Clinical Localization of Acute Stroke

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Clinical Localization

- Topographic diagnosis:
  - Determination of where in the nervous system damage has occurred
  - Basis of neurologic diagnosis developed in the pre-imaging era
- Precedes and complements neuroimaging
  - Prevents overlooking pertinent clinical findings, especially in patients with more than one etiology for their presentation
  - Directs appropriate use of imaging technologies (i.e. monocular blindness = interrogate the ICA)
  - Noncontrast CT is insensitive to acute ischemia making clinical localization an essential element to diagnosing stroke; directs attention to subtle – otherwise less noticeable – changes on noncontrast CT

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History of Localization

- Oldest known document relating to clinical localization pertains to aphasia
  - Recorded on Egyptian papyrus and dates to the age of the Pyramids (~3000-2500 BC)
  - Describes invasive injury in the region of the left temple affecting an ability to understand verbal content, but an inability to verbally express oneself with language

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History of Localization

- In Greece, dating to the time of Hippocrates, there is evidence of knowledge that injury to the left part of the brain results in motor weakness of the right side of the body
  - Rationale for this given at the time was that paired organ systems (e.g. 2 brain hemispheres) had identical functions

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History of Localization

- Paul Broca (1824-1880) provided the first glimpse of clarity that challenged the thinking around "paired organs" in his description of lateralization of language to the left hemisphere
  - Used the term "aphemia" to describe what is now called Broca's aphasia (1865)
  - Localized the area of the brain involved to the third, left frontal convolution (inferior frontal gyrus)
- Carl Wernicke (1848-1905) described a broader area for language localization in 1874:
  - Broca's area in the frontal lobe was defined as housing "motor language"
  - Enlarged the area housing language function to include areas extending posteriorly to parieto-temporal and occipital regions that were associated with "word images"
    - Attributed the function of these areas to be related to auditory and visual reception of language, noting that injury to the brain anywhere between the triangle connecting Broca's area to the parieto-temporal or occipital language areas will result in an aphasia of different mechanisms

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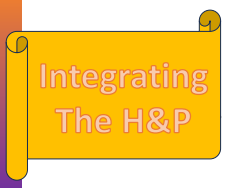

- History:
  - Contributes information about the temporal evolution of the deficits
  - Sudden onset: Suspect neurovascular origins or epilepsy
  - Development over the course of several days: Suspect infection or demyelinating disease
    - Development over the course of weeks or months: Suspect tumor or degenerative disease

Physical:

- Localization to area in the brain
- Vascular territories implicated, OR whether findings are likely not associated with a specific vascular territory (focal, multi-focal, or diffuse)
- Authenticity of symptoms

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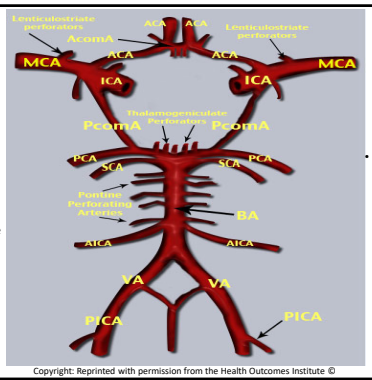
Historically, up to 50% of the population were thought to have an incomplete circle of Willis

Recent CTA study showed that in stroke patients about a third have an incomplete circle of Willis; most common variant = fetal origin of the PCA



- Poorly or incompletely developed segments are common at:
  - A1 branch of the ACA
  - P1 branch of the PCA
  - Posterior communicating arteries

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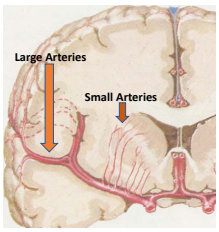
Large Arteries & Small Perforating Arteries



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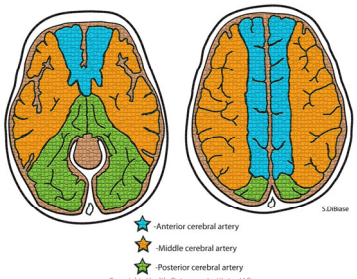
Cerebral Cortex and Subcortex Vascular Distribution



-Anterior cerebral artery
-Middle cerebral artery
-Posterior cerebral artery

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Cerebral Cortex (Telencephalon)

