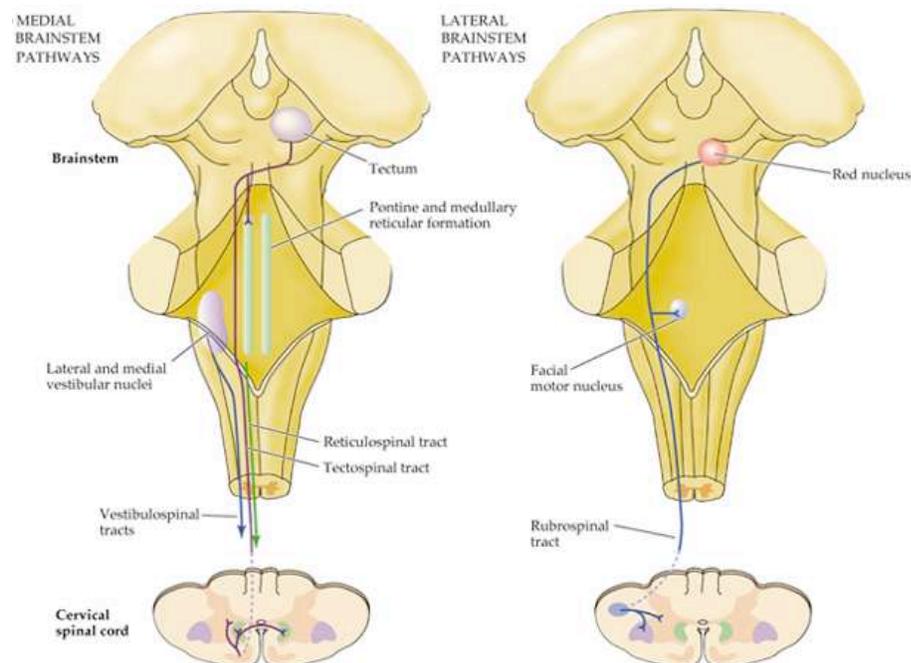


## VI. MOTOR SYSTEM

### CATEGORIES OF DESCENDING SYSTEMS:

- Traditional Categories: Pyramidal and Extrapyrarnidal. There are problems with this categorization:
  - It grossly overemphasizes the importance of the Pyramidal system. You can lesion the pyramidal tract without intractable motor deficits.
  - It artificially groups together systems that are functionally unrelated.
- DORSOLATERAL: Tracts located in the dorsolateral fasciculus of the spinal cord. *They control distal muscles and are largely **crossed**. They run close to the distal alpha-motoneuron which also lie laterally.*
  - **CORTICOSPINAL TRACT**: The traditional pyramidal system. 90% crossed (Lateral CST) and 10% uncrossed (Anterior CST)
    - **CORTICOSPINAL TRACT**: Produces *loss of independent finger movement and distal weakness*. Mild deficits, especially if the red nucleus is intact.
    - **MOTOR CORTEX LESION**: Highly **incapacitating**. Severe paralysis of contralateral muscles.
    - **INITIALLY: Flaccid Paralysis**
    - **LATER: Spastic (UMN) Paralysis** kicks in as spinal reflexes become hypersensitive.
  - **RUBROSPINAL TRACT**:
    - **RED NUCLEUS LESION** (as opposed to the tract) will cause cerebellar-type disturbances.



- VENTROMEDIAL: Tracts located in the ventromedial fasciculus of the spinal cord. *The control proximal (axial) muscles and are largely **uncrossed**. They run close to the proximal  $\alpha$ -motoneurons which also lie medially.*
  - LESIONS: Lesions of this system are very **incapacitating**. Inability to maintain upright posture.
  - **RETICULOSPINAL TRACT**:
    - These neurons are involved with coordination of limb movement.
    - **CENTRAL PATTERN GENERATOR (CPG)**: In the brain stem (Reticular Formation), it mediates the signals that are necessary for **locomotion** (walking and running). These signals are carried through Reticulospinal Tract.
  - **VESTIBULOSPINAL TRACT**:
    - These neurons are important for maintaining an upright posture.
- Interstitial Spinal and Tectospinal Tracts: They are involved in control of head and neck muscles. Other than that don't worry about them.

