Stroke
Intracranial hypertension
Cerebral edema

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Books

• Silbernagl 356
• Other book 667
Brain

- The most complex structure in the body
  - Anatomically
  - Functionally

- Signals to and from various part of the body are controlled by very specific areas within the brain

- Brain is more vulnerable to focal lesions than other organs
  - Renal infarct does not have a significant effect on kidney function
  - Brain infarct of the same size can produce complete paralysis on one side of the body
Brain

- 2% of body weight
- Receives 1/6 of resting cardiac output
- 20% of oxygen consumption
A. Cerebrospinal Fluid (CSF) Flow

- Sagittal sinus
- Arachnoid villi
- Dura mater
- Bone
- Lateral ventricle
- Choroid plexus
- 3rd ventricle
- Aqueduct
- 4th ventricle
- Foramen of Luschkae
- Foramen of Magendii
- Arachnoid villi
- Interventricular foramina

Malformations, scarring, tumors

CSF flow obstructed

Hydrocephalus (non-communicating)

- Thrombosis, sinus occlusion, cardiac failure
- Meningitis, subarachnoid hemorrhage, etc.

Obstruction of arachnoid villi

Venous outflow

Hydrocephalus (communicating)

CSF absorption

Tumors, infections

Protein concentration

Hydrocephalus in newborn
Blood-brain barrier

C. Blood-Brain-Barrier

1. Normal cerebral capillary
   - Lipid-soluble substances
   - Astrocytes
   - Closed tight junctions
   - Electrolytes, proteins, cells
   - Selective carrier

2. Abnormal
   - Tumors
   - Bacterial meningitis
   - Infusions
   - Defective astrocytes
   - Osmolarity ↑
   - Open tight junctions
Mechanisms of brain injury

• Various causes:
  • trauma
  • tumors
  • stroke
  • metabolic dysbalance

• Common pathways of injury:
  • Hypoxia
  • Ischemia
  • Cerebral edema
  • Increased intracranial pressure
Hypoxia

• Deprivation of oxygen with maintained blood flow
• Causes:
  • Exposure to reduced atmospheric pressure
  • Carbon monoxide poisoning
  • Severe anemia
  • Failure to oxygenate blood
• Well tolerated, particularly if chronic
  • Neurons capable of anaerobic metabolism
  • Euphoria, listlessness, drowsiness, impaired problem solving
• Acute and severe hypoxia – unconsciousness and convulsions
• Brain anoxia can result to cardiac arrest
Ischemia

- Reduced blood flow
- **Focal / global** ischemia
- Energy sources (glucose and glycogen) are exhausted in **2 to 4 minutes**
- Cellular ATP stores are depleted in **4 to 5 minutes**
- 50% - 75% of energy is spent on mechanisms for maintenance of ionic gradients across cell membrane (Na, K, Ca ions)
- Different sensitivity of various brain regions to hypoxic-ischemic injury (glutamate levels)
Glutamate

• The **main excitatory neurotransmitter**
• Responsible for higher-order functions – memory, cognition, movement, sensation
• Excess extracellular concentrations are removed and **actively transported into astrocytes and neurons**
• In ischemia, these transport mechanisms become ineffective, causing **accumulation of extracellular glutamate**
Glutamate

• **High extracellular glutamate** – uncontrolled opening of NMDA receptor-operated ion channels – increase in extracellular calcium – activation of calcium cascade

• **Neuroprotectants** interfere with this pathway to reduce brain cell injury
CSF pressure, cerebral edema

A. Volume Changes of Brain Compartment

- Skull
- Intracellular (~80%)
- Interstitial (<10%)
- CSF (~10%)
- Intravascular (~1–3%)
- Exchange of metabolites

1. Cranial volumes
2. Pulse-synchronous vessel dilation

- Cell swelling
- CSF space collapsed
- Vessel narrowing
- Cerebral perfusion

3. Cell swelling
4. Acute CSF obstruction

- Outflow obstruction
- CSF pressure
- Vessel narrowing
- Cerebral perfusion

5. Chronic CSF obstruction
- Death of neurons
• 1. **Intrac**ellular. Cause – energy deficiency (hypoxia, ischemia, hypoosmotic states - water intoxication)

• 2. **Inter**cellular. Cause – increased permeability (infections, tumors, bleeding, abscess, poisoning)