- The anterior circulation receives 80% of the brain's blood flow
- Consists of 2 hemispheres separated by the longitudinal fissure and joined by the corpus callosum; separated from the cerebellum by the transverse fissure
- Largest part of the brain (80% of the brain's weight)
- Thin outer layer of gray matter = cortex
  - $^2/_3$ of the cortex is buried in convolutions (gyri)
  - Contains the cell bodies of neurons
  - White matter situated below gray matter and is made up of neuronal axons

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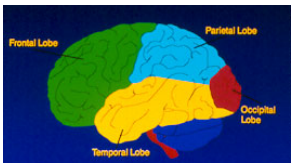
Cerebral Cortex

- Fissures and sulci (less deep divisions) separate the cerebrum into 6 distinct lobes:
  - Frontal
  - Parietal
  - Temporal
  - Occipital
  - Insula (island of Reil)
  - Rhinencephalon (limbic)
- Main functions:
  - Association – intellect, memory storage, language, linkage of motor/sensory areas
  - Motor – all voluntary and some involuntary motor function
  - Sensory – interpretation of environment



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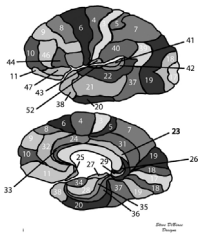
Brain Cytoarchitecture



- Emerged in1909; developed by Korbinian Brodmann, German neurologist
- System is incomplete, yet offers a method to label and localize physiology
- Both discreet and indiscreet functional boundaries are detailed

"Vergleichende Lokalisationslehre der Großhirnrinde in ihren Prinzipien dargestellt auf Grund des Zellenbaues" (Comparative Localization Studies in the Brain Cortex, its Fundamentals Represented on the Basis of its Cellular Architecture); 1909.

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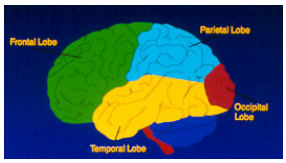
Frontal Lobe Localization



- Voluntary motor function
- Language expression
- Higher intellectual function

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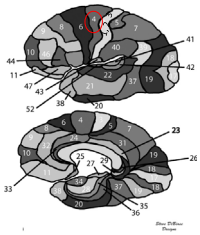
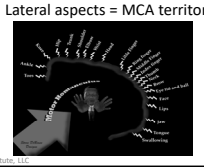
Brodmann Area 4: Voluntary Motor Function



- Precentral gyrus of the frontal lobe
  - Motor homunculus
  - Medial superior aspects = ACA territory
  - Lateral aspects = MCA territory



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Corresponding Assessments

- Facial symmetry:
  - Variety of facial expressions examined
  - MCA distribution primarily affects the lower face
    - Central VII loss
    - Commonly spares the upper face (scant direct cortical innervation provided to the upper face)
  - Suspect pontine stroke with loss of both upper and lower face (nucleus of CN VII affected)
  - Normal face with weak extremities likely suggests a stroke mimic diagnosis
- Extremity motor grades:
  - 0/5 = no movement
  - 1/5 = flicker of movement
  - 2/5 = cannot overcome gravity
  - 3/5 = can overcome gravity, but cannot overcome resistance
  - 4/5 = overcomes resistance but weak
  - 5/5 = normal power

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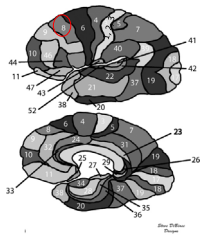
Brodmann Area 8: Frontal Eye Fields

- The frontal eye fields are responsible for saccadic eye movements and head turning
  - Allows the eyes to rapidly move together to surveil visual content
- Interruption of this area of the brain causes conjugate eye deviation toward the affected MCA territory
- In contrast, brainstem stroke may cause more unusual eye deviation patterns
  - Lateral deviation, ptosis and pupillary dilation (CN III; oculomotor)
  - Upward migration and vertical diplopia that worsens when attempting to look down (CN IV; trochlear)
  - Midline eye deviation with limited lateral gaze producing diplopia (CN VI; abducens)



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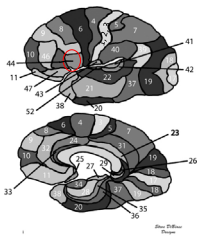
Brodmann Area 44: Language Expression

