Hemorrhagic and Ischemic Stroke

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Hemorrhagic v. Ischemic Strokes

15% Hemorrhagic
85% Ischemic

Etiology and Subtypes of Stroke

Stroke Statistics

- Approximately 795,000 Americans suffer a stroke each year with nearly 77% being first time strokes.
- Stroke recently dropped to the 5th leading cause of death in the US but remains the leading cause of serious, long-term disability.
- On average in the US, every 40 seconds someone has a stroke and every 4 minutes someone dies.
- 87% of all strokes are ischemic, 10% are intracerebral hemorrhage, and 3% are subarachnoid hemorrhage.

Stroke Statistics in Minnesota

- Over 97,000 Minnesotans have had a stroke
- Approximately 11,500 new strokes occur each year
- Stroke is the 5th leading cause of death and the leading cause of long-term disability
- In 2011, $414 million was spent on hospital care for stroke

Stroke Statistics

- Cost of Stroke in the United States:
  - 2012 Estimates:
    - Direct cost (provision of care) = $71.55 billion
    - Indirect cost (lost of productivity) = $33.65 billion
    - Total cost = 105.2 billion
  - Projected 2030 Estimates*:
    - Direct cost = $183.13 billion
    - Indirect cost = $56.54 billion
    - Total cost = $239.67 billion

* 2030 estimates calculated based on current stroke rates and aging of population. It is projected that by 2030, 3.88% of the population in the U.S. will have had a stroke.
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Modifiable Risk Factors

- Hypertension
- Atrial fibrillation
- Diabetes
- Hyperlipidemia
- Smoking
- Heart Disease
- Carotid Artery Disease
- Sleep Apnea
- Oral contraceptives
- Clotting Disorders
- Vasculitis
- Lifestyle: obesity, diet, exercise, alcohol, illicit drugs

Uncontrollable Risk Factors

- Age – risk doubles every decade after age 55
- Gender
  - Women have greater number of strokes every year due to longer life span, but men have greater number of strokes in the younger population.
- Race – risk related to incidence of stroke risk factors
  - African American have twice the risk
  - Increased risk in Hispanic and Native American population
- Family history

Stroke Warning Signs

- Sudden weakness or numbness of the face, arm or leg, especially on one side of the body
- Sudden confusion, trouble speaking or understanding
- Sudden trouble seeing in one or both eyes
- Sudden trouble walking, dizziness, loss of balance or coordination
- Sudden, severe headaches with no known cause (for hemorrhagic stroke)

F.A.S.T.

- F = Face  Ask the person to smile.
- A = Arms  Ask the person to raise both arms to check for a drift.
- S = Speech Ask the person to repeat a simple sentence.
- T = Time  Take action and alert immediately.

Stroke Types

- **Ischemic**
  - Atherothrombotic
  - Embolic
  - Transient Ischemic Attack
- **Hemorrhagic**
  - Intracerebral Hemorrhage
  - Subarachnoid Hemorrhage

Ischemic Stroke

- **Embolic**
  - Caused by a blood clot dislodged from a distant source
    - Atrial fibrillation
    - Heart valve replacement
- **Thrombotic**
  - Caused by occlusion of a cerebral vessel from atherosclerosis or plaque formation
Ischemic Stroke: Embolic-vs-Atherothrombotic

Atherothrombotic

- Atherosclerotic changes in the vessel wall, typically causing stenosis (narrowing)
- Irregular wall shape catches platelets as they circulate in the blood
- Additional platelets are attracted to the site leading to thrombus formation and occlusion

Atherothrombotic

- Atherosclerotic plaque may rupture (sometimes called ulcerative plaque)
- Platelets and other clotting factors are attracted to the injured area and a combination of plaque and thrombus occlude the vessel

Most Common Causes of Cardioembolic Stroke

- Atrial Fib
- Dilated Cardiomyopathy
- Infective Endocarditis
- Rheumatic Heart Disease
- Prosthetic Valves
- Atrial Septal Defects

Ischemic Stroke: Artery to Artery Emboli

Ischemic Stroke

- Reduced blood flow distally
- Small or large vessel disease
  - Large vessel more common
  - Small vessel aka “lucunar infarction”
- Slightly higher incidence in men versus women
- TIA’s in 30-50% of cases
Important Facts About TIAs

- The prevalence of transient ischemic attacks (TIA) increases with age. (Cerebrovasc Dis. 1996; 6[suppl 1]:26–33.)
- About 15 percent of strokes are preceded by a TIA. (Cerebrovasc Dis. 1996; 6[suppl 1]:26–33.)
- About half of patients who experience a TIA fail to report it to their healthcare providers. (Neurology. 2003;60:1429–1434.)

Stroke Mimics

- Hypoglyemia
- Migraine
- Seizures
- Syncope
- Transient global aphasia
- Peripheral nerve disorders
- Intracranial hemorrhage, tumor, abscess
- Psychogenic episodes
- Metabolic disturbances

Diagnostic Evaluation

- Physical Exam/History/Time of onset
- Brain imaging (CT or MRI)
- Neurovascular imaging of head and neck (CTA, MRA)
- EKG - 12-lead and continuous monitoring
- Lab Work
- Further Cardiac Work-Up
- Cerebral Angiography

Treatment of Ischemic Stroke

- Reperfusion Strategies
  - Intravenous tPA – 0-4.5 hour Tx window
  - Intra-arterial thrombolysis (tPA) – 0-6 hour Tx window
  - Mechanical thrombectomy
    - Stent Retrievers
    - Thrombo-aspiration
    - 0-6 hour Tx window in anterior circulation
    - 0-24 hour Tx window in posterior circulation
    - Considered for moderate to severe “Wake-Up” strokes
    - On-going research in 6-16 hour window

Penumbra™ Thrombo-aspiration Device
Tissue Plasminogen Activator (tPA)

- Fibrinolytic that assists with dissolving clots.
- Produced endogenously by endothelial cells.
- Converts proenzyme plasminogen to activated enzyme plasmin. (Dissolves fibrin clots.)
- Serum ½ life is 4-6 minutes but when bound to the fibrin of a clot this time is lengthened.
- Criteria for administration is very stringent.

Post Recanalization Considerations

- Frequent vital signs (q 15 min x 2 hours, then q 30 minutes x 6 hours, then hourly x 16 hours)
- Frequent neuro checks with VS
- No anticoagulation/antiplatelet agents for 24 hours t-PA
- Bleeding Precautions
- Avoid invasive procedures (IVs, urinary catheters, NG tube) for first 2 hours after infusion.

Times to Symptomatic ICH

<table>
<thead>
<tr>
<th>Hours from Start of Treatment</th>
<th>Number of SICH</th>
</tr>
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<tbody>
<tr>
<td>0-3</td>
<td>5</td>
</tr>
<tr>
<td>4-6</td>
<td>3</td>
</tr>
<tr>
<td>7-12</td>
<td>2</td>
</tr>
<tr>
<td>13-24</td>
<td>4</td>
</tr>
<tr>
<td>24-36</td>
<td>2</td>
</tr>
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TPA Outcomes

0-3 Hour Treatment Window
tPA Outcomes:
3-4.5 Hour Treatment Window

0-4.5 Hour Treatment Window

Recent Endovascular Trials

<table>
<thead>
<tr>
<th>Trial</th>
<th>IV tPA given</th>
<th>TICI 2b/3</th>
<th>Control Group</th>
<th>IA Therapy Group</th>
<th>Control Group</th>
<th>IA Therapy Group</th>
<th>Control Group</th>
<th>IA Therapy Group</th>
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</thead>
<tbody>
<tr>
<td>MR CLEAN</td>
<td>90%</td>
<td>59%</td>
<td>19%</td>
<td>33%</td>
<td>6%</td>
<td>8%</td>
<td>23%</td>
<td>21%</td>
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<tr>
<td>ESCAPE</td>
<td>76%</td>
<td>72%</td>
<td>29%</td>
<td>53%</td>
<td>3%</td>
<td>4%</td>
<td>19%</td>
<td>10%</td>
</tr>
<tr>
<td>EXTEND-IA</td>
<td>100%</td>
<td>86%</td>
<td>40%</td>
<td>71%</td>
<td>6%</td>
<td>0%</td>
<td>20%</td>
<td>9%</td>
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<tr>
<td>SWIFT-PRIME</td>
<td>98%</td>
<td>88%</td>
<td>36%</td>
<td>60%</td>
<td>3%</td>
<td>0%</td>
<td>12%</td>
<td>9%</td>
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<tr>
<td>REVASCAT</td>
<td>73%</td>
<td>66%</td>
<td>28%</td>
<td>44%</td>
<td>2%</td>
<td>2%</td>
<td>16%</td>
<td>18%</td>
</tr>
</tbody>
</table>

Antithrombotic Medications

- If reperfusion therapy, defer 24 hours
- If no reperfusion therapy, start by end of hospital day 2 (core measure) to reduce in-hospital mortality
- IV heparin is rarely indicated except as bridge for oral anticoagulation. Some feel it is indicated for carotid or vertebral dissection
- Prescribe at discharge for secondary stroke prevention (core measure)
- Antiplatelet Medication
  - aspirin, clopidogrel, extended release aspirin plus dipyridamole
- Anticoagulation Medication (core measure if a-fib)
  - warfarin, rivaroxaban, dabigatran apixaban, or endoxaban

Ischemic Stroke in the ICU

- Most patients with ischemic stroke are in the ICU are there for observation after recanalization therapy
- Cerebellar Stroke
- Malignant MCA Syndrome
- Brainstem Stroke
Cerebrovascular Anatomy

Cerebral Arteries

- Cerebral Arteries
  - Internal Carotid (ICA)
  - Middle cerebral (MCA)
  - Anterior cerebral (ACA)
  - Posterior cerebral (MCA)
  - Basilar
  - Vertebral

Middle Cerebral Artery

Clinical Presentation

- Small Vessel (Lacunar) Infarct
  - Most common in areas supplied by lenticulate striate (branches of MCA)
  - Lacunes – small holes deep within brain
  - Associated with hypertension and diabetes
  - Result in
    - Pure motor stroke
    - Dysmetria-clumsy hand syndrome
    - Ataxic hemiparesis
    - Pure sensory stroke

Clinical Presentation

- Central Small Vessel Infarcts
  - Thalamus – sensory loss to face, arm, leg and trunk (sensory dysfunction to trunk is almost exclusive to thalamic infarct)
  - Basal Ganglia – Altered integration of motor activities. May appear to be weakness.
  - Internal Capsule – Unilateral motor and/or sensory loss is possible

Clinical Presentation

- Large Vessel Infarct
  - Middle Cerebral Artery
    - Contralateral weakness, face and arm > leg
    - Contralateral sensory loss
    - Expressive and/or Receptive aphasia if in dominant hemisphere (left), and neglect if in non-dominant hemisphere (right)
    - May also see homonymous hemianopsia (contralateral), gaze preference away from affected side, decrease LOC, headache and difficulty reading
Clinical Presentation

• Anterior Cerebral Artery
  – Contralateral weakness leg > hand and face
  – Contralateral sensory loss (may be worse in lower extremities)
  – Flat affect
  – Cognitive impairment
  – Perseveration
  – Urinary incontinence
• Internal Carotid Artery
  – Symptoms of both MCA and ACA infarct. Weakness and sensory loss even throughout.
  – Altered LOC, headache more common due to size of infarct

Clinical Presentation

• Cerebellar Infarct (Vertebrobasilar Insufficiency)
  – Vertigo
  – Impaired Balance
  – Impaired Coordination (Ataxia)
  – Impaired gait
  – High risk of severely impaired swallowing
• Brainstem Infarct (Vertebrobasilar Insufficiency)
  – When due to large vessel occlusion is often lethal
• Posterior Cerebral Artery
  – Visual deficits including peripheral vision
  – Memory (supplies part of temporal lobe)
  – Supplies part of thalamus and brain stem so may cause symptoms from those areas.

Stroke Symptoms Related to area of Brain Involvement

• Right Brain Involvement
  • Left sided hemiplegia
  • Left sided neglect
  • Spatial-perceptual deficits
  • Tends to minimize/deny problems
  • Impulsive, safety problems
  • Rapid performance/short attention span
  • Impaired judgment
  • Impaired time concepts

• Left Brain Involvement
  • Right sided hemiplegia
  • Impaired right/left discrimination
  • Aware of deficits
  • Depression, anxiety, emotional lability, easily frustrated
  • Slow performance, cautious, disorganized
  • Impaired comprehension related to language and math
  • Impaired speech/language aphasias
  • Right neglect possible

Principles and Pearls

• Know your patient’s neurologic baseline and assess for changes
• Know vascular distribution affected and anticipate neuro changes
• Know what reperfusion interventions were used and assess your patient’s response
• Keep the brain perfused
• Keep blood pressure in the target range

Principles and Pearls

• Monitor and optimize cardiac and respiratory function
• Address safety and functional issues
• Educate
• Assess the emotional response of the patient and family
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**Hemorrhagic Stroke**

- Non-traumatic rupture of a cerebral vessel resulting in an intracranial bleed
- Includes all “spontaneous” bleeds including ruptured vascular malformations such as aneurysm or AVM
- Hemorrhages related to a tumor, recent surgery or treatment effect from whole brain radiation are not considered strokes
- Head CT is considered the gold standard for initial diagnosis

**Intracerebral v. Subarachnoid Hemorrhage**

- **Intracerebral Hemorrhage**
  - Rupture or leak of blood vessel
  - Most common type of hemorrhagic stroke
  - Mortality rate is 35 – 55%
  - Causes
    - Hypertension (most common cause)
    - Substance Abuse
    - Amyloid angiopathy (seen only in older population)
    - Vascular Malformation

- **Subarachnoid Hemorrhage**
  - 80% of non-traumatic SAH is caused by ruptured aneurysm
  - 5% is caused by arteriovenous malformation (AVM)
  - The other 15% is due to a variety of causes including hypertension, vasculitis, tumor and clotting abnormalities

**Hemorrhagic Stroke**

- **ICH**
  - Bleeding into the tissue of the brain
  - Typically caused by leakage of a small artery – usually deep
  - Also known as an intraparenchymal hemorrhage

**Intracerebral Hemorrhage**

- **Subarachnoid Hemorrhage**
  - 80% of non-traumatic SAH is caused by ruptured aneurysm
  - 5% is caused by arteriovenous malformation (AVM)
  - The other 15% is due to a variety of causes including hypertension, vasculitis, tumor and clotting abnormalities
Hemorrhagic Stroke

- SAH
  - Blood enters subarachnoid space from ruptured aneurysm.

Hemorrhagic Stroke

- Arteriovenous Malformation (AVM)
  - Defects in the vascular system
  - Tangles of abnormal blood vessels

ICH and SAH Symptoms

- SAH patient typically presents with “the worst headache of their life”

ICH patient may have sudden severe HA or mild/moderate HA that worsens over time

Other Symptoms of Hemorrhagic Stroke

- Altered or loss of consciousness
- Visual disturbance common with SAH (“blinding white light”, “flashing light”)
- Focal deficits vary depending on size and location of hemorrhage

Diagnostic Evaluation

- Physical Exam/History/Time of onset
- CT/CTA or MRI/MRA
- Cerebral Angiography
- Lumbar puncture if history suggestive of SAH but CT negative and delay in imaging

ICH Treatment

- Current evidence-based guidelines recommend aggressive care x 24 hours before considering comfort measures
- Primary medical management
  - Airway management
  - Blood pressure control
  - Reversal of anticoagulation or coagulopathy
  - ICP management/minimize secondary brain injury
  - Avoid complications
- Surgical Intervention
  - Decompressive craniotomy
  - Hematoma evacuation (only indicated if hematoma is near brain surface or in cerebellum)
<table>
<thead>
<tr>
<th>SAH Facts</th>
<th>Risk Factors</th>
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</thead>
<tbody>
<tr>
<td>• Affects 30,000 Americans annually</td>
<td>• Hypertension</td>
</tr>
<tr>
<td>• Mortality as high as 50%</td>
<td>• Obesity</td>
</tr>
<tr>
<td>• Significant morbidity</td>
<td>• Cigarette smoking</td>
</tr>
<tr>
<td>• Incidence increases with age</td>
<td>• Alcohol</td>
</tr>
<tr>
<td>– Most commonly between ages 40 and 60</td>
<td>• Polycystic kidney disease</td>
</tr>
<tr>
<td>• Gender and racial differences</td>
<td>• Connective tissue disease</td>
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<table>
<thead>
<tr>
<th>Causes of SAH</th>
<th>Treatment of Ruptured Aneurysm</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Vascular malformations</td>
<td></td>
</tr>
<tr>
<td>– Aneurysms</td>
<td></td>
</tr>
<tr>
<td>– Arteriovenous malformations</td>
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<table>
<thead>
<tr>
<th>Coiling and Clipping</th>
<th>Neurosurgical Management</th>
</tr>
</thead>
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Coiling

Stent Assisted Coiling

Balloon Assisted Coiling

Balloon Assisted Coiling

Pipeline™ Stent

AVM Management
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Embolization of AVM

Embolization of AVM

Surgical Removal of AVM

Complications Associated with Aneurysmal Subarachnoid Hemorrhage

### Hunt and Hess Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
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<tbody>
<tr>
<td>0</td>
<td>Unruptured aneurysm</td>
</tr>
<tr>
<td>1</td>
<td>Asymptomatic, mild headaches, slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Cranial nerve deficits, moderate to severe headache, nuchal rigidity</td>
</tr>
<tr>
<td>3</td>
<td>Mild to moderate focal deficits</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, hemiparesis, early decerebrate rigidity</td>
</tr>
<tr>
<td>5</td>
<td>Deep coma, decerebrate rigidity</td>
</tr>
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</table>
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Miller-Fisher Grading System

<table>
<thead>
<tr>
<th>Grade</th>
<th>Blood on CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No subarachnoid blood detected</td>
</tr>
<tr>
<td>II</td>
<td>Diffuse, thin layers of SAH &lt; 1 mm thick</td>
</tr>
<tr>
<td>III</td>
<td>Localized clot(s) &gt; 3x5 mm in the cisterns or transverse planes and/or vertical layers of SAH &gt; 1 mm thick</td>
</tr>
<tr>
<td>IV</td>
<td>Intracerebral/intraventricular clot with diffuse of no SAH</td>
</tr>
</tbody>
</table>

Rebleed

- 20% rebleed within first 2 weeks (4% within first 24 hours)
- 50% of patients with rebleed will die
- Prevention
  - Rapid intervention (coiling, clipping)
  - Blood pressure management
  - Subarachnoid “Precautions”

Hydrocephalus

- Acute Hydrocephalus
  - Occurs within first 24 hours
  - Often presents as abrupt onset of stupor or coma
- Subacute Hydrocephalus
  - Develops in a few days to a week
  - Onset is usually gradual with decreasing LOC
- Delayed Hydrocephalus
  - Develops 10 days or more after SAH

Vasospasm

- Angiographically evident vasospasm is believed to exist in up to 80% of patients
- Symptomatic vasospasm occurs in 35-40% of aneurysmal SAH
- Onset is between day 4 and 14 with peak incidence at day 7-8.
- Mortality rate associated with symptomatic vasospasm is 25-35%
- The vessel diameter is narrowed by > 50% in symptomatic vasospasm

Risk Factors for Vasospasm

- Miller-Fisher grade III (one study showed 96% developed symptomatic vasospasm)
- Hypotension (SBP < 180 mmHg at presentation to hospital)
- Hypovolemia
- Site of aneurysm (anterior circulation aneurysms, particularly at bifurcations of ICAs and MCAs
- Acute hydrocephalus
- Higher Hunt and Hess grade (3,4,5)
- Smoking

Signs and Symptoms of Vasospasm

- Decreased level of consciousness
- Confusion/disorientation
- Focal neurological deficits
  - Symptoms depend on which vessel is in spasm
  - The vessel in spasm is often in proximity to the hemorrhage but may be remote if cisternal clot is present
  - MCA is most common vessel to spasm
    - Contralateral hemiparesis/plegia
    - Contralateral sensory alteration
    - Speech alteration (if dominant hemisphere)
    - Contralateral neglect (if non-dominant hemisphere)
Diagnosis of Vasospasm

- Acute neurologic deterioration after day 3
- Exclusion of structural causes (hemorrhage, hydrocephalus, cerebral edema)
- Absence of other explanations (electrolyte abnormalities, seizure, hypoxia)
- Transcranial Doppler (increased velocities)
- Perfusion CT
- Angiography

TCD Values in Vasospasm

<table>
<thead>
<tr>
<th>Vessel</th>
<th>Normal Velocity</th>
<th>Vasospasm</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA 35-65</td>
<td>80-120 = probable mild spasm</td>
<td>&gt; 160 = severe spasm</td>
</tr>
<tr>
<td>ACA 35-55</td>
<td>&gt; 120 = probable spasm</td>
<td>&gt; 140 = definite spasm</td>
</tr>
<tr>
<td>BA 25-45</td>
<td>60-90 = probable spasm</td>
<td>&gt; 90 = definite spasm</td>
</tr>
</tbody>
</table>

Prevention of Vasospasm

- Nimodipine
  - Must be started within 72 hours
  - Continued for 21 days
  - Must be given orally or via feeding tube
- Maintain “high” euvolemia
- Permissive hypertension
- Evidence suggests that maintaining Magnesium level > 2.0 may decrease incidence of vasospasm

Treatment of Vasospasm

- Nimodipine
- Triple “H” Therapy
  - High Euvolemia (take insensible loss into consideration)
  - Hypertension (target SBP 160-180)
  - Hemodilution (Ideal Hct ~ 32%)
- Arterial Dilation Procedures
  - Smooth muscle relaxants (papaverine, nicardipine)
  - Angioplasty

Angioplasty of vasospasm

Hyponatremia

- Transient SIADH in the first 1-2 days (mild)
- Cerebral Salt Wasting Syndrome (CSWS)
  - Peak incidence 5-6 days after SAH
  - Caused by release of natriuretic peptides (mechanism is unknown)
Cerebral Salt Wasting Syndrome

- **Signs/Symptoms**
  - Serum Sodium < 135
  - Signs of hyponatremia (LOC, N/V, confusion, seizures)
- **Prevention**
  - Avoid hypotonic solutions
  - Maintain euvolemia
    - Isotonic maintenance solution
    - Hypertonic saline for volume expansion
- **Treatment**
  - Measures used for prevention AND give sodium (NS, NaCl tabs, hypertonic saline)

EKG Abnormalities

- Occurs in 35-50% of patients
- Peak incidence is day 2-3
- Primarily due to catecholamine release and adrenal stimulation
- Most common abnormality is prolonged QTc (61%) but arrhythmias, conduction abnormalities and ischemic changes may all occur
- No known prevention, treat using standard treatment for identified abnormality, avoid drugs & factors that may exacerbate.

Fever

- **Etiology**
  - Hemolysis of subarachnoid blood (peak day 4)
  - Infection
  - Pituitary/hypothalamic dysfunction
- **Treatment**
  - Antipyretic agents
  - Cooling measures (avoid shivering!)

Seizures

- Risk of seizures for first 3-5 days
- Prophylactic antiepileptic drugs (AEDs) are not recommended if patient is seizure free after day 5
- Phenytoin/fosphenytoin has been found to worsen cognitive and functional outcomes following SAH and should be avoided.
- Levetiracetam is now often being used as a prophylactic AED.

Changing the Perception of Stroke

- Stroke is unpreventable
- Cannot be treated
- Strikes only the elderly
- Recovery ends 6 months after a stroke
- Stroke is largely preventable
- Requires urgent treatment
- Can happen to anyone
- Stroke recovery can continue throughout life

QUESTIONS??

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Anterior Cerebral Artery

- Supplies medial surfaces of the cerebral hemispheres
- Complete occlusion is the least common cause of stroke

ACA Occlusion

- Contralateral hemiplegia greatest in the foot and thigh, shoulder
- Mild sensory loss which follows the pattern of the weakness
- Personality, behavioral changes

Middle Cerebral Artery

- Supplies large portion of the lateral surfaces of the frontal, parietal, and temporal lobes and basal ganglia
- Most common vessel for stroke to occur

MCA Occlusion

- Contralateral hemiplegia greater in face and arm
- Contralateral sensory impairment varying in severity
- Left: severe aphasia
- Right: neglect, poor motivation, constructional apraxia

Alterations in sensation and perception

- Neglect
  - Seen more with right hemisphere involvement
  - Disorder of attention
  - Typically unilateral
<table>
<thead>
<tr>
<th>Posterior Circulation: Vertebral Arteries</th>
<th>Basilar Artery</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Occlusion or stenosis may not cause neurologic deficit if it does not extend into the basilar artery and does not impair blood flow into any of the tributaries</td>
<td>• Supplies brainstem, primarily the pons</td>
</tr>
<tr>
<td>• Retrograde flow following crossover from one vertebral to the other provides adequate blood supply</td>
<td>• Branches to form right and left posterior cerebral arteries – supplies midbrain and diencephalon</td>
</tr>
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<table>
<thead>
<tr>
<th>Basilar Artery Occlusion</th>
<th>Basilar Artery Occlusion</th>
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</thead>
<tbody>
<tr>
<td>• Quadraparesis or quadraparalysis possible</td>
<td>• Inability to move eyes</td>
</tr>
<tr>
<td>• Flaccidity</td>
<td>• Decreased level of consciousness</td>
</tr>
<tr>
<td>• May change to increase tone or hyporeflexia</td>
<td>• “Locked in”</td>
</tr>
<tr>
<td>• Normal sensation</td>
<td></td>
</tr>
<tr>
<td>• Cranial nerve impairment</td>
<td></td>
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</table>

<table>
<thead>
<tr>
<th>Posterior Cerebral Artery</th>
<th>PCA Occlusion</th>
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<tbody>
<tr>
<td>• Supplies midbrain, thalamus, occipital lobes and medial-posterior portions of the temporal lobes</td>
<td>• Hemiplegia</td>
</tr>
<tr>
<td></td>
<td>• Hyperesthesia</td>
</tr>
<tr>
<td></td>
<td>• Decreased level of consciousness</td>
</tr>
</tbody>
</table>