• 3. **Inter**cellular. Cause – high interstitial osmolarity (drop in blood glucose, urea, Na+)

• 1 is in grey matter; 2 and 3 are vasogenic and occur in white matter
Brain edema

1. **Cytotoxic** – grey matter
   - Cause – energy deficiency (hypoxia, ischemia, hypoosmotic states - water intoxication)
   - Accumulation of Na+ in the cell, influx of water in the cell
   - Vascular endothelium, smooth muscle cells, astrocytes, oligodendrocytes, neurons
   - Stupor, coma

2. **Inter**cellular vascular
   - Cause – increased permeability (infections, tumors, bleeding, abscess, poisoning)
   - White matter

3. **Inter**cellular interstitial
   - Cause – high interstitial osmolarity (drop in blood glucose, urea, Na+)
   - White matter
Increased intracranial pressure

- Cranial cavity:
  - Blood (10%)
  - Brain tissue (80%)
  - CSF (10%)

- Monro-Kellie hypothesis – reciprocal compensation in volume changes of the respective compartments

- Normal ICP: 0-15 mmHg in lateral ventricles

- Changes in any of the three compartments can lead to change in ICP
Increased intracranial pressure

- Impact of increase in **blood, brain tissue** and **CSF** on ICP depends on:
  - Amount of increase
  - Effectiveness of compensatory mechanisms
  - Compliance of brain tissue
Cerebral perfusion pressure

• The **pressure perfusing the brain**
• Determined by the pressure gradient between the **internal carotid artery** and **subarachnoid veins**
• = mean arterial blood pressure – ICP
• Frequently monitored in patients with brain conditions that increase ICP
• 1. 70 – 100 mmHg: normal
• 2. below 70 – 50 mmHg: ischemia
• 3. ICP exceeds MABP: cellular hypoxia, neuronal death
Increased intracranial pressure

- **Early and reliable sign:** decrease in consciousness level

- **Late reflex:** CNS ischemic response triggered by ischemia in vasomotor center in brain stem – Cushing reflex

  - Neurons produce increase in MABP (up to 270 mmHg)
  - Widening of the pulse pressure
  - Reflex slowing of the heart rate
Increased intracranial pressure

• Common pathway for different types of insults

• Consequences:
  • Obstruction of cerebral blood flow
  • Damage to brain cells
  • Displacement of brain tissue (brain herniation)
  • Other type of damage to brain structures
C. Effects of Increased Intracranial Pressure

1. Papilledema

2. Additional effects
   - Headache
   - Nausea
   - Vomiting
   - Coma
   - Bradycardia
   - Hypertension
   - Squint
   - Fixed pupils

3. Herniation

Skull

- Cerebellum
- C
- b
- a
# TABLE 37-4
Descending Levels of Consciousness and Their Characteristics

<table>
<thead>
<tr>
<th>Level of Consciousness</th>
<th>Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Confusion</td>
<td>Disturbance of consciousness characterized by impaired ability to think clearly, and to perceive, respond to, and remember current stimuli; also disorientation</td>
</tr>
<tr>
<td>Delirium</td>
<td>State of disturbed consciousness with motor restlessness, transient hallucinations, disorientation, and sometimes delusions</td>
</tr>
<tr>
<td>Obtundation</td>
<td>Disorder of decreased alertness with associated psychomotor retardation</td>
</tr>
<tr>
<td>Stupor</td>
<td>A state in which the person is not unconscious but exhibits little or no spontaneous activity</td>
</tr>
<tr>
<td>Coma</td>
<td>A state of being unarousable and unresponsive to external stimuli or internal needs; often determined by the Glasgow Coma Scale</td>
</tr>
</tbody>
</table>

(Data from Bates D. [1993]. The management of medical coma. *Journal of Neurology, Neurosurgery, and Psychiatry* 56, 590)

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# TABLE 37-5
The Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Test</th>
<th>Score*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye Opening (E)</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To call</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td><strong>Motor Response (M)</strong></td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td>Normal flexion (withdrawal)</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion (decorticate)</td>
<td>3</td>
</tr>
<tr>
<td>Extension (decerebrate)</td>
<td>2</td>
</tr>
<tr>
<td>None (flaccid)</td>
<td>1</td>
</tr>
<tr>
<td><strong>Verbal Response (V)</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused conversation</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
</tbody>
</table>