### GENERAL PROPERTIES OF DESCENDING SYSTEMS:

- **ALPHA-GAMMA CO-ACTIVATION**: Descending systems will send excitatory branches to both alpha and gamma neurons at the same time. Since gamma neurons ultimately increase alpha activity, this helps to amplify the excitatory signal.

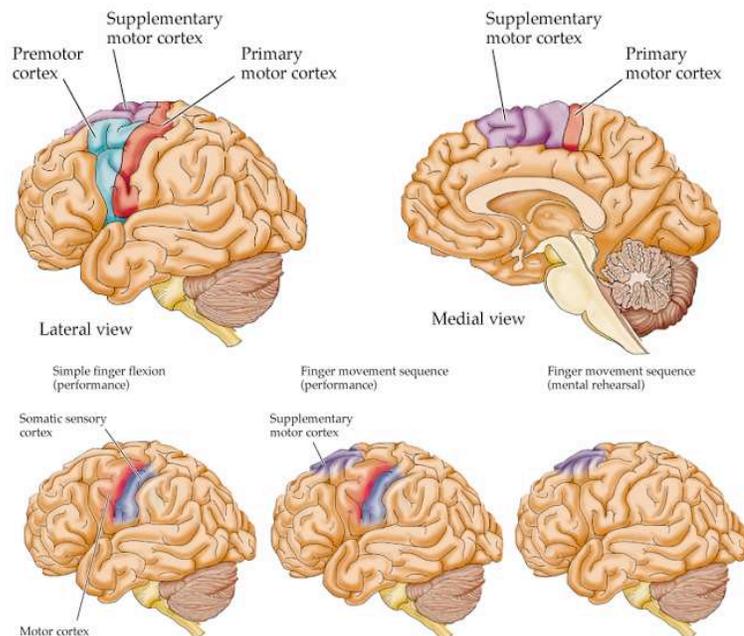
- **AUTOMATIC LOAD COMPENSATION:** Muscles will automatically adjust their tension to accommodate the “load” on the muscle.
  - INCREASED LOAD will stretch a muscle that is contracting.
  - This stretching will trigger Ia Spindle Afferents -----> Increase  $\alpha$ -activity even further -----> increase muscle tension -----> overcome the increased load.
  - Gamma co-activation (which is unaffected by Spindle Afferents) also occurs. This further helps to modulate automatic load compensation.
- HIGHER INFLUENCE on SPINAL REFLEXES:
  - INVERSE STRETCH REFLEX: We have higher control over sensitivity of the Inverse Stretch Reflex.
- **“PYRAMIDAL SENSORY” SYSTEM:** The Corticospinal system does have “sensory” neurons that serve to *screen out routine stimuli*, by putting inhibition on sensory nuclei in the brainstem and spinal cord.
  - PATHWAY: Descending fibers originate from **Primary Somatosensory Cortex** -----> *Dorsal Column Nuclei and Dorsal Horn* -----> excite or inhibit the dorsal column nuclei.

**BABINSKI REFLEX:** Test for UMN Lesions.



- TEST: Stroke the ventrolateral aspect of the foot (the sole of the foot).
  - Normal Response: Toes should point down - *Plantar flexion*.
  - Pathological Response: Toes point upward and *fan out*; *Dorsiflexion*. This indicates a problem with the Corticospinal Tract in adults.
- INFANTS normally show a positive Babinski until two years of age.

**THE MOTOR (PRE-CENTRAL) CORTEX: BRODMANN’S AREA 4**



- **SOMATOTOPIC ORGANIZATION:**
  - **MEDIAL:** Nearest the top of the cortex. Contains upper neurons for the FOOT
  - **LATERAL:** Nearest the temporal lobe. Contains upper neurons for the HEAD and NECK
- *Muscle Groups capable of the most skilled movements (Ocular muscles, facial muscles, and hands) have the largest representation of neurons in the Motor Cortex.*
- **FORCE TRANSDUCTION:** *There is a linear relationship between the force of muscle contraction and the firing rate of Upper Motor Neurons*
  - There is a **70 - 100 msec** time difference between the initiation of the UMN signal and contraction of the target muscle.
- **LESION:** *A motor cortex is much more severe than pyramidal tract lesions.* In pyramidal tract lesions, the motor cortex is still intact and can still influence the extrapyramidal systems to maintain some motor control.
  - **INITIALLY:** Motor Cortex lesions produce a **Flaccid Paralysis**, as there is a huge loss of excitatory input on alpha-neurons -----> hyperpolarization.
  - **LATER** (after ~2 months): **Spastic Paralysis and Hyperreflexia**, as the alpha-neurons compensate for the lost excitatory input by becoming more sensitive and firing spontaneously.

#### SECONDARY CORTICAL MOTOR AREAS:

- **SUPPLEMENTARY MOTOR AREA (SMA):** BRODMANN'S 6 (Pre-Motor Cortex), most medially.
  - **FNXN:** Involved with executing pre-programmed or planned motor movements (such as playing the piano or typing).
  - **EXPT: MEASURE CEREBRAL BLOOD FLOW** (which indicates neuron activity) in human patients performing motor tasks.
    - **MOTOR CORTEX** lit up because of movement of fingers
    - **SOMATOSENSORY CORTEX** lit up because of touching the fingers.
    - **MOTOR and SOMATOSENSORY CORTICES** lit up as before.
    - **SMA** also lit up: **CONCLUSION** = this suggests that the SMA is involved in executing the complex sequences.
    - **SMA** was heavily activated while the **MOTOR CORTEX** and **SOMATOSENSORY CORTEX** were not, because the fingers weren't actually moving.
- **PREMOTOR CORTEX:** BRODMANN'S 6 (Pre-Motor Cortex), most laterally.
  - **FNXN:** Involved with assembly of new motor programs and "learning" of repeated motor sequences.
- **PARIENTAL AREAS 5 and 7: Selective Attention** to areas in **EXTRA PERSONAL SPACE**. *Extra personal space is the space external to our bodies, but within reach.*
  - **PARIENTAL AREA 5:** It helps control movement of the **limbs** in response to extra personal space.
  - **PARIENTAL AREA 7:** It helps control movement of the **eyes** in response to extra personal space.
    - *Saccade Related*
    - *Fixation Related*
    - *Smooth Pursuit Related*
  - **NEGLECT SYNDROME:** Unilateral lesions of Parietal Cortex, especially to the non-dominant hemisphere.
    - There may also be contralateral hemiparalysis.
    - **Apraxia:** Difficulty drawing objects in 3D, such as drawing a clock that is completely round.
    - **Astereognosis:** Failure to recognize objects placed in contralateral hand.
    - **Anosognosia:** Denial of symptoms

#### EYE MOVEMENTS AND VESTIBULAR SYSTEM

**SACCADES:** Extremely rapid movement of eyes, creating an instantaneous change in gaze, from one location in space to a different location.

- *It is not possible to voluntarily move the eyes in a continuous, smooth path across space, from one side of the room to the other.*
- **VISUAL FIELD:** There is *no blur of the visual field* with saccades, i.e. you can't see the field move as you move your eyes. This is because:
  - The movement is too rapid for the visual apparatus to process.
  - **COROLLARY DISCHARGE:** The same signal that produces the saccade also *inhibits the Lateral Geniculate Nucleus* from processing visual stimuli during the eye movement.
- **VOLUNTARY SACCADE:** The signal originates in the **Frontal Eye Field (Area 8)**.
  - **PATHWAY:** *Frontal Eye Field (Area 8) -----> Superior Colliculus -----> Reticular Formation ----> ---> Oculomotor Neurons -----> Saccade movement.*

