Neurovascular Anatomy and Physiology
Objectives

- Identify normal vascular anatomy and physiology of the brain
- Define Stroke and sub-types
- Recognize clinical presentations of strokes in different vascular distributions
- Understand differences between imaging and blood flow test
- Understand the NIH Stroke Scale
- Understand the impact of aSAH
- Know the factors associated with aneurysmal growth and rupture
- Recognize the symptoms and presentation of aSAH
- Review the diagnostic work-up of SAH
- Describe the pathophysiology of SAH and its complications
- Understand the basis for SAH management
The Brain

- Word production
- Problem solving
  Planning
- Behavioural control
  Emotion
- Memory
  Emotion
  (not visible)
- Vision
- Word understanding
The Brain

- Brain is divided into lobes based on their location and function. One hemisphere is usually dominant in relation to the other. The majority of right handed individuals are left brain dominant.
- The frontal lobe controls executive function, personality, problem solving, planning skills, language areas and primary motor cortex.
- Parietal lobe for primary sensory cortex, visual spatial orientation, association cortex, and attention to the world around us.
The Brain

- The temporal lobe is primarily involved in memory.
- Occipital control interpretation of visual stimuli.
- Cerebellum coordinates our movement.
- Brain stem is the major relay area between the brain and the body. Brain stem is responsible for unconscious activity such as breathing and the beating of the heart.
The Coverings of the Brain

The brain is protected by many layers including the skin, skull Meninges (dura mater, arachnoid, and pia mater)
The Ventricles

- Within the cranial vault, the brain is bathed by CSF. CSF is produced within the ventricles and flows over the surface of the brain.
- The ventricles make the skull buoyant.
Axial Section of Brain

- Very often, we look at axial pictures of the brain on neuro-imaging.
Vascular irrigation of the brain

- The brain is supplied by 4 major vessels; Two internal carotid arteries anteriorly and 2 vertebral arteries posteriorly.
Vascular irrigation of the brain
Diagnostic Tests
Diagnostic Tests

- Diagnostic tests examine how the brain looks, works and gets blood supply. There are 2 categories:
  - Imaging Tests
  - Blood Flow Tests
Imaging tests

CT (Computed Tomography) scan or CAT scan give valuable information about the cause of the stroke, the location and extent of brain injury
Brain CT

Normal Brain CT

Ischemic Stroke

Hemorrhagic Stroke
Imaging tests

- CTP Quantitative determination of cerebral blood flow, can be performed quickly, allows to make distinction between the irreversibly damaged infarct core and the penumbra.
MRI/MRA (Magnetic Resonance Imaging) gives sharper and more detailed image than CT scan so it’s often used to diagnose small, deep injuries. Some contraindications (metal clips, pacemakers, claustrophobia)
Brain MRI

Normal brain MRI

Ischemic Stroke
Normal MRA
Vascular Imaging

- Ultrasound: carotid Duplex and transcranial Doppler. Non-invasive & cheap

- CTA: good correlation with angiography, uses contrast, therefore some contraindications (renal failure, allergy iodine)
Vascular Imaging

- Angiography most exact but carries some risk:
  - Groin
  - Hematoma
  - Stroke
  - Renal failure
  - Allergy iodine
STROKE

Acute focal neurological deficit due to spontaneous disruption of blood supply to the brain

Ischemic 70-80%
  Anterior circulation
  Posterior Circulation

Hemorrhagic 20-30%
  ICH
  SAH
  Other
Ischemic Strokes

- Occurs when blood vessels to the brain become narrowed or clogged thereby cutting off blood flow to brain cells
- 70-80% of all strokes are ischemic
- TIA or “mini stroke” may give some warning of a major ischemic stroke
- High blood pressure is most important modifiable risk factor for ischemic stroke
Ischemic Stroke: Thrombotic