*GCS Score = E + M + V. Best possible score = 15; worst possible score = 3.
Cerebrovascular disease

• Stroke
• Transient ischemic attack
• Aneurysmal subarachnoid hemorrhage
• Arteriovenous malformations
Stroke

• WHO definition: "neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours"

• 24 hour limit divides stroke from transient ischemic attack
Epidemiology

• **Second** most frequent cause of death (after heart disease)

• Incidence increases exponentially from 30 years of age

• 95% of strokes in people age 45 and older

• 2/3 of strokes in people age 65 and older

• Men are 25% more likely to suffer stroke, but 60% of deaths occur in women
Risk factors

• Age, gender, race, heart disease, hypertension, high cholesterol, smoking, prior stroke, diabetes
• Sickle cell disease, polycythemia, excess alcohol use, drug use, obesity, sedentary lifestyle
• Women-specific risk factors: pregnancy, childbirth, menopause
• Genetic tendency for stroke, shared lifestyle
History

- Stroke reported in **2nd millennium BC** in ancient Mesopotamia and Persia
- **Hippocrates (460 to 370 BC)** described the phenomenon of sudden paralysis – introduced the term „apoplexy“ (struck down with violence)
- The term „stroke“ used first in 1599
- **1658 Johann Jacob Wepfer** identified the cause of hemorrhagic stroke – people who died of apoplex had bleeding in their brains
- Wepfer also identified ischemic stroke as cerebral infarction
- **Rudolf Virchow** described the mechanism of thromboembolism as a major factor
- The term „cerebrovascular accident“ introduced in 1927
- The term „brain attack“ introduced and used since 1990
Pathophysiology

- **Occlusion or rupture** of arteries results in deficits in surrounding regions
- Poor blood flow to the brain region results in cell death
- The basic mechanism is always **energy deficiency** caused by **ischemia**
- What about bleeding?
- Inhibition of NA+/K+-ATPase – depolarization – accumulation of CL- - swelling – cell death

- **Ischemic** (88%)
- **Hemorrhagic** (12%)
Ischemic Stroke

Clot occluding artery
85%

Intracerebral Hemorrhage

Bleeding into brain
10%

Subarachnoid Hemorrhage

Bleeding around brain
5%
A. Effects of Abnormal Cerebral Perfusion

- Ischemia
- Bleeding

O$_2$↓ → K$^+$↑ → ATP → Na$^+$↑ → Ca$^{2+}$↑ → Glutamate → Necrosis

- Depolarization
- Water (H$_2$O)

Nerve cell → Cell swelling

Brain tissue → Inflammation

Lesion of cells at margin of ischemic region → Thromboxan → Vasoconstriction → Intracranial pressure↑

1. Dysfunction
   - Onset of anoxia: 4-6s
   - Paralysis: 10-20s
   - Irreversible damage: 3-5min
   - Cell death
Ischemic stroke

• Interruption of blood flow caused by thrombosis or emboli
• 5 subtypes
  • 1. Large artery atherosclerotic disease (20%)
  • 2. Small vessel or penetrating artery disease – lacunar stroke (25%)
  • 3. Cardiogenic embolism (20%)
  • 4. Cryptogenic stroke – undetermined cause (30%)
  • 5. Other – unusual causes (5%)
1. Large vessel thrombotic stroke

- **Atherosclerotic plaques** mostly at arterial bifurcations
  - origins of internal carotid and vertebral arteries
  - junctions of the basilar and vertebral arteries
- Infarction due to:
  - **Acute local thrombosis and occlusion** (with or without embolization)
  - **Critical perfusion failure** distal to stenosis
1. Large vessel thrombotic stroke

- Often affects cortex – aphasia or neglect, visual field defects
- Usually a single cerebral artery and its territories are affected
- In older persons
- Accompanied with atherosclerotic heart or peripheral arterial disease
- Not associated with physical activity
- May occur at rest
2. Small vessel (lacunar) stroke

- Small (1.5 to 2.0 cm) to very small (3 to 4 mm) infarcts in deeper noncortical parts or brain stem
- In single penetrating arteries supplying internal capsule, basal ganglia or brain stem
- Occlusion of small branches of large arteries, commonly the middle and posterior cerebral arteries
- In the process of healing, small cavities – lacunae (lakes) are formed
- Usually no cortical deficits – aphasia, apraxia
2. Small vessel (lacunar) stroke