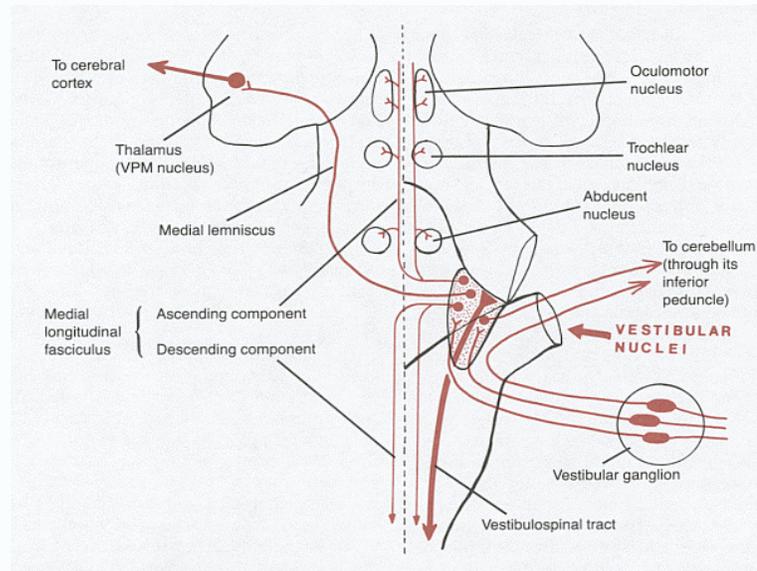
- **REFLEX SACCADDE:** Involuntary saccades made to a **novel stimulus**, such as an unexpected flash of light that appears in the visual field.
  - The reflex involves the **SUPERIOR COLLICULUS** directly and does not utilize input from the Frontal Eye Field.
  - PATHWAY: *Novel Stimulus* -----> *Retinal Ganglion Cells* -----> *Superior Colliculus* -----> *Reticular Formation* -----> *Oculomotor Neurons* -----> *Saccade response to stimulus*
  - SOMATOTOPIC ORGANIZATION: Superior Colliculus has somatotopic organization across two layers:

**SMOOTH PURSUIT:** Eye movements involved in maintaining fixation on a moving target while the head is stationary.

- **VELOCITY** of movement is less than that for saccades: 30 / sec maximum. You can't keep up with a really fast moving object and hence it will appear as a blur.
- **INVOLUNTARY:** These actions are an **involuntary reflex**. You cannot prevent smooth pursuit movements (such as following your finger with your eyes) without shifting your gaze elsewhere.
- **PATHWAY:** *Retinal stimulus* (indicating slip of focus) -----> *Retinal Ganglion Cells* -----> **Lateral Geniculate Nucleus** -----> *Visual Cortex* -----> *Reticular Formation* -----> *Oculomotor Neurons* ----> *Smooth Pursuit movement*.

**VESTIBULO-OCULAR REFLEX (VOR):** Maintaining gaze on a fixed object while moving your head. *The direction of eye movement will be equal and opposite to that of the head, i.e. the eyes will turn medially.*

Central connections of the vestibular system



- **PATHWAY:** DISYNAPTIC. *Vestibular Hair Cell Deflection* -----> *VIIIth nerve* -----> **Ipsilateral Medial Vestibular Nucleus increases firing rate** -----> it SYNAPSES with two different nerves
  - **EXCITATORY SYNAPSE** to ipsilateral **Oculomotor Nucleus** -----> **ipsilateral eye turns medially**, in opposite direction as original head movement.
  - **INHIBITORY SYNAPSE** to ipsilateral **Abducens Nucleus** -----> *prevent eyes turning laterally*
- **CONTRALATERAL SIDE:** The exact converse will be going on: *Contralateral Medial Vestibular Nucleus is INHIBITED* -----> *decrease Oculomotor firing and increase Abducens firing* -----> **contralateral eye turns laterally**, in the opposite direction as original head movement.
- **MEDIAL LONGITUDINAL FASCICULUS (MLF)** also helps to coordinate the gaze between the two eyes. It works in synergy with the VOR reflex.

**SEMICIRCULAR CANALS:** Detect head turns and keep them in balance; detect angular acceleration.

- **FUNCTIONAL PAIRS:** Semicircular Canals are divided in **PAIRS** of canals that counter each other in terms of their response to head turns (one will be stimulated while the other will be inhibited to the same degree).
  - **Left Horizontal Canal** <====> **Right Horizontal Canal**
  - **Left Anterior Canal** <====> **Right Posterior Canal**
  - **Left Posterior Canal** <====> **Right Anterior Canal**

- ANATOMY:
  - The Semicircular Canals are attached to the Utricle.
  - They contain **Endolymph** to make the hair cells move. Endolymph is continuous throughout the Utricle and Semicircular Canals.
  - **AMPULLA**: The enlargement at one end of each canal, where the hair cell receptors are located.
  - **CUPULA**: In the Ampulla, the gelatinous mass into which the hair cells insert.
- HAIR CELLS: **Scarpa's Ganglion** contains the cell bodies of the hair cells, which form the VIIIth (Vestibular) nerve.
  - **STEREOCILIA** insert into the Cupula and move in response to head movements.
  - **KINOCILIUM** is the end cilium, larger than the other cilia.
- VIIIth NERVE STIMULATION.
- ORIENTATION OF KINOCILIUM.
- EXAMPLE: TURN HEAD RIGHT, The endolymph fluid will move left initially (due to inertia). Hair cells in the Horizontal Canals will deflect to the left.
  - **RIGHT HORIZONTAL CANAL**: *Stereocilia move toward Kinocilium -----> activate VIIIth nerve afferents.*
  - **LEFT HORIZONTAL CANAL**: *Stereocilia move away from Kinocilium -----> inhibit VIIIth nerve afferents.*

#### OTOLITH ORGANS:

- UTRICLE
- SACCULE

**VESTIBULO-POSTURAL REFLEX:** Compensation for turning the body to the right or the left.

Example: TURN BODY RIGHT, and two responses happen

- RIGHT SEMICIRCULAR CANALS ACTIVATE -----> *Activate Right VIIIth nerve afferents -----> activate right Vestibulospinal Tract -----> excite extensors on the right -----> net extension on right side.*
- LEFT SEMICIRCULAR CANALS DEACTIVATE -----> *Inhibit Left VIIIth nerve afferents -----> inhibit left Vestibulospinal Tract -----> inhibit extensors on the left -----> net flexion on left side.*
- RESULT: Turn the body right, and the body attempts to fall (flex) toward the left for compensation.

**NYSTAGMUS REFLEX:** The reflexive response of *eye movements* to continual rotation in one direction.

SPIN BODY TO THE RIGHT, and the eyes will do the following:

- **Vestibulo-Ocular Reflex (VOR):** Initially there will be slow rotation to the left. However, as rotation continues, the eyes will no longer be able to turn left, so there must be a resetting of gaze.
- **Involuntary Saccade:** The eyes will instantaneously reset the gaze *toward the right*, i.e. in the same direction as rotation. This reflex is not VOR but rather is an involuntary saccade.
- NYSTAGMUS is the combination of the two reflexes above, in alternating order with each other (A, then B, then A, then B). This is the eye's response to continual rotational movement.
- *The direction of nystagmus, by convention, is the direction of the reflexive saccade.* Thus: **Spinning the head right results in a "rightward" nystagmus reflex.**

**BARANY CHAIR TEST:** Rotate a person in a chair really fast to the **right** for about 30 sec; stop the chair, and then test for **leftward acceleration reflexes** of the eyes.

- **Post-rotational Component:** After stopping the chair, you will be testing for *deceleration* to the right, which is the same as testing reflexes for *leftward acceleration*.
  - What the semicircular canals detect is the rate of change of velocity of the fluid (*angular acceleration*), not the movement of the fluid itself (*angular velocity*). Net angular acceleration will be to the *left* in this case.
- EXPECTED RESULTS: You would thus expect a **leftward Nystagmus** once the chair is stopped.
  - So, you look for the patient rotating his eyes toward the right (VOR reflex), and then instantaneously shifting them left (reflex saccade), then rotating eyes rightward again, etc.

**INNER NUCLEAR OPHTHALMOPLEGIA:** Lesion in the MLF, as often occurs with Multiple Sclerosis.

- SYMPTOM: You lose coordinated gaze of the **medial rectus (CN III)**.
- RIGHTWARD ROTATION VOR: Your right eye would be able to move laterally just fine, but your left eye would not move medially.
- So with this condition, lateral movements are fine but medial movements in the VOR reflex are impaired.

**UNILATERAL LABYRINTHECTOMY:** Unilateral lesion of VIIIth Nerve.

- VIIIth nerve on both sides has a high basal level of activity.
- **LESION the RIGHT VIIIth NERVE:** It will produce an effect similar to **LEFTWARD ROTATION**, i.e. net stimulus of the Left VIIIth nerve:
  - **SYMPTOM:** Leftward Nystagmus

**CALORIC STIMULATION:** Clinical test for functionality of semicircular canals.

- Tilt head back 60 so that the horizontal canals are oriented vertically.
- Irrigate inner ear with water of different temperature, then, **NORMAL RESPONSES:**
  - **Warm Water:** Nystagmus toward the ear being irrigated.
  - **Cold Water:** Nystagmus toward the opposite ear (cold water will inhibit the vestibular receptors)

## THE CEREBELLUM AND BASAL GANGLIA

**CEREBELLAR FIBERS AND CELL TYPES:**

- **AFFERENT FIBERS:**
  - **MOSSY FIBERS:** They make excitatory synapses with **Granule Cells**. They will ultimately influence a large number of Purkinje Cells via the Granule Cell output.
    - Spinocerebellar Tract (motor info about the extremities)
    - Pontine Nuclei (motor info about the head and neck)
  - **CLIMBING FIBERS:** They make *powerful, obligatory* excitatory synapses on a very **SMALL NUMBER of Purkinje Cells**
- **GRANULE CELLS**, also called **PARALLEL FIBERS**, are strictly within the cerebellum, in the folia. They are interneurons in the cerebellar circuit.
  - They make excitatory synapses with a **LARGE NUMBER of Purkinje Cells**
  - They are called parallel fibers because their fibers run parallel to the folia (folds) of the cerebellar hemispheres.
  - Granule cells will also send excitatory signals to the following inhibitory interneurons:
- **PURKINJE CELLS:** EFFERENT FIBERS
  - They are the only “efferent” fibers in the cerebellar cortex. They will make synapses on the Deep Cerebellar Nuclei -----> transmit info to the rest of the CNS.
  - Purkinje cell input onto the Deep Cerebellar Nuclei is **always inhibitory**, using neurotransmitter **GABA**.
- **DEEP CEREBELLAR NUCLEI:** They will project to Thalamus -----> *excitatory* connection to Motor Cortex -----> *excitatory influence* on descending systems.
  - FNXNS:
  - NUCLEI:

**VESTIBULOCEREBELLUM:** Archicerebellum.

- **STRUCTURE: FLOCCULONODULAR LOBE** is the small lobe on the posterior inferior aspect of cerebellum. It modulates VOR and Postural Reflexes.
- **FNXNS:**
  - **MODULATE VESTIBULO-POSTURAL REFLEX:** Cerebellum does fine-tuning of postural reflexes, adjusting the magnitude, strength, and range of movement.
    - Excitatory signals to Vestibulospinal will excite (modulate) the extensors.
    - Excitatory signals to Reticulospinal will excite (modulate) the flexors.
    - Fastigial Nucleus does the calculations for these adjustments, although some fibers also go directly to the descending tracts without going through fastigial nucleus.
  - **SUPPRESS VOR REFLEX:** If you want to follow a moving an object while moving your head too, then you don't want the VOR reflex to move your eyes. Under these circumstances the vestibulocerebellum will suppress that reflex.
  - **ADJUST REFLEX GAIN:** If you put on **reversing prisms** (which reverse the movement of the visual field), your VOR reflex will compensate for the new visual input within a few days. *This change in the VOR reflex is dependent on the Cerebellum and Inferior Olive.*
- **LESIONS:** Caused by a **medulloblastoma** in children, or by chronic **alcoholism** → Produce 2 defects:
  - **Nystagmus** is produced from unmodulated VOR reflex.
  - **Disequilibrium**, an inability to maintain posture, from unmodulated postural reflexes.
- **REPAIR SHOP THEORY:** Based on reversing prisms experiments and others, this theory says that the cerebellum is responsible for continually responding to the changing behavior of neurons with regard to voluntary movements and reflexes.

## **SPINOCEREBELLUM:** Paleocerebellum

- **STRUCTURES**
  - **VERMIS:** Midline structure, receives input from Vestibular Nuclei and Spinocerebellar Tract.
  - **PARAVERMIS:** On either side, receives input only from Spinocerebellar Tract.
- **FNXN:** *Continual correction of movements, comparing actual motor movement with motor cortex "intentions" of movement.*
  - **INPUT:** Spinocerebellum receives input from **motor cortex** and **spinocerebellar tract**.
- **SOMATOTOPIC ORGANIZATION:** The spinocerebellum has somatotopic organization with regard to the regions of the body it is "comparing." Head and neck is generally nearest the center, with extremities in the periphery.

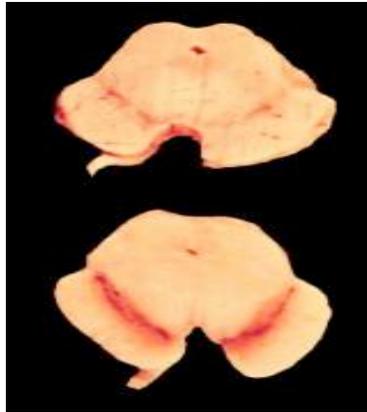
## **CEREBROCEREBELLUM:** Neocerebellum

- **STRUCTURE:** The large lateral hemispheres that make up most of the cerebellum.
- **FNXN:** *Programming of repeated movements.* Calculating the "metrics" of movements, such as reaching for an object in space. We don't consciously think about these details of conscious movement.
  - **INPUT:** *Pontine Nuclei from Cerebral Cortex.*
  - **OUTPUT:** **Dentate Nucleus** -----> **Thalamus** -----> **Motor Cortex**
- **PATHWAY of MODULATION:** **Limbic System** initiates the drive or desire to move
  - *Limbic System* -----> *Frontal Cortex* (conscious or subconscious judgment and intention) -----> *Pontine Nuclei* → *Cerebellum* → **Dentate Nucleus** → *Thalamus* → *Motor Cortex* → *movement is executed*
- **LESION: CEREBELLAR SYNDROME.** This is a lesion of both Spinocerebellum and Cerebrocerebellum, as there is rarely (i.e. never) a lesion of only one or the other.
  - **Hypotonia:** From reduced gamma neuron activity, due to loss of extrapyramidal excitatory input.
  - **Ataxia, Asynergia:** Lack of coordinated movement. Most important, errors in METRICS of movement (reaching arm too far or too close to target)
  - **INTENTION TREMOR, Dysmetria:** A problem terminating movements, or a tremor that only present *when moving*. This is different than Resting Tremor, which occurs with Parkinson's.
  - **Adiadochokinesis:** Inability to make rapid alternating movements, (Pronation & Supination of hand).
- **LESION: IPSILATERAL DEFICIT.** *All lesions of the cerebellum produce ipsilateral deficit because the system is double-crossed.*
  - Deep Nuclei cross the midline to opposite motor cortex.
  - Upper motor neurons cross again through pyramidal decussation.

## **BASAL GANGLIA:** Components

- **STRIATUM:** Uses **GABA** as inhibitory transmitter; it projects to -----> Globus Pallidus to have inhibitory influence.
  - **PUTAMEN**
  - **CAUDATE NUCLEUS**
- **GLOBUS PALLIDUS:**
  - It receives inhibitory input from the Corpus Striatum.
    - *Corpus Striatum* -----> *GPe* -----> *Subthalamic Nucleus* -----> *GPi*
  - **FNXN:** It projects **inhibitory** neurons to the thalamus to modulate motor function.
  - **INTERNAL SEGMENT (GPi):** It is functionally continuous with the Substantia Nigra, Pars Reticularis (SNr)
    - *In Parkinson's Disease, it is overactive.*
  - **EXTERNAL SEGMENT (GPe)**
- **SUBTHALAMIC NUCLEUS:** *The subthalamic nucleus normally stimulates the GPi.*
  - *In Parkinson's Disease, it is overactive.*
  - *In Huntington's and Hemiballismus, it is under active.*
- **SUBSTANTIA NIGRA**
  - **PARS RETICULARE (SNr):** Ventral part of the Substantia Nigra. It is functionally continuous with the Internal Globus Pallidus (GPi)
  - **PARS COMPACTA (SNc): Dopamine-containing neurons.**
    - **CIRCUIT:** SNc -----> *excitatory* on Striatum -----> (Substance P) Inhibitory on GPi -----> *result is inhibition of Thalamic projections* -----> Cerebral cortex doesn't receive the Thalamic projections.
    - **CIRCUIT:** SNc -----> *inhibitory* on Striatum -----> (Enkephalins) inhibitory on GPe -----> -----> *inhibitory to Subthalamic Nucleus* -----> GPi ----> *result is inhibition of Thalamic projections* ----> -----> Cerebral Cortex doesn't receive the Thalamic projections.
- **BASAL GANGLIA Basic Circuitry:** *Cerebral Cortex* -----> **Striatum** -----> **GPi + SNr** -----> -----> *Thalamus* -----> *Motor cortical output*

**PARKINSON'S DISEASE:** A lesion of the Dopamine-containing **Nigrostriatal Tract**, producing a dopamine deficit.



**Parkinson Disease**

**Normal**

- SYMPTOMS: Parkinson's Disease is a **hypokinetic** disorder.
  - **Lead Pipe Rigidity:** Resistance to movement.
  - **RESTING TREMOR:** To be distinguished from an intention tremor (as in Cerebellar Syndrome). This is a tremor when there is no movement.
  - **Akinesia / Bradykinesia:** Inability to initiate movement, or slow initiation of movement.
  - Postural Instability
  - Cognitive Problems
- ETIOLOGY: Deficiency of dopamine exerts its effects through two pathways. *Both pathways ultimately result in over activity of the Globus Pallidus Interna -----> Over suppression of Thalamus -----> fewer thalamic projections to the Motor Cortex.*
  - In Parkinson's Disease, **the subthalamic nucleus is overactive.**
  - Dopamine can get to 20% below normal before Parkinsonian symptoms will occur.
- PROGRESSION OF DISEASE: Cognitive loss and eventual death from respiratory failure.
- TREATMENT:
  - **L-DOPA** is drug of choice.
    - **ON-OFF Phenomenon:** Suddenly therapy is ineffective, periodically.
    - **Freezing Phenomenon:** All of the sudden become rigid and stop, unable to initiate movement.
    - The drug becomes less effective with long-term use.
    - **Dyskinesias:** Involuntary movements (another form of tremors, hyperkinetic) result from the excessive dopamine.
  - **THALAMOTOMY:** Surgical lesion of Ventral Lateral Thalamus, *for the treatment of tremors.* this surgery does not alleviate the original problem, so it is only symptomatic treatment.
    - Must have no cognitive deficiencies and be able to respond well to surgery.
    - This is a surgical alternative to outright Thalamotomy, leaving the VL Thalamus intact and only inhibiting it as necessary.
  - **PALLIDOTOMY:** Surgical lesion of Globus Pallidus Interna. This surgery has been shown to be the most effective.
- HISTORICAL STUFF:
  - **Stereotactic Surgery:** Early attempts to place a brain lesion in specific place, using a *pineal calcification* (on a skull film) as a reference point.

**HUNTINGTON'S CHOREA:** Lesion of the Corpus Striatum, involving GABA and **Enkephalin** neurons

- ETIOLOGY: Loss of Corpus Striatum GABA/Enk neurons -----> Disinhibition of the Globus Pallidus Externa -----> *Excessive inhibition of the Subthalamic Nucleus.*
  - **The subthalamic nucleus is deficient**
- SYMPTOMS: Huntington's is a **hyperkinetic** disorder.
  - The result is a lesion similar to the effects of a Subthalamic Nucleus lesion (i.e. Hemi-ballismus).

**HEMIBALLISMUS:** Lesion of Subthalamic Nucleus, ruins the GPi inhibitory neurons that project to the thalamus -----> over excitation.

- SYMPTOM: Violent, involuntary movement of contralateral limb.
- ETIOLOGY: Usually created by a vascular lesion (stroke) specific to the Subthalamic Nucleus.
  - **The subthalamic nucleus is deficient**