Neuro Nugget: Stroke

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Neuro Nugget: Stroke

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Stroke: Definitions & Background

- Stroke is also called **Cerebrovascular Accident** (CVA)
- Stroke is the clinical designation for a rapidly developing loss of brain function due to an interruption in the blood supply to all or part of the brain.
- WHO defines a stroke as a "neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours"
- If < 24 hrs then it is a **Transient Ischemic Attack** (TIA)
- If > 24 hrs of deficits and they resolve, called a **Reversible Neurological Ischemic Deficit** (RIND)
    - Prior to 24 hrs when it is unknown if stroke or TIA, treat as stroke
    - TIA was developed for research purposes. Stroke, TIA and RIND are the same disease process
    - 1/3 of pts with TIA will have a stroke within 5 years
    - In some med centers stroke-in-progress is called a "brain attack" analogous to a "heart attack. They have "Brain Attack Teams" working out of Neurology Dept.
- Strokes are Medical Emergency - intervene early: "**Time is Brain**" refers to the increasing loss of brain tissue the longer the delay in treatment.
- Stroke is the third most common cause of death in US and industrialized West.
- Stroke Types are divided into:
    - **Ischemic Strokes** – 80% of all strokes
    - **Hemorrhagic Strokes** – 20% of strokes

Stroke: Risk Factors

Modifiable risk factors

- High blood pressure
- Cigarette smoking
- Transient ischemic attacks
- Heart disease
- Diabetes mellitus
- Hypercoagulopathy
- Carotid stenosis
- Cocaine -doubles stroke risk

Non-modifiable risk factors

- Age
- Gender
- Race
- Prior stroke
- Heredity

Strokes: Ischemic

- Ischemic Stroke Categories
    - Thrombotic (artery narrowed by thrombus formation)
    - Embolic (occlusion of artery, partial or complete, by a traveling clot or plaque)
    - Systemic hypoperfusion – leads to watershed (or Borderzone) strokes
- Ischemic strokes have three sources of pathology
    - Cardiac (pump)

- Vascular (vessel)
- Hematologic (fluid)

- The Ischemic Cascade: This is how the brain dies during ischemia
  - Ischemic neuron depolarizes and calcium floods the cell
  - Calcium leads to release of neurotransmitters, including excitatory ones.
  - This leads to further depolarization of surrounding neurons
  - Destructive enzymes are released leading to cell membrane destruction and death of cell
  - Within hours, certain genes are activated in the cells in the ischemic penumbra, which form cytokines.
  - This leads to an inflammatory response that compromises the microcirculation of these surrounding regions
  - Thus, the cells in this penumbra begin to die. Thus, the penumbra joins the infracted core.
  - The goal of treatment is to abort this cascade and save the penumbra, thus minimizing the brain damage.
  - Below are descriptions of the specific types of strokes.

Strokes: Thrombotic

- Thrombotic clots usually form at site of pre-existing arteriosclerotic plaque
- They cut off blood supply to distal brain tissue, depriving of glucose (brain food) and oxygen
- Onset: Since thrombus formation is often gradual, **thrombotic stroke symptom onset may be gradual**
  - Note that thrombus can break off, leading to embolic stroke distal to thrombus
- Thrombotic strokes divided into two types:
  - **Large Vessel Disease**: These occur in the internal carotid and the large vessels off the circle of Willis. The following disease states (incomplete list) cause formation of thrombi
    - **Atherosclerosis** (chronic inflammatory response due to deposition of lipoproteins; three parts: atheroma – macrophages, cholesterol crystals, calcifications)
    - **Vasoconstriction** (internal response or triggered by substances – cocaine)
    - **Dissection** (tearing of artery wall caused by HTN or connective tissue disease)
    - **Takayasu Arteritis** (inflammatory, cause unknown, in Asian females ages 15-30, signs: wreath-like formation of new blood vessels in retina; symptoms: night sweats, fever, weight loss, fatigue
    - **Giant Cell Arteritis** or Temporal Arteritis: (Inflammatory disease with giant cells found on bx, effecting medium and large arteries of the head, mostly temporal artery)
    - **Moyamoya Syndrome** (rare except in Japan, cause unknown, progressive stenoses of Circle of Willis; leads to strokes, migraine-like HA's)
    - **Fibromuscular Displasia**
  - **Small Vessel Disease**
    - **Lipohyalinosis** (lipid hyaline build-up secondary to HTN and aging) and fibrinoid degeneration (deposits of acidophilic fibrin-like material leading to small strokes known as lacunar infarcts)
    - **Microatheromas** (atheromas that extend from larger arteries into the smaller arteries)

