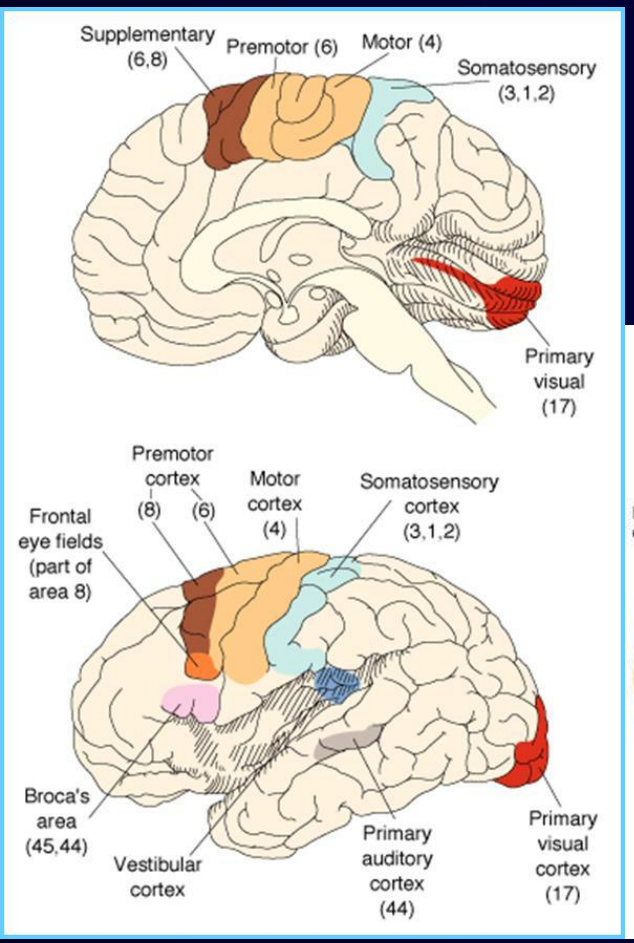


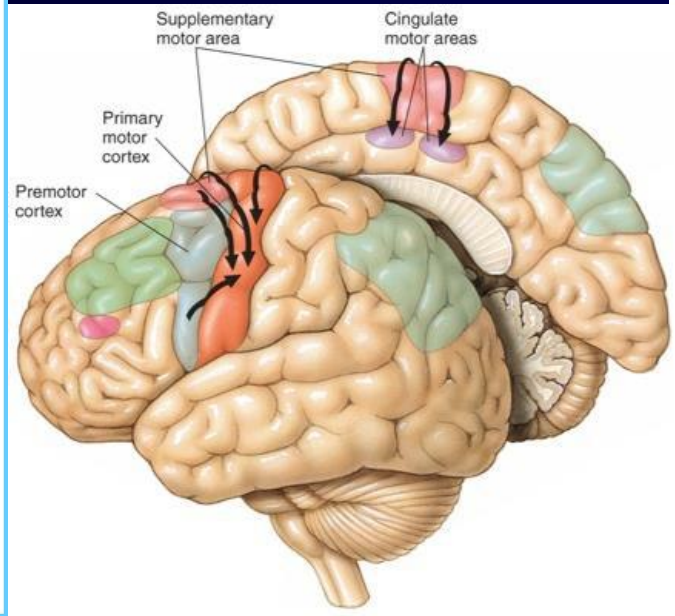


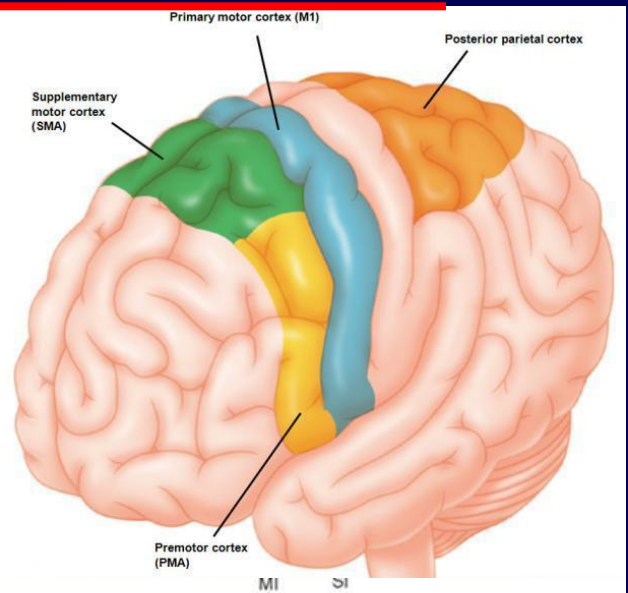

Motor area
Sensory area
Hip
Knee
Ankle
Toes
Trunk
Shoulder
Elbow
Wrist
Hand
Little
Ring
Middle
Index
Thumb
Neck
Brow
Eyelid/eyeball
Face
Lips
Tongue
Swallowing
Chewing
Salivation
Vocalization
Hip
Leg
Foot
Toes
Genitals
Trunk
Head
Neck
Shoulder
Arm
Elbow
Forearm
Wrist
Hand
Little
Ring
Middle
Index
Thumb
Eye
Nose
Face
Upper lip
Lips
Lower lip
Teeth, gums, jaw
Tongue
Pharynx
Intra-abdominal

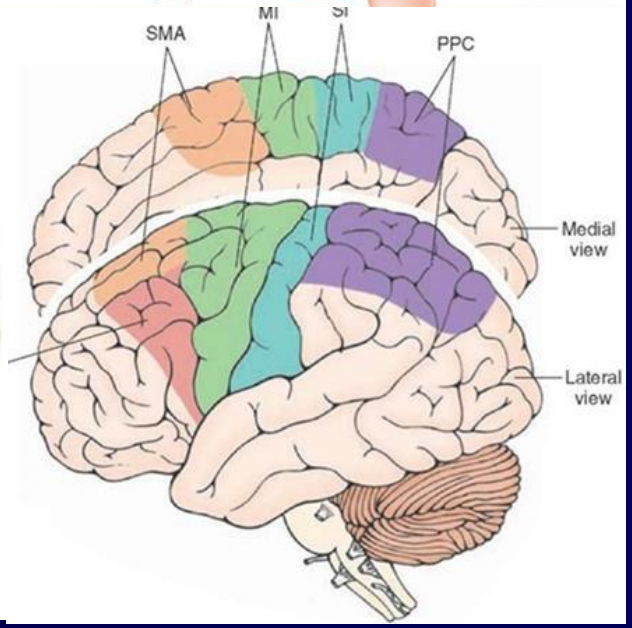
Cortical motor – associated areas

- Primary motor cortex (BA4)
- Suplementary (BA6,8)
- Premotor cortex (BA 6)
- Frontal assotiation cortex
- Frontal eye field (6)
- Broca speech area (BA 44)
- Parietal eye field
- Parietal association cortex
- Writing, painting, speech









Cortical representation of motor (left, MI, precentral gyrus, area 4) and sensory (right, SI, postcentral gyrus, areas 1,3,2) supply (based on prioneering human data by Penfield and others)

Additional skeletomotor areas

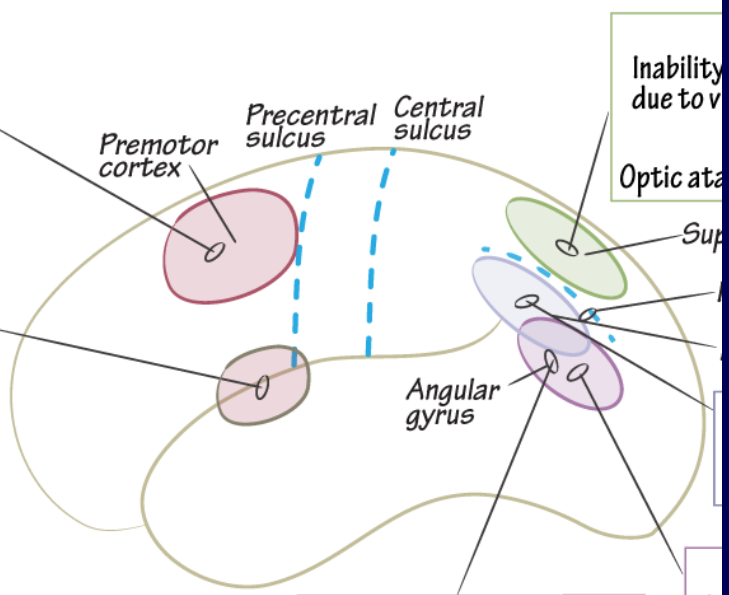
APRAXIA & NEGLECT

- **Apraxia**
  - ✓ *Inability to perform a purposeful action in the setting of preserved overall neurologic function, and is more common with LEFT brain injuries.*
- **Hemispatial Neglect**
  - ✓ *Neglect is a lack of regard for objects in the contralateral hemisphere and is more common with RIGHT brain injuries.*

EXECUTIVE APRAXIAS

Limb-Kinetic Apraxia
Impairment in finely-graded finger movements and awk-wardness of the arm and hand.

Speech Apraxia
Slowing and incoordination of speech in the presence of otherwise normal language.

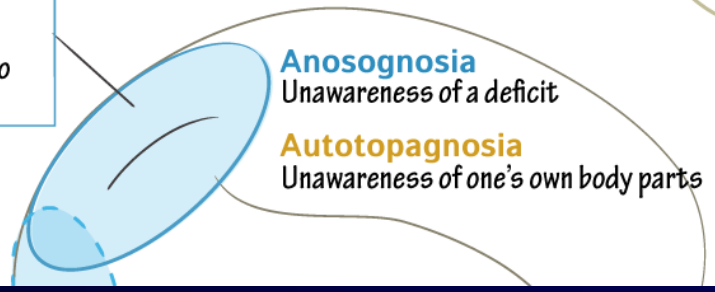


Gerstmann Syndrome
Left /right disorientation
Finger agnosia, Dyscalculia
Dysgraphia

HEMISPATIAL NEGLECT

Mild – Extinction to double simultaneous stimulation.

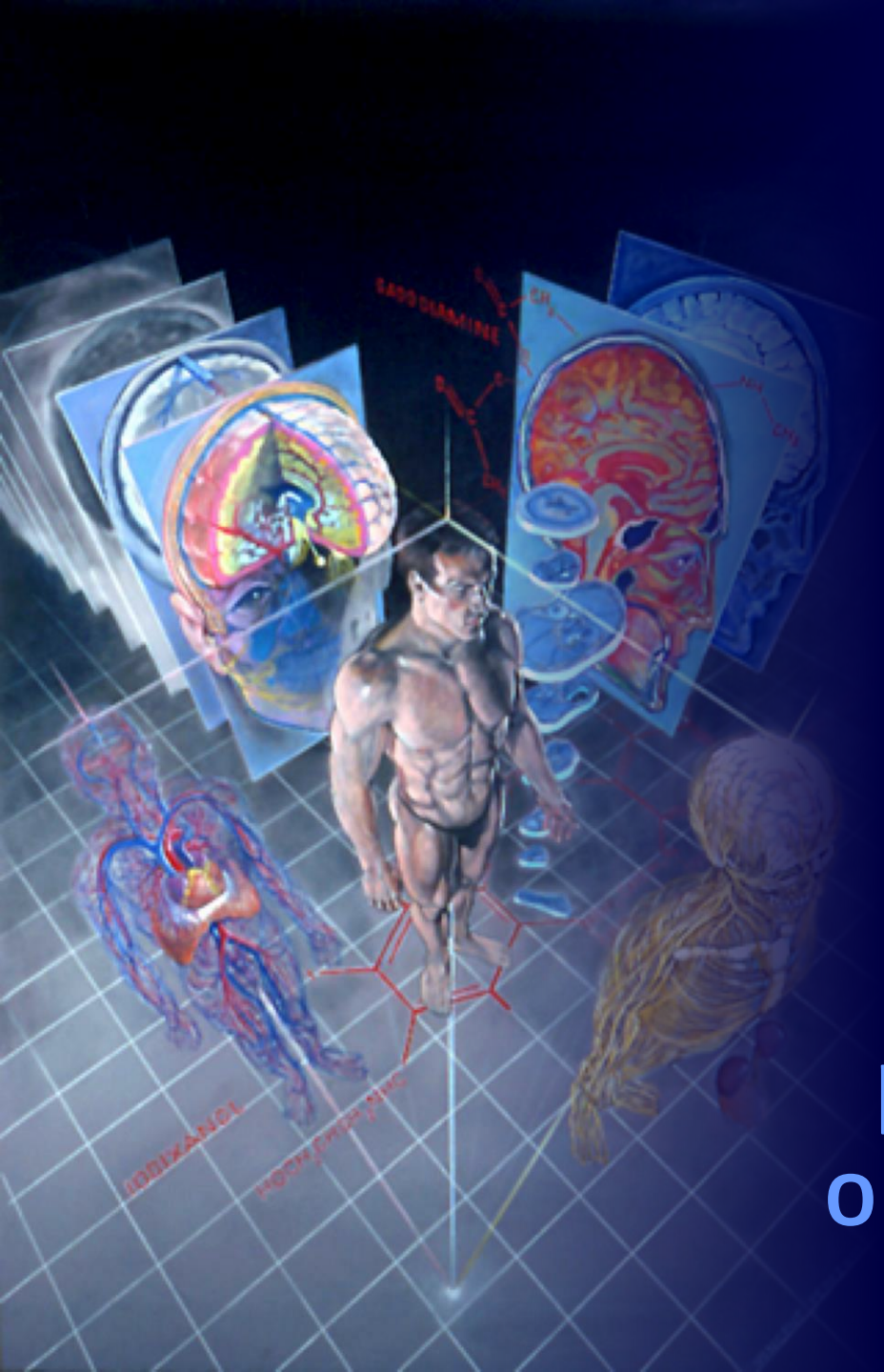
Severe – Failure to attend to the left half of the world.



Anosognosia
Unawareness of a deficit

Autotopagnosia
Unawareness of one's own body parts

Area (Brodmann)	Role in movement / praxis	Damage effects
Inferior parietal lobule (BA 39 + 40) Angular g. (39) + su pramarginal g. (40)	Storage of “movement formulas” / praxis engrams; action recognition, sequencing, tool-use knowledge; sensory-motor integration	Left **> right** hemisphere **Ideomotor apraxia** (cannot imitate or pantomime on command); **ideational/conceptual apraxia** (cannot sequence multi-step actions or use tools conceptually); gesture discrimination I
Superior parietal lobule (BA 5 + 7) intraparietal sulcus; medial/lateral parietal	Somatosensory association; visuomotor integration; spatial body schema; online correction of movements	Right more neglect + constructional apraxia **Optic ataxia** (cannot accurately reach under visual guidance); constructional apraxia (drawing/copying deficits); **Tactile/motor agnosia** (astereognosis); dressing apraxia; part of Balint’s syndrome (with simultanagnosia)
Parieto-occipital junction / posterior parietal BA 7/19/39; intraparietal sulcus	Grasping, reaching, spatial attention for action	**Optic ataxia** + **visuomotor apraxia**; impaired tool grasping and action means Often co-occurs with inferior parietal lesions
Insula (not strict Brodmann; ~BA 13–16) lateral fissure;	Praxis of speech/orofacial movements; tool-use knowledge; action recognition	**Left insula** **Speech apraxia** (orofacial/limb-kinetic); impaired common tool use and action recognition; strongly linked to apraxia of speech
Corpus callosum (white-matter disconnection, not pure cortex)	Anterior body or splenium Inter-hemispheric transfer of praxis commands	**Callosal apraxia** (left-hand only ideomotor apraxia; right hand spared) **Disconnection syndrome;** parietal-frontal fibers affected



2

Clinical pathophysiology of motor disorders

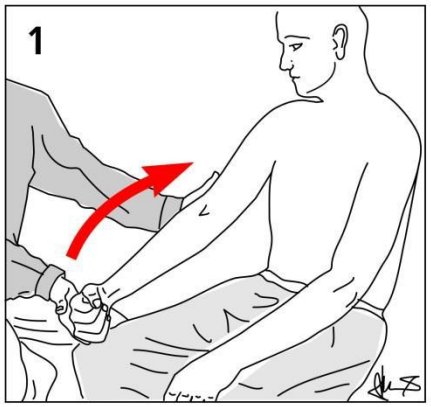
Clinical evaluation and terminology

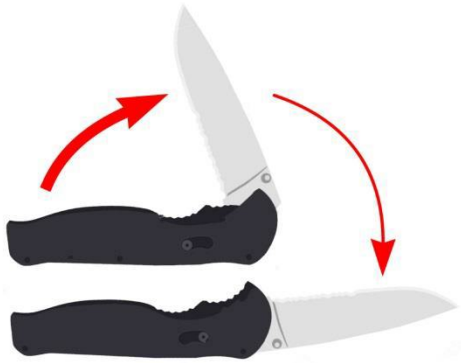
- Ability to move, muscle force:
  - Muscle weakness (Paresis)
  - Paralysis (Plegia, Palsy)
  - Distribution of these:
    - Hemiparesis, quadruparesis, monoparesis, biplegia, paraparesis, etc.
- Appropriate amount or pattern of movement:
  - Hypokinesia, bradykinesia
  - Hyperkinesia, dyskinesia
    - Chorea, athetosis, tics, ballism, tremor, akathisia, myotonia, myokymia, myorhythmia
- Muscle tone:
  - Hypotonia - flaccidity
  - Hypertonia
    - Spasticity and rigidity
  - Dystonia and spasms
- Gait – standing, walking (narrow base, wide-base:
  - Ataxia (dystaxia)
    - Spinal (posterior collums)
    - Cerebellar, Frontal
    - Vestibular ataxia
- Reflexes:
  - Hyporeflexia,
  - Hyperreflexia

Muscle tone

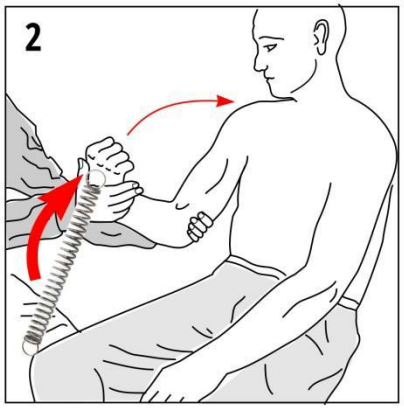
- Def: Resistence felt by examiner while trying to move(flex) extremities passively
- Hypotonia - flaccidity dead hand, foot; dropping foot (flaccid paralysis)
- Hypertonia - Spastic rigidity (uppermotor neuron syndrome; multiple sclerosis)
  - Plastic rigidity (extrapyramidal dis.; parkinsonism)

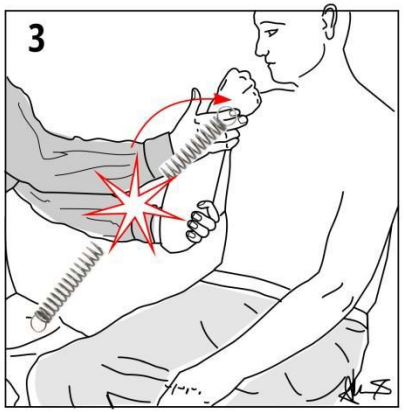
Clasp knife phenomenon



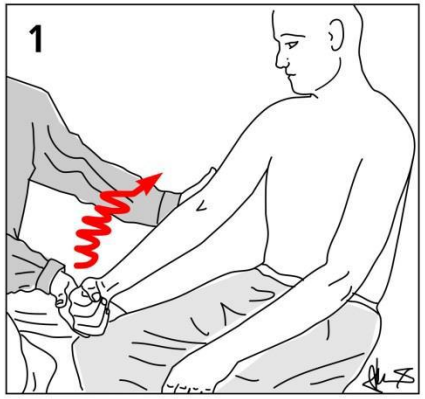


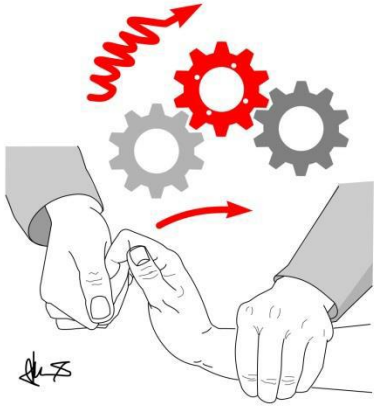
Spring like rigidity



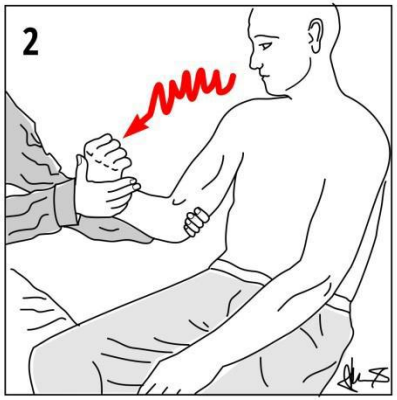


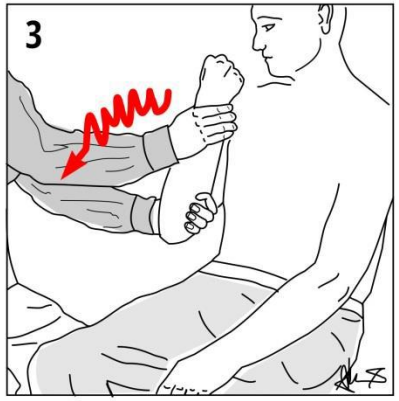
Cog-wheel phenomenon





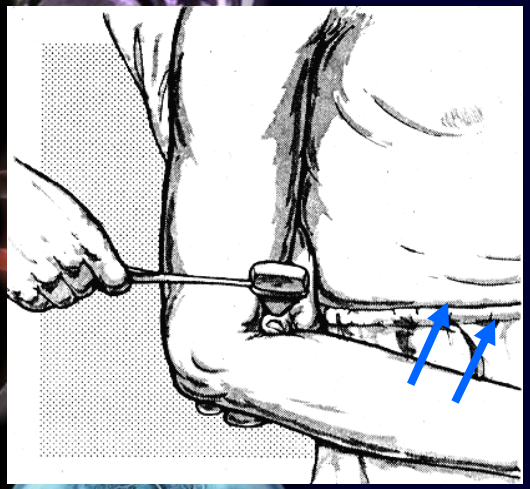
Plastic regidity



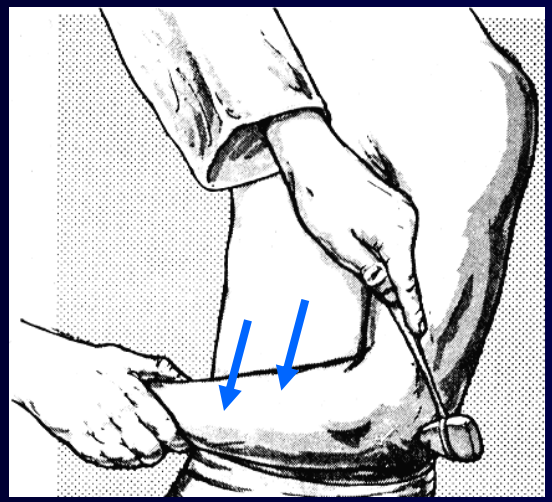


Deep tendon reflexes

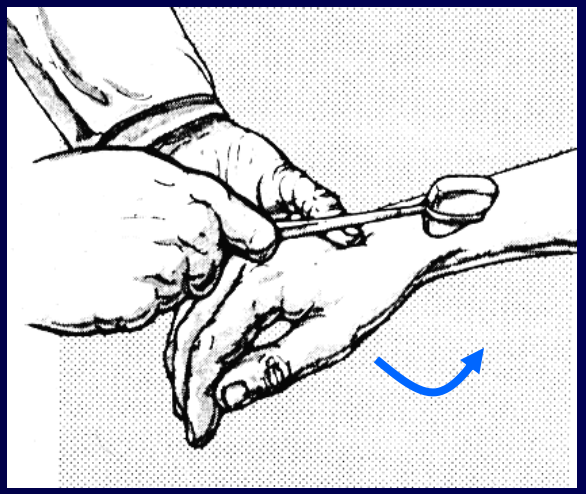
Bicipital reflex C5-C6

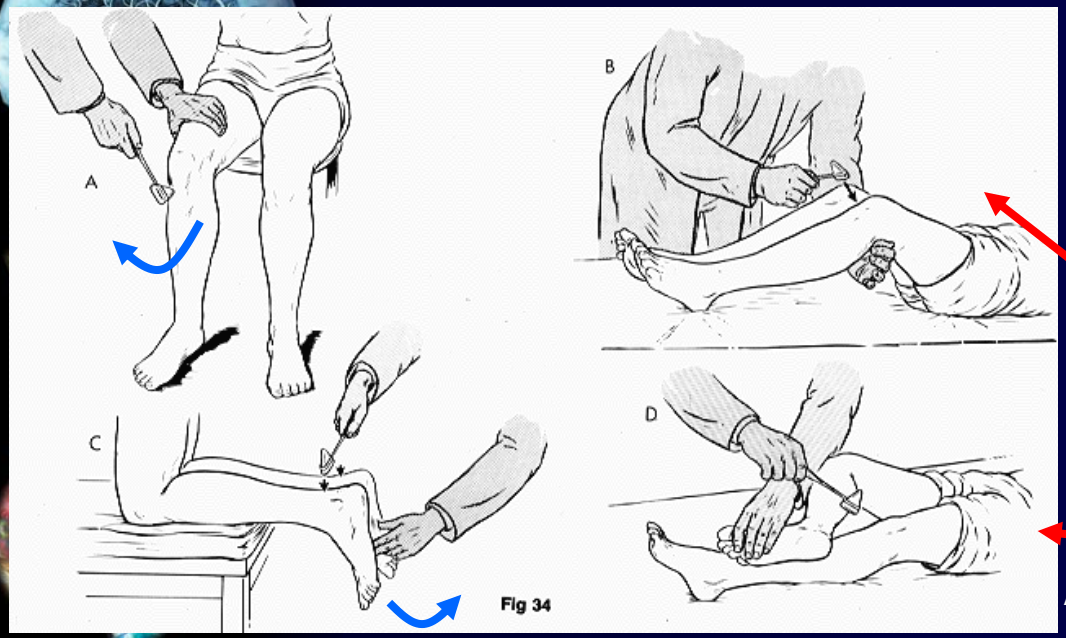


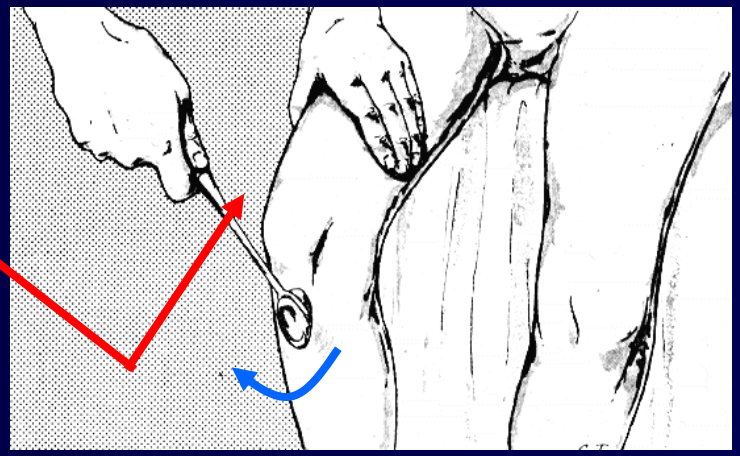
Tricipital reflex C7-C8



Reflex brachioradialis C5-C6







Patellar reflex L2-L4

Achilles ankle tendon reflex S1- S2

Cerenie zubov
Clenching the teeth

Uzatvorenie úst
Closing mouth

Špúlenie pier
Snouting

Zatvorenie očí
Closing eyes

Zatvorenie očí proti odporu
Closing eyes againts resistence

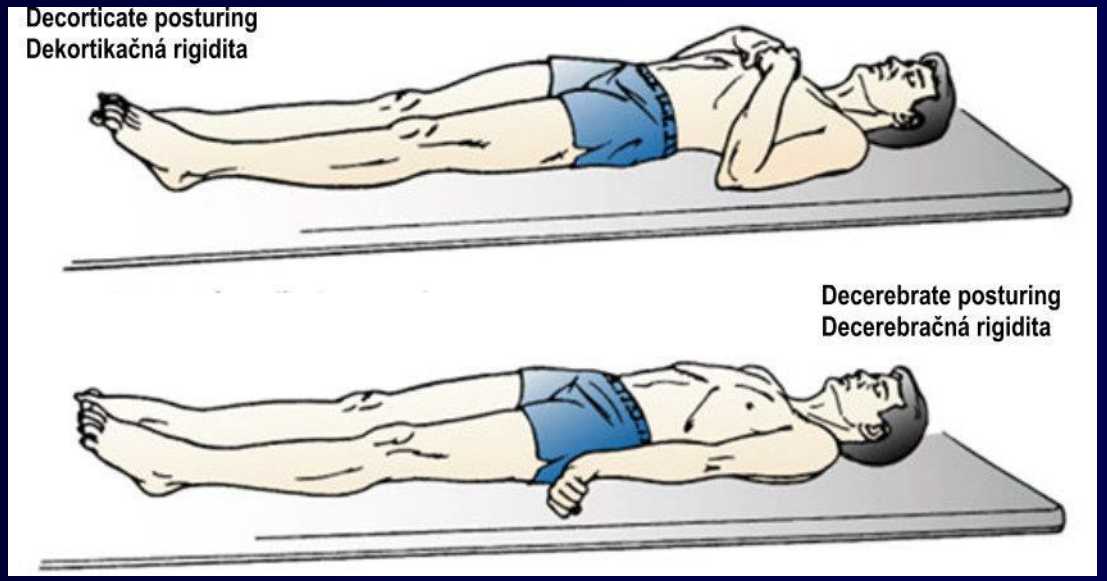
Dvíhanie obočia, vraštenie čela
Eyebrow raising, forehead wrinkling

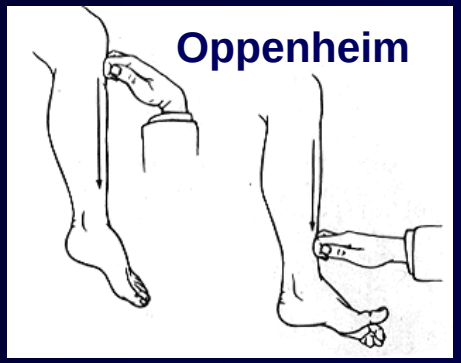
Usmievanie
Smiling

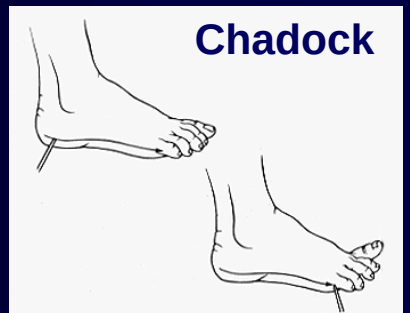
Jaw jerk
Maseterový reflex

Deliberačné javy = axiálne reflexy (mentolabiálny, nazolabiálny)

Vyšetrovanie patologických odpovedí







Hoffmanov reflex (reakcia)

Gait disturbances

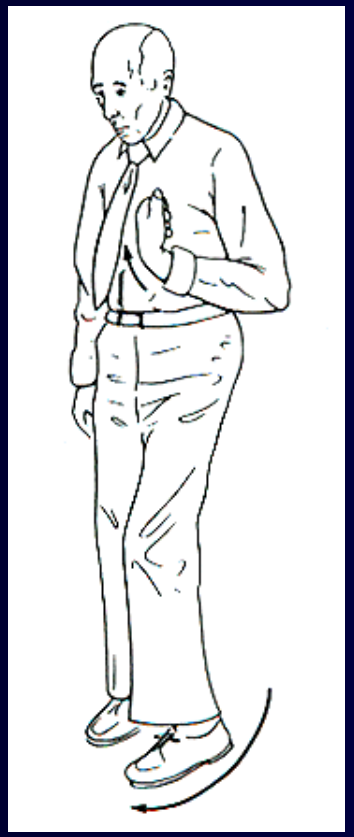
- Spastic Gait (Hemiparetic or Scissor) damage to pyramidal tract upper motor neurons (e.g., stroke or Cerebral Palsy).
  - Hemiparetic: Sy: One side is stiff. The patient cannot flex the hip or knee properly, so they swing the leg out in a semi-circle (Circumduction) to move forward.
  - Scissor (Diplegic): Sy: Both legs are affected. The thighs press together, and the knees cross over each other like scissors while walking.
- Parkinsonian Gait (Festinating) cause: Parkinson's disease; SyStooped posture, small shuffling steps, and a lack of arm swing. Festination—the patient's center of gravity is too far forward, so they start walking faster and faster in small steps as if trying to "catch up" with their own body to avoid falling.
- Waddling Gait (Myopathic) cause: muscular dystrophy or severe hip weakness; Sy: The pelvis drops on the side of the leg being lifted. The entire trunk sways from side to side, resembling a duck's walk, because the "Gluteus Medius" muscle is too weak to stabilize the hip.
- Trendelenburg gait - cause: /paralysis of the hip abductor muscles, primarily the gluteus medius and gluteus minimus. Sy: In a healthy person, these muscles contract when standing on one leg to keep the pelvis level. In a patient with a Trendelenburg gait, the "pump" fails, causing the pelvis to tilt.

Gait disturbances

- Steppage Gait (Equine/Neuropathic) cause: "Foot Drop" Peroneal nerve palsy or L5 radiculopathy). Sy: The patient lifts their knee extra high to prevent the toes from dragging on the ground. When the foot finally hits the floor, it makes a distinctive "slap" sound.
- Sensory Ataxic Gait (Stamping) damage to posterior columns; The patient watches the ground and their feet constantly. Stamping. They slam their feet down hard to create a vibration they can actually feel. This gait worsens significantly in the dark.
- **Cerebellar gait** cause: damaged (e.g., Stroke, Multiple Sclerosis, or Alcoholism). Sy: : Wide-based stance (legs far apart) and staggering. The patient is unsteady and "veers" from side to side, appearing intoxicated.

Gait disturbances

Spastic hemiparetic

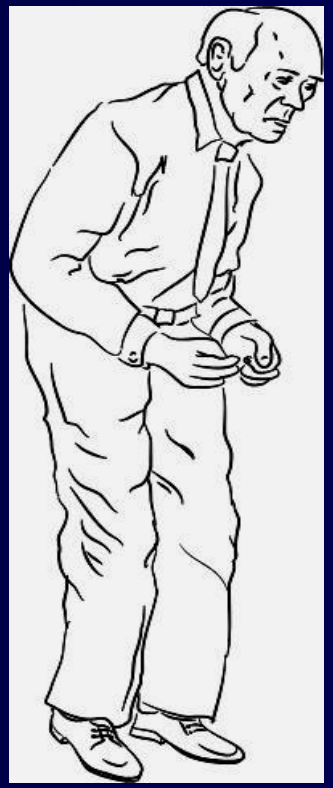


Spastic paraparetic

- Cerebral, subcortical, or myelopathiclesons. Stroke, MS, or tumor atmultiple sites: frontal lobe, internalcapsule, pontine, or brainstem andcervical spinal cord, particularly spinalstenosis.

Gait disturbances

Rigid parkinsonic



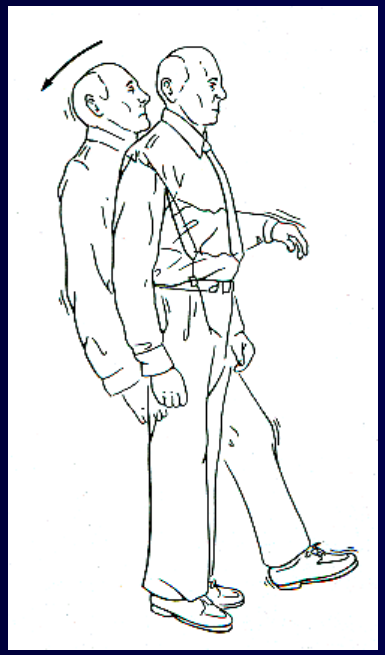
Light: unilateral; mask face; rigid right arm held, tremor

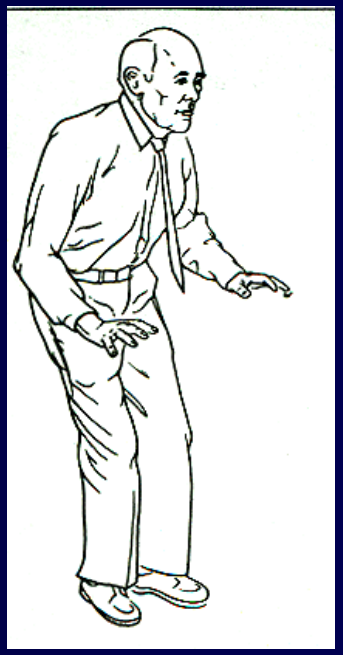
Mild: bilateral; mask face; stooped posture, shuffling, short steps (petit pas)

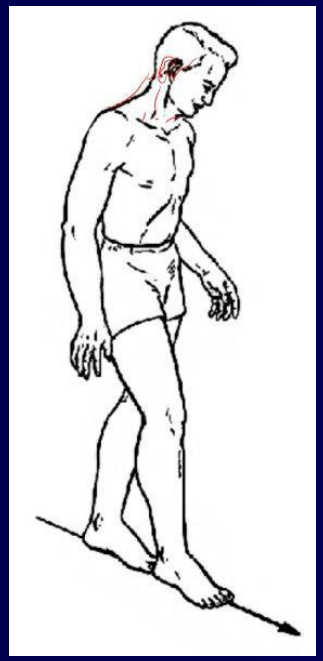
Severe: instability, falling bilateral; inability to start to move (hypokinesia) shuffling, short steps arm, hand !robot“ flection

Peroneal steppage (peroneal n. lesion)

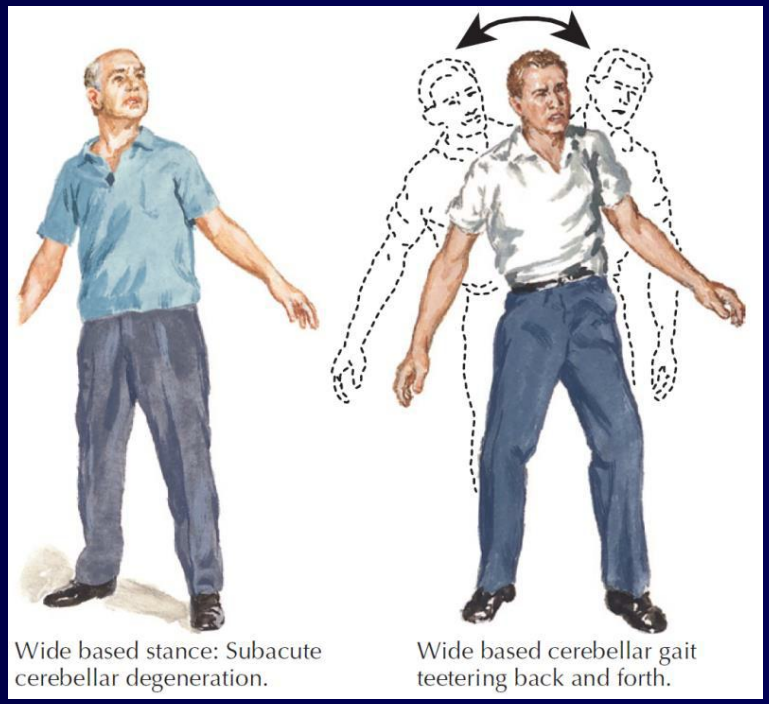
Painful burning feet withnumbness and tingling.Foot drop.







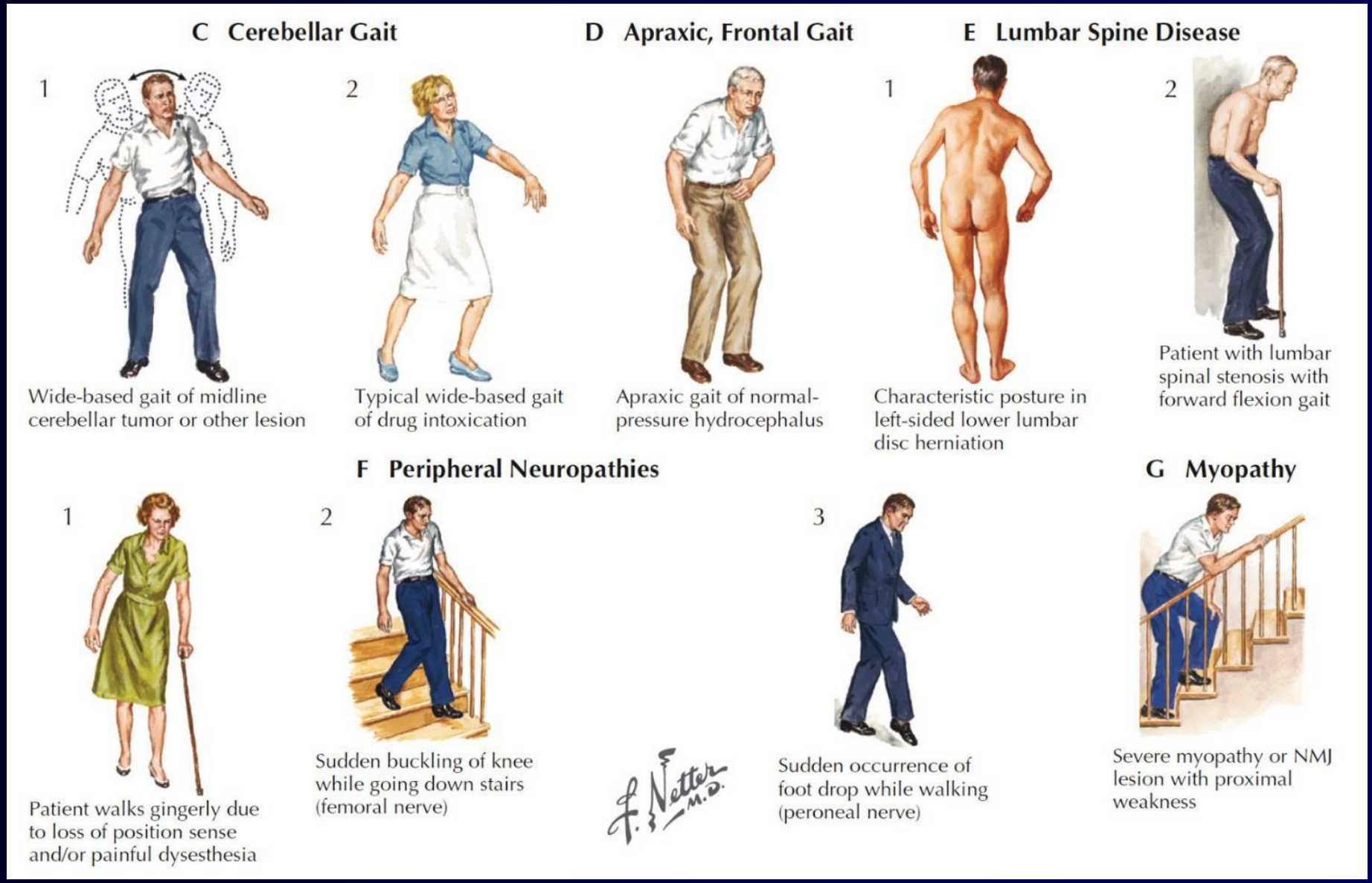
Retropulsions	Frontal/cortical
(vestibular dis.)	Subcortical-hypokinetic
Falling to back	Subcortical-hyperkinetic
Frontal ataxia	Pyramidal
	Cerebellar
	Vestibular
	Neuropathic
	Myopathic
	Orthopedic

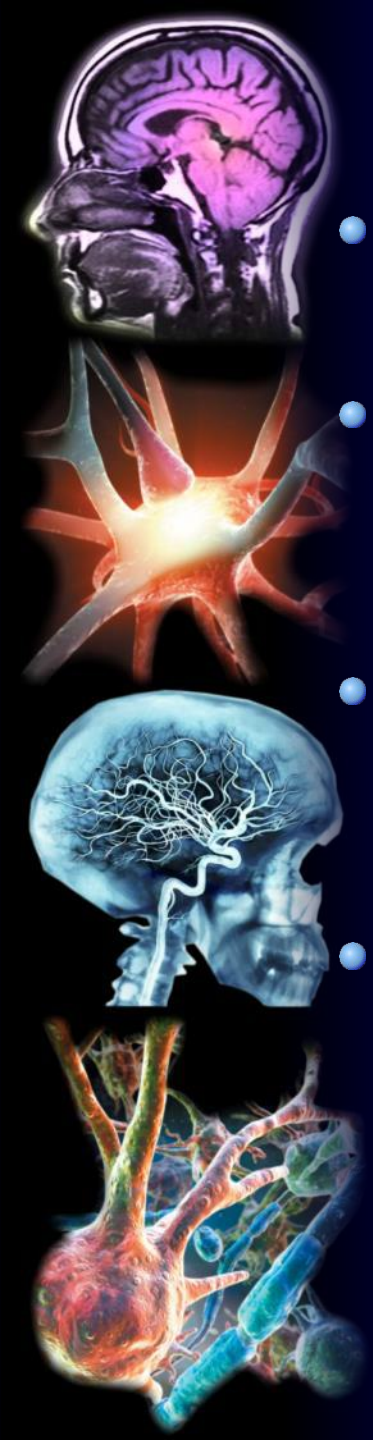


Wide based stance: Subacute cerebellar degeneration.

Wide based cerebellar gait teetering back and forth.

Gait disturbances

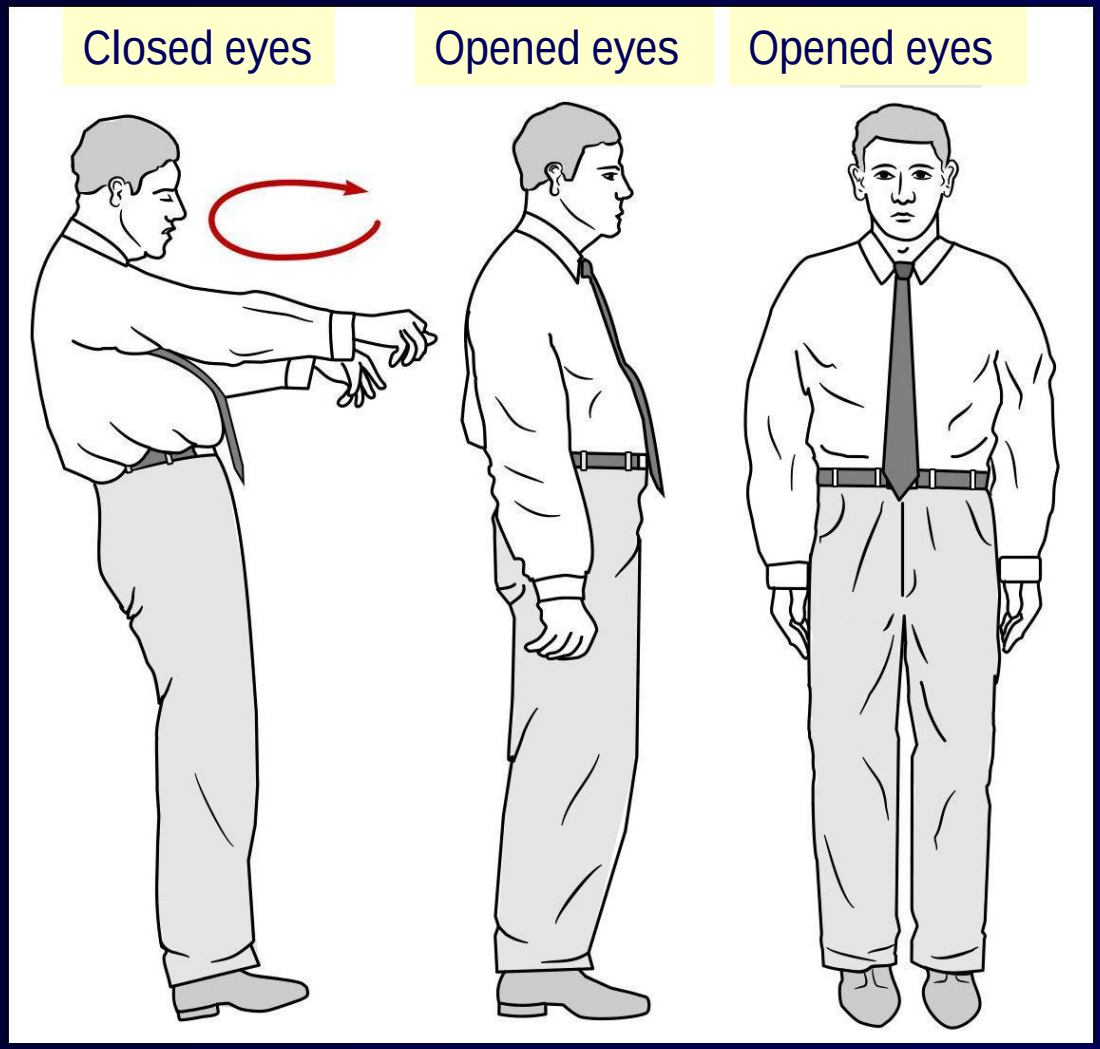




Ataxia

- Def.: loss of coordination and balance, difficulty in speech and swallowing, unsteady walking, hand clumsiness, and affected eye movements.should be differentiated from dizziness.
- Sympt:
  - walking is unsteady or with the feet set wide apart to maintain better balance
  - difficulty with fine motor tasks (eating, writing, stitching, tying laces)
  - change/difficulty in speech (dysarthria), difficulty in swallowing,
- Etio: damage to cerebellum) or its neural connections from spinal cord, vestibular nerve, cortex
  - Neurologic damage (focal lesions) vs. Systemic disorders (general lesions)
  - Acquired cases vs hereditary causes.
- Acquired focal:
  - Kids - Cerebral palsy (damage to a child's brain during early development),
  - Children, adults - Brain tumor (causing pressure on cerebellum), Head injury, Multiple sclerosis, Stroke,Brain abscess, Multiple system atrophy)

Romberg test



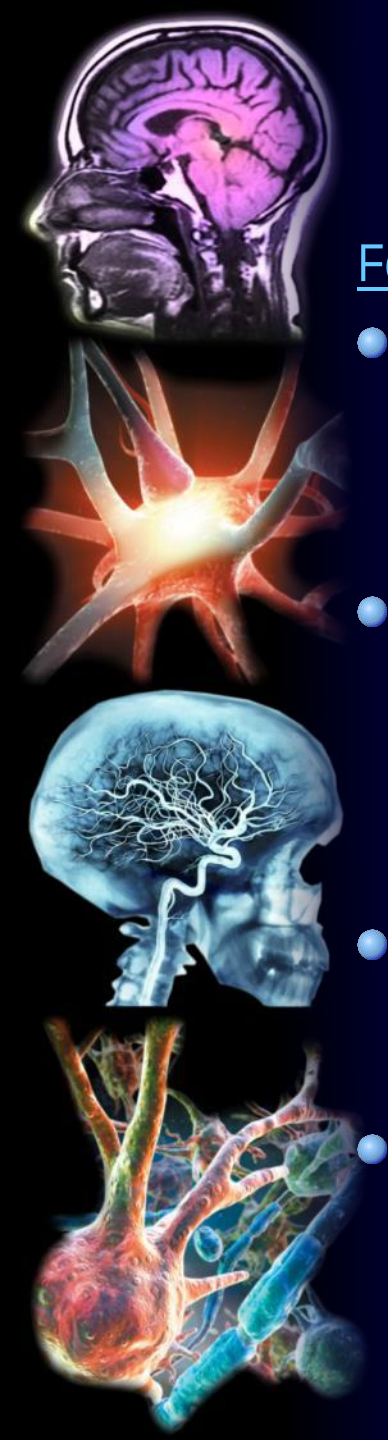
Ataxia

- Acqiuired general: a) Alcohol (axute intoxication + alcoholism = long-term excess intake), b) Vitamin deficiency (B1, B12, B6), c) Medications (side effects - barbiturates (phenobarbital), benzodiazepines (diazepam); antiepileptics (phenytoin), chemotherapy) d) Infections (usually viral – exanthems, COVID, HIV), e) Toxins (metal poisoning -lead, mercury, solvents, thinners) f) Thyroid problems g) Autoimmune diseases or paraneoplastic syndromes (abnormal immune response to cancer).

- Hereditary causes: genetic abnormalities of proteins in the cerebellum
- a) Autosomal dominant : spinocerebellar ataxia (progressive degenerative); episodic ataxia (brief episodes triggered by stress, sudden movement and startling, associated with muscle twitching, and weakness).

b) Autosomal recessive:

- Friedreich's ataxia (most common) – < 25 y. of age; difficulty in walking, arch deformities of the feet, deformities of spine (scoliosis), abnormal arm-trunk coordination, abnormal speech (slurred), weakness, and involuntary eye movements. Cause: damage to the cerebellum, spinal cord, peripheral nerves



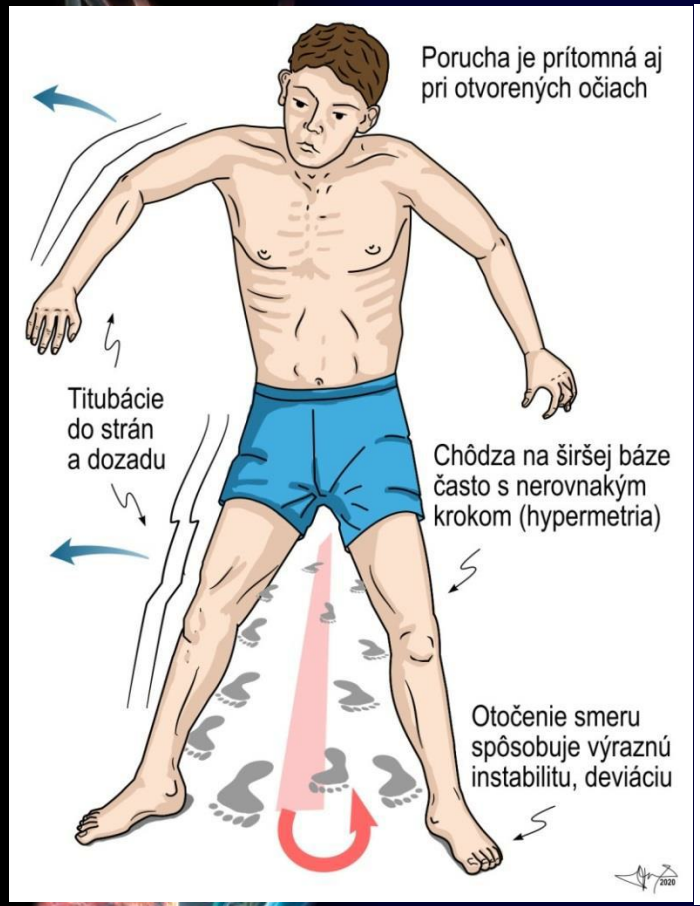
Ataxia

Forms:

- Cerebellar ataxia - most common form, Cause: damage to the cerebellum).Symptoms: "Drunk-like" gait, stumbling, and difficulty with fine motor tasks.Speech: "Scanning" speech—staccato-like, where the person breaks words into individual syllables.
- Sensory (spinal) ataxia - loss of "proprioception" (the sense of where your limbs are in space). Cause: Damage to the spinal cord or peripheral nerves (Vitamin B12 deficiency, advanced diabetes). Symptoms: „ High-Steppage Gait: Patients often "stomp" to increase the vibration and sensory feedback from the floor. They usually fail the Romberg Test.
- Vestibular Ataxia Cause: damage to inner ear or vestibular nerve. Symptoms: Vertigo, nausea, and a tendency to veer toward one side while walking.
- Frontal (apraxic) ataxia Cause: cortical degneneration- dementia; Symptoms: hesitating, unsure gait; leaning against the surrounding walls, furniture

Gait disturbances

Cerebellar ataxia



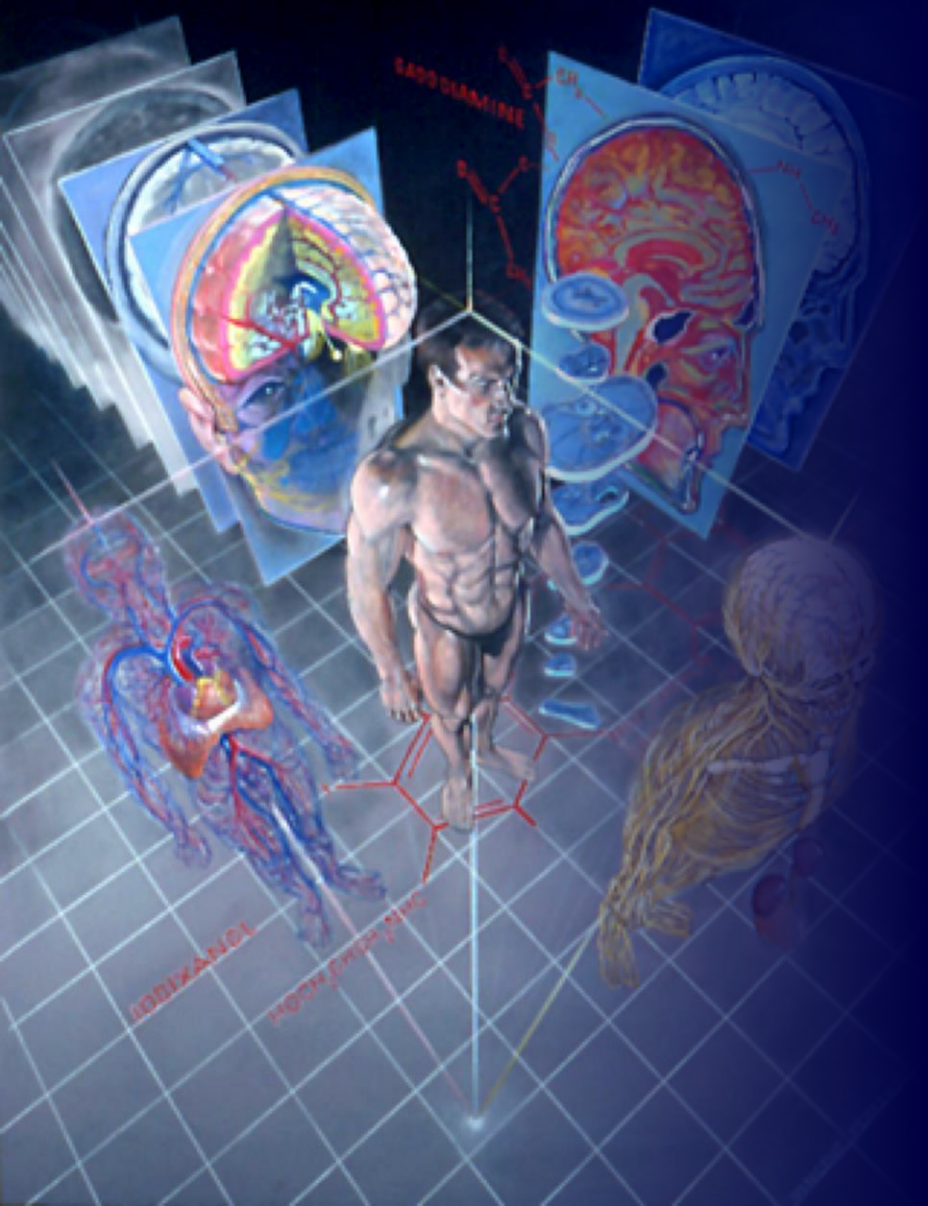
Wide base, titubations, turning back problem, open eyes

Vestibular ataxia

Tilted head, wide base, side deviation, open/ closed eyes

Spinal ataxia

Tilted head, wide base, side deviation, open/ closed eyes, Steppage Gait



3

PARALYSIS (PALSY)

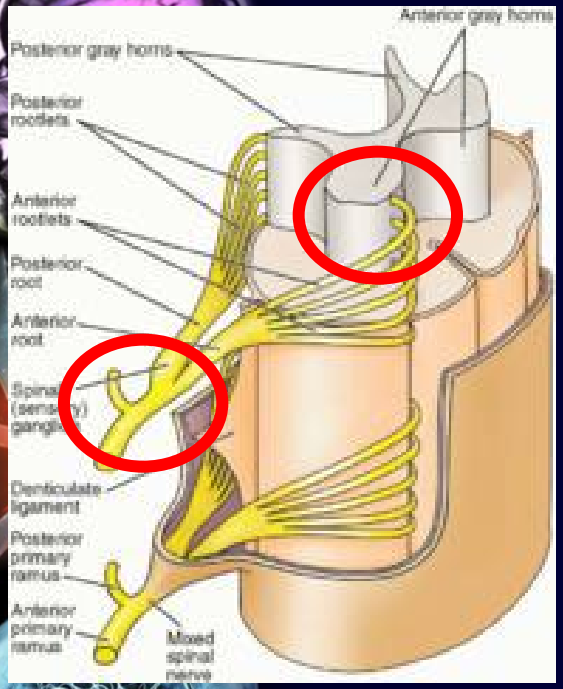
1. Lower motoneuron syndrome – peripheral palsy (weakness)

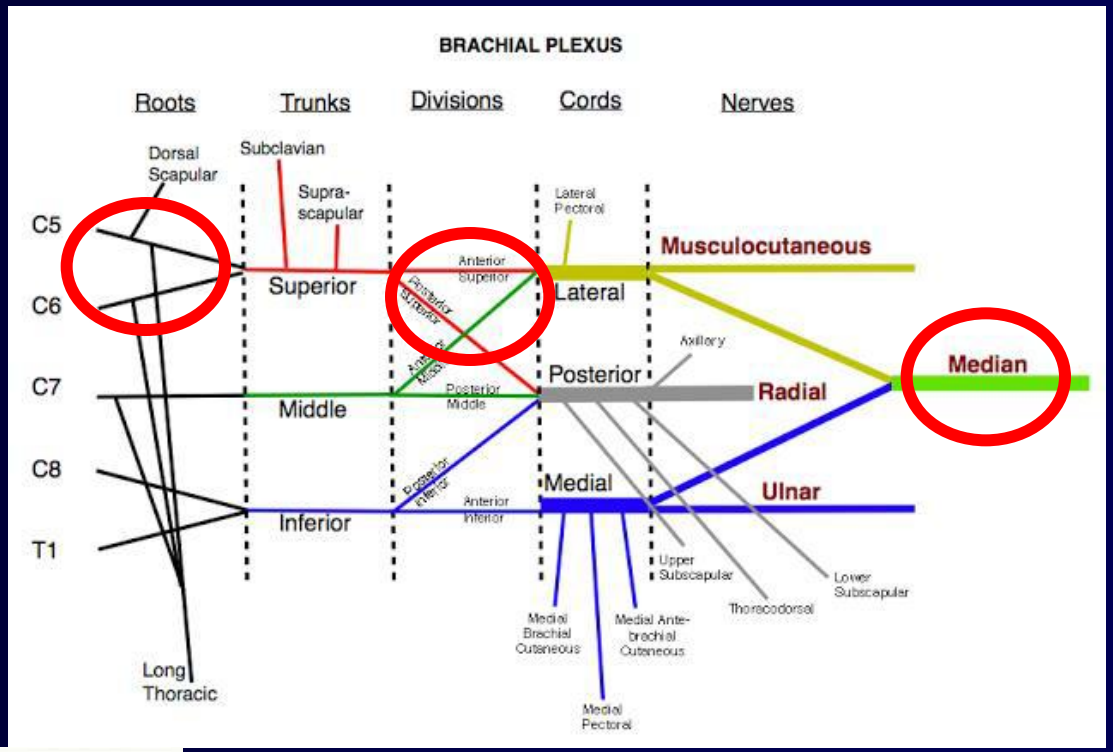
- Weakness (palsy) in one or more muscles, groups
  - watershed of nerve, plexus, root, anterior horn
- Hypotonia, atonia (flaccidity) - floppy
- Hyporeflexia, areflexia
- Muscle atrophy
- Fasciculations, fibrillations
  - mostly under motoneuronal damage (cord, brainstem)
- Spasms, cramps
  - in unaffected antagonistic muscles

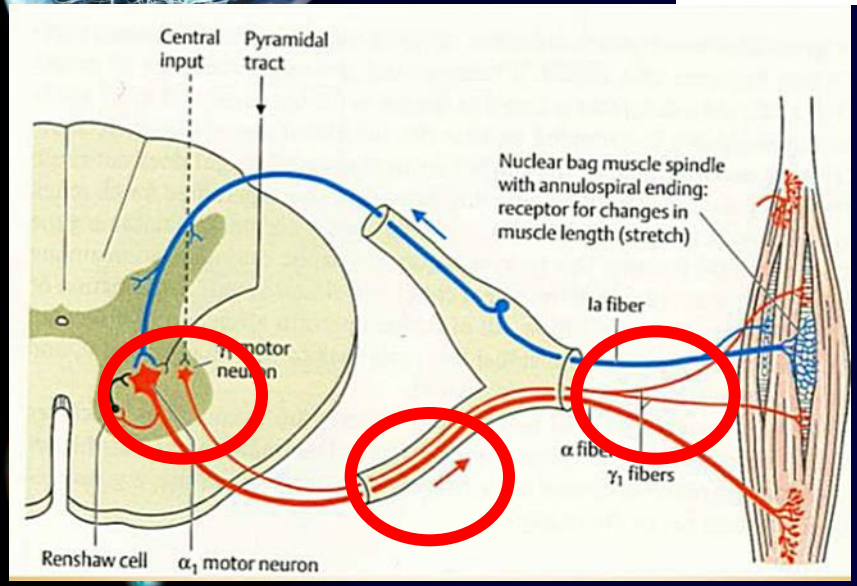
Causes:

- damage to the nerve, plexus,
- damage to the anterior horn of spinal cord (trauma, ischaemia) or ventral roots

Symptoms are homolateral to the site of damage



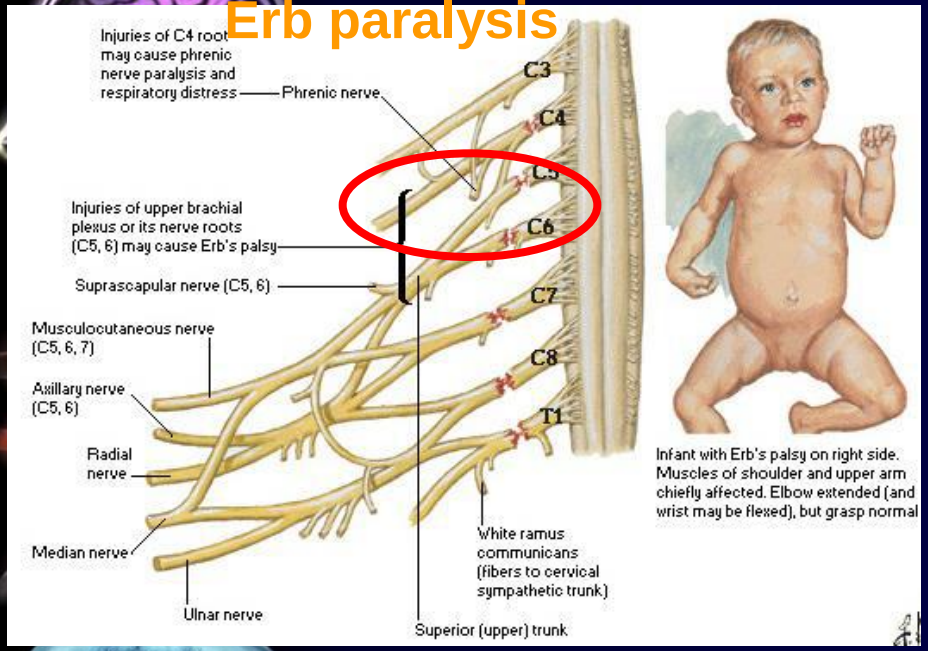


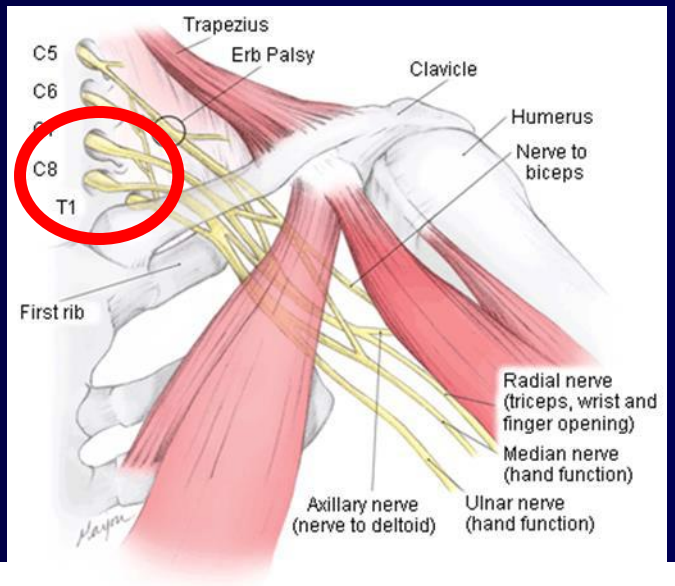


- Manifeztattion

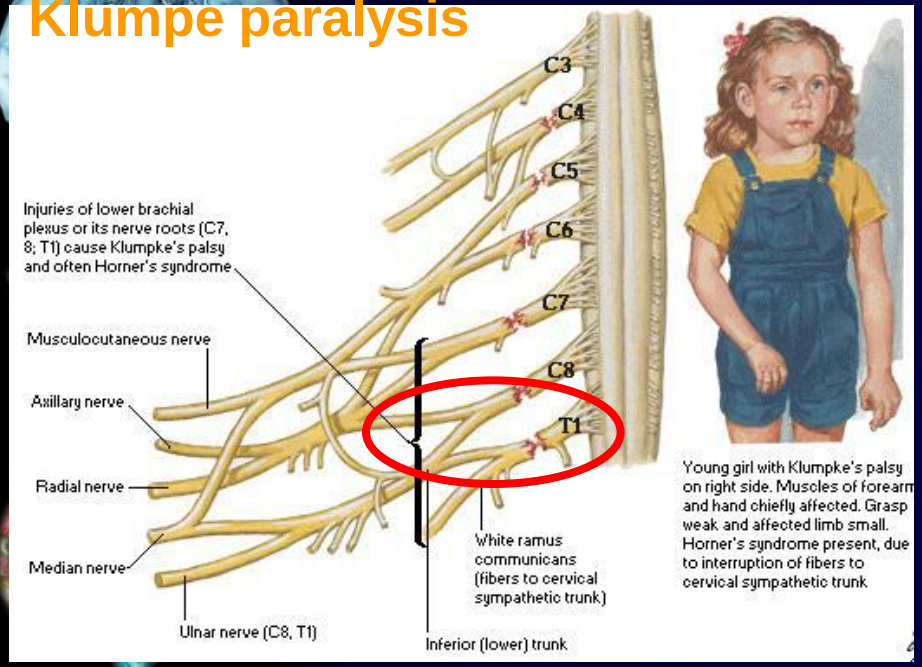
Damage to cervical roots and plexuses

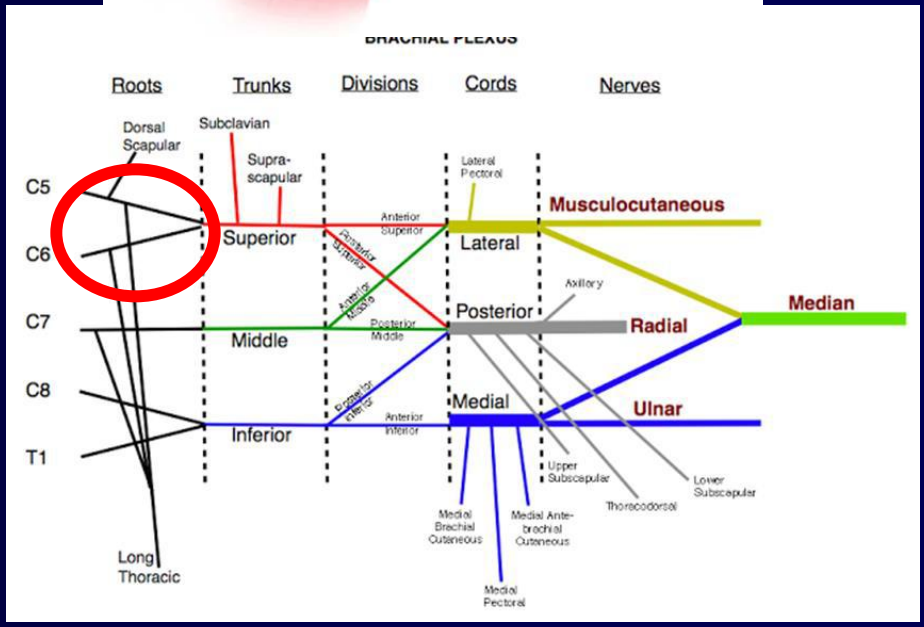
Erb paralysis





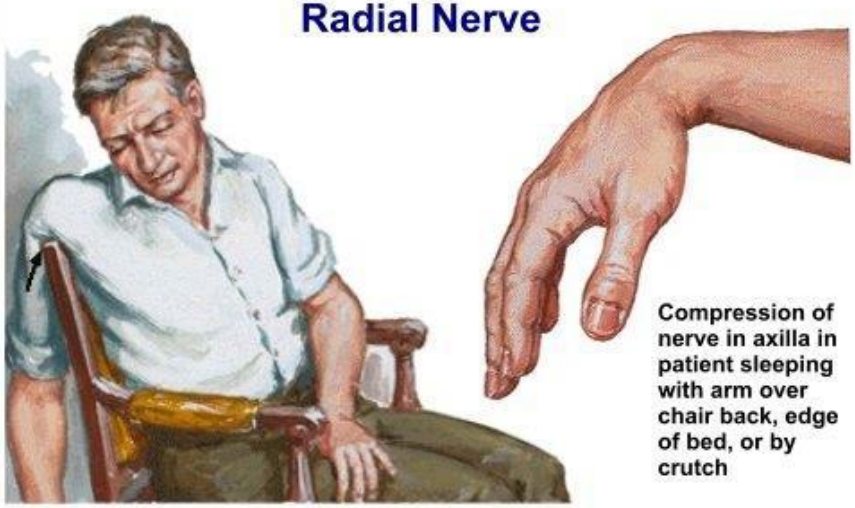
Klumpe paralysis





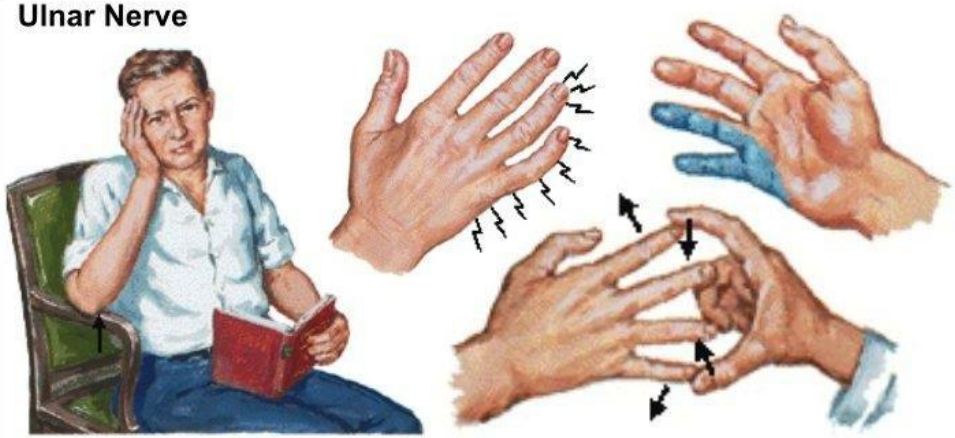
Upper extremity – nerve damage

Radial Nerve



Compression of nerve in axilla in patient sleeping with arm over chair back, edge of bed, or by crutch

Ulnar Nerve



Compression of nerve on hard surface (chair, desk, table, etc.)

Numbness, tingling in ulnar nerve area. Interosseous muscle wasting between tumb and index finger, abduction and adduction by interasseous muscles diminishes or los

Patient awakened by tingling and/or pain in thumb, index and middle fingers

Difficulty in rising arm to brush hair

Median Nerve

Atrophy of thenar muscles due to long-standing compression of median nerve

Gradual numbness of fingers while driving

Lower extremity – nerve damage

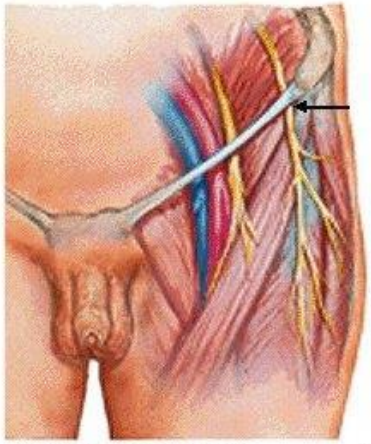
N. gluteus inferior

Difficulty in arising from chair is often an early complaint

Difficult stepping into bus

Difficulty in climbing stairs is often an early symptom due to weakness of pelvic girdle muscles

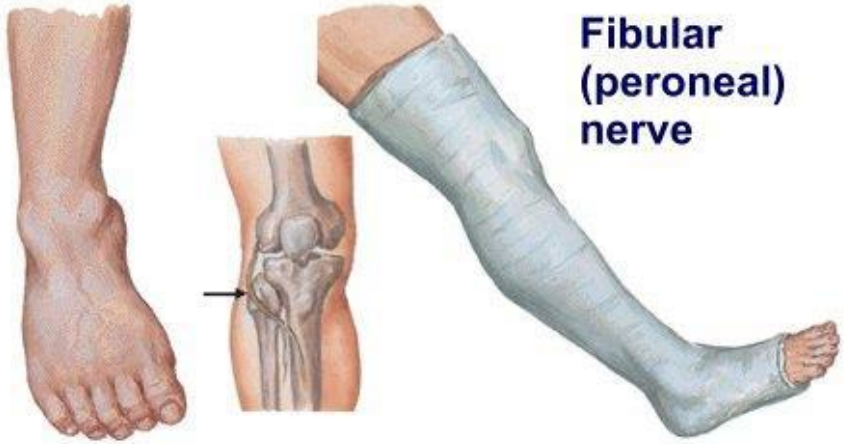
Lataral paralysis of n.femoralis



Entrapment of nerve under inguinal ligament

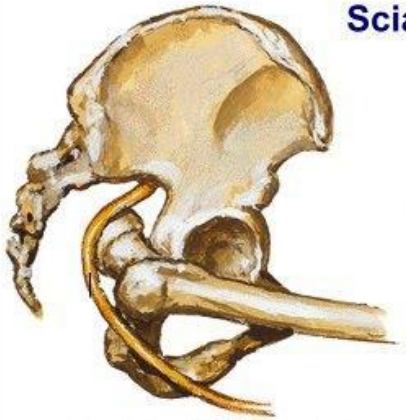
Numbness and dysesthesia in lateral thight meragia paraesthetica

Fibular (peroneal) nerve



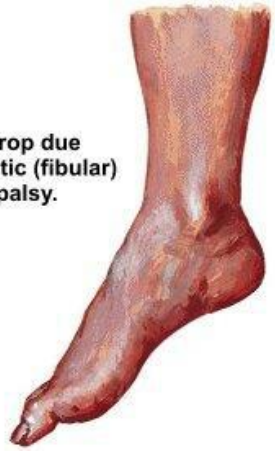
Compression of common fibular nerve over fibular head, e.g. sitting with legs crossed, or sleeping on side on hard suface

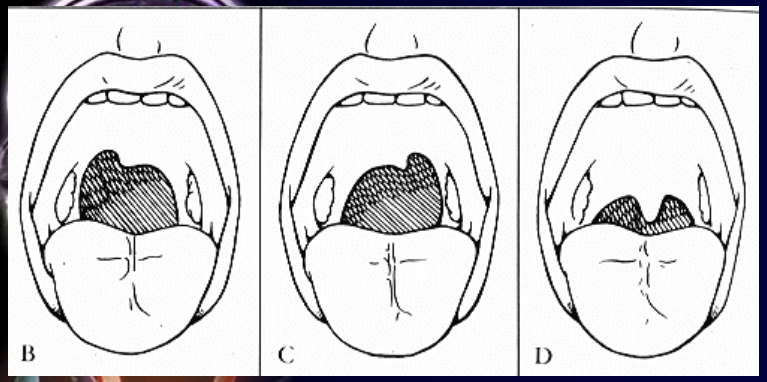
Sciatic Nerve



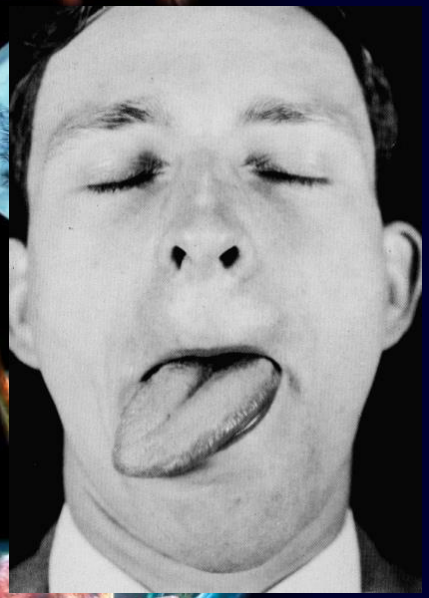
Posterior dislocation of hip. Femoral head may impinge on sciatic nerve, leading to palsy.

Foot drop due to sciatic (fibular) nerve palsy.

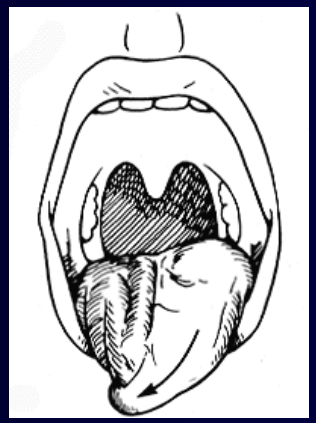


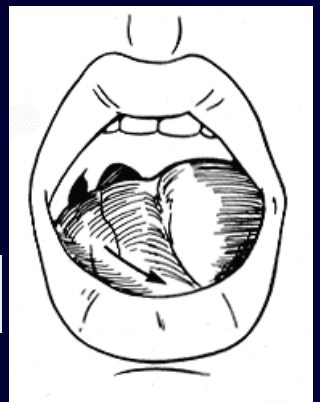


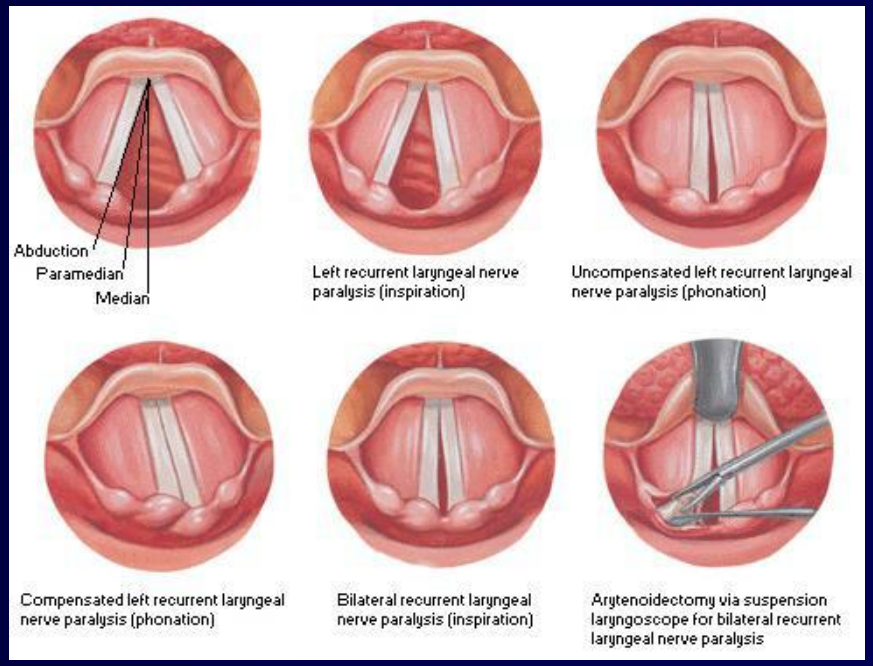
Soft palate drop
Nuclear palsy IX, Xn



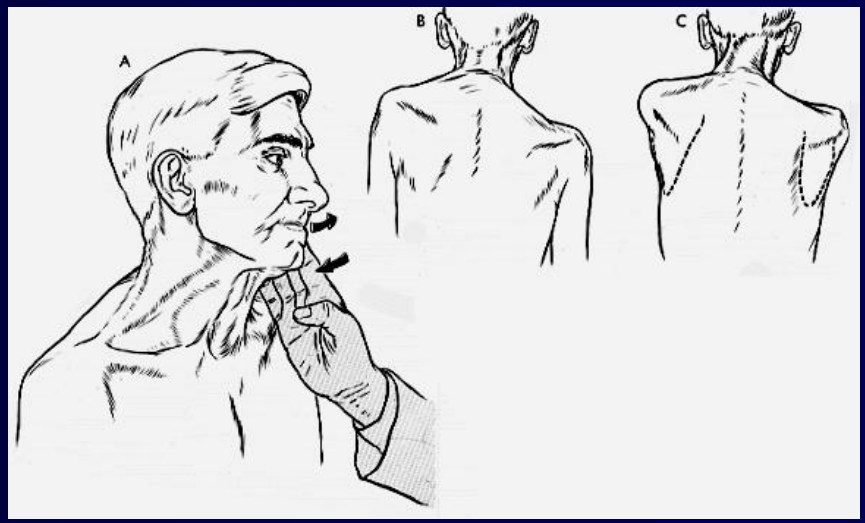
Nuclear palsy of XIIn







Vocal cord paralysis



Nuklear palsy of XIn (acessorius n.)

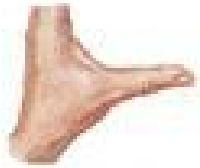
Neurology of cervical radiculopathies (herniation syndromes)

Herniated disc compresses nerve root

Myelogram (AP view) shows prominent extradural defect at C6-7

Level	Motor signs (weakness)	Reflex signs	Sensory loss
C5		0	
C6			
C7			
C8			

Spurling's maneuver. Hyperextension of neck and rotation away from side of lesion cause radicular pain in arm and neck

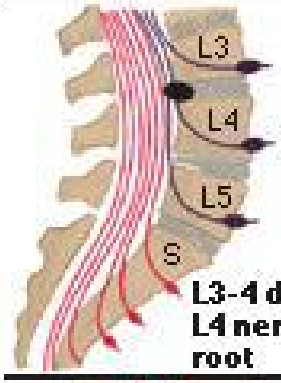
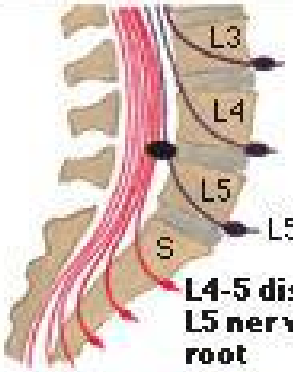
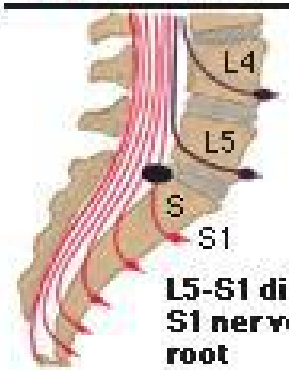
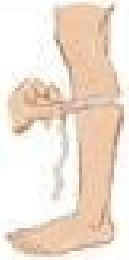


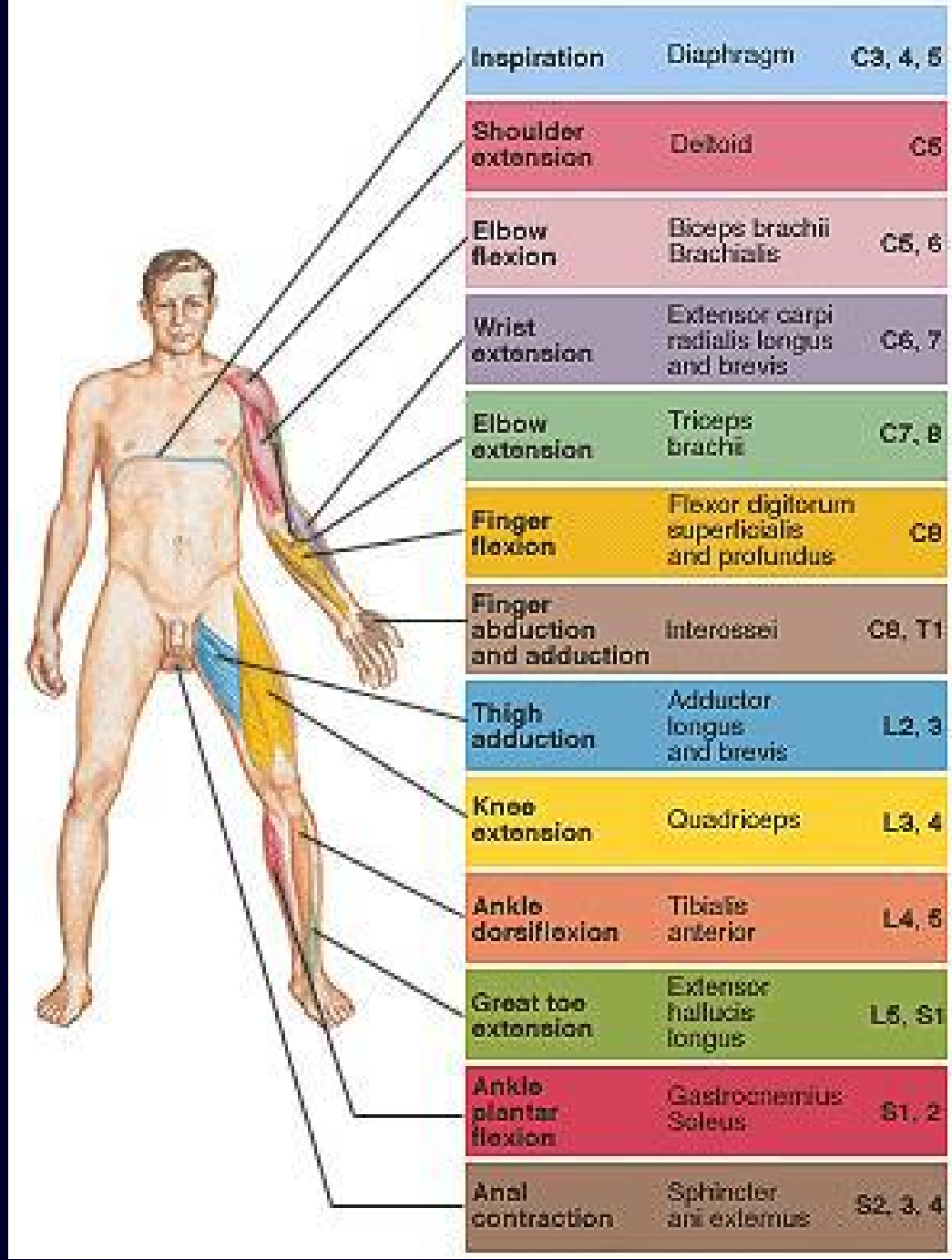
Thumb extension. Extensor pollicis longus. C6

Finger extension. Extensor digitorum. C7

Finger flexion. Flexor digitorum superficialis and profundus. C8

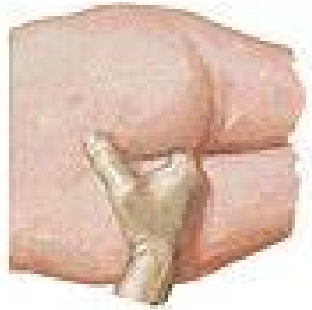
Neurology of lumbar radiculopathies (herniation syndromes)

Level of herniation	Pain	Numbness	Weakness	Atrophy	Reflexes
 **L3-4 disc; L4 nerve root**	 Lower back, hip, posterolateral thigh, anterior leg	 Anteromedial thigh and knee	 Quadriceps	 Quadriceps	 Knee jerk diminished
 **L4-5 disc; L5 nerve root**	Above sacroiliac joint, hip, lateral thigh, and leg 	 Lateral leg, first three toes	 Dorsiflexion of great toe and foot; difficulty walking on heels; foot drop may occur	Minor or nonspecific	Changes uncommon in knee and ankle jerks; posterior tibial reflex diminished or absent
 **L5-S1 disc; S1 nerve root**	Over sacroiliac joint, hip, posterolateral thigh, and leg to heel 	 Back of calf, lateral heel, foot and toe	Plantar flexion of foot and great toe may be affected; difficulty walking on toes	 Gastrocnemius and soleus	 Ankle jerk diminished or absent



Inspiration	Diaphragm	C3, 4, 5
Shoulder extension	Deltoid	C5
Elbow flexion	Biceps brachii Brachialis	C5, 6
Wrist extension	Extensor carpi radialis longus and brevis	C6, 7
Elbow extension	Triceps brachii	C7, 8
Finger flexion	Flexor digitorum superficialis and profundus	C8
Finger abduction and adduction	Interossei	C8, T1
Thigh adduction	Adductor longus and brevis	L2, 3
Knee extension	Quadriceps	L3, 4
Ankle dorsiflexion	Tibialis anterior	L4, 5
Great toe extension	Extensor hallucis longus	L5, S1
Ankle plantar flexion	Gastrocnemius Soleus	S1, 2
Anal contraction	Sphincter ani externus	S2, 3, 4

Level	Motor signs (weakness)
C5	Deltoid
C6	Biceps brachii
C7	Triceps brachii
C8	Interossei



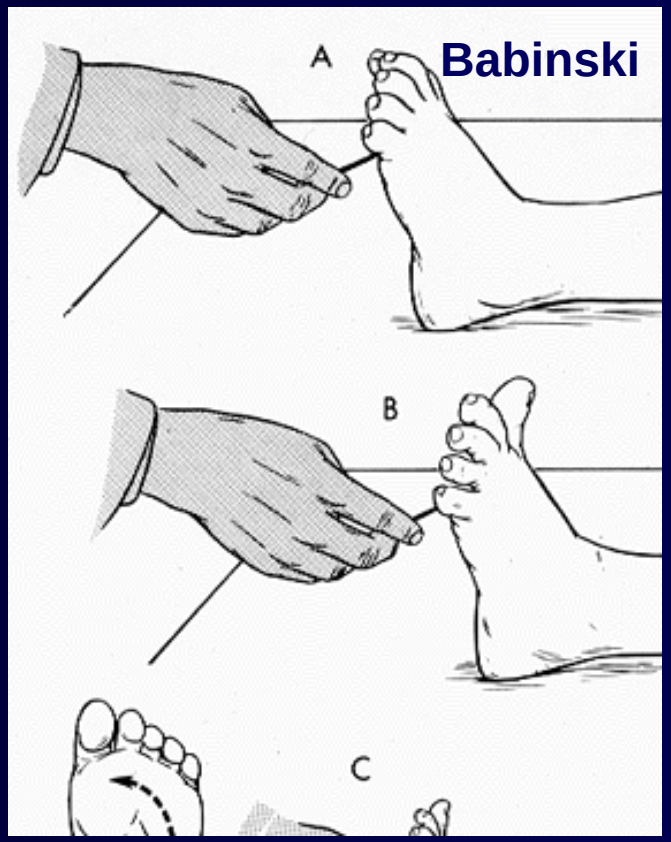
2. Upper motoneuron syndrome – central paralysis (weakness)

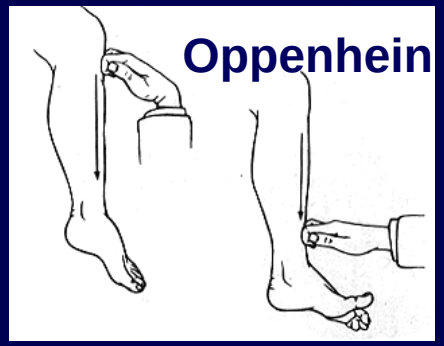
A. Acute stage, B. Chronic stage – after 7-14 d.

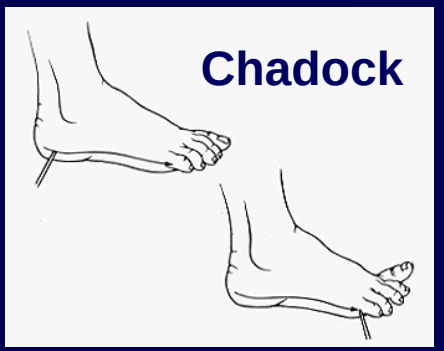
- Hemiparesis(-plegia), paraplegia, monoparesis
  - Loss of fast, delicate, (flexor) movements
  - Face (mouth, tongue), hand (arm swing, grasp, fingers, internal rotat.), foot – external, dorsal flex.)
- Hypertonia – spasticity (clasp-knife)
  - acutely little evident, mostly late sign
  - always occurs in groups of muscles, not individual m.
- Hypereflexia – brisk UE a LE reflexes
- Spasms in affected muscles
- Pathological reflexes – Babinski - extensor plantar response
  - main finding in acute stage compared to LMNS

Causes: damage to motor cortex and along the pyramidal pathway (capsula interna, brainstem, spinal cord)

Symptoms are contralateral to damage

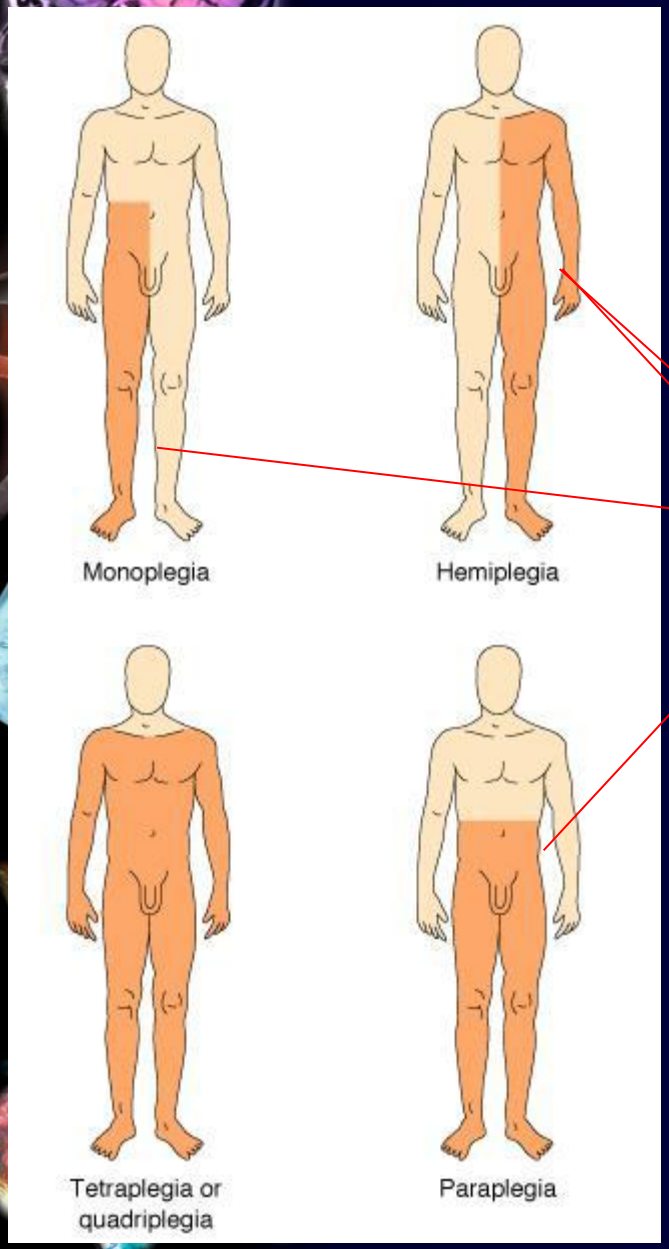

Babinski
A
B
C


Oppenhein


Chadock

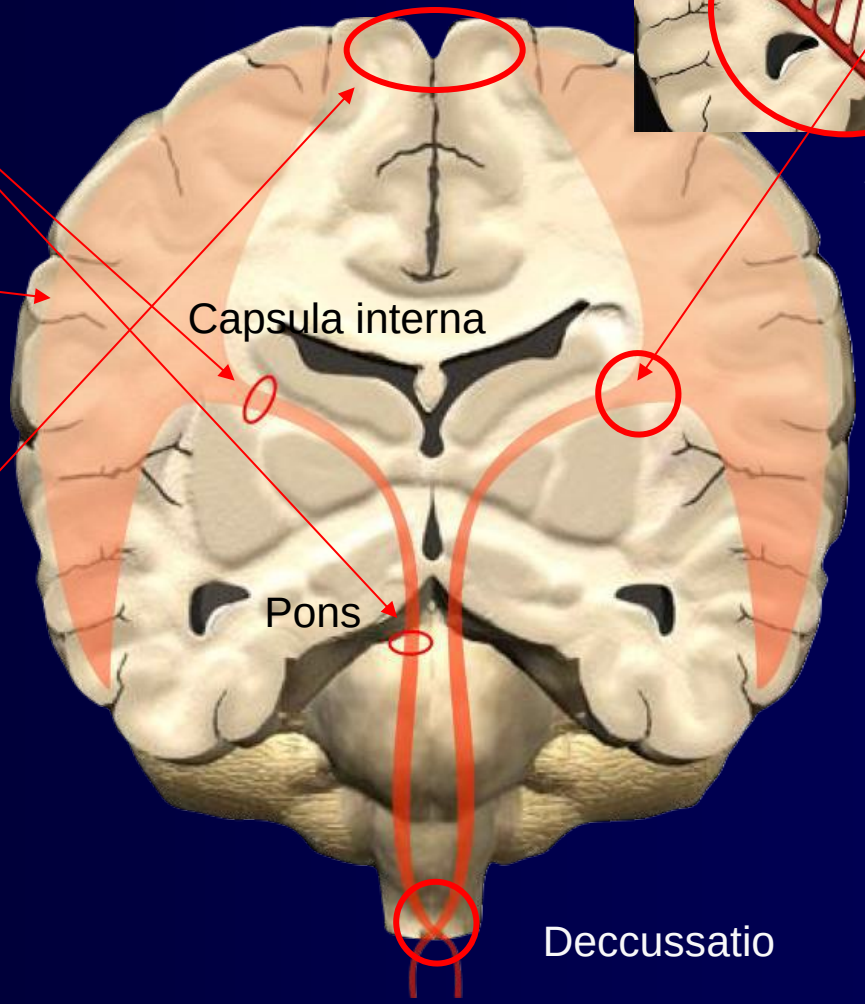

Gordon

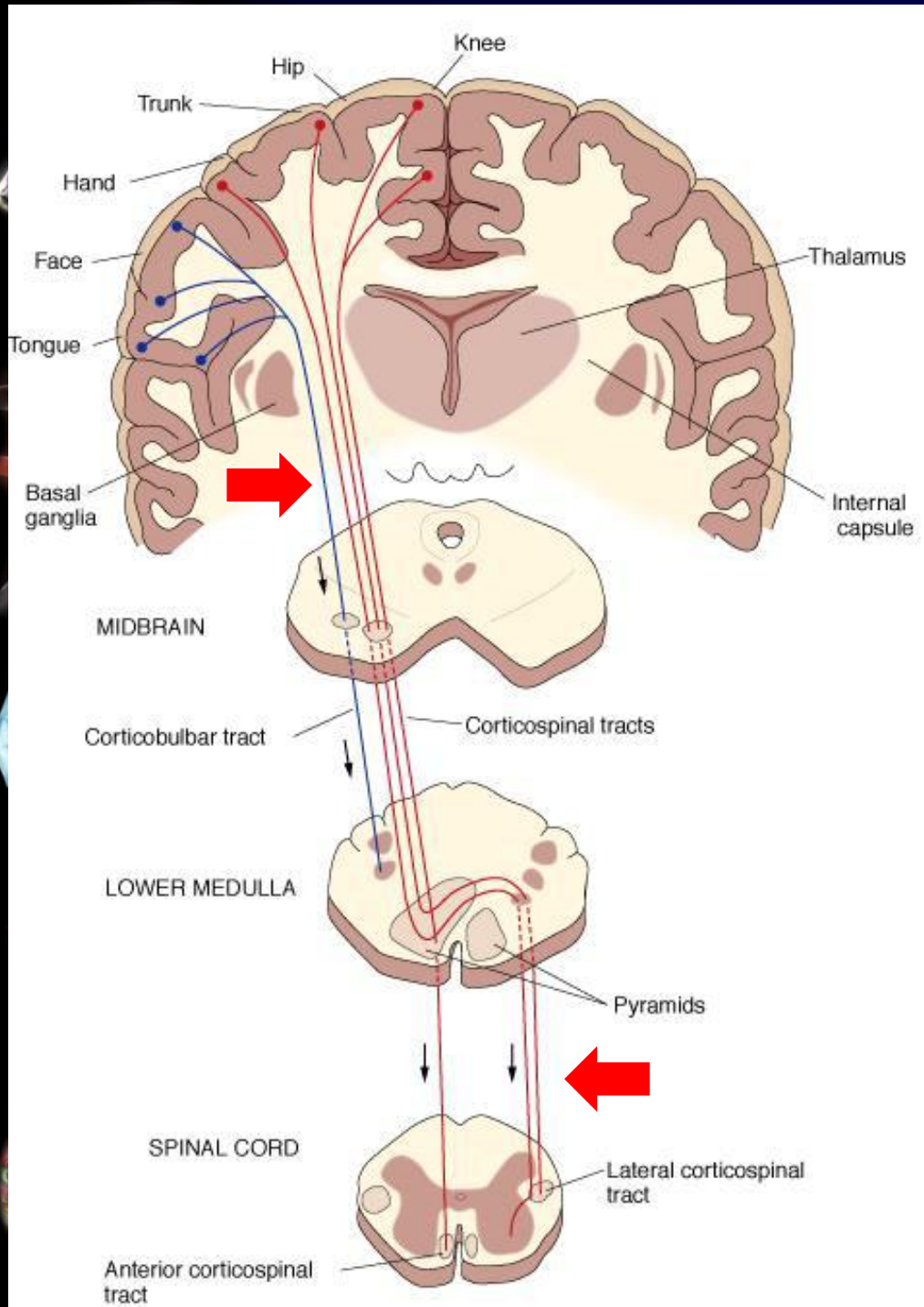
UMNS manifestation

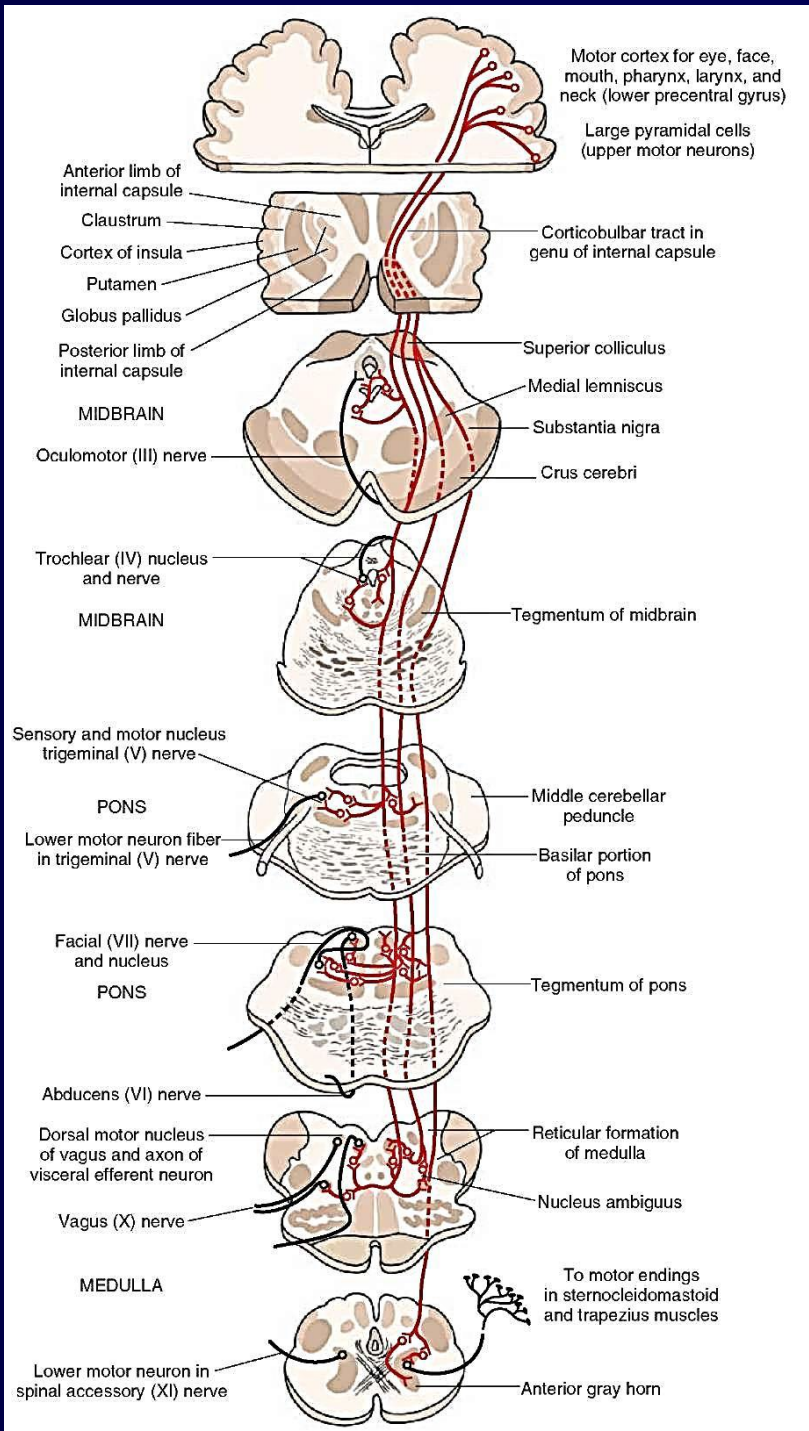


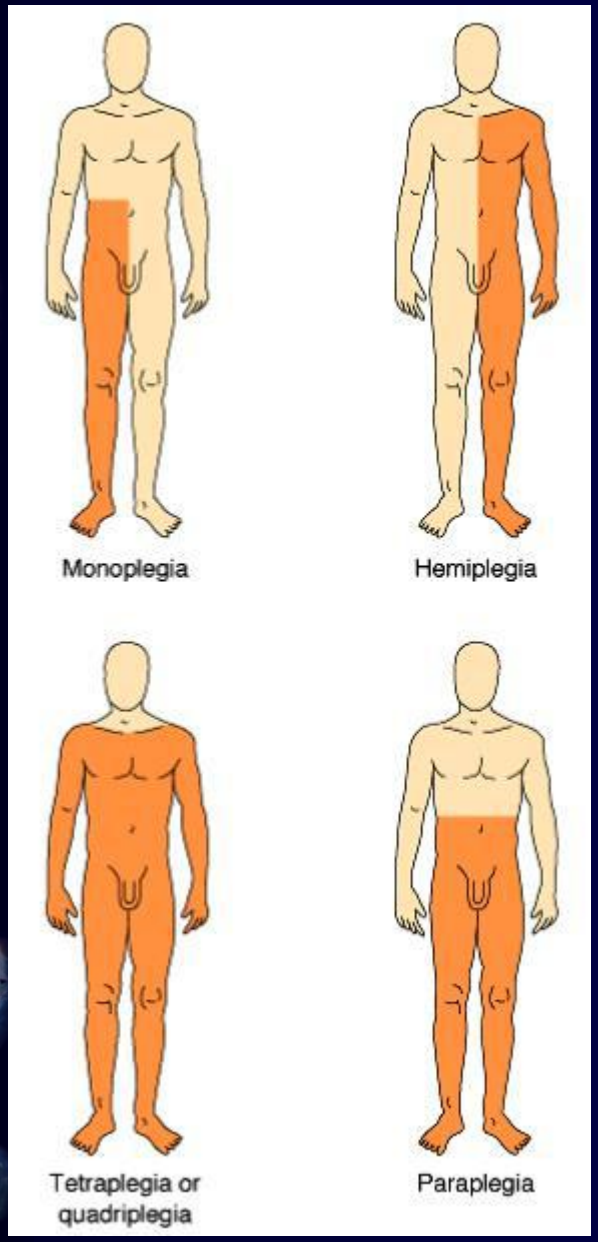
Water-shed of anterior cerebral artery

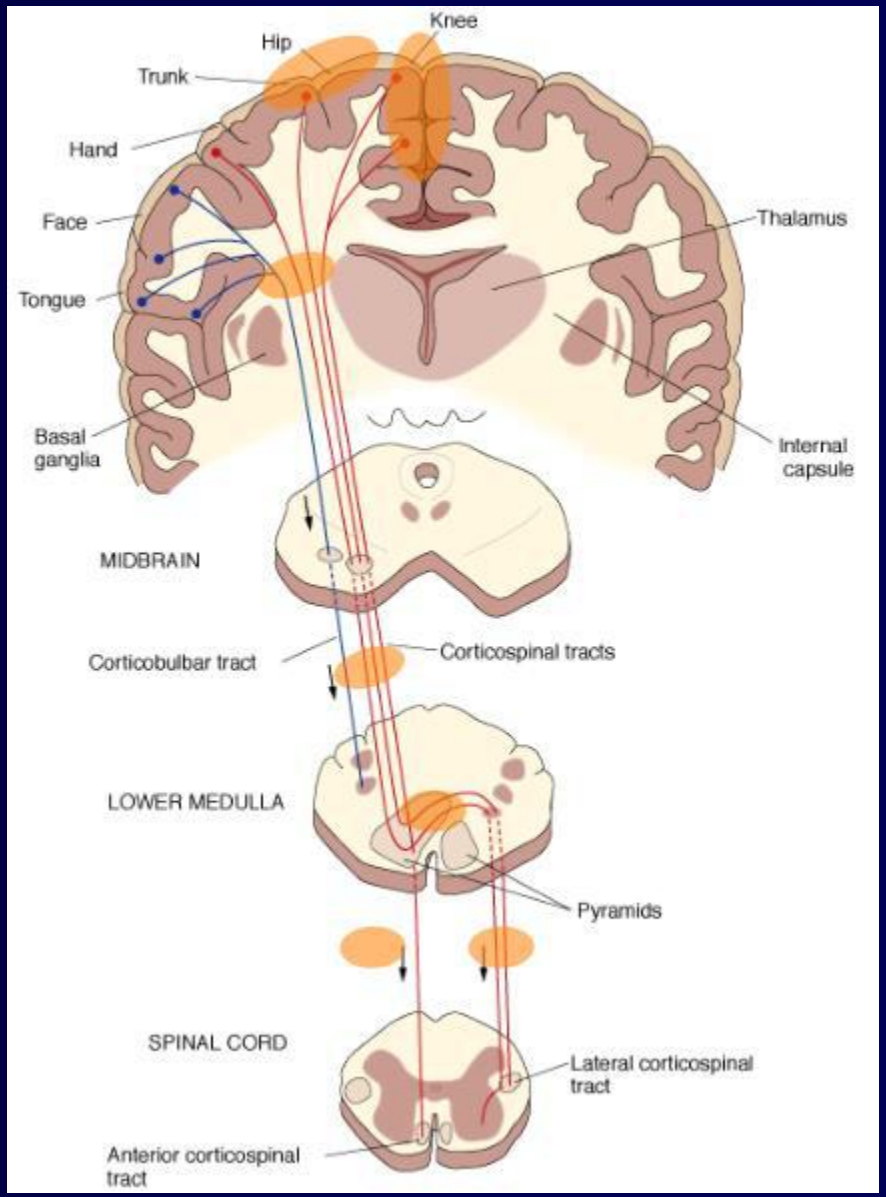
Water-shed of medialcerebral artery



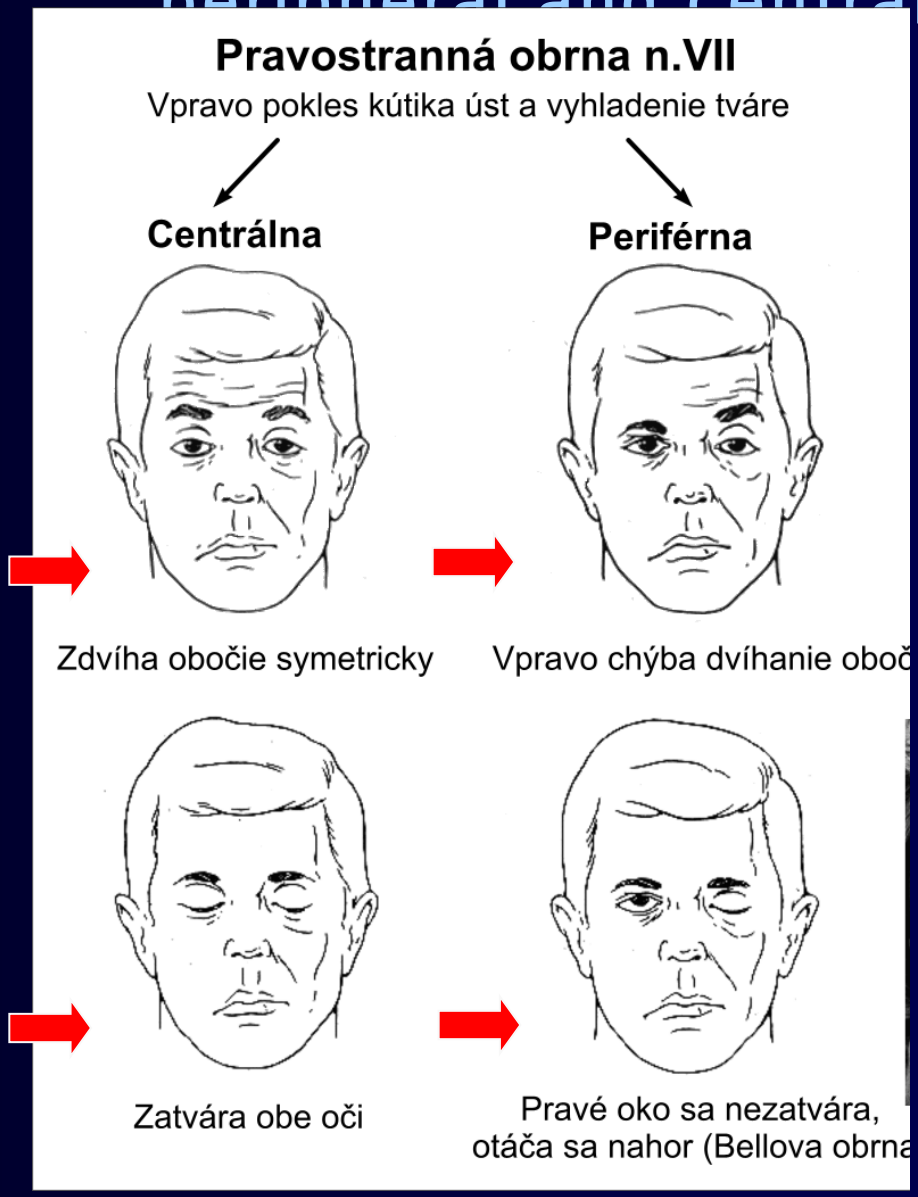

Knee
Hip
Trunk
Hand
Face
Tongue
Thalamus
Basal ganglia
Internal capsule
MIDBRAIN
Corticobulbar tract
Corticospinal tracts
LOWER MEDULLA
Pyramids
SPINAL CORD
Lateral corticospinal tract
Anterior corticospinal tract


Motor cortex for eye, face, mouth, pharynx, larynx, and neck (lower precentral gyrus)
Large pyramidal cells (upper motor neurons)
Anterior limb of internal capsule
Claustrum
Cortex of insula
Putamen
Globus pallidus
Posterior limb of internal capsule
Corticobulbar tract in genu of internal capsule
Superior colliculus
Medial lemniscus
MIDBRAIN
Substantia nigra
Oculomotor (III) nerve
Crus cerebri
Trochlear (IV) nucleus and nerve
MIDBRAIN
Tegmentum of midbrain
Sensory and motor nucleus trigeminal (V) nerve
PONS
Middle cerebellar peduncle
Lower motor neuron fiber in trigeminal (V) nerve
Basilar portion of pons
Facial (VII) nerve and nucleus
PONS
Tegmentum of pons
Abducens (VI) nerve
Dorsal motor nucleus of vagus and axon of visceral efferent neuron
Reticular formation of medulla
Nucleus ambiguus
Vagus (X) nerve
MEDULLA
To motor endings in sternocleidomastoid and trapezius muscles
Lower motor neuron in spinal accessory (XI) nerve
Anterior gray horn


Monoplegia
Hemiplegia
Tetraplegia or quadriplegia
Paraplegia

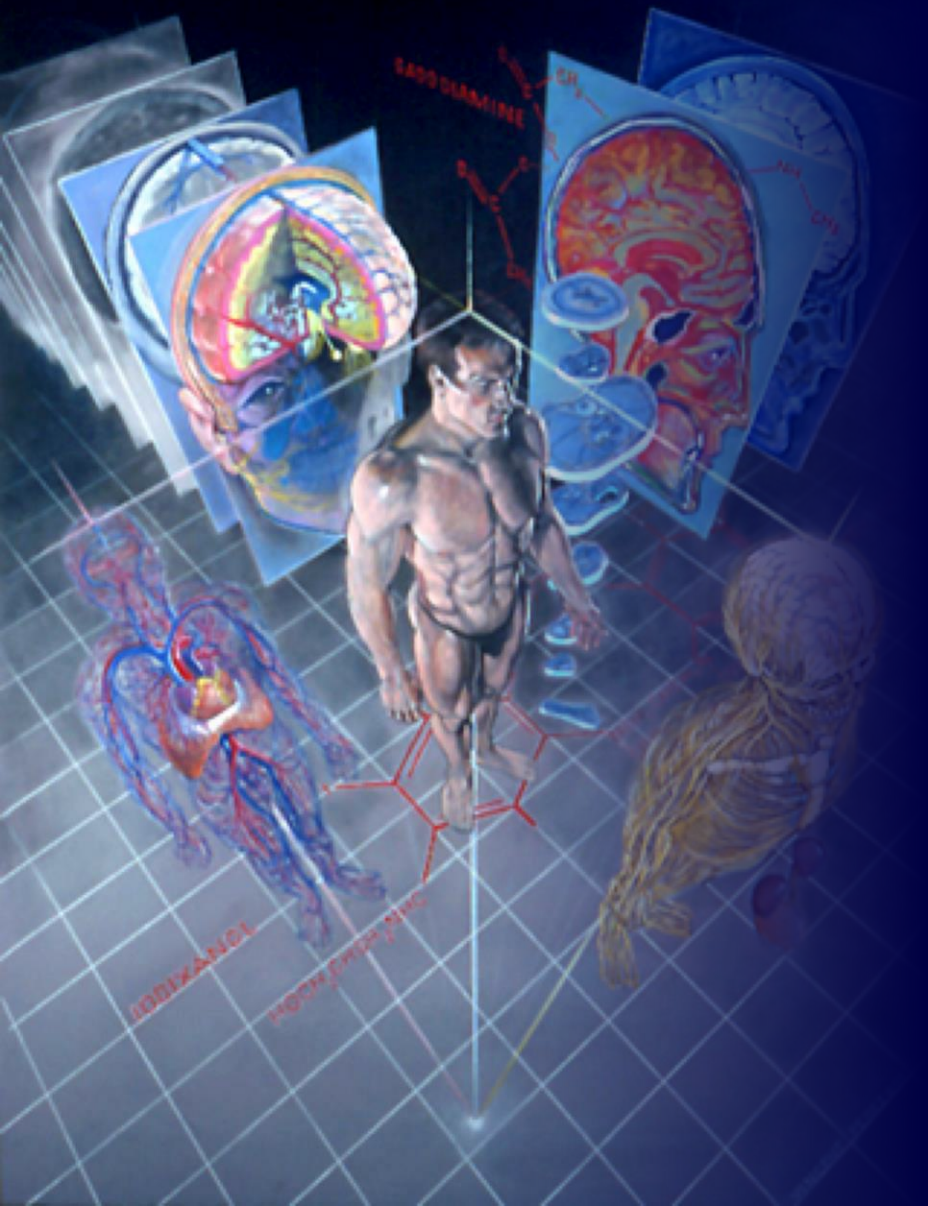

Knee
Hip
Trunk
Hand
Face
Tongue
Basal ganglia
Thalamus
Internal capsule
MIDBRAIN
Corticobulbar tract
Corticospinal tracts
LOWER MEDULLA
Pyramids
SPINAL CORD
Lateral corticospinal tract
Anterior corticospinal tract

Facial nerve paresis – comparison of peripheral and central laesion



UMNS vs. LMNS

UMNS	LMNS
Corticospinal (bulbar) + subcorticospinal tr.	Mononeurons + axons
Muscles are normal	Muscles atrophy
Hypertonia (spasticity)	Atonia (flaccidity)
Reflexes are brisk	Weak reflexes
Spasms, cramps in affected muscles	Spasms, contractures in unaffected muscles
No irritation signs	Irritation signs – fasciculations
Pathological reflexes	No pathological reflexes

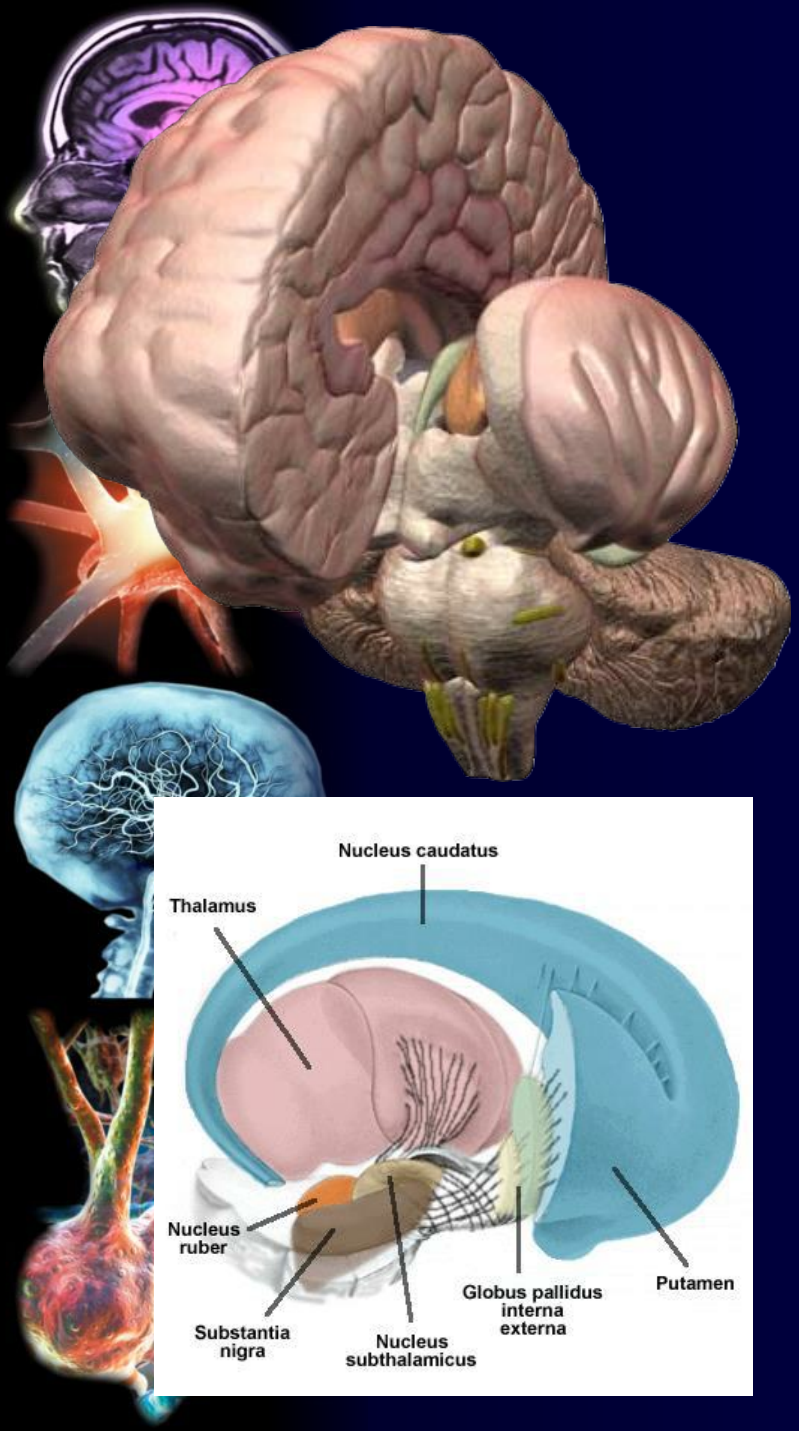


4

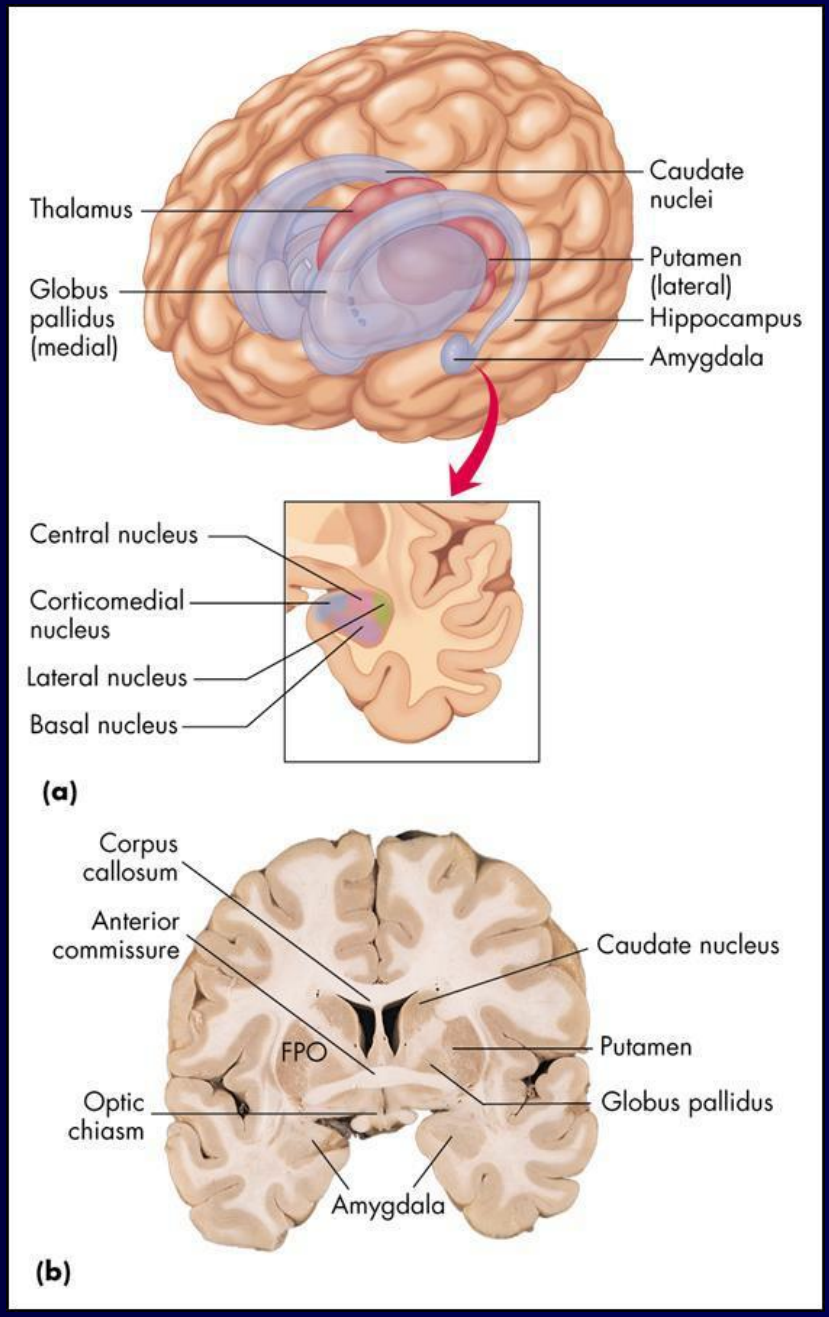
EXTRAPYRAMIDAL DISODERS (Basal ganglia) (Cerebellum)

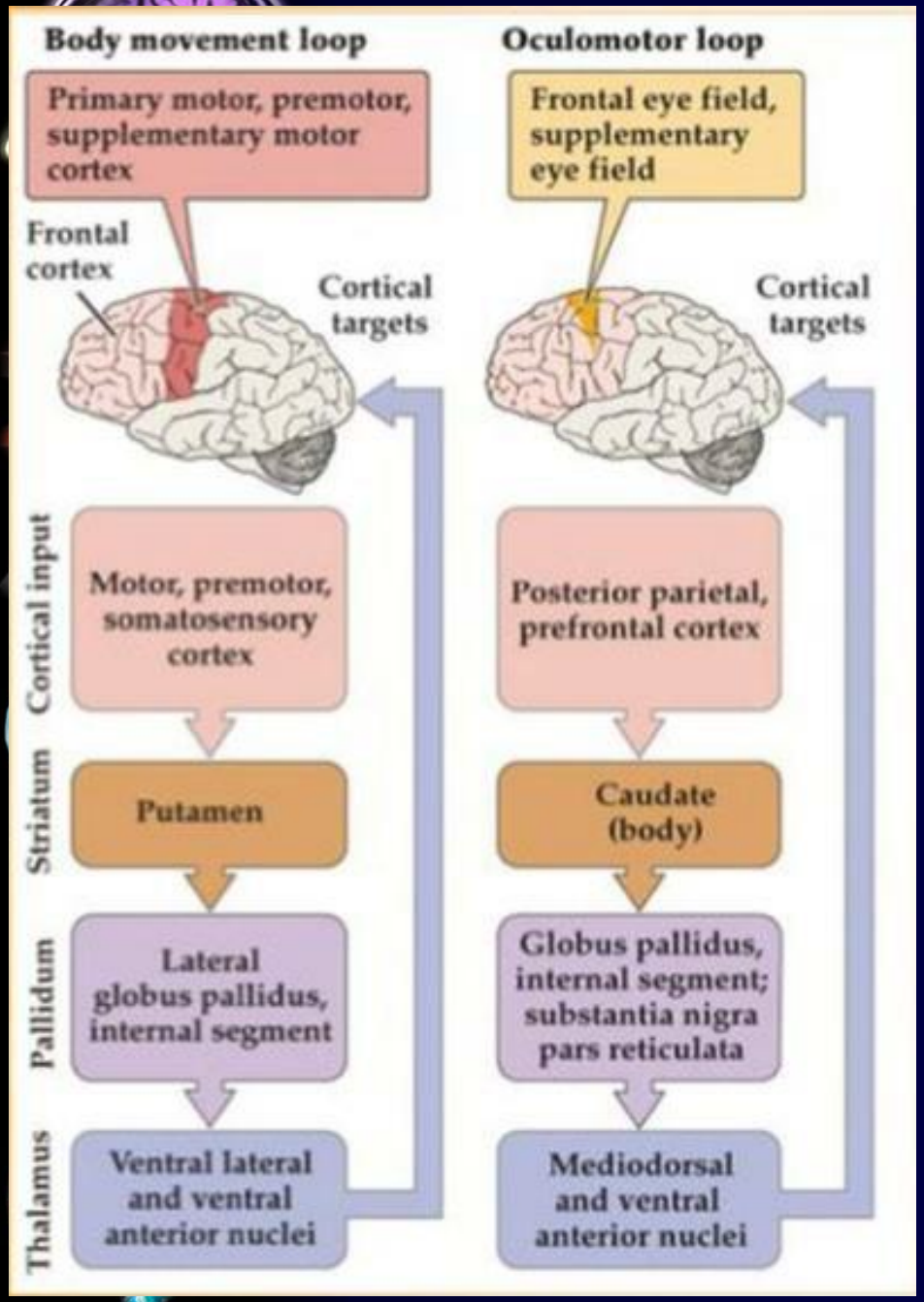
BASAL GANGLIA DISORDERS

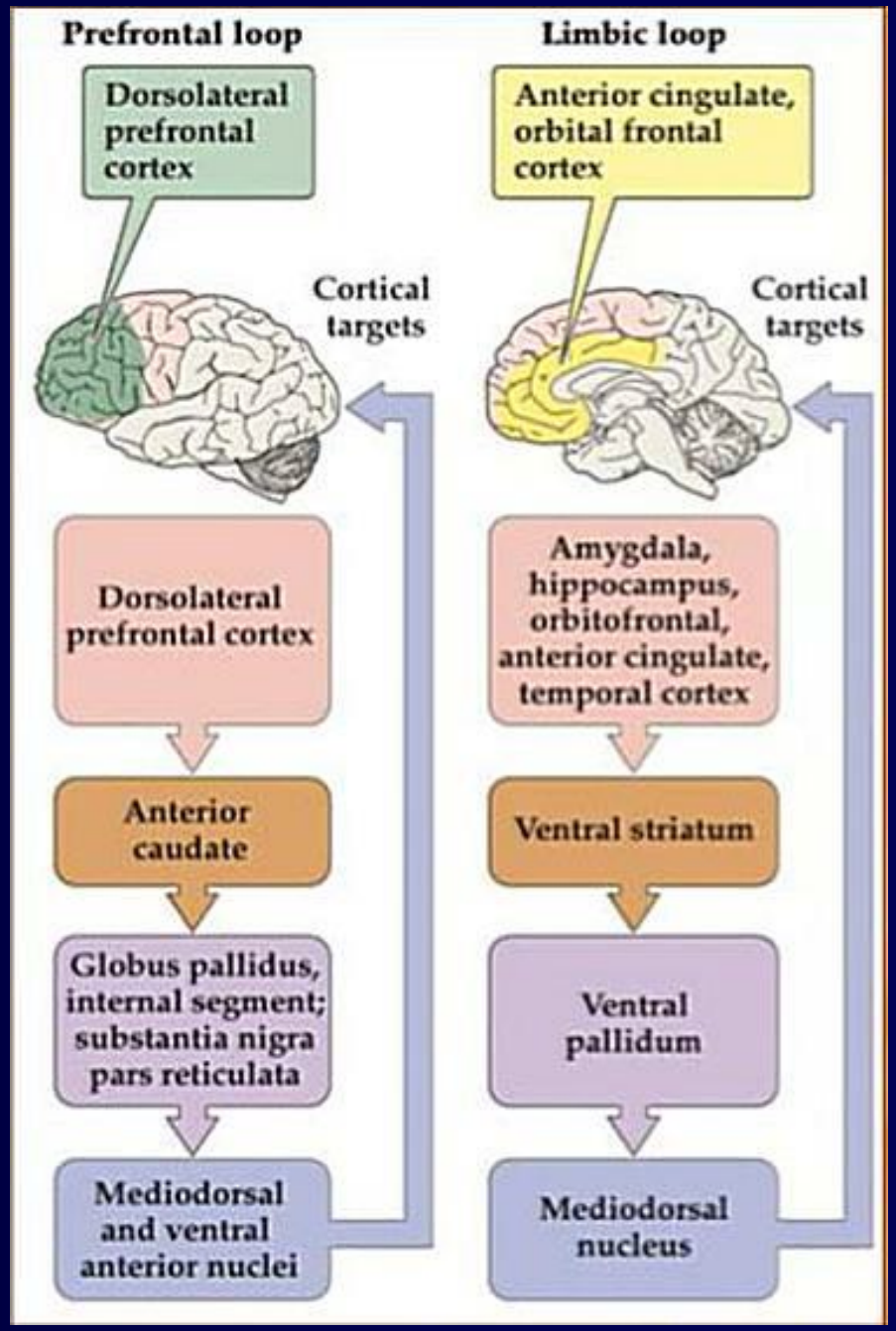
- Structures:
  - Caudate nucl., putamen, globus pallidus, substantia nigra, subthalamic nucl., red nucleus, nucl. campi Forell
- Function of BG:
  - Initiation of movement, Muscle tone
  - Complex motor programs, implicit memory
- Clinical manifestations:
  - No palsy or weakness, problem is rather in amount, fluency, smoothness, timing
  - Goal-directed movements are interrupted, uncoordinated, slow or infiltrated by escaped movement intrusions
  - Muscle tone is always affected – hypotonia, dystonia, hypertonia
  - Difficulties are ameliorated at rest, omitted in sleep, perpetuated by motion tasks, worsened by good or bad emotional excitement



Nucleus caudatus
Thalamus
Nucleus ruber
Substantia nigra
Nucleus subthalamicus
Globus pallidus interna externa
Putamen

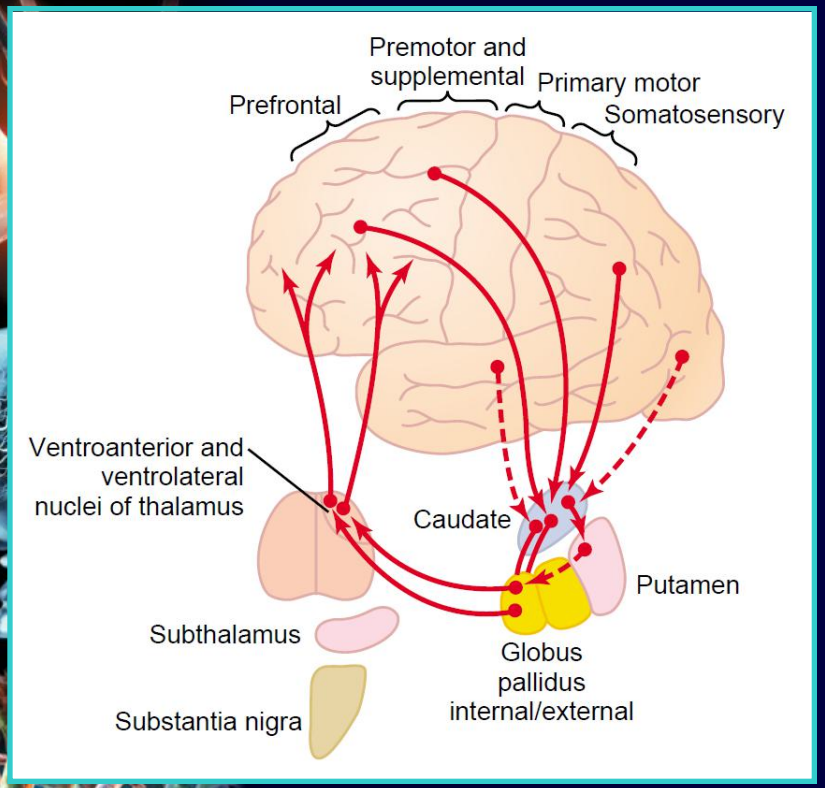

Thalamus
Caudate nuclei
Globus pallidus (medial)
Putamen (lateral)
Hippocampus
Amygdala
Central nucleus
Corticomedial nucleus
Lateral nucleus
Basal nucleus
(a)
Corpus callosum
Anterior commissure
Caudate nucleus
FPO
Putamen
Optic chiasm
Globus pallidus
Amygdala
(b)


Body movement loop
Primary motor, premotor, supplementary motor cortex
Frontal cortex
Cortical targets
Cortical input
Motor, premotor, somatosensory cortex
Striatum
Putamen
Pallidum
Lateral globus pallidus, internal segment
Thalamus
Ventral lateral and ventral anterior nuclei
Oculomotor loop
Frontal eye field, supplementary eye field
Cortical targets
Posterior parietal, prefrontal cortex
Caudate (body)
Globus pallidus, internal segment; substantia nigra pars reticulata
Mediodorsal and ventral anterior nuclei


Prefrontal loop
Dorsolateral prefrontal cortex
Cortical targets
Dorsolateral prefrontal cortex
Anterior caudate
Globus pallidus, internal segment; substantia nigra pars reticulata
Mediodorsal and ventral anterior nuclei
Limbic loop
Anterior cingulate, orbital frontal cortex
Cortical targets
Amygdala, hippocampus, orbitofrontal, anterior cingulate, temporal cortex
Ventral striatum
Ventral pallidum
Mediodorsal nucleus

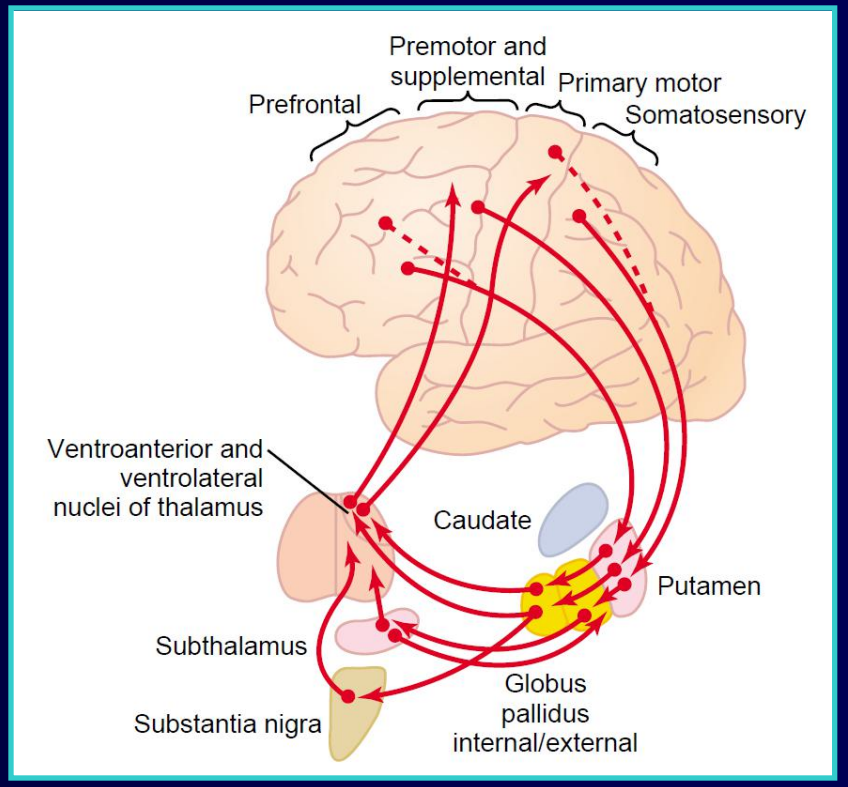
Basal ganglia circuits

Body movement loop1



Caudate circuit for cognitive planning of sequential and parallel patterns to achieve specific conscious goals.

Body prefrontal movement loop2



Putamen circuit for subconscious execution of learned patterns of movement.

BASAL GANGLIA DISORDERS

- HYPERKINETIC - DYSTONIC MOVEMENT DISORDERS
  - TREMOR - a rhythmical predictable oscillation of a body part
  - DYSTONIA - an involuntary muscle contraction causing a sustained twisted or abnormal posture
  - MYOCLONUS - a lightening like jerk of a body part.
  - STEREOTYPY - any patterned, stereotypic movement.
  - TICS - a stereotypic or patterned movement that is frequently preceded by an urge to need to move, transient suppressibility, and post movement relief.
  - CHOREA - random, purposeless, fleeting movements, spreading from one body part to another.
  - HEMIBALISMUS - A high amplitude flailing of the limbs on one side of the body.
- HYPOKINETIC - HYPERTONIC MOVEMENT DISORDERS
  - PARKINSONISM
  - Stiff Man Syndrome, Akinetic mutism
  - Psychomotor retardation
- Biochemistry
  - Ach> dopamine -> hypokinesia
  - Dopamine> Ach -> hyperkinesia

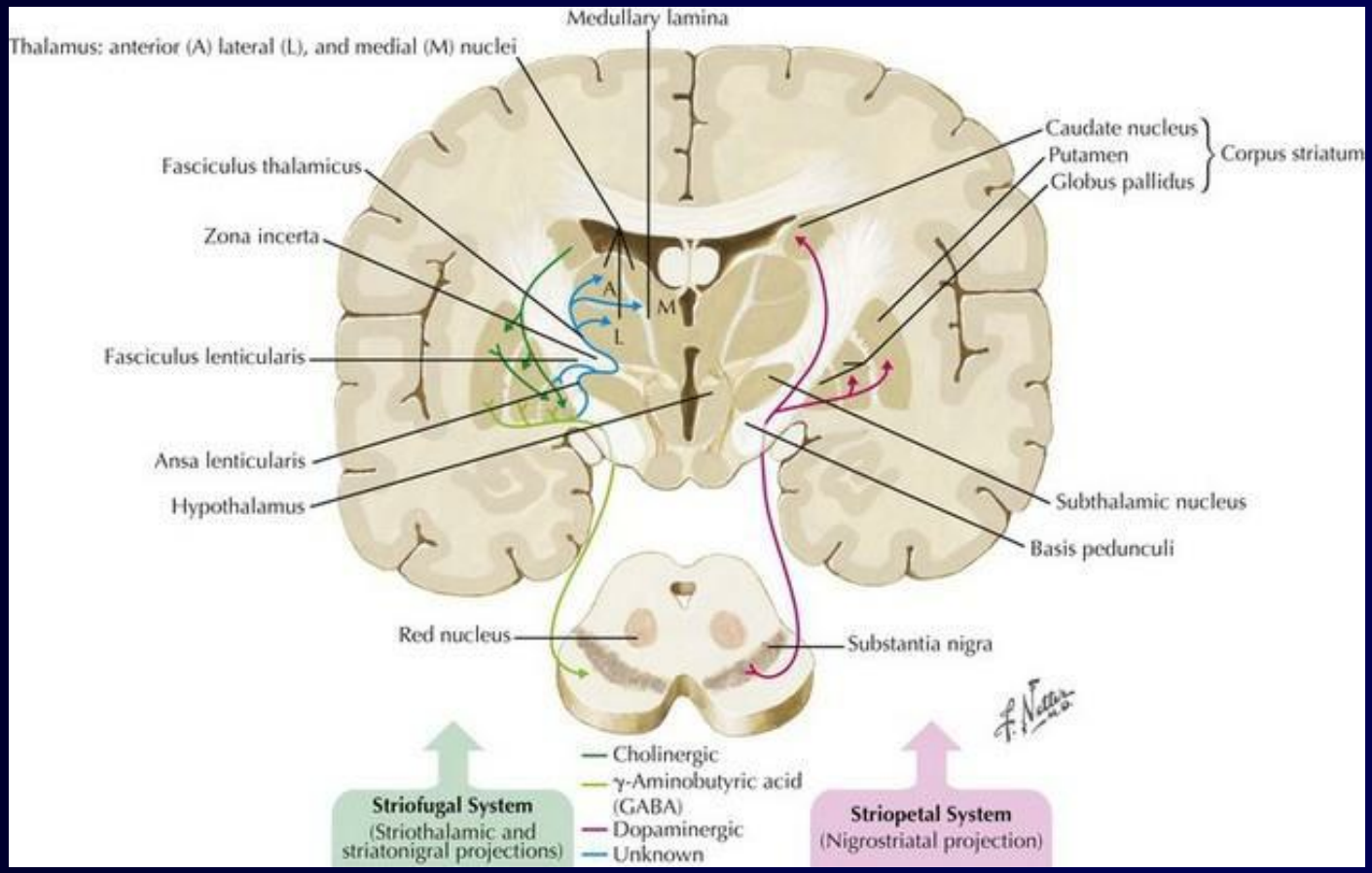
Parkinsonism

- Manifestation:
  - Positive symptoms - behaviors normally inhibited
    - Tremor (80%) – mostly in rest (hands, feet, chin, tongue), sparing the head; thumb slides back and forth on the index finger - „pill rolling“
    - Rigidity (stiffness) - loss of arm swing with walking, lack of facial expression, fatigue, muscle pain, "cog wheel"
  - Negative symptoms – missing but normally present
    - Hypokinesia
    - Disorders of posture - inability to maintain a part of body in normal position,
    - Disorders of equilibrium - difficulty in standing or sitting; Disorders of righting - inability to get up
    - Disorders of locomotion - difficulty to start and to maintain the movement (slowing down), Disorders of speech - telegraphic slurred speech, soft voice,
    - Akinesia, bradykinesia delayed & slowed movements - facial mask, shuffling gait, messy illegible writing, drooling (wet pillow) due to difficult swallowing (50%), freezing or sudden loss of movement

Parkinson' disease

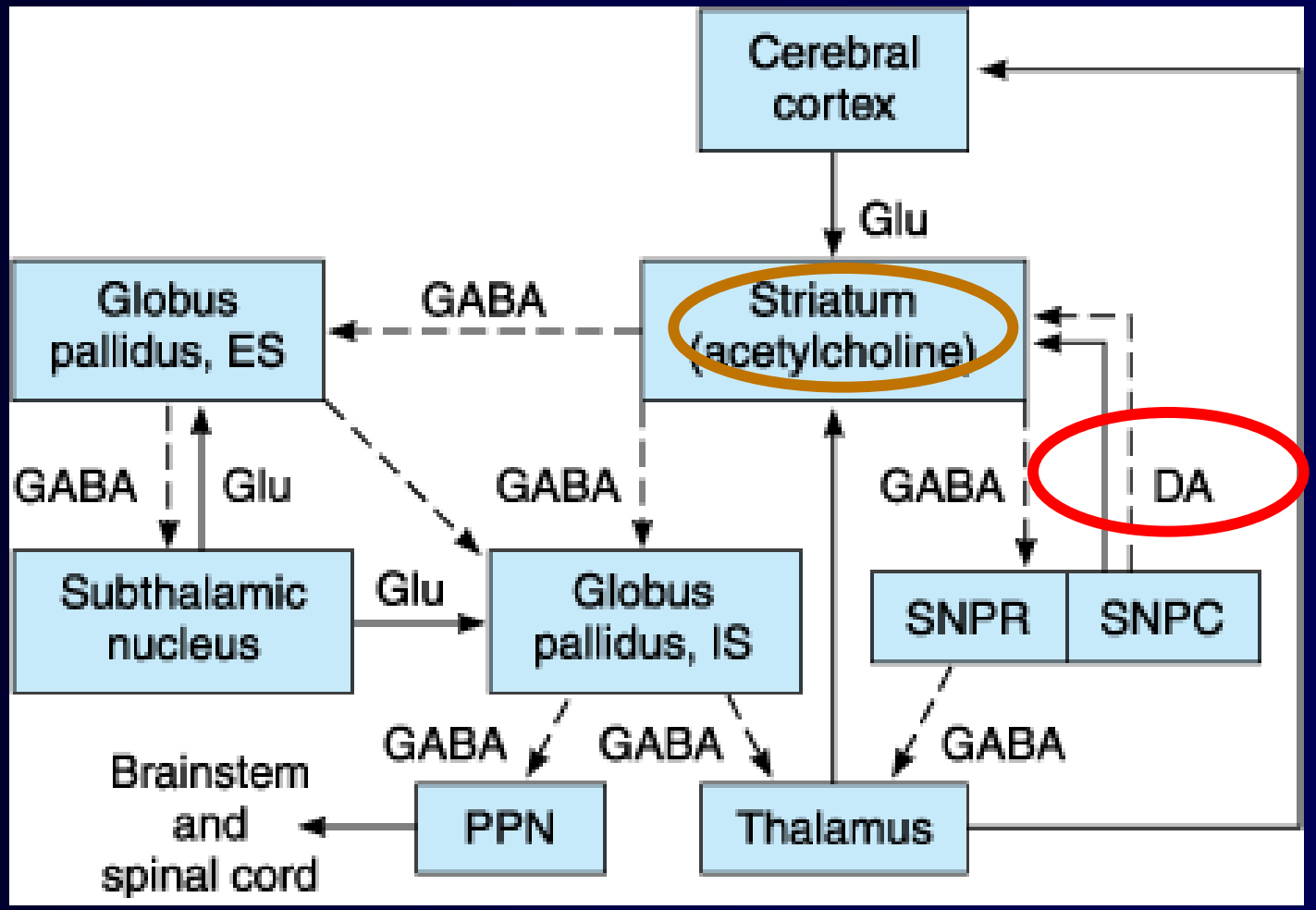
- Mechanism:
  - lack of dopamine in striatum (degeneration within substantia nigra)
- Manifestation:
  - Parkinsonism: variety in different patients
  - Cognitive defects: in some progressive cases
    - impaired ability to spontaneously generate efficient strategies when relying on self-directed, task-specific planning
    - deficits associated with frontal lobe damage
      - prefrontal caudate circuit, frontal cortex receives direct dopaminergic input from basal forebrain
    - motor planning deficits
      - increased response time in choice response tasks relative to controls, even accounting for initial difference in simple response time
  - Dementia syndrome: memory loss, mental changes
  - Vegetative dysfunctions: sexual dysfunction constipation, seborrhoea, fatigability, urinary incontinency,
- Occurrence:
  - 0.1-1% of population, 3rd most common neurological disease
  - Occurs after 50; may progress 10-20 y

Parkinsonism - Transmisssion

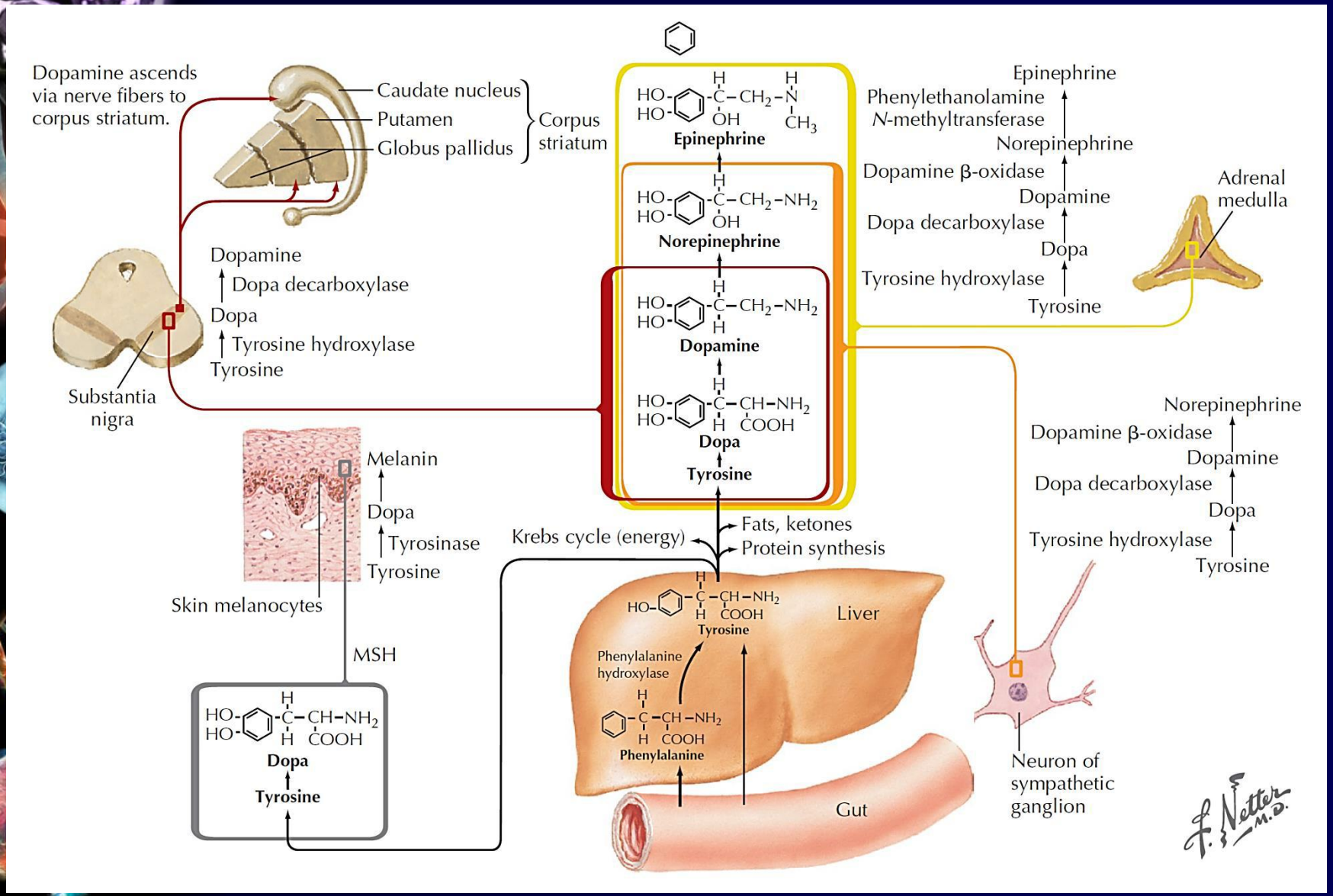


- Defect of dopaminergic projections from substantia nigra into striatum ((nigrostriatal)
- Abundance of cholinergic activity in stratum towards pallidum

Parkinsonism- Mechanisms



Parkinsonism- Mechanisms



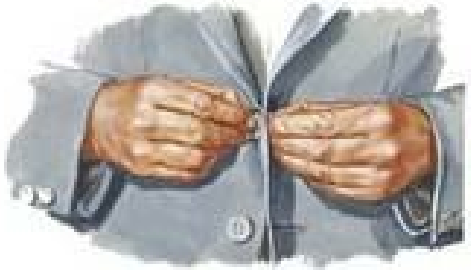
Parkinsonism - Manifestations

Tremor

Tremor of one hand is an early manifestation of parkinsonism

Tremor often improves or disappears with purposeful function

Bradykinesia



Difficulty in performing simple manual functions may be initial symptom

Rigidity and Gait Disorders

Stage 1: unilateral involvement; blank facies; affected arm in semiflexed position with tremor; patient leans to unaffected side

Stage 2: bilateral involvement with early postural changes; slow shuffling gait with decreased excursion of legs

Stage 3: pronounced gait disturbances and moderate generalized disability; postural instability with tendency to fall

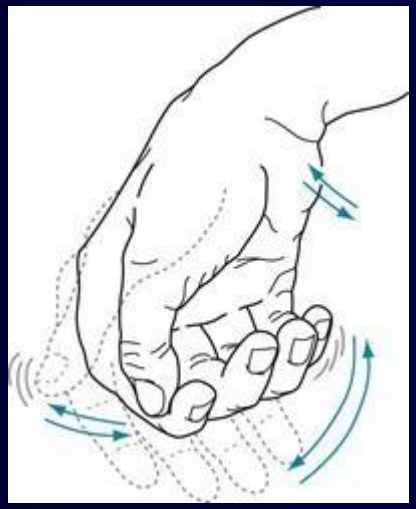
Late Stage Disabilities

Stage 4: significant disability; limited ambulation with assistance

Stage 5: complete invalidism; patient confined to bed or chair; cannot stand or walk even with assistance

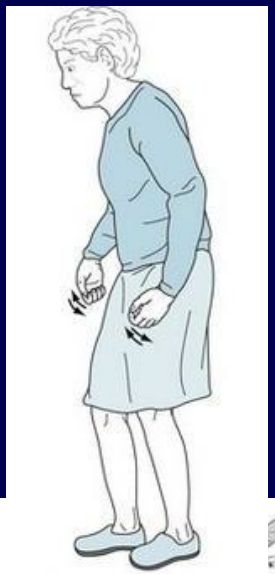
Parkinsonizmus

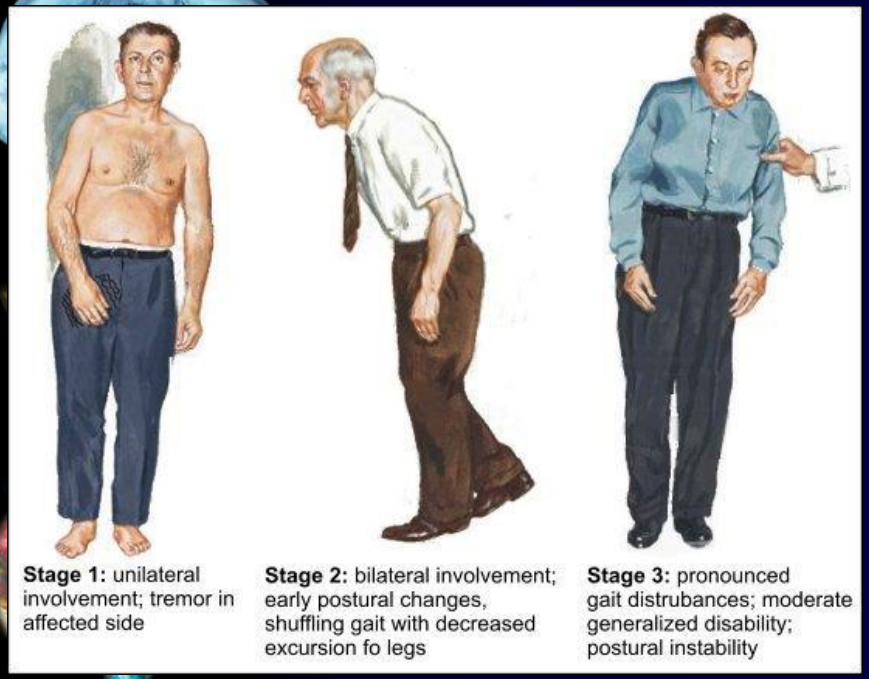
Mask - like face
Maskovitá tvár

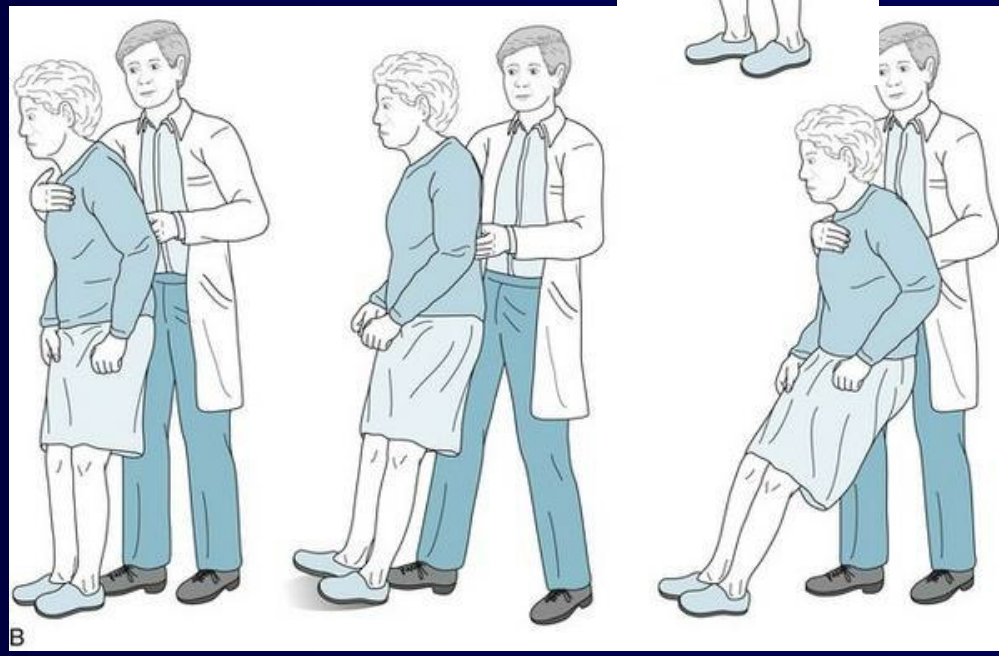


Money counting tremor
Rest 4-6 Hz

Rigidita prevodového systému (Cog wheel)

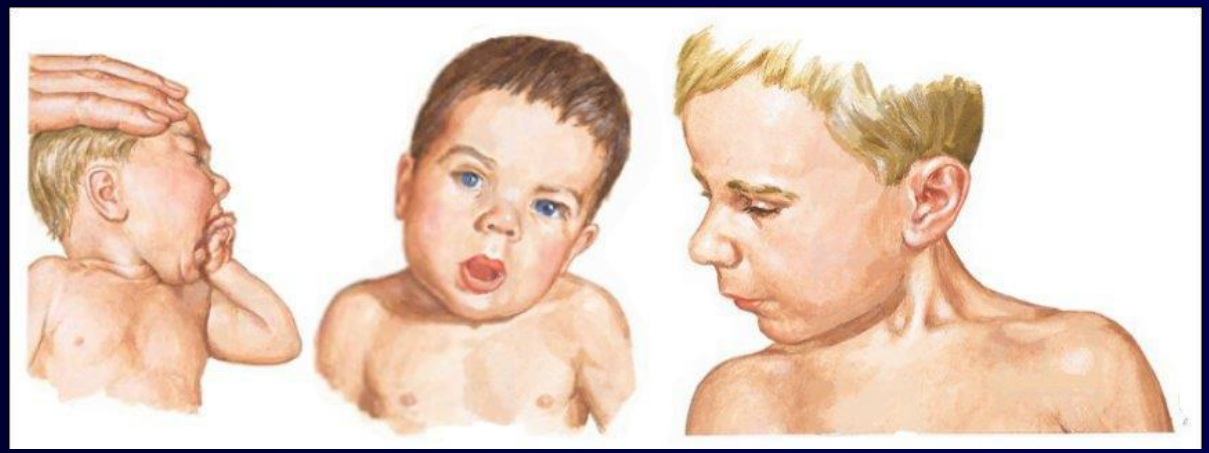


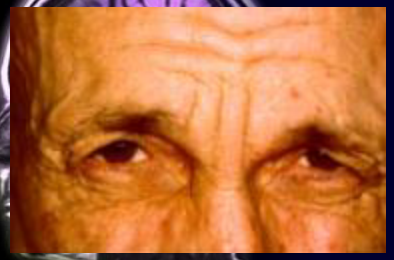




Dystonia

- Manifestation:
  - sustained, irregular, involuntary contractures
  - Focal - cervical dystonia (torticollis), writer's cramp), blepharospasm, oromandibular dystonia, Meige's syndrome
  - Generalized – torsiospasm
- Causes:
  - hereditary (focal) vs. acquired (central lesions), occupational
- Mechanism:
  - cholinergic excess in striatum (anticholinergic therapy)
- Tretment:
  - Anticholinergics, botulinum toxin injections



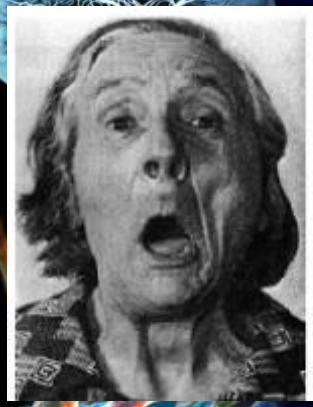


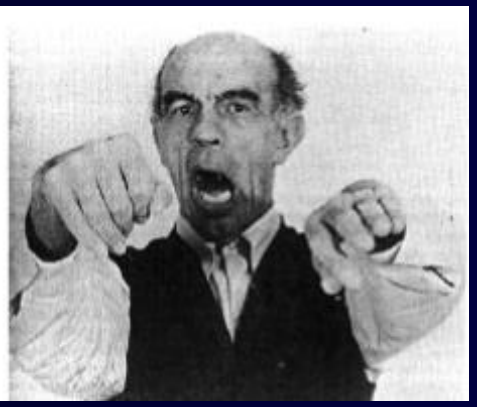
Blefarospazmus

Brachial dystonia
(writers cramp)

Limb
dystonia

Torticolis





Oromandibular dystonia

Torsiospasmus

Chorea (choreos = dancing)

- Manifestations: distal limbs, head
  - Irregular, fast jerky extra-movements in distal muscles – hands, head, feet;
  - Steps are overswinging, staggering
  - Obeisance - like poses, gestures by hands, fingers), head turns and grimasing, unrest, jitterning in legs, unsettled appearance
- Causes:
  - Hungtington's disease + other hereditary dis.
  - Sydenham's chorea – acute rheumatic fever
  - Cerebral palsy, pregnancy, etc.
- Mechanism:
  - Loss of cholinergic & abundance of dopaminergic effects in striatum

Huntington' disease

- George Huntington – 1872 (first systematic study)
- Occurrence:
  - 1.6 per million per year death rate; more common in caucasian Europeans; rare in Asians or Africans
    - village of Bures in England in 1630 - individuals thought to be witches
    - brought to US in 1630 among passengers of John Winthrop fleet
    - Lake Maracaibo - large incidence - one women whose father, an English sailor, carried the gene - > 3000 decendents, 100 with Huntington's disease,
- Etiology:
  - hereditary AD- transmitted disease (discovered in1993) studying 75 families from Lake Maracaibo
  - defective huntingtin protein (Ch4) trinucleotide repeate mutation; CAG triplet occurs 11-34 times in the normal gene, from 35 to 100 or more times in mutant

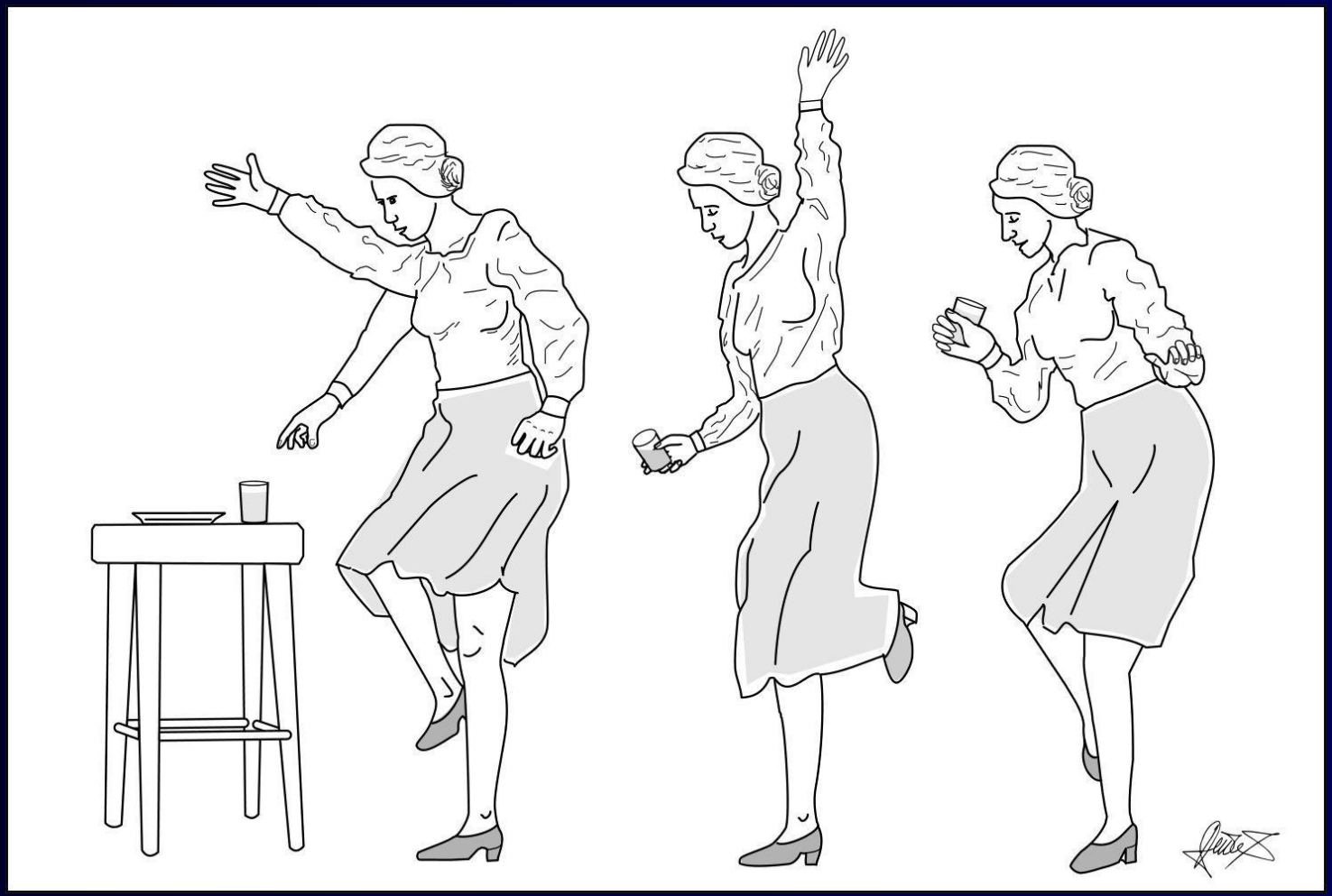
Huntington' disease

- Pathogenesis:
  - Degeneration of caudate nucleus - loss of cholinergic and GABA-ergic neurons in basal ganglia; relative excess of DA
  - Modelled in nonhuman primates by excitatory neurotoxins injected in the striatum
- Manifestation:
  1. Middle-age onset (40-50y) subtle start: absentminded, irritable, depressed, fidgeting, clumsiness
  2. Progressive chorea: violent uncontrolled overbursts - until individual confined to bed
  3. Dementia: cognitive impairment, speech is slurred, incomprehensible and finally stops; death after 15-20y

Athetosis (athetos = fidgety)

- **Manifestations:**
  - **Irregular, twisting, revolving,** turbulent, widely bursting, fidgety extra-movements of extremities
  - **Walking** – interrupted, staggering, swinging, rolling
  - Poses, obeisance, head turns, grimacing hands, fingers (gestures), legs (shaking)
- **Causes:**
  - Cerebral palsy, pregnancy, etc.
- **Mechanism:**
  - **Loss of cholinergic & abundance of dopaminergic effects in striatum**

Athetosis



Myoclonia

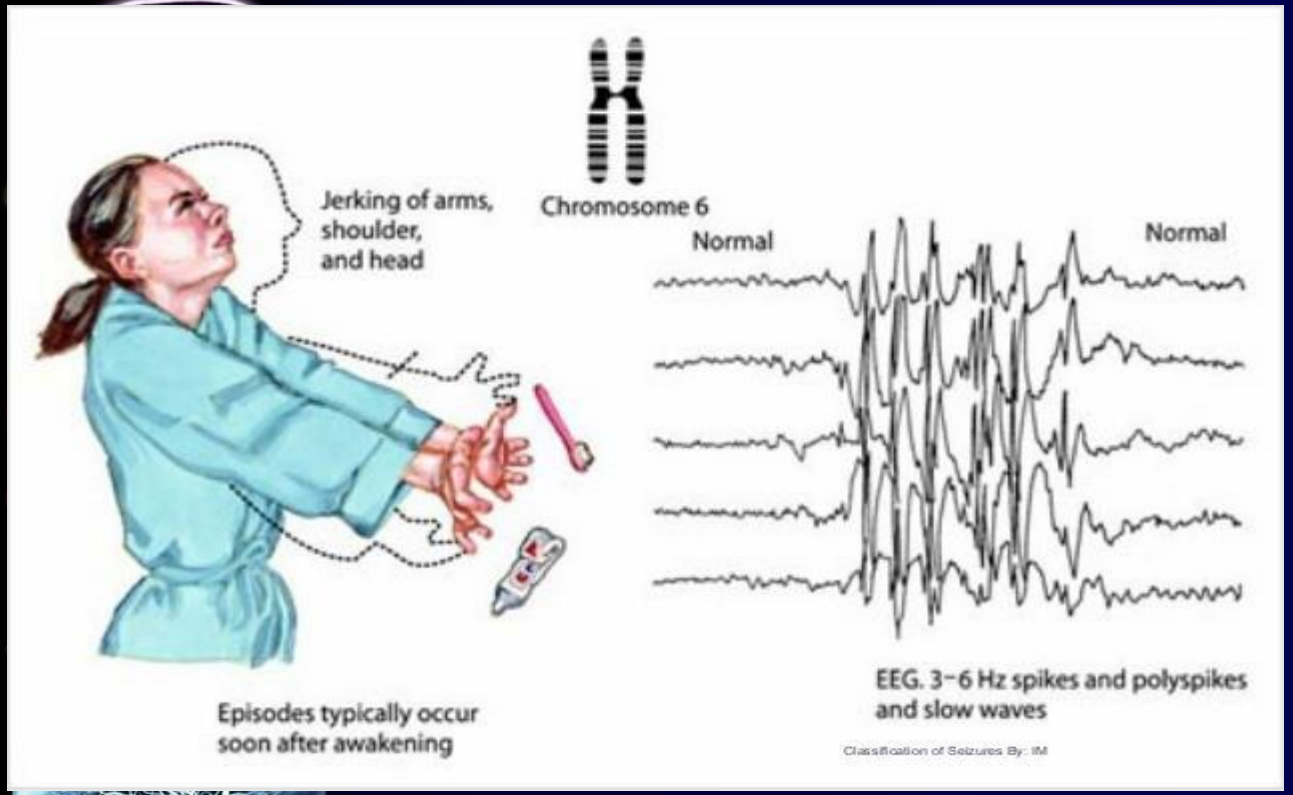
- Def.: Deliberalized very fast (50 - 150 c/min), fine clonic twitches in muscles of fine kinetic function
- Ptg: damage (loss of function) of nucl. ruber, tr. cerebello-rubralis, tr. rubro-olivalis

Myoclonia (myoklonus) lightening fast irregular twitches

- Frequency fluctuates from unique twitch to several dozen drains per minute; enhance with emotionsm disappear in sleep.
- Tongue, Larynx, pharynx,,soft palate - interruption of swallowing, speech but also ventilation.
- Limb muscles, oculomotor, respiratory muscles, abdominal muscles and sfincters.

Myorythmia - weaker, more regular (rhythmic), frequency may vary

- always localized in a particular muscle area; they appear during sleep and waking. soft palate (nystagmus veli palatini),
- muscles of pharynx and larynx (cause dysartria, voice frightening), mimic muscles (rhythmical singultus)

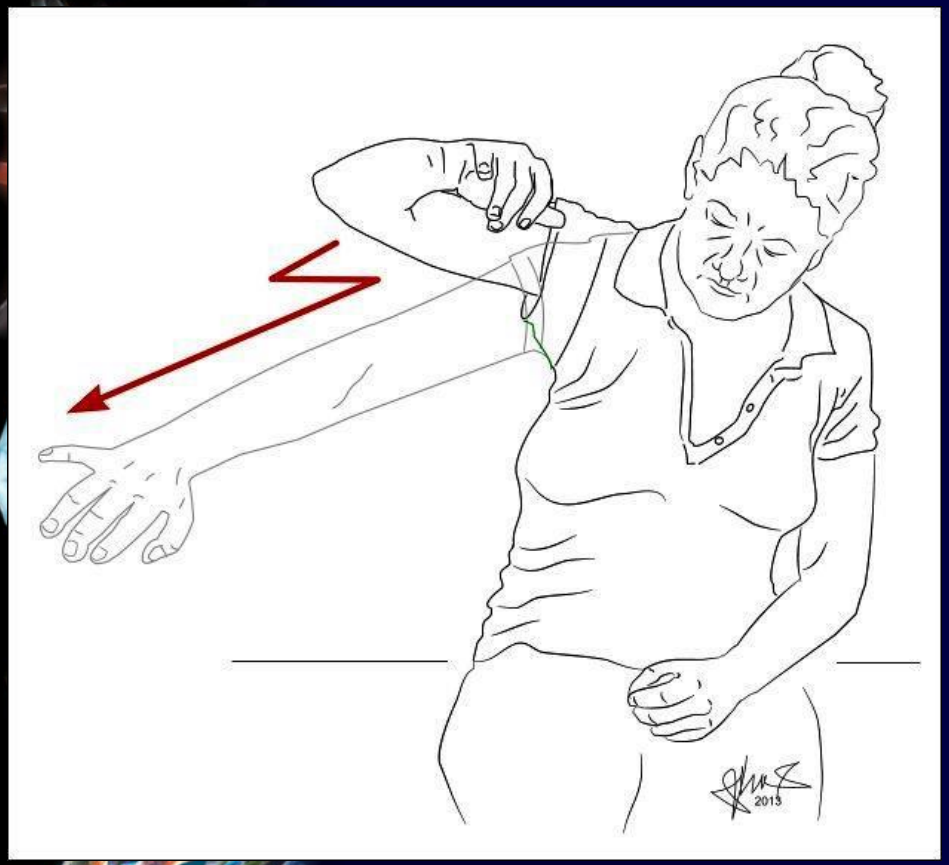


Myoclonic epilepsy - sudden twitches (jerks) or "cramps" by locality of affections (head, arms, shoulder, han) after awaking

Myoclonic status: generalized mutifocal jerks damage at level of pons and mesencephalon in coma, very poor prognosis

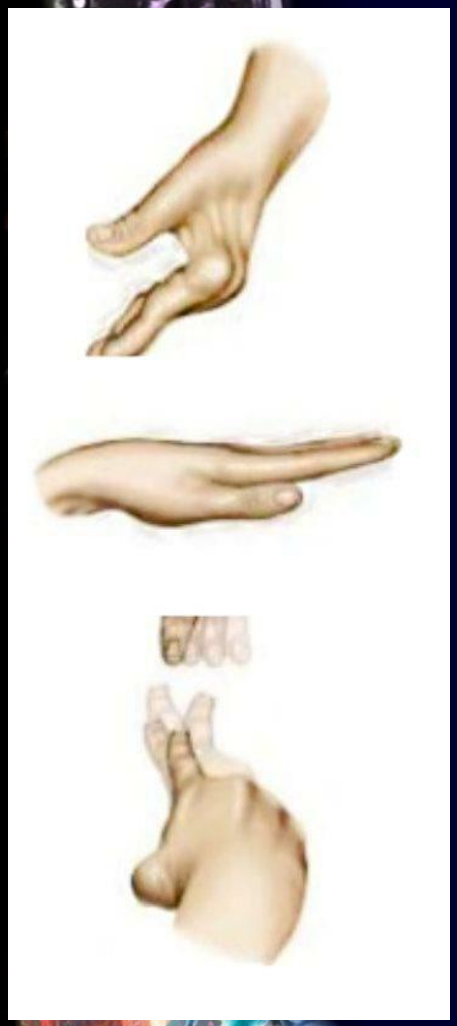
Physiological Myoclonus: Singultus
Sleep myoclonus (legs of hands), after exercise (limbs), benign infantile myoclonia (children)

Hemiballism



- Manifestation:
  - Sudden, violent, purposeless, excessive, throwing movements, gyrations (ball = throw; ballistic rocket)
  - Limb an trunk involuntary movements
- Causes:
  - Stroke in subthalamic nucleus
- Mechanism:
  - cortical escape from basal ganglia control

Tremor



- Resting 4-6 Hz (occurs with limb inactivity, chin, hand)
  - Parkinsonism, heavy manual work, emotional distress, midbrain stroke Treatment: dopaminergic agonists, pill-rolling, 5 c/sec
  - disappear with voluntary, movement, slow, fine,
- Action (intention) 3-4 Hz (absent at rest, often get worse when touching object
  - Cerebellar disease, midbrain stroke, sclerosis multiplex
- Postural (fine, rapid, antigravity posturing, fingers of outstretched arms, protruded tongue), worsen with intention
  - Exaggerated physiologic 10-12 Hz
    - Catecholamines, sympaticus,
  - Essential 4-10 Hz
    - 50% inherited familial tremor, treatment: beta-blockers, primidone
    - Hyperparathyroidims,, anxiety, fatigue, familiar

Tremor – Etiology

1. Rest tremor	M. Parkinson, parkinsonský sy., M. Wilson, hepatocerebral degenerationm, damage of nucl. ruber, esential tremor
2. Static tremor	Physiological (stress, anxiety) drugs (teofilín, amfetemín, neuroleptics, antagonists of DA2 receptors, antidepresants,), Intoxication (coffein, alcohol, Hg, Pb, As), neuropaties (Roussy-Lévyho sy.), esencial tremor
3. Intentional tremor	Cerebellar tremor (neocerebellum) , hepatolenticular degeneration, M. Wilson, trauma, brainstem bleeding Pb- poisoning
4. Functional tremor	Psychoses, neurose
5. Similar dieorders	Asterix, clonus, spazmus mutans, nystagmus, myoklonia, myorytmia

Tics

- Manifestation:
  - Brief, stereotypic, predictible, suppresible jerks worsening with stress
  - Vocalisations, grimasing,swearing, rising eye brows, gestures, grining
  - Worsening with stress
- Mechanism: Dopamine excess causing disinhibition of limbic circuit

Winking

Eyebrowing, frowning

Clenching, licking, tongue sticking out

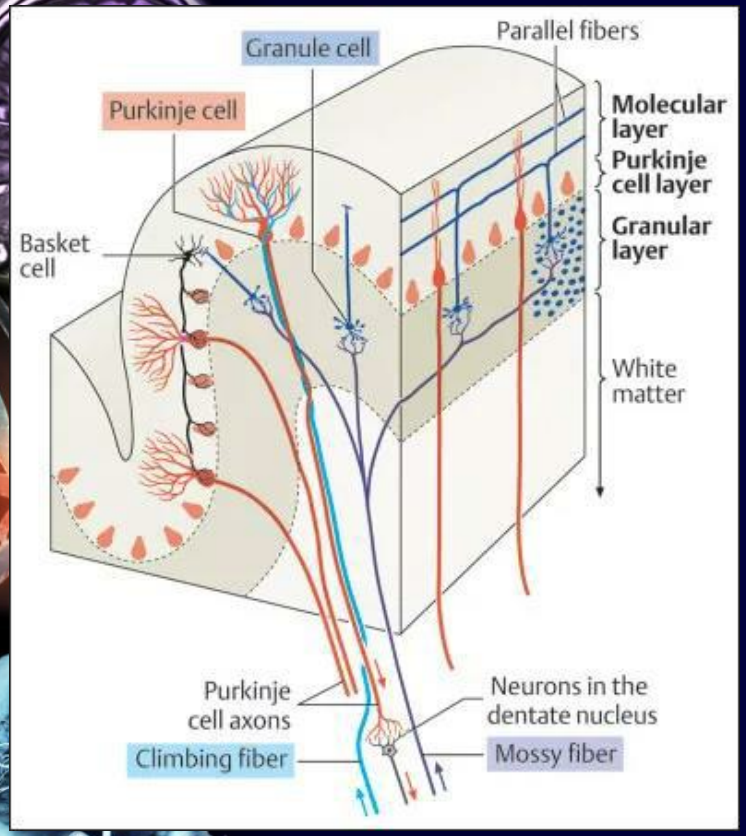
Tardive dyskinesia

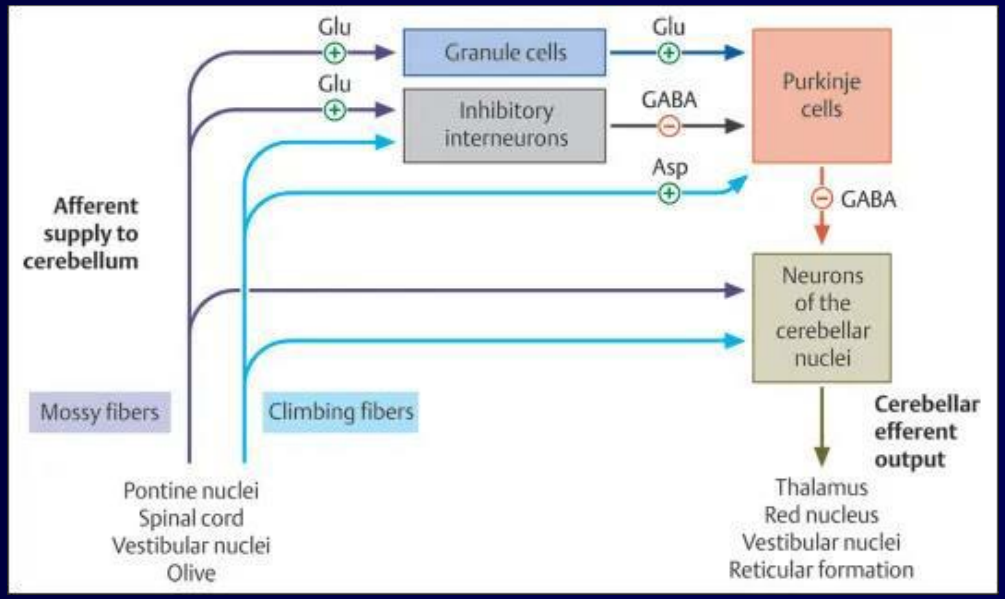
- Etiopathogenesis:
  - Chronic treatment (> 6 weeks) by dopamine antagonists, neuroleptics
  - Hypersensitivity of striatal DA - receptors
- Manifestation:
  - Orofacial repetitive movements, Limb an trunk involuntary movements

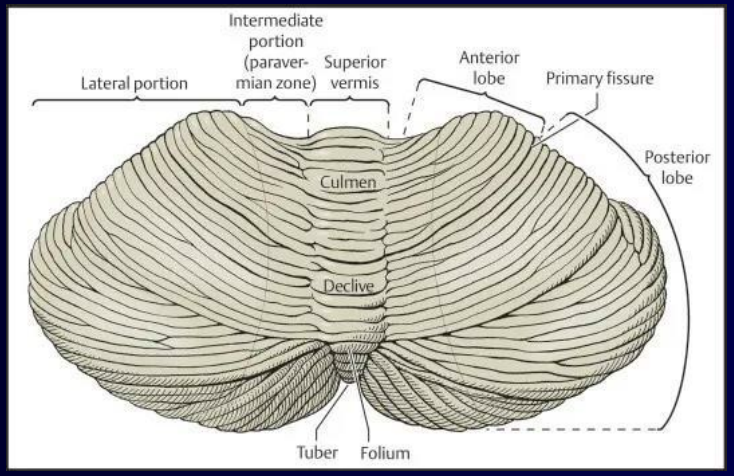
Repeated sticking out and rolling of the tongue, opening the mouth, etc

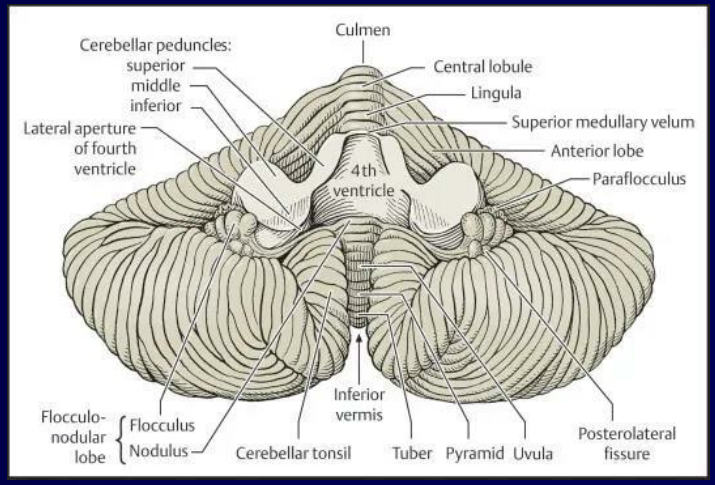
Cerebellar disorders

- Manifestation:
  - Hypotonia - pendular knee reflex
  - Loss of elementar postural reflexes,
  - Asynergy
  - Cerebellar ataxia – zig –zag walking, titubations, falling to back or sides
  - Adiadochokinesis
  - Hypermetria, dysmetria
  - Intentional tremor
- Causes:
  - Trauma, ischemia, haemorrhage, tumors, degeneration, demyelinisations affecting cerebellum, 4th- ventricle, ponto-cerebellar angle, pathways into and from

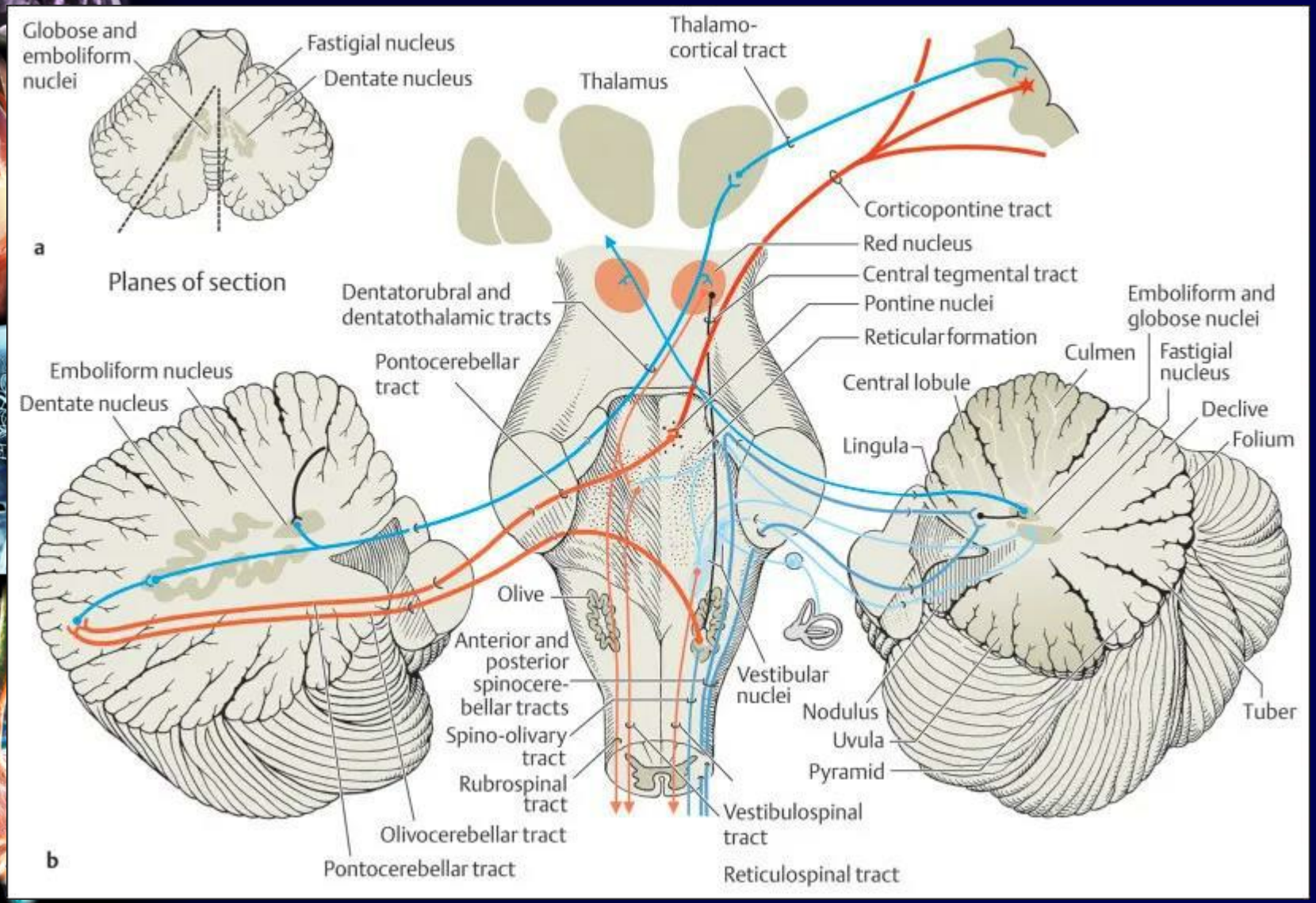

Granule cell
Parallel fibers
Purkinje cell
Molecular layer
Purkinje cell layer
Granular layer
Basket cell
White matter
Purkinje cell axons
Neurons in the dentate nucleus
Climbing fiber
Mossy fiber

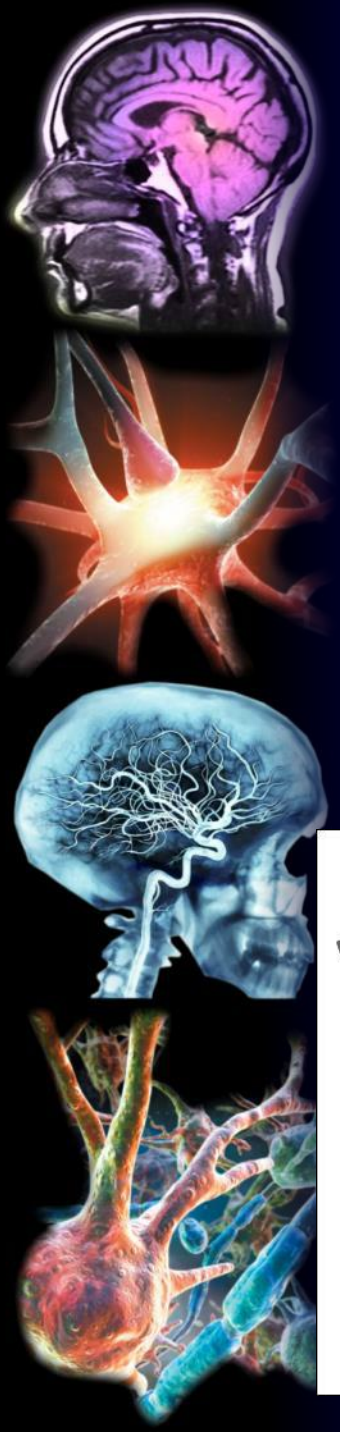

Glu
Granule cells
Glu
Purkinje cells
Glu
Inhibitory interneurons
GABA
Asp
GABA
Afferent supply to cerebellum
Neurons of the cerebellar nuclei
Mossy fibers
Climbing fibers
Cerebellar efferent output
Pontine nuclei
Spinal cord
Vestibular nuclei
Olive
Thalamus
Red nucleus
Vestibular nuclei
Reticular formation

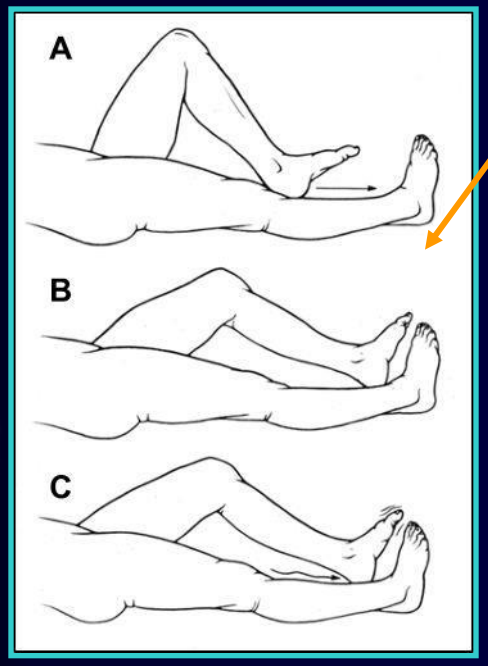

Intermediate portion (paraver-mian zone)
Lateral portion
Superior vermis
Anterior lobe
Primary fissure
Posterior lobe
Culmen
Declive
Tuber
Folium


Culmen
Cerebellar peduncles: superior middle inferior
Central lobule
Lingula
Superior medullary velum
Lateral aperture of fourth ventricle
Anterior lobe
4th ventricle
Paraflocculus
Inferior vermis
Flocculo-nodular lobe
Flocculus
Nodulus
Cerebellar tonsil
Tuber
Pyramid
Uvula
Posterolateral fissure

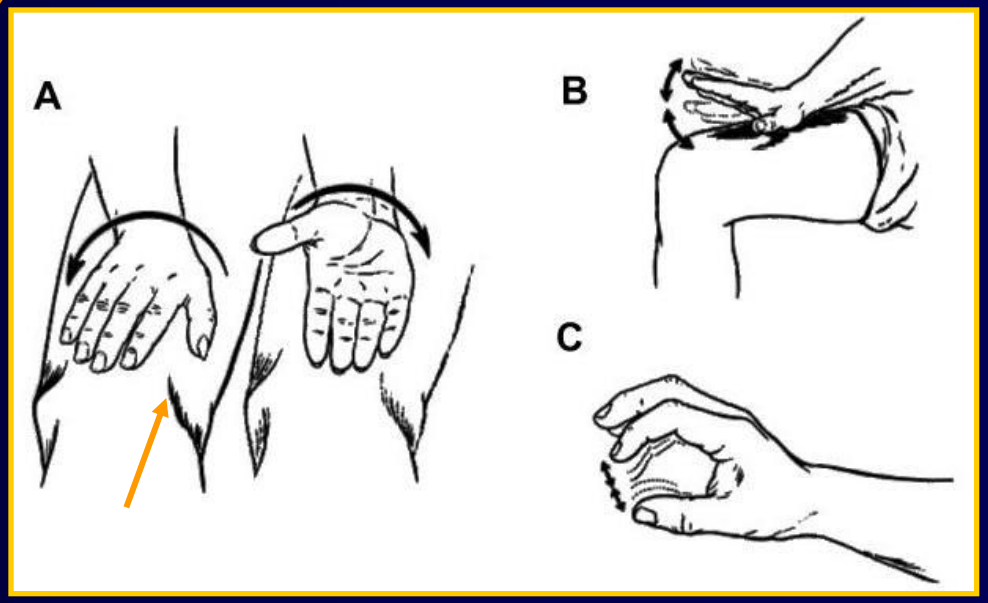
Cerebellar disorders





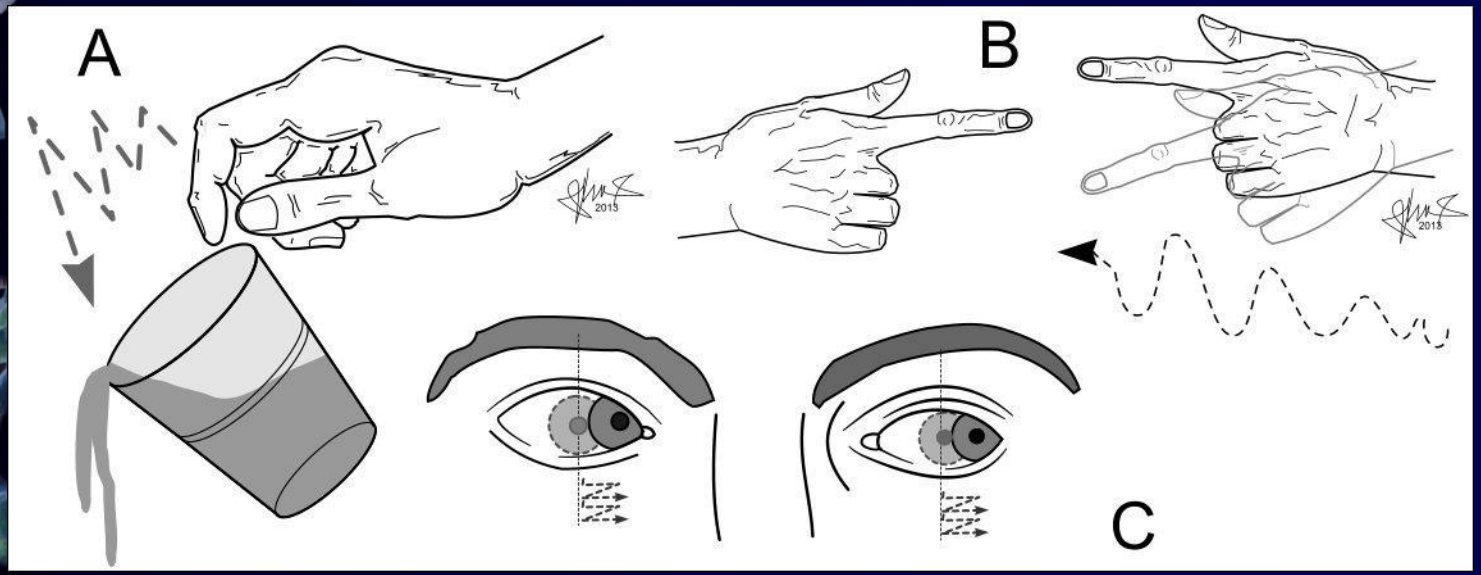


Test heel – knee. A. normal, B. abnormal

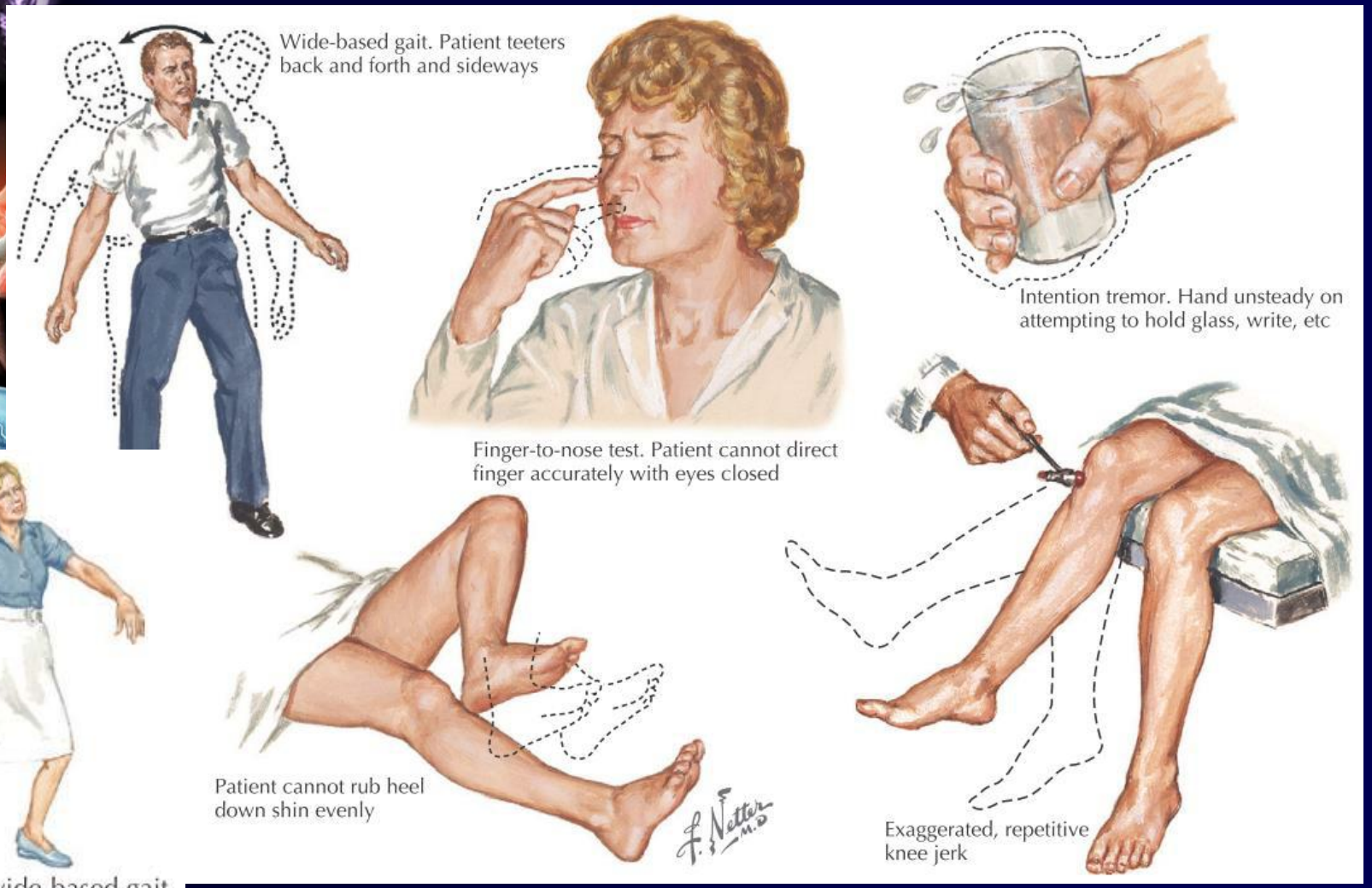


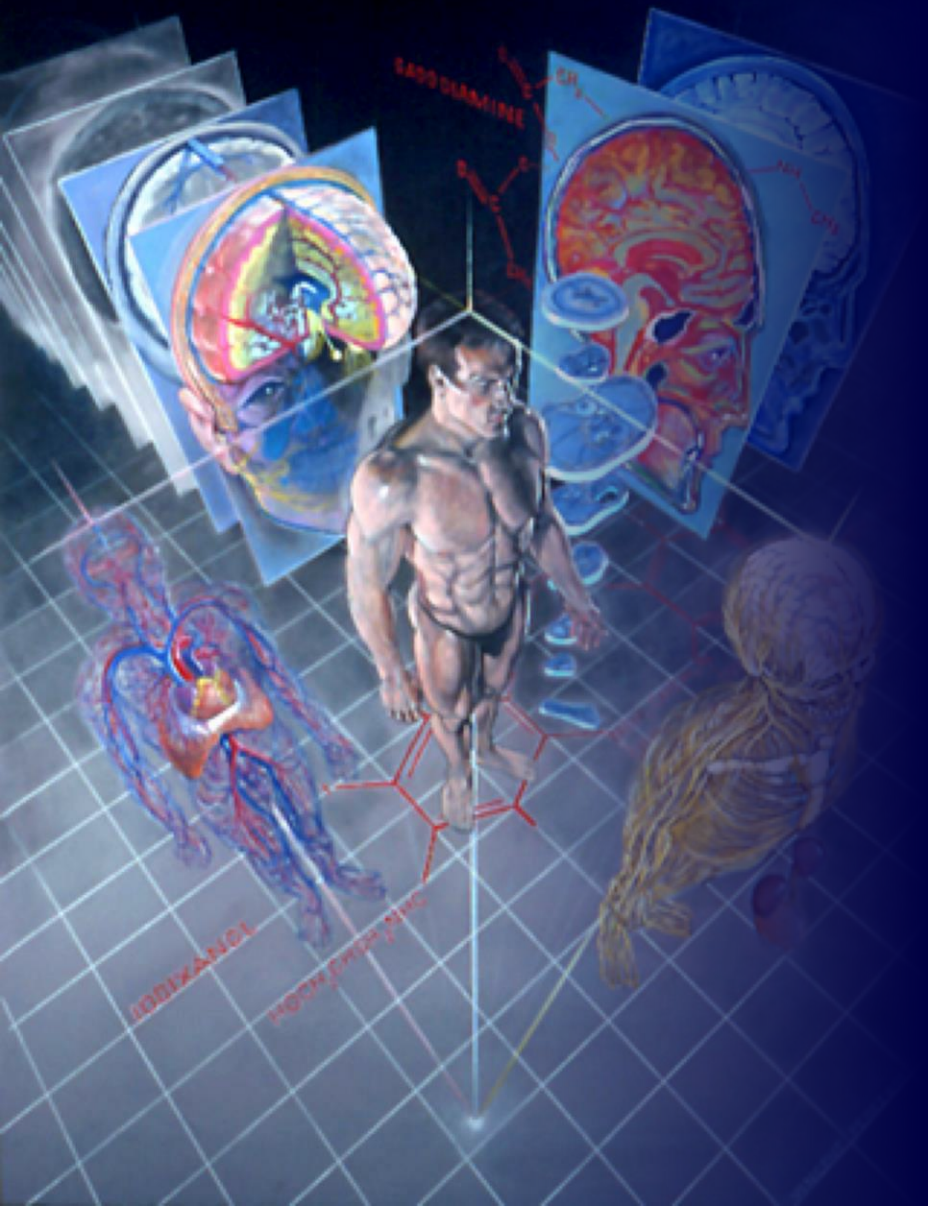
Dysdiadochokinesia:

A. turning of hand B. tapping, waving C. thumb- index



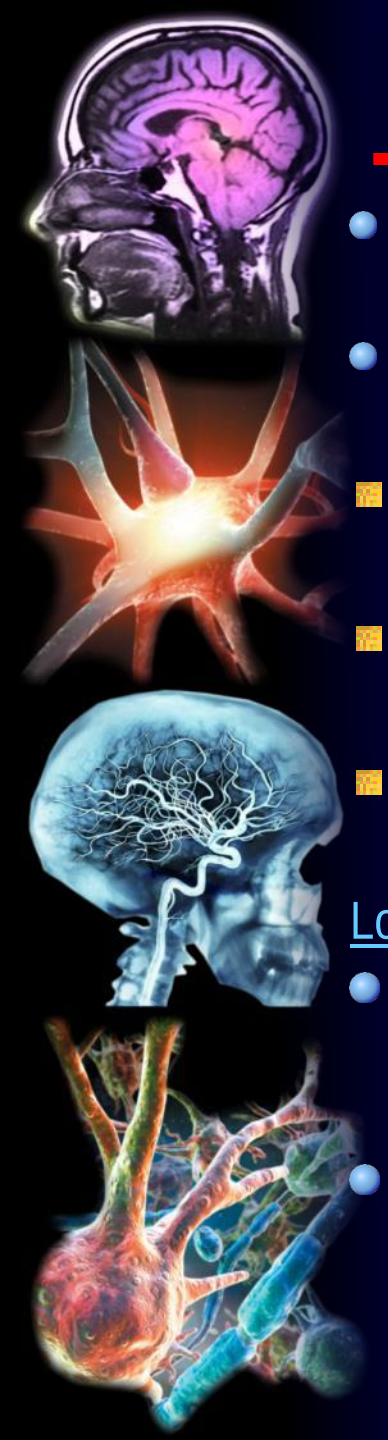
Cerebellar manifestations





5

Apraxias



Apraxia

- Def: Liepmann (1900): inability to produce a movement that is not due to paresis (paralysis) - specific loss of skill
- various forms depending on site of damage (not complete agreement on designations and criteria)
  - Ideational apraxia = misuse of objects due to disturbance of identification (agnosia)
  - Ideomotor apraxia = simple movements can be executed, but complex movements cannot
  - Limb kinetic apraxia = inability to make movements or use objects or the purpose intended by the will

Location:

- Premotor cortex (area 6 - lateral)
  - inability to produce indirect trajectories
  - ablation of premotor cortex impairs conditional motor behavior
- Supplementary motor area (area 6 - medial)
  - Brinkman, bimanual coordination deficit
  - absence of speech

Apraxia 2

- Prefrontal
  - deficits in delayed response tasks
    - dorsolateral - spatial
    - ventral - object

- Posterior parietal cortex (areas 5, 7)
- Gerstmann's syndrome
  - Following left parietal (in normal right hand dominant)
  - left-right confusion, finger agnosia
  - dysgraphia , dyscalculia, apraxia

- Balint's syndrome
  - Following bilateral damage
  - unable to make voluntary eye movements into affected hemifield
  - optic ataxia - deficit in visually guided reaching, deficit in visual attention

- Neglect
  - Following right (nondominant) hemisphere damage Constructional apraxia