- MCA territory; regarded as Broca's territory
- Most commonly in the left (dominant) hemisphere, even if left handed
- Written and spoken language
- Assess word finding
- May be accompanied by dysarthria (Brodmann Area 4)



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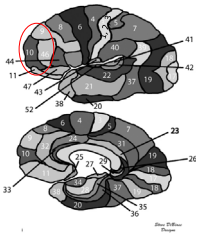
Brodmann Areas 9-11: Cognition



- ACA territory
- Specific components:
  - Orientation – time, place, person
  - Memory – short term, long term
  - Insight – understanding the significance of things
  - Judgment – decision making
  - Arithmetic and abstraction
- Extensive connections with the thalamus and hypothalamus – exerts strong influence on visceral activities and emotional responses to stimuli

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Localizing Findings to the Frontal Cortex

Clinical Findings	Neurovascular Territory
Arm and facial weakness	Contralateral MCA
Pure leg weakness (rare)	Contralateral ACA
Co-extensive arm, face and leg weakness	Contralateral distal ICA or proximal M1 MCA
Loss of language fluency	MCA (usually on the left)
Binocular eye deviation toward the side of the brain affected by stroke	MCA
Cognitive loss (rare)	ACA

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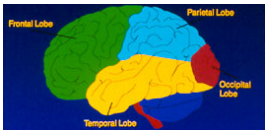
Parietal Lobe Localization

- Primary tactile sensory and spatial sensation function
  - Receives sensory information from sensory receptors in the skin and joints
  - Information is relayed to the parietal lobe by way of the thalamus
- True discrimination:
  - Position of body in space
  - Size, shape and texture
  - Intensity and locality
  - Tests are "classically" performed with the eyes closed



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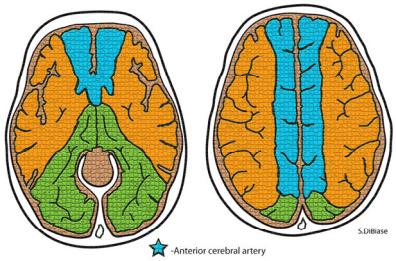

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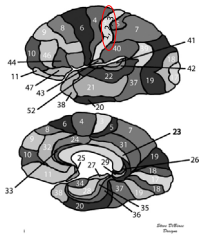
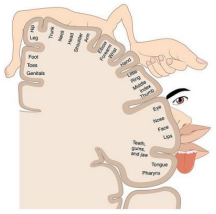
Brodmann Areas 1, 2, 3: Primary Somatosensory Cortex

- Tactile sensation:
  - Pinprick
  - Vibration
  - Temperature
  - Position sense
  - Touch





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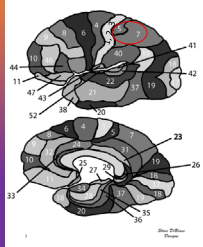
Brodmann Areas 5 & 7: Somatosensory Association



- Deciphers/interprets sensory stimuli at a deeper level:
  - Double simultaneous stimulation testing
  - Graphesthesia testing
  - Stereognosis testing

Greater distribution of perceptual function most commonly resides in the right cortical hemisphere; distinguish gaze preference associated with neglect, from gaze deviation

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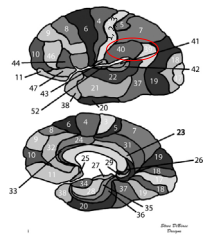
Brodmann Areas 39 & 40: Receptive Language

- MCA territory; regarded as constituting Wernicke's area for receptive language
- Significant networking:
  - Auditory (temporal)
  - Visual (occipital)
  - Intellect (frontal)
  - Somatosensory cortex
  - Alerting (limbic)
- Area 39 = reading center and arithmetic functions; dysfunction causes dyslexia, dysgraphia, and/or dyscalculia (can be inherited)
- Area 40 = connects to the somatosensory association area; dysfunction causes confusion with left and right (can be inherited)
- Gerstmann's syndrome = acquired neurologic insult causing area 39/40 dysfunction; symptoms as above as well as apraxia



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Localizing Findings to the Parietal Lobe

Clinical Findings	Neurovascular Territory
Arm and facial sensory loss	Contralateral MCA
Pure leg sensory loss (rare)	Contralateral ACA
Co-extensive arm, face and leg sensory loss with same distribution motor loss	Contralateral distal ICA or proximal M1 MCA
Loss of language reception (fluent aphasia)	MCA (usually on the left)
Spatial neglect	MCA (usually on the right)
Global aphasia	MCA (usually on the left with loss of arcuate fasiculus connection between Wernicke's and Broca's areas)
Gerstmann's syndrome	Dyslexia, dysgraphia, dyscalculia, left-right confusion, apraxia