Stroke: Embolic Stroke

- Emboli sources: broken off blood clots, atherosclerotic plaques, bacterial emboli (endocarditis), cancerous emboli, fat or bone marrow emboli (following injury)
- Onset: Since an embolus lodges quickly, **embolic stroke onset is sudden and maximal at onset**

- Emboli from the anterior circulation most often occlude the Middle Cerebral Arteries, since 85% of anterior circulation blood flow goes there.
- Treatment Approach
    - Treat embolism
    - Identify source of embolus and treat source
- Categories: embolic strokes may be divided into 4 categories based on clinical presumption of source
    - **Cardiac Source**: Atrial fib, rheumatic mitral or aortic valve disease, mechanical valve disease, atrial or ventricular thrombus, sick-sinus syndrome, recent MI, CABG, infective endocarditis, nonbacterial endocarditis such as from adenocarcinoma leading to valvular vegetations
    - **Potential Cardiac or Aortic Source:** patent foramen ovale
    - **Arterial Source:** aortic dissection
    - **Unknown Source**

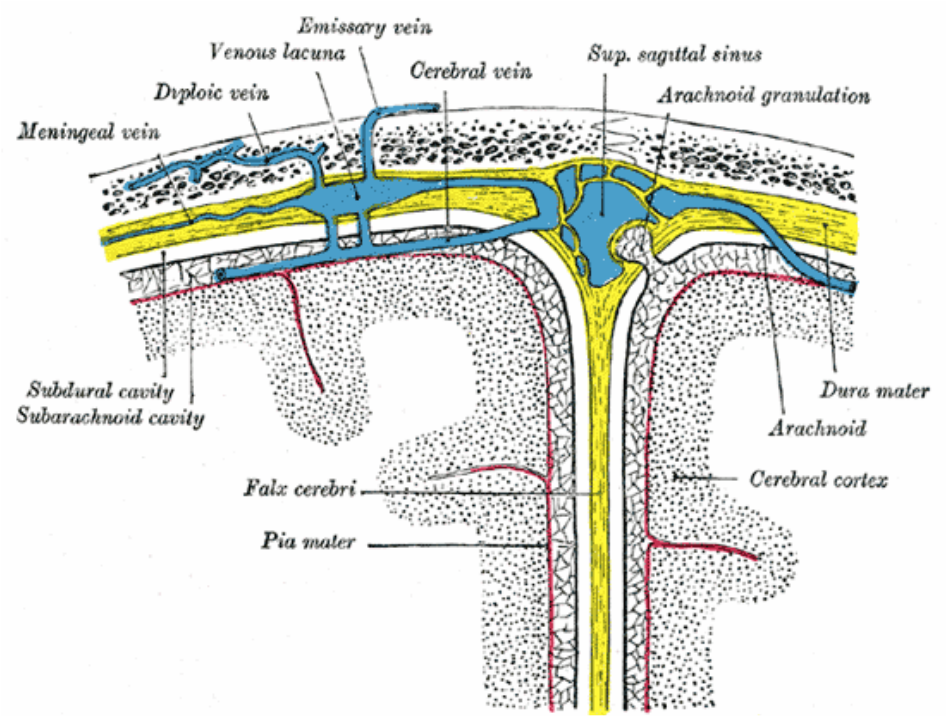
Stroke: Watershed Stroke

- Caused by **systemic hypoperfusion**: Cardiac arrest, arrhythmia, MI, CHF, bodily injury, surgery
- Borderzone brain areas are the areas that are most distal, in between the arterial territories.