• Lacunar symptoms:
  • Pure motor hemiplegia
  • Pure sensory hemiplegia
  • Dysarthria with clumsy hand syndrome
3. Cardiogenic embolic stroke

- Caused by moving blood clot from left heart of carotid arteries
- Predisposing factors:
  - Rheumatic heart disease
  - Atrial fibrillation
  - Recent myocardial infarction
  - Ventricular aneurysm
  - Bacterial endocarditis
- Larger proximal cerebral arteries
- Most often – middle cerebral artery (least resistance)
Ischemic stroke

• Based on the extent of the symptoms:
  • Total anterior circulation stroke (TAC)
  • Partial anterior circulation stroke (PAC)
  • Lacunar stroke (LAC)
  • Posterior circulation stroke (POC)

• These four entities predict:
  • extent of the stroke
  • area of the brain affected
  • underlying cause
  • prognosis.
Ischemic stroke

- Central core of dead tissue
- Ischemic area of minimally perfused tissue – penumbra (i.e. halo)
- Survival of penumbra cells depends on:
  - successful timely return of adequate circulation
  - volume of toxic products released by dying cells
  - degree of cerebral edema
  - alteration in local blood flow
Transient ischemic attack

- Transient episode of neurologic dysfunction caused by ischemia without acute infarction (tissue death)
- Focal ischemia of brain, spinal cord or retina
- Related syndrome of stroke symptoms that resolve completely within 24 hours
- Symptoms: paralysis, weakness, numbness, dimming, loss of vision, aphasia, dysarthria, mental confusion
- Cerebral infarct that lasts longer than 24 hours, but less than 72 hours is reversible ischemic neurologic deficit (RIND)
Hemorrhagic stroke

- **Rupture of a blood vessel** leads to edema, compression of brain or spasm of adjacent vessels
- Risk factors: **age**, **hypertension**, trauma, aneurysm, tumors, AV malformations, coagulopathy, vasculitis, drugs
- Occurs **suddenly**, when the person is **active**
- Most commonly: hemmorhage into **basal ganglia**, which results in **contralateral hemiplegia**
- Often progresses to coma and death
Hemorrhagic stroke

- 2 types
- Cerebral hemorrhage
  - Intraparenchymal
  - Intraventricular
- Subarachnoid hemorrhage – outside of the brain tissue, but inside the skull
  - Between arachnoid and pia mater
Manifestations of stroke

- Determined by:
  - artery that is affected
  - area of brain that is supplied by the vessel
  - collateral circulation
- Sudden, focal, one-sided
- Most common – unilateral weakness of face, arm or leg
- Unilateral numbness, vision loss, aphasia, dysarthria, sudden loss of balance or ataxia
- Early recognition: FAST – face, arm, speech, time
When Stroke Strikes, Act F.A.S.T.

FACE
Smile.
Does one side of the face droop?

ARMS
Raise both arms.
Does one arm drift downward?

SPEECH
Repeat a sentence.
Are they able to speak clearly? Can they repeat the sentence?

TIME
Time is critical.
Call 911. Get to the hospital immediately. Brain cells are dying. Every Minute Counts!
B. Vascular Occlusion as Cause of Ischemia

Hemiparesis, hemianesthesia, apraxia, apathy

Hemianesthesia, hemiparesis, conjugate deviation, visual field defect, aphasia, apraxia, hemineglect

Hypokinesia
Hemiparesis
Hemianopsia

Hemianesthesia

Hemianopsia

III, VII, XII, Parkinson’s dis., hemiparesis

V, VI, VII, hypacusis, ataxia, nystagmus, Horner’s syndrome

Tetraplegia, pseudobulbar paralysis

V, X, nystagmus, Horner’s syndrome, hemiataxia, hiccough, hemianesthesia, ageusia, hypakusis

XII, hemiparesis, Hemihypesthesia
Frequent cause

• Occlusion of the **middle cerebral artery:**
  • Contralateral muscle weakness and spasticity (damage to postcentral gyrus)
  • Sensory deficits (damage to precentral gyrus)
  • Ocular deviation (damage to visual motor area)
  • Hemianopsia
  • Motor and sensory speech disorders (Broca and Wernicke areas of dominant hemisphere)
  • Damaged spatial perception
  • Apraxia
  • Hemineglect
Causes