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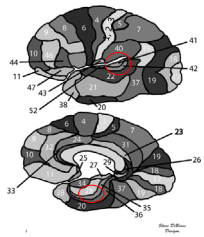
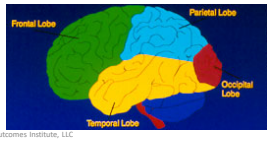
Temporal Lobe Localization



- Vascular territory: MCA
- Area 41 = Primary auditory reception
- Area 28 = Primary olfactory area (rhinencephalon)



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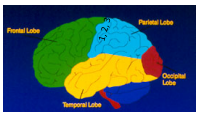
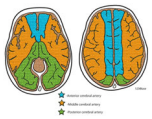
Occipital Lobe Localization

- Primary visual lobe
  - Receives input from the optic nerves and special nuclei of the thalamus to visually interpret findings
  - PCA territory
  - Visual impulses travel through the MCA territory to the occipital lobe
    - Large MCA strokes may cause visual field deficits
    - Differentiate MCA visual defects from pure PCA infarction by the addition of common MCA stroke findings (motor and sensory loss, aphasias, neglect)





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The Course of Visual Stimuli

Retinal ganglionic cells for vision

⇩

Optic nerve fibers

⇩

Chiasm

⇩

Optic tract

⇩

Lateral geniculate bodies

⇩

Brodmann area 17 ⇨ Brodmann area 18

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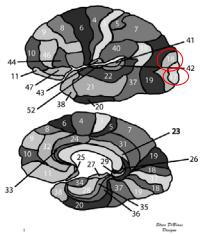
Brodmann Areas 17 & 18

- Area 17 (Calcarine):
  - Primary visual cortex
  - Receives all visual information from the neurons of the lateral geniculate body
- Areas 18:
  - Visual association cortex
  - Integrates all visual information received
- Right occipital cortex receives impulses from the left half of each eye; left occipital cortex receives impulses from the right half of each eye
- The central 10 to 15 degrees of vision constitute 50-60% of the total surface area of the occipital cortex



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Visual Localization Rules

1. Confrontation must be used in patients with poor attention, cognitive limitations that affect cooperation, or in cases of altered consciousness
2. Defects of one eye (monocular) = retina or optic nerve; interrogate the ipsilateral ICA
3. Binasal hemi- or quadrantinopia is usually due to intraocular disease or third ventricular enlargement in hydrocephalus
4. Defects of both eyes (binocular) = chiasm or beyond; homonymous hemianopias are always beyond the chiasm
5. Presence of a Marcus Gunn pupil suggests optic tract involvement, instead of lateral geniculate, optic radiation or occipital locations
6. Bitemporal field defects are not of vascular origin; suspect compressive mass of the optic chiasm (i.e. pituitary adenoma)
7. Patients with purely occipital infarction are usually aware of their field defect, while those with more anterior involvement affecting the parietal lobe and visual association cortex may remain unaware of the loss of a visual field: Anton's syndrome (hemianoptic anosognosia)
8. Diplopia is almost always caused by lesions affecting the ocular movement system in the brainstem (CN III, IV, VI)

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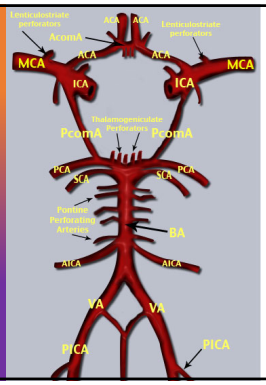

Cortical Blindness

- May result from a single or consecutive events
- Most often the result of simultaneous or successive PCA occlusions
- Sudden onset of cortical blindness in a patient with previously normal visual fields suggests top of the basilar occlusion

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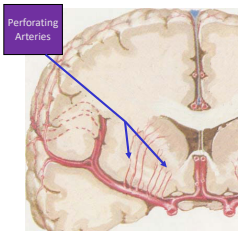
Subcortex of the Brain

- The subcortex is packed with ascending sensory fibers and descending motor fibers, and the basal nuclei
- The diencephalon (thalamus and hypothalamus) sits posterior to the internal capsule
- Lesions affecting the subcortical region commonly result in either:
  - Pure sensory deficits;
  - Pure motor deficits; or,
  - Combined sensori-motor deficits
- Uncommonly, aphasia resulting from subcortical infarction may occur with thalamic infarct intersecting motor speech pathways



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Cerebellum

- The cerebellum is the 2nd largest part of the brain, representing 10% of the total mass
- Consists of 2 lateral hemispheres and a connecting portion, the vermis
- Receives motor cortex stimuli and spinocerebellar tract proprioceptive feedback related to the position of the body in space; uses these data to make appropriate postural adjustments
- Function of the cerebellum:
  - Fine coordination of muscle movements
  - Maintenance of muscle tone and posture
  - Impulses from the vestibular apparatus in the inner ear are continually delivered and processed to maintain equilibrium

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Cerebellar Dysfunction

- Lesions cause feedback deficits that affect alterations in equilibrium and coordination, but are less debilitating than primary motor strip or motor pathway lesions
- Ataxia – Inability coordinate motor function
  - Loss of gross motor coordination
  - Loss of fine motor coordination
  - Dysmetria with over and under-shooting of motor responses
- Cerebellar ipsilateral limb ataxia is a classic finding
- Often associated with brainstem findings

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Clinical localization of Cerebellar Stroke

- Typically vertiginous presentation
- Pure cerebellar stroke typically presents with an alteration in motor coordination that is not associated with motor weakness, sensory loss, or cranial nerve dysfunction
- Large cerebellar strokes are associated with significant swelling and are deemed a neurosurgical emergency
- Cerebellar stroke often occurs in association with brainstem stroke
  - Perforators from all the cerebellar arteries have branches that penetrate the brainstem
  - Cranial nerve dysfunction, motor/sensory loss suggest brainstem dysfunction

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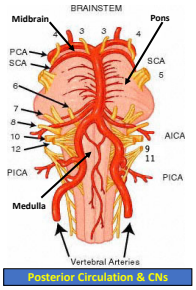
Brainstem

- Major control centers:
  - Level of consciousness (ascending reticular formation)
  - Cardiac
  - Respiratory
  - Vasomotor
  - Vomiting
  - Sneezing
- Sensory (ascending) and motor (descending) pathways
- Cranial nerves III-XII



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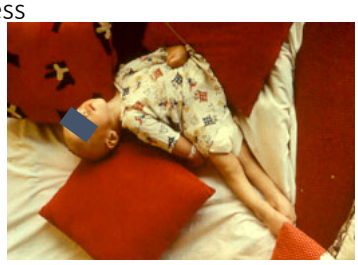
Level of Consciousness: Degree of Wakefulness

- Alert: Wide awake
- Lethargic: Sleepy, dull, indifferent
- Obtunded: Deep sleep, difficult to arouse
- Stupor: Unable to arouse; responds normally to noxious stimuli
- Coma: Unable to arouse; doesn't respond or responds abnormally to noxious stimuli:
  - Decorticate - abnormal flexion
  - Decerebrate - abnormal extension



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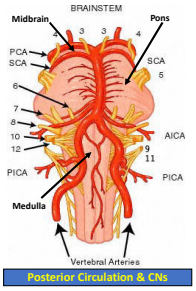
Brainstem Vascular Supply

- Midbrain:
  - BA
  - P1-PCA (primary source)
- Pons:
  - BA paramedian branches (midline)
  - BA short circumferential branches (lateral aspects)
  - BA long circumferential branches (dorsolateral aspects)
  - AICA (dorsolateral aspects)
  - SCA (rostral aspects)
- Medulla:
  - VA
  - PICA (lateral aspects)
  - Spinal arteries (caudal aspects)



Posterior Circulation & CNs

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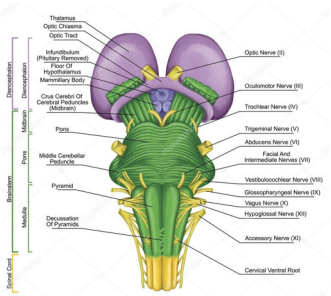
Midbrain (Mesencephalon)