Stroke: Hemorrhagic Strokes

- **Intracerebral Hemorrhage**
    - Arterial bleed
    - Causes symptoms by compressing surrounding brain tissue, causing ischemia, and by direct destruction of tissue.
    - S/S: severe headache, decreased LOC, deficits do not follow arterial distribution
- **Subarachnoid Hemorrhage**
    - Arterial bleed
    - Causes effects through 1. elevating intracranial pressure and 2. toxic effects of the subarachnoid blood on the brain tissue, and 3. secondary vasospasm leading to ischemia
    - S/S: usually presents with headache rather than focal neurological deficits
- **Subdural & Epidural Hemorrhages**
    - Venous bleed
    - Causes a mass lesion that compresses brain
    - Often cause is head trauma
    - S/S: present with headaches, altered LOC, including coma, or if not acute, with neuropsychiatric symptoms

Illustration of Dura, Arachnoid, and Venous Sinuses



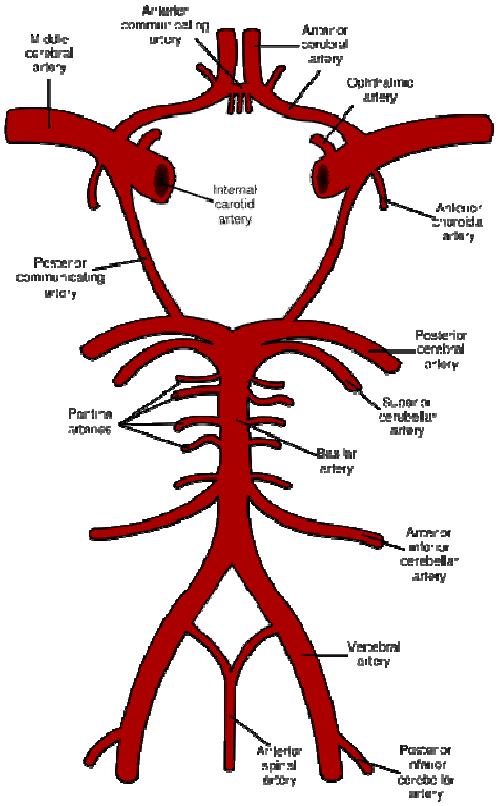
Stroke: Localization

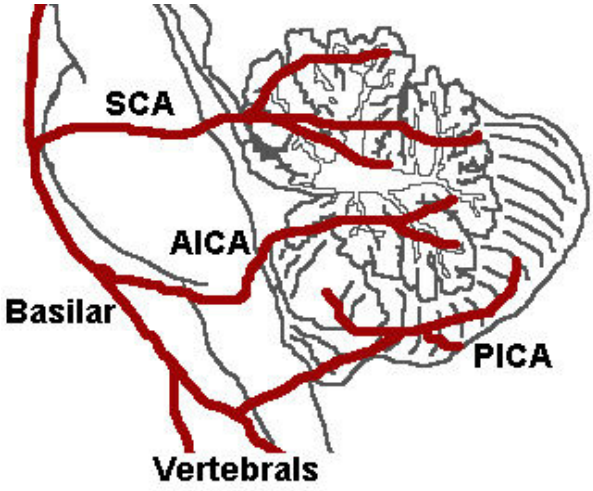
Arterial Supply	Structures Supplied	Clinical Stroke Syndrome
Anterior Circulation		
1. Anterior Choroidal	Hippocampus, globus pallidus, lower internal capsule	1. hemiplegia, 2. hemianesthesia, 3. homonymous hemianopia
2. Anterior Cerebral	Medial frontal & parietal cortex, anterior corpus callosum	1. Contralateral leg 2. bladder center
3. Middle Cerebral	Lateral frontal, parietal, occipital, temporal cortex	
3a. Superior Division	Motor-sensory cortex & expressive language	1. Contralateral **face & arm** 2. Contralateral hemianesthesia No homonymous hemianopia Broca's aphasia (dominant)
3b. Inferior Division	Visual Radiations, macular visual cortex, receptive language	1. **Wernicke's aphasia** (dominant), 2. **Gerstmann Syndrome** of dominant parietal lobe (agraphia, acalculia, right-left confusion, finger agnosia, ideomotor apraxia), 3. Homonymous hemianopia
3c. Lenticulostriates	Caudate nucleus, putamen, **internal capsule**, deep white matter	1. Contralateral **face, arm, & leg** 2. Extrapyramidal mov't s/s

Combined MCA Occlusions: Occlusion at MCA Bifurcation (combined superior and inferior division syndromes); Occlusion at stem of MCA (combined superior and inferior division and lenticulostriate syndromes)		
Posterior Circulation		
1. Basilar	Posterior occipital and medial temporal lobes, thalamus, brain stem, cerebellum	**Top of the Basilar Syndrome**: 1. paralysis of all mov't except lateral eye gaze and eyelid opening, 2. intact sensation, 3, intact consciousness
1a. Posterior Cerebral	Medial occipital & temporal cortex, post. corpus callosum	**Anton Syndrome:** bilateral lesions: 1. Cortical blindness (pupils still react to light) 2. denial of blindness **Weber Syndrome**: 1. Contra arm & leg weakness, 2. Ipsi CN III (lateral gaze) palsy
Thalamoperforates	Thalamus	**Dejerine-Roussy or Thalamic Pain Syndrome:** 1. hemisensory loss in all modalities, 2. hemibody pain
Thalamogeniculates	Thalamus	Thalamic Syndromes
1b. Superior Cerebellar	Midbrain, pons, cerebellum	
1c. Anterior Inferior Cerebellar	Pons, cerebellum	**Marie-Foix Syndrome**: 1. Ipsi arm & leg ataxia, 2. Contra arm & leg weakness, 3. Contra hemisensory loss
2. Vertebral		
2a. Posterior Inferior Cerebellar	Medulla, cerebellum	**Wallenberg syndrome:** 1. dysarthria, 2. dysphagia, 3. hoarseness 4. contra pain/ temperature from body, 5. Ipsi pain/temperature from face; 5. Ipsi Horner's, 6. vestibular signs – vertigo, nystagmus, nausea
2b. Vertebral branches	Medial Medulla	**Dejerine Syndrome:** 1. Contra arm & leg weakness, 2. Contra hemisensory loss position and vibration, 3. tongue weakness (CNXII)
Strokes NOT Associated with major cerebral artery distributions		
Lacunar strokes	Small vessels supplying deep brain and white matter;	

Ipsi = ipsilateral, Contra = contralateral

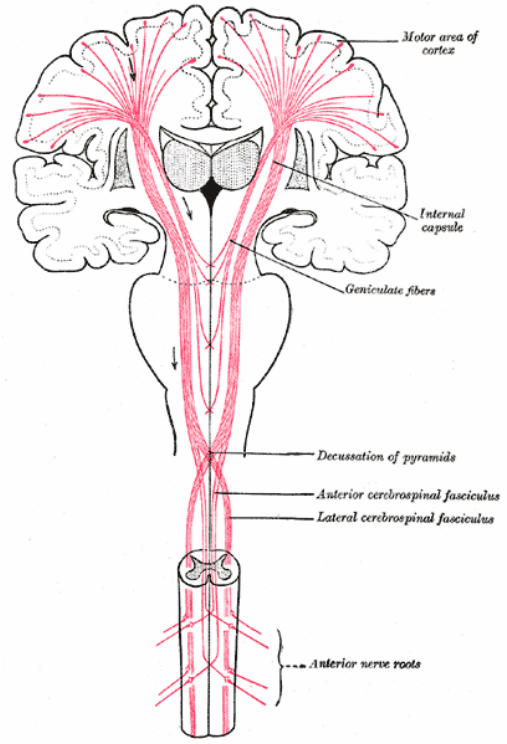
Stroke: Blood Supply





Note: Crossed Stroke Syndromes

Crossed stroke syndromes occur with brain stem strokes (posterior circulation). This is due to the Cerebrospinal that controls movement of the contralateral side of the body, crossing at the decussation of the pyramids at the level of the medulla (see below).



Stroke: Evaluation & Treatment

Stroke is a medical emergency. Therefore reducing time to treatment is crucial. Experts at an NIH recommended the following approach to Stroke Evaluation and Treatment

Detection: Early recognition
Dispatch: Early EMS activation
Delivery: Transport & management
Door: ED triage
Data: ED evaluation & management
Decision: Specific therapies
Drug: Thrombolytic & future agents

EMS Evaluation and Transport

- Conduct ABC's
- Recognize Stroke
- Establish time of stroke onset
- Perform neurological evaluation
- Check glucose
- Notify hospital early
- Transport rapidly

Stroke-Team Stroke Evaluation & Treatment

- Protect ABC's
- Maintain appropriate blood pressure

- Control glucose (hyperglycemia is associated with poor outcome
- Control fever
- Monitor for seizures
- Monitor for cerebral edema and treat with hyperventilation and mannitol
- Work-UP
    - Perform Physical Exam
        - Perform extracranial exam for source of stroke and stroke-mimics
        - Perform head exam for evidence of trauma, infection, meningeal signs
        - Perform fundiscopic exam
        - Perform neuro exam for localizing signs (see table above)
    - Complete Stroke Scale (example below)
    - Neuroimaging – CT
        - **CT results are a fundamental branch point in stroke eval – either ischemic stroke or hemorrhagic**.
        - Look for evidence of hemorrhage and distribution of stroke
        - Often no changes seen on non-contrast CT in early hours post-stroke. It is used to rule-out hemorrhages.
    - Labs
        - CBC (polycythemia, thrombocytosis, thrombopenia, leukemia)
        - Chem panel (electrolyte abnlties)
        - Coagulation studies
        - Cardiac enzymes
    - EKG
    - Other studies used under specific circumstances
        - Transcranial Doppler to view intracranial large vessels
        - Carotid Duplex scanning when carotid occlusion suspected
        - Echocardiogram when cardiac source of emboli suspected
        - Angiography when ischemic distribution needs to be clearly defined

Thrombolytic Agent (rT-PA) Treatment Decision:

The MAIN treatment decision is whether or not to give a thrombolytic agent (recombinant tissue-type plasminogen activator (rT-PA)). This decision is based on whether there is enough evidence that the stroke is hemorrhagic. In such cases, rT-PA is contraindicated because it would lead to a further bleed (see exclusions below).

- rT-PA used for strokes with measurable neurological deficit
- Evidence shows decrease in deficits in pts treated with tpa at 3 month interval for all types of ischemic stroke.
- Converts plasminogen->plasmin->hydrolyze fibrin, fibrinogen.
    - Must give intravenously within **3hrs of stroke symptoms. (6% risk of ICH)**
    - Intra arterial tpa can be given within **6hrs of stroke symptoms (higher risk of ICH-20%)**

Exclusions to Thrombolytic Therapy

- Stroke or head trauma within 3 months
- Major surgery within 14 days
- Systolic BP > 185 mm Hg

- Diastolic BP > 110 mm Hg
- Rapidly improving or minor symptoms
- Glucose < 50 or > 400 mg/dl
- Seizures at the onset of stroke
- Evidence of Hemorrhages
    - Any history of intracranial hemorrhage
    - Symptoms suggestive of subarachnoid hemorrhage
    - GI hemorrhage within 21 days
    - Urinary tract hemorrhage within 21 days
    - Arterial puncture at non-compressible site past 7 days
- Patients taking oral anticoagulants
- Heparin within 48 hours AND an elevated PTT
- PT >15 / INR >1.4
- Platelet count <100 X 10/L

Initial Neurologic Examination - NIH Stroke Scale

As an example, here is a commonly used Stroke Assessment Scale

Item	Description	Range
1a	Level of Consciousness	0 – 3
1b	LOC Questions	0 – 2
1c	LOC Commands	0 – 2
2	Best Gaze	0 – 2
3	Best Visual	0 – 3
4	Facial Palsy	0 – 3
5	Motor Arm Left	0 – 4
6	Motor Arm Right	0 – 4
7	Motor Leg Left	0 – 4
8	Motor Leg Right	0 – 4
9	Limb Ataxia	0 – 2
10	Sensory	0 – 2
11	Neglect	0 – 2
12	Dysarthria	0 – 2
13	Best Language	0 – 3