• Occlusion of the **anterior cerebral artery**:
  • Contralateral hemiparesis and sensory deficits (loss of the medial portion of precentral and postcentral gyri)
  • Speech difficulties
  • Apraxia of left arm (when anterior corpus callosum is impaired – connection from dominant hemisphere to the right motor cortex)
  • Bilateral occlusion – apathy (damage to limbic system)
Causes

• Occlusion of the **posterior cerebral artery**:  
  • Partial contralateral hemianopsia (damage to primary visual cortex)  
  • Bilateral occlusion – blindness  
  • Memory losses (lower temporal lobe)
Causes

• Occlusion in **carotid** or **basilar artery**:
  • Deficits in the supply area of the anterior and middle cerebral arteries

• Occlusion in **anterior choroid artery**:
  • Basal ganglia (hypokinesia)
  • Internal capsule (hemiparesis)
  • Optic tract (hemianopsia)

• Occlusion in posterior communicating artery to the thalamus:
  • Sensory deficits
<table>
<thead>
<tr>
<th>Cerebral Artery</th>
<th>Brain Area Involved</th>
<th>Signs and Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior cerebral</td>
<td>Infarction of the medial aspect of one frontal lobe if lesion is distal to communicating artery; bilateral frontal infarction if flow in other anterior cerebral artery is inadequate</td>
<td>Paralysis of contralateral foot or leg; impaired gait; paresis of contralateral arm; contralateral sensory loss over toes, foot, and leg; problems making decisions or performing acts voluntarily; lack of spontaneity, easily distracted; slowness of thought; aphasia depends on the hemisphere involved; urinary incontinence; cognitive and affective disorders</td>
</tr>
<tr>
<td>Middle cerebral</td>
<td>Massive infarction of most of lateral hemisphere and deeper structures of the frontal, parietal, and temporal lobes; internal capsule; basal ganglia</td>
<td>Contralateral hemiplegia (face and arm); contralateral sensory impairment; aphasia; homonymous hemianopia; altered consciousness (confusion to coma); inability to turn eyes toward paralyzed side; denial of paralyzed side or limb (hemiatention); possible acaulclia, alexia, finger agnosia, and left–right confusion; vasomotor paresis and instability</td>
</tr>
<tr>
<td>Posterior cerebral</td>
<td>Occipital lobe; anterior and medial portion of temporal lobe</td>
<td>Homonymous hemianopia and other visual defects such as color blindness, loss of central vision, and visual hallucinations; memory deficits, perseveration (repeated performance of same verbal or motor response)</td>
</tr>
<tr>
<td>Thalamus involvement</td>
<td></td>
<td>Loss of all sensory modalities; spontaneous pain; intentional tremor; mild hemiparesis; aphasia</td>
</tr>
<tr>
<td>Cerebral peduncle involvement</td>
<td></td>
<td>Oculomotor nerve palsy with contralateral hemiplegia</td>
</tr>
<tr>
<td>Basilar and vertebral</td>
<td>Cerebellum and brain stem</td>
<td>Visual disturbance such as diplopia, dystaxia, vertigo, dysphagia, dysphonia</td>
</tr>
</tbody>
</table>

*Depend on hemisphere involved and adequacy of collaterals.*
Diagnosis

• Complete history
• Physical examination
• Neurologic evaluation

• Determine the presence of hemorrhage or ischemia
CT vs. MRI

- For diagnosing ischemic stroke in the emergency setting:
  - **CT scans** (without contrast enhancements)
    - sensitivity = 16%
    - specificity = 96%
  - **MRI scan**
    - sensitivity = 83%
    - specificity = 98%

- For diagnosing hemorrhagic stroke in the emergency setting:
  - **CT scans** (without contrast enhancements)
    - sensitivity = 89%
    - specificity = 100%
  - **MRI scan**
    - sensitivity = 81%
    - specificity = 100%
• A CT showing early signs of a middle cerebral artery stroke with loss of definition of the gyri and grey white boundary
**Acute (4 hours)**
Infarction

Subtle blurring of gray-white junction & sulcal effacement

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**Subacute (4 days)**
Infarction

Obvious dark changes & “mass effect” (e.g., ventricle compression)
• CT scan of the brain showing a right-hemispheric ischemic stroke.
• CT scan of an intraparenchymal bleed (bottom arrow) with surrounding edema (top arrow)
Diagnosis

• Additional methods to determine the underlying cause:
  • ultrasound/Doppler of carotid arteries (carotid stenosis)
  • ECG or ECHO (arrhythmias)
  • Holter monitor (abnormal heart rhythms)
  • angiogram (aneurysm of malformations)
  • blood tests (high cholesterol, tendency to bleed)
Scoring system

- **National Institutes of Health Stroke Scale (NIHSS)**
- A tool to quantify the impairment caused by stroke
- 11 items, score 0-4
- Consciousness, eye movement, visual field, facial palsy, motor arm, motor leg, limb ataxia, sensory, language, speech, inattention

<table>
<thead>
<tr>
<th>Score</th>
<th>Stroke severity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No stroke symptoms</td>
</tr>
<tr>
<td>1-4</td>
<td>Minor stroke</td>
</tr>
<tr>
<td>5-15</td>
<td>Moderate stroke</td>
</tr>
<tr>
<td>16-20</td>
<td>Moderate to severe stroke</td>
</tr>
<tr>
<td>21-42</td>
<td>Severe stroke</td>
</tr>
</tbody>
</table>
Treatment

Royal College of Physicians

National clinical guideline for stroke
Prepared by the Intercollegiate Stroke Working Party
Fifth Edition 2016

AHA/ASA Guideline
Guidelines for the Early Management of Patients with Acute Ischemic Stroke

Guidelines
Cerebrovasc Dis 2013;35:93–112
DOI: 10.1159/000346087

European Stroke Organization Guidelines for the Management of Intracranial Aneurysms and Subarachnoid Haemorrhage
Management

• Airway – maintain tissue oxygenation

• Blood pressure
  • The goal is to maintain cerebral perfusion!!
  • Higher BP goals with ischemic stroke
  • Lower BP goals with hemorrhagic stroke (avoid hemorrhagic expansion, especially in AVMs and aneurysms)
BP-AIS relationship

- BP increase is due to arterial occlusion (i.e., an effort to perfuse penumbra)
- High BP is a response, not a cause
- Lowering BP starves penumbra, worsens outcomes
Supportive therapy

• Glucose Management
  • Infarction size and edema increase with acute and chronic hyperglycemia
  • Hyperglycemia is an independent risk factor for hemorrhage when stroke is treated with t-PA

• Antiepileptic Drugs
  • Seizures are common after hemorrhagic CVAs
  • ICH related seizures are generally non-convulsive and are associated with higher NIHSS scores and tend to predict poorer outcomes
Hyperthermia

• Treat fevers!
  • Evidence shows that fevers > 37.5°C that persists for > 24 hrs correlates with ventricular extension and is found in 83% of patients with poor outcomes
Stroke algorithm

- Time goals set by the National Institute of Neurological Disorders (NINDS) for in-hospital management
- Immediate general assessment by a stroke team, emergency physician, or other expert within **10 minutes of arrival**, including the order for an **urgent CT scan**
- **Neurologic assessment** by stroke team and **CT scan performed** within **25 minutes** of arrival
- **Interpretation of CT scan** within **45 minutes** of ED arrival
- **Initiation of fibrinolytic therapy**, if appropriate, within **1 hour** of hospital arrival and **3 hours** from onset of symptoms Door-to-admission time of 3 hours
tPA

- Fast Facts
- Tissue plasminogen activator
- “clot buster”
- IV tpa window 3 hours
- IA tpa window 4.5 hours
- Disability risk ↓ 30% despite ~5% symptomatic ICH risk

- Contraindications
- Hemorrhage
- SBP > 185 or DBP > 110
- Recent surgery, trauma or stroke
- Coagulopathy
- Seizure at onset of symptoms
- NIHSS > 21
- Age?
- Glucose < 50
Mechanical thrombolyis

- Often used in adjunct with tPa
- **MERCI (Mechanical Embolus Removal in Cerebral Ischemia)** Retrieval System is a corkscrew-like apparatus designed to remove clots from vessels
- **PENUMBRA** system aspirates the clot
Prevention

- Secondary prevention
- Anti-platelet
- Anti-coagulant

AHA/ASA Guideline

Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack
A Guideline for Healthcare Professionals From the American Heart Association/American Stroke Association
Control questions

• What is TIA
• Types of stroke
• Early signs of stroke
• Therapeutic options
• Common stroke signs and symptoms
• Ischemic stroke series