- Extends from the pons to the diencephalon and contains aspects of the substantia nigra and red nucleus
- Visual reflex centers for head and eye movement in response to visual and auditory stimuli
- Nuclei of CNs III (oculomotor), IV (trochlear); Edinger-Westphal pathway of III CN for pupillary constriction
- Midbrain stroke:
  - EOM dysfunction: Loss of calorics; absent doll's eyes
  - Pupillary dilation
  - Decreased LOC including mass response
  - Motor/sensory dysfunction



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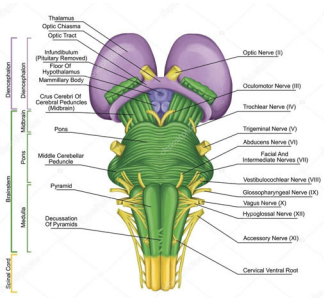
Pons (Metencephalon)

- Lies inferior to the midbrain and above the medulla oblongata, with its posterior aspects forming a wall of the 4th ventricle
- Corticospinal tract in ventral pons – may produce "pure motor" stroke
- CNs V (trigeminal), VI (abducens), VII (facial)
- Horizontal gaze center: medial longitudinal fasciculus (MLF) extends from the pons to the midbrain, connecting nuclei of CNs III, IV & VI with vestibular and pontine paramedian reticular formation
- Apneustic center – lower pons; pneumotaxic center – upper pons
- Pontine stroke:
  - EOM dysfunction; absent calorics/doll's eyes
  - Decreased LOC including mass response
  - CN and motor/sensory dysfunction, including "locked in syndrome"
  - Respiratory dysfunction with central hyperventilation or apneustic gasping



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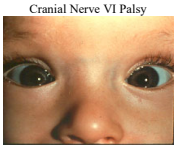
Extraocular Movement Dysfunction

- Isolated CN III, IV or VI nuclear lesions are rare; most commonly, other structures situated in close anatomical proximity to these lower motor brainstem nuclei will likely be involved
- Brainstem stroke causes unusual eye deviation patterns:
  - Lateral deviation, ptosis and pupillary dilation (CN III; oculomotor)
  - Upward migration and vertical diplopia that worsens when attempting to look down (CN IV; trochlear)
  - Midline eye deviation with limited lateral gaze producing diplopia (CN VI; abducens)

Cranial Nerve III Palsy

Cranial Nerve VI Palsy



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Diplopia

- Most common subjective complaint elicited by lesions in the ocular motor system
- Occurs most frequently with lesions of the extraocular muscles or ocular motor nerves than with supranuclear brainstem lesions that more commonly cause gaze palsies
- Results from lack of visual fusion with the perceived object projected to non-corresponding points of the retina, causing either a double image, or more commonly, a blurry image

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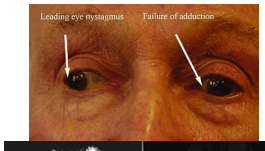
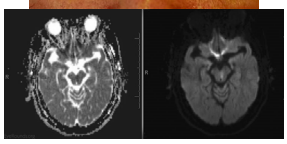
Internuclear Opthalmoplegia (INO)

- Dysconjugate gaze palsy also called, medial longitudinal fasciculus (MLF) syndrome
- Usually monocular in ischemic stroke; binocular presentation is most common in MS
- Presentation:
  - Ipsilateral adduction weakness due to MLF lesion; may have transient clockwise nystagmus
  - Monocular nystagmus is present in the normal/leading eye when abducting and is likely reflexive in nature
  - May have no symptoms; others may have diplopia with or without oscillopsia





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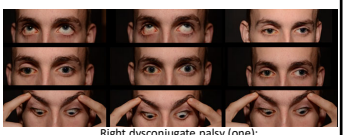
One and a Half Syndrome

- Another form of dysconjugate gaze palsy:
  - Dysconjugate gaze palsy to one side (one)
  - Impaired adduction upon looking to the other side (half)
  - The only horizontal movement remaining is abduction of one eye, which also shows nystagmus during abduction
- Involves the MLF and abducens
- When facial palsy is present the syndrome is called, "eight and a half" syndrome
- MS, ischemic infarction, hemorrhage, AVMs and tumors are common causes

Left dysconjugate palsy (one); limited right adduction (half); right eye abduction remains intact



Right dysconjugate palsy (one); limited left adduction (half); left eye abduction remains intact

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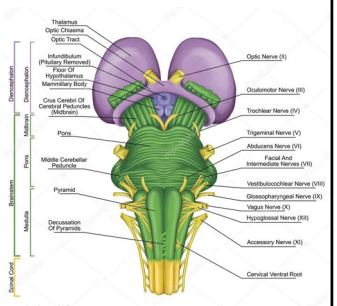
Medulla Oblongata (Mylencephalon)

- Most inferior aspect of the brainstem, continuous caudally with the spinal cord.
- Pyramids are prominent anterior bulges of white matter lie laterally on each side and contain the fibers of the corticospinal (UMN) tract.
- Nuclei of CNs VIII, (vestibulocochlear-acoustic) IX (glossopharnygeal), X (vagus), XI (spinal accessory), and XII (hypoglossal).
- Cardiac, vasomotor and respiratory centers.
- Medullary stroke:
  - Respiratory dysfunction; cardiac and vasomotor instability
  - CN dysfunction with dysphagia
  - Motor/sensory dysfunction
  - Decreased LOC including mass response



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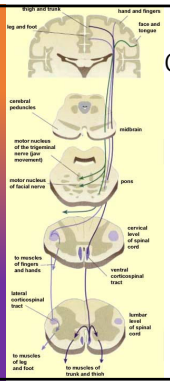
Corticobulbar & Corticospinal Tracts



- Two descending cortical tracts for voluntary motor function:
  - Corticobulbar tract – originates in the lower third of Brodmann area 4 and terminates at brainstem motor nuclei for cranial nerves innervating the face, tongue, jaw (V, VII, IX, X, XI, XII)
  - Corticospinal tract – decussation in the pyramids of the medulla oblongata; terminal point is lower motor neurons in the spinal cord
- Bilaterality:
  - Pontine stroke may produce ipsilateral facial weakness (upper and lower face) and contralateral arm and leg weakness

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Basilar Artery Thrombosis

- Most commonly misdiagnosed of all ischemic infarction presentations
- Findings may include any/all of the following:
  - Vertigo
  - Hiccups
  - Shivering
  - Motor/sensory loss
  - Cranial nerve palsies
- Most severe symptoms include:
  - Respiratory failure
  - Decreased level of consciousness, including coma
  - Locked-in syndrome
  - Cortical blindness with bilateral PCA occlusions

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Brainstem/Cerebellar Stroke Syndromes

- Lateral Medullary Syndrome (Wallenberg's Syndrome):
  - First described in 1895; vascular territory = vertebral or PICA
  - Typified by vertigo, nausea/vomiting, dysphagia, nystagmus and ipsilateral hemi-ataxia. Other findings may include:
    - Ipsilateral headache
    - Contralateral body hemianalgesia with ipsilateral facial hemianalgesia
    - Ipsilateral palate, vocal cord weakness (hoarseness)
    - Uncontrollable hiccups
  - Key: Motor, tongue, and dorsal column function spared because these structures lie medially in the medulla
- Anterior Inferior Cerebellar Artery syndrome:
  - Vertigo, unilateral ipsilateral deafness (labyrinthine artery ischemia), ipsilateral facial weakness, and ataxia
- Superior Cerebellar Artery syndrome:
  - Ipsilateral cerebellar ataxia, nausea and vomiting, dysarthria, loss of pain and temperature over the opposite side of the body
  - May also present with auditory loss, upper extremity tremor and Horner's syndrome

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Differentiating Findings

- Loss of sensory and/or motor loss that is all on the same side involving the face and extremities indicates a lesion in the cortex, subcortex, thalamus or upper brainstem
- Cardinal brainstem stroke findings = Bilaterality; cranial nerve deficit on the same side as the infarct with opposite side extremity weakness and/or sensory loss
- Uncommon stroke findings may indicate a brainstem lesion: Auditory loss; vertigo; diplopia; dysconjugate gaze; other CN findings
- Sudden loss of consciousness in non-hemorrhagic stroke may indicate brainstem stroke
- Lower facial findings with preservation of the upper face are suggestive of either upper brainstem, subcortical and cortical findings
- Loss of both upper and lower hemi-facial motor function is suggestive of pontine stroke
- Associated aphasia and/or neglect suggest cortical stroke
- Visual loss must be carefully distinguished from visual neglect; neglect suggests cortical stroke, whereas visual loss could be posterior circulation or anterior circulation stroke depending on other associated symptoms

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