- Caused by blood clot (thrombus) in artery to the brain
- Clots usually form in arteries damaged by arteriosclerosis
- Accounts for 60% of all strokes
Ischemic Stroke: Embolic

• About 20% of all strokes caused by a “wandering clot” (embolus) that’s formed usually in the heart or neck arteries
• Clot travels and clogs a blood vessel in or leading to the brain
Ischemic Penumbra

- Penumbra is defined as: “potentially salvageable region of brain tissue surrounding the core infarct in which there is enough blood flow to survive, but not enough to function”

Imaging Ischemic Penumbra
Stroke

Acute focal neurological deficit due to spontaneous disruption of blood supply to the brain

Ischemic 70-80%
- Anterior circulation
- Posterior Circulation

Hemorrhagic 20-30%
- ICH
- SAH
- Other
Anterior Circulation

- Anterior cerebral
- Middle cerebral
- Ophthalmic
- Posterior cerebral
- Basilar
- Right internal carotid
- Left internal carotid
- Right common carotid
- Right vertebral
- Right subclavian
- Brachiocephalic
- Aortic arch
- Left vertebral
- Left subclavian
- Left common carotid
STROKE SYNDROMES

• **Left Hemisphere**
  – Right HemiPLEGIA
  – Impaired language = aphasia
  – Intellectual impairment
  – Slow cautious behavior with little spontaneous movements
  – Right visual field loss
  – Left gaze deviation
STROKE SYNDROMES

- **Right Hemisphere**
  - LEFT HemiPLEGIA
  - Left hemisensory loss
  - Impaired Spatial Perception
  - Quick impulsive behavior
  - Poor Judgment
  - Denial-Neglect
  - Right-gaze preference
  - Left visual field deficit
Stroke

Acute focal neurological deficit due to spontaneous disruption of blood supply to the brain

Ischemic 70-80%

Anterior circulation

Posterior Circulation

Hemorrhagic 20-30%

ICH

SAH

Other
Posterior Circulation
Top of the Basilar Syndrome

Infarction of

- **Midbrain**
  - Unilateral or bilateral paralysis of upward/downward gaze
  - Impaired convergence, skew deviation of the eyes and pupillary abnormalities

- **Thalamus**
  - Sensory deficits
  - Seizures
  - Agitated delirium

- **Temporal and occipital lobes**
  - Visual defects, cortical blindness, Balint’s syndrome
  - Behavioral abnormalities
  - Somnolence, memory disturbances and agitated delirium, hallucinations
STROKE SYNDROMES

• BRAINSTEM
  – Crossed Signs- ipsilateral face and contralateral body
  – Sensory loss in all 4 limbs- BASILAR
  – Quadriparesis
  – Hemiparesis
  – Hemisensory loss
Cerebellar Stroke

• **Symptoms**
  – Vertigo, dizziness, nausea, vomiting, gait unsteadiness, limb clumsiness, headache, dysarthria, diplopia and decreased alertness

• **Signs**
  – Limb and gait ataxia, dysarthria, nystagmus, altered mental status
Cerebellar stroke
Stroke

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Ischemic 70-80%
  - Anterior circulation
  - Posterior Circulation

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  - ICH
  - SAH
  - Other
Hemorrhagic Stroke

- **Intracranial**
  - Extra-parenchymal
    - Subarachnoid
    - Subdural
    - Epidural
    - Intraventricular
  - Intra-parenchymal
    - Intracerebral
US Burden of ICH

- Annually 70,000 cases of ICH.
- Disproportionately affects minorities.
- Typically younger than ischemic stroke
- 20% functionally independent at 6 months
- Least treatable stroke subtype
## Pathophysiology of ICH

<table>
<thead>
<tr>
<th>Time course</th>
<th>Event</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seconds</td>
<td>Vascular rupture</td>
<td>Chronic micro vascular changes</td>
</tr>
<tr>
<td>1st hour</td>
<td>Hematoma formation</td>
<td>Blood pressure, coagulation abnormalities, local tissue distortion and shearing</td>
</tr>
<tr>
<td>1-6 hours</td>
<td>Hematoma growth</td>
<td>Blood pressure, peri hematoma vascular and tissue injury</td>
</tr>
<tr>
<td>1-72 hours</td>
<td>Peri-hematoma edema</td>
<td>Cellular, humeral toxicity, blood formation and degradation</td>
</tr>
</tbody>
</table>

Modified from F Rincon, SA Mayer: Current Opinion in Critical Care, April 2004
Causes

<table>
<thead>
<tr>
<th>Sub Cortical</th>
<th>Vs.</th>
<th>Lobar</th>
</tr>
</thead>
<tbody>
<tr>
<td>• HTN</td>
<td></td>
<td>• Trauma</td>
</tr>
<tr>
<td>• Amyloid angiopathy</td>
<td></td>
<td>• Venous thrombosis</td>
</tr>
<tr>
<td>• Bleeding diathesis</td>
<td></td>
<td>• Vascular malformations</td>
</tr>
<tr>
<td>• Recreational drugs</td>
<td></td>
<td>• Others</td>
</tr>
<tr>
<td>• Brain tumor</td>
<td></td>
<td>• Hyper-perfusion</td>
</tr>
</tbody>
</table>

![Brain scan images](image-url)
Hemorrhage

- **Clinical features**
  - Acute neurological deficit based on location
  - Additional features
    - Headache
    - Nausea, vomiting
    - Decreased level of alertness
    - Lethargy, coma
Cerebellar Hematoma

Before surgery

After surgery

Treatment with drainage alone rather than surgical evacuation is not recommended

(Class III;C)

(New recommendation)
STROKE

Acute focal neurological deficit due to spontaneous disruption of blood supply to the brain

Ischemic 70-80%
- Anterior circulation
- Posterior Circulation

Hemorrhagic 20-30%
- ICH
- SAH
- Other
Subarachnoid Hemorrhage
Subarachnoid Hemorrhage

- **Subarachnoid hemorrhage (SAH)** or subarachnoid hemorrhage, is bleeding into the subarachnoid space surrounding the brain, the area between the arachnoid membrane and the pia mater.

- The bleeding may occur spontaneously, usually from a cerebral aneurysm, or may result from trauma. Regardless of the cause, it is considered a medical emergency.

- Symptoms include an intense headache with a rapid onset, vomiting, and an altered level of consciousness.[1]
Causes SAH

- Common causes:
  - Trauma
  - Aneurysm

- Other
  - Cerebral venous thrombosis
  - Peri-mensencephalic SAH without aneurysm
  - Others
Symptoms of SAH

Meningeal irritation
• Severe “worse” headache
• Photophobia
• Nuchal rigidity, back pain
• Seizures

Increased Intracranial Pressure
• Vomiting
• Altered mental status
• Sudden loss of consciousness
• Double vision
Subarachnoid Hemorrhage

SAH can lead to death or severe disability even if recognized and treated at an early stage.

Treatment is with close observation, medication and early neurosurgical investigations and treatments. Subarachnoid hemorrhage causes between 1 and 7% of all strokes.

Of all people with SAH, 10-15% die before arriving in hospital, and average survival is 50%.[1]
SAH Signs: Third Nerve Palsy

Severe HA + double vision: Always consider aneurysm!

- Compressive CN III palsy by a P-com aneurysm:
- Ptosis (eyelid droop)
  Lateral deviation of the eye
- Pupillary dilatation (midriasis)
SAH Assessment: Clinical

- **Hunt & Hess scale** classifies severity SAH
  
  Grade 1 - Asymptomatic, mild headache, slight nuchal rigidity
  
  Grade 2 - Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
  
  Grade 3 - Drowsiness / confusion, mild focal neurological deficit
  
  Grade 4 - Stupor, moderate-severe hemi paresis
  
  Grade 5 - Coma, decerebrate posturing
SAH Assessment: CT

- **Fisher scale**: CT scale useful in predicting vasospasm:
  - Group 1 - No blood detected
  - Group 2 – SAH layer <1 mm
  - Group 3 - Localized clots, layer >1 mm
  - Group 4 – ICH or IVH
Case 1

- 71 year old Right Handed Male
  - 20 pack/year history of smoking
  - Uncontrolled HTN, DM
  - Recurrent episodes of loss of vision in his L eye
  - Did not present to medical attention
  - Brought to emergency room by family because of R face/arm weakness, unable to speak
Case 1

- Exam
  - T 97  PR 88  BP 218/116  RR 16  Glu 180mmol/L
  - CVS: High pitched carotid bruit audible over the L ICA
  - Awake, Alert, Grunted only, no words, follows no commands but will mimic actions.
  - Pupils 3mm, blink to threat bilaterally present, R NLF weakness (sparing of forehead) No R arm movement, He is able to lift the R leg off the bed.
  - NIHSS= 13
NIH Stroke Scale

- Is a 15-item systematic neurologic examination that provides a quantitative measure of stroke-related neurologic deficit
- Ratings for each item are scored with 3 to 5 grades with 0 as normal, and there is an allowance for un-testable items.
<table>
<thead>
<tr>
<th>NIH STROKE SCALE ITEM</th>
<th>Scoring Definitions</th>
<th>Score</th>
</tr>
</thead>
</table>
| **1a. LOC**           | 0=alert and responsive  
1=arousable to minor stimulation  
2=arousable only to painful stimulation  
3=reflex responses or unarousable |       |
| **1b. LOC Questions**--Ask pt’s age and month. Must be exact. | 0=Both correct  
1=One correct (or dysarthria, intubated, foreign lang)  
2=Neither correct |       |
| **1c. Commands**--open/close eyes, grip and release non-paretic hand, (Other 1-step commands or mimic ok) | 0=Both correct (ok if impaired by weakness)  
1=One correct  
2=Neither correct |       |
| **2. Best Gaze**--Horizontal EOM by voluntary or Doll’s. | 0=Normal  
1=partial gaze palsy; abnl gaze in 1 or both eyes  
2=Forced eye deviation or total paresis which cannot be overcome by Doll’s. |       |
| **3. Visual Field**--Use visual threat if nec. If monocular, score field of good eye. | 0=No visual loss  
1=Partial hemianopia, quadrantanopia, extinction  
2=Complete hemianopia  
3=Bilateral hemianopia or blindness |       |
| **4. Facial Palsy**--If stuporous, check symmetry of grimace to pain. | 0=Normal  
1=minor paralysis, flat NLF, asymm smile  
2=partial paralysis (lower face=UMN)  
3=complete paralysis (upper & lower face) |       |
<table>
<thead>
<tr>
<th><strong>NIHSS</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>5. Motor Arm</strong>—arms outstretched 90 deg (sitting) or 45 deg (supine) for 10 secs. Encourage best effort. Circle paretic arm in score box</td>
</tr>
</tbody>
</table>
| 0=No drift x 10 secs  
1=Drift but doesn’t hit bed  
2=Some antigravity effort, but can’t sustain  
3=No antigravity effort, but even minimal mvt counts  
4=No movement at all  
X=unable to assess due to amputation, fusion, fx, etc. |
| L or R |
| **6. Motor Leg**—raise leg to 30 deg supine x 5 secs. |
| 0=No drift x 5 secs  
1=Drift but doesn’t hit bed  
2=Some antigravity effort, but can’t sustain  
3=No antigravity effort, but even minimal mvt counts  
4=No movement at all  
X=unable to assess due to amputation, fusion, fx, etc. |
| L or R |
| **7. Limb Ataxia**—check finger-nose-finger; heel-shin; and score only if out of proportion to paralysis |
| 0=No ataxia (or aphasic, hemiplegic)  
1=ataxia in upper or lower extremity  
2=ataxia in upper AND lower extremity  
X=unable to assess due to amputation, fusion, fx, etc. |
| L or R |
| **8. Sensory**—Use safety pin. Check grimace or withdrawal if stuporous. Score only stroke-related losses. |
| 0=Normal  
1= mild-mod unilateral loss but pt aware of touch (or aphasic, confused)  
2=Total loss, pt unaware of touch. Coma, bilateral loss |
| **9. Best Language**—Describe cookie jar picture, name objects, read sentences. May use repeating, writing, stereognosis |
| 0=Normal  
1=mild-mod aphasia; (diff but partly comprehensible)  
2=severe aphasia; (almost no info exchanged)  
3=mute, global aphasia, coma. No 1 step commands |
| **10. Dysarthria**—read list of words |
| 0=Normal  
1=mild-mod; slurred but intelligible  
2=severe; unintelligible or mute  
X=intubation or mech barrier |
| **11. Extinction/Neglect**—simultaneously touch patient on both hands, show fingers in both vis fields, ask about deficit, left hand. |
| 0=Normal, none detected. (vis loss alone)  
1=Neglects or extinguishes to double simult stimulation in any modality (vis, aud, sens, spatial, body parts)  
2=profound neglect in more than one modality |
Case 1

71 yo RHM 20 pk year history of smoking and uncontrolled HTN, DM

Baseline NIHSS

Consciousness (0-3): 0
Orientation (0-2): 0
Commands (0-2): 0
Gaze (0-2): 0
Visual fields (0-3): 0
Facial Palsy (0-3): 2

Total: 13
Unilateral R face/ arm weakness > leg
Non fluent Aphasia
L carotid bruit
Case 1: Initial CT

- Features of early stroke on CBT
  - Hyperdense artery sign of a clot
  - Insular ribbon sign
  - Loss of grey white junction
Case 1: MRI, DWI
Case 1
What is the diagnosis?

L MCA Stroke
MRA

Normal

Abnormal
MCA Stroke

- **Left side:**
  - Right face and arm weakness
  - **Broca’s aphasia.**
  - Right face/arm sensory loss

- **Right side:**
  - Left face/arm weakness
  - Left **hemi-neglect** (variable extent)
  - Left face/arm sensory loss.
Case 2

- 56 year old Hypertensive man with prior history of drug abuse, presents with:
  - Vitals signs T 98.6 PR 84 BP 225/130 RR 20
  - CVS no murmurs or bruits,
  - Drowsy, dysarthric and with major headache
  - Pupils 3mm ERL
  - Severe nausea and vomiting
  - Gait deferred because of drowsiness
Case 2 : CT
Case 2
What is the diagnosis?
ICH
Medical Management of Acute Cerebrovascular Syndromes

Dr. Gillian L. Gordon Perue
Acute Cerebrovascular Syndromes

- New term introduced by AHA in 2013
  - Acute focal neurological deficit due to spontaneous disruption of the blood supply to the brain.
- Encompasses all subtypes of stroke
  - Acute ischemic stroke
  - Transient Ischemic attack
  - Acute intracerebral hemorrhages
  - Acute Subarachnoid hemorrhages
- Emphasizes early recognition to facilitate timely intervention.
- Treatment strategies are as varied as the different cause and depends on establishment of underlying cause.
Stroke Diagnosis

• Critical to determine the type of stroke in progress because treatment is different for ischemic stroke or a hemorrhagic stroke

• **Time = Brain**

• Based on Medical History, physical neurological examination, blood tests, diagnostic tests

• Rule out conditions with similar symptoms like: seizures, fainting, migraine, heart problems
CT Scan is Important in Acute Stroke Care
ACUTE ISCHEMIC STROKE MANAGEMENT PROTOCOL
Ischemic Penumbra

- Normal Brain: 50 cc/100g/min
  - Grey matter: 100 cc/100g/min
  - White matter: 20 cc/100g/min
- Penumbra: 10-20 cc/100g/min
  - 20-40% normal flow
- Irreversible injury: <10 cc/100g/min
  - <20% normal flow
## Loss of cerebral elements in acute stroke

*Time is Brain*

<table>
<thead>
<tr>
<th>Loss of</th>
<th>Neurons</th>
<th>Synapses</th>
<th>Myelinated fibers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Per infarct</td>
<td>1,200 million</td>
<td>8,300,000 million</td>
<td>7140 kilometers</td>
</tr>
<tr>
<td>Per hour</td>
<td>120 million</td>
<td>830,000 million</td>
<td>714 kilometers</td>
</tr>
<tr>
<td>Per minute</td>
<td>1.9 million</td>
<td>14,000 million</td>
<td>12 kilometers</td>
</tr>
<tr>
<td>Per second</td>
<td>32,000</td>
<td>230 million</td>
<td>200 meters</td>
</tr>
</tbody>
</table>

Based on average supratentorial stroke (54 ml)  
JL Saver. Stroke 2006;37:263
Steps to Recovery

Objectives of acute ischemic stroke therapy

- Early Reperfusion
  - Intravenous thrombolysis
  - Endovascular therapies
- Neuroprotection strategies
- Save the penumbra
  - Promote collateral flow
- Prevent complications
  - Complications ultimately causes progression of infarct
- Secondary prevention
- Early Rehabilitation
AHA/ASA Acute ischemic stroke guidelines

Assess
CT / EKG/ Lab
10 15 25 45
Results
IV t-PA
60 60+
IA…
Timing of arterial recanalization and outcome

Christou, I. et al. Stroke 2000;31:1812
Mechanism of Action of tPA

Plasminogen

\[ \text{tPA} \rightarrow \text{Plasmin} \]

\[ \text{PAI} \rightarrow \text{Plasmin} \]

\[ \text{Plasmin} \rightarrow \text{alfa2} \]

\[ \text{Plasmin} \rightarrow \text{antiplasmin} \]

Fibrinogen \[ \rightarrow \] Fibrin
IV rtPA in Acute Stroke
Recommended use

- Acute ischemic stroke within 3 (4.5) hours
- Significant neurological deficit
- “Normal” CT
- Keep SBP <180, DBP <110
- No historical or laboratory contraindication
- 0.9 mg/kg, 10% bolus, rest over 1 hour
- No antithrombotics for 24 hours

Arterial/lumbar puncture < 7d
Surgery <14 d
GI/GU hemorrhage < 21d
MI/Stroke <3 m
Cerebral Hemorrhage
Cerebral Aneurysm
Platelets <100 K
INR >1.7
Glucose <50 mg/dL
Thrombolysis 3-4.5 hours: ECASS 3

- IV alteplase (0.9 mg/kg) vs. placebo
- 1° endpoint: mRS 0-1 at 90 d
- Safety: death, symptomatic ICH, other SAE
- N=821
- ECASS 3 specific exclusion criteria
  - Age >80
  - NIHSS >25
  - Combination of previous stroke + diabetes
  - Oral anticoagulants regardless of INR

Hacke et al. ECASS 3. NEJM 2008; 359:317
Treatment for eligible patient with IV-TPA or other acute reperfusion intervention

• In patients eligible for IV TPA, hypertension needs to be controlled <185/110 before starting the administration of the thrombolytics
  • Labetalol 10-20 mg IV *1
  • Nicardipine 5mg/h IV max of 15hr
Management of blood pressure during and after treatment with IV-TPA or other acute reperfusion intervention

- BP management during and after treatment with IV-TPA
- Monitor blood pressure:
  - Every 15 minutes during treatment and then for another 2 hours, then
  - Every 30 minutes for 6 hours, and then
  - Every 1 hour for 24 hours
Example of endovascular therapy

DWI

PWI

MRA
Endovascular Rx for Acute Stroke

Strategies:
- Thombectomy
  - Removal of thrombus occluding the artery
- Thrombus disruption
  - Fragmentation of thrombus
- Augmented fibrinolysis
  - Mechanical enhancement of native fibrinolytic mechanism
- Multi-modal
MERCI RETRIEVER
PENUMBRA CATHETER

Solitaire Retrievable Stent
Cerebral Angiogram

Left terminal ICA occlusion

MERCI Thrombus Retrieval
Endovascular

2 cm firm thrombus retrieved
Cerebral Angiogram

3 passes and 5 mg IA t-PA
Guidelines Acute Stroke Thrombolysis at JMH

0-3 h
- IV rtPA 0.9 mg/kg
  - Improvement
    - Stop
  - No improvement
    - Neuroimaging
      - No salvageable tissue
        - Open large vessels
      - Salvageable tissue
        - Endovascular approaches

3-4.5 h
- Age <80
  - NIHSS <25
    - No Prior stroke +DM
  - NIHSS >25
    - Prior stroke +DM

4.5-8 h
- Age >80
  - NIHSS >25
    - Prior stroke +DM

1

2

3
Steps to Recovery

Objectives of acute ischemic stroke therapy

- Early Rehabilitation
- Secondary prevention
- Prevent complications
  Complications ultimately causes progression of infarct
- Save the penumbra
  Promote collateral flow
  Neuroprotection strategies
- Early Reperfusion
  Intravenous thrombolysis
  Endovascular therapies
BLOOD PRESSURE MANAGEMENT IN ACUTE ISCHEMIC STROKE

(Based on: Guidelines for early management of adults with ischemic stroke. Stroke. 2013;44:870-947)
BLOOD PRESSURE MANAGEMENT IN ACUTE ISCHEMIC STROKE

- If patient is not eligible for treatment with intravenous TPA or other acute reperfusion intervention, then it is not necessary to reduce blood pressure so aggressively.
Patient not eligible for IV rt-PA:

- Only treat Systolic >220 mm Hg or diastolic >120 mm Hg
  - Labetalol 10 mg IV over 1 to 2 minutes, may repeat every 10 to 20 minutes, maximum dose of 300 mg;
  - or Labetalol 10 mg IV followed by an infusion at 2 to 8 mg/min
Patient not eligible for IV rt-PA

- Systolic >220 mm Hg or diastolic 121 to 140 mm Hg
  - or Nicardipine infusion, 5 mg/h, titrate up to desired effect by increasing 2.5 mg/h every 5 minutes to maximum of 15 mg/h
Patient not eligible for IV rt-PA

- Systolic >230 mm Hg or diastolic 121 to 140 mm Hg
  - If blood pressure not controlled, after labetalol or nicardipine, consider sodium nitroprusside
Steps to Recovery

Objectives of acute ischemic stroke therapy

- Early Reperfusion
  Intravenous thrombolysis
  Endovascular therapies

- Save the penumbra
  Promote collateral flow
  Neuroprotection strategies

- Prevent complications
  Complications ultimately causes progression of infarct

- Secondary prevention

- Early Rehabilitation
Prevent Complications

- Aspiration Pneumonia
  - Dysphagia screen prior to any PO intake (medications)

- Deep Vein Thrombosis
  - No role for full anticoagulation
  - Mechanical: SCDs
  - Pharmacological
    - Heparin subcutaneous, initiate 24 hours after intravenous thrombolytic therapy
  - Early Ambulation
Stroke Unit

- Coordinated multidisciplinary team
- Staff with expertise in stroke monitoring
- BP, glycemic, temperature management
- Early mobilization
- DVT, GI prevention
- Training & education programs

<table>
<thead>
<tr>
<th></th>
<th>Patients Treated on General Wards</th>
<th>Patients Treated on Stroke Unite</th>
<th>P</th>
<th>Relative Risk*</th>
<th>95% CI</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case-fatality rate (30 days)</td>
<td>69 (23%)</td>
<td>161 (17%)</td>
<td>0.03</td>
<td>0.45</td>
<td>0.28 to 0.71</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Died during hospital stay</td>
<td>89 (29%)</td>
<td>214 (23%)</td>
<td>0.01</td>
<td>0.50</td>
<td>0.34 to 0.74</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>6-month mortality</td>
<td>106 (35%)</td>
<td>258 (28%)</td>
<td>0.01</td>
<td>0.57</td>
<td>0.39 to 0.82</td>
<td>0.002</td>
</tr>
<tr>
<td>1-year mortality</td>
<td>120 (39%)</td>
<td>300 (32%)</td>
<td>0.01</td>
<td>0.59</td>
<td>0.42 to 0.84</td>
<td>0.003</td>
</tr>
<tr>
<td>5-year mortality</td>
<td>216 (71%)</td>
<td>597 (64%)</td>
<td>0.02</td>
<td>0.60</td>
<td>0.42 to 0.85</td>
<td>0.003</td>
</tr>
</tbody>
</table>

Aspirin in Acute Ischemic Stroke

- Aspirin should be initiated soon after stroke
- If thrombolysis, delay antithrombotics 24 h
  - Aspirin 325 mg within 24-48 h recommended for most patients (Class I, Level of Evidence A)
- Caution in malignant infarction, anticipated decompression

CAST Study:
- 20,000 patients <48 hours
- ASA 160 mg/day vs. placebo for 4 weeks
- 0.6% decrease death (3.3-3.9%)
- 0.5% decrease recurrent strokes (1.6-2.1%)
- 0.2% increase hemorrhagic strokes 1.1-0.9%

CAST. Lancet 1997: 349:1641
ACUTE ISCHEMIC STROKE MANAGEMENT PROTOCOL

• Temperature management:
  – Sources of fever should be treated and antipyretic medications should be administered to lower temperature in febrile patients.

• Glucose management:
  – Regular insulin sliding scale to treat glucose concentrations >121 mg/dL
ACUTE ISCHEMIC STROKE MANAGEMENT PROTOCOL

- If indicated: MRI, angiography, TEE, 24-hour holter monitor, lipid panel, hemoglobin A1C, hypercoagulability profile.
- Physical therapy/occupational therapy/speech therapy (with dysphagia evaluation)
- Deep vein thrombosis prophylaxis
- Secondary stroke prevention
- GI prophylaxis
ACUTE ISCHEMIC STROKE MANAGEMENT PROTOCOL

- After initial therapy, a stroke work up is done in order to determine the cause of the stroke, and prevent recurrence:
  - Holter
  - Echocardiogram
  - CT brain (plain) at 24 hours.
  - Transcranial Doppler Ultrasound (TCD)
  - Carotid duplex US
  - Advanced imaging
Acute Stroke Therapy Summary

- Treatment in a multidisciplinary Stroke Unit
- IV rtPA < 3 hours from onset has established benefit
- Data and recommendations support IV rtPA up to 4.5 hours
- Age is not an absolute contraindication to IV rtPA < 3 hours
- Time to treatment is most important predictor of outcome
- Avoid very high and very low BP
- Avoid hyperthermia and hyperglycemia
- Start aspirin soon if no contraindication
Steps to Recovery

- Objectives of acute ischemic stroke therapy

  - Early Reperfusion
    - Intravenous thrombolysis
    - Endovascular therapies
  - Save the penumbra
    - Promote collateral flow
    - Neuroprotection strategies
  - Prevent complications
    - Complications ultimately causes progression of infarct
  - Secondary prevention
  - Early Rehabilitation
Secondary prevention

- Blood pressure control
- Blood sugar control
- Antithrombotic therapy
  - Starts by day two or earlier (t-PA)
- Lipid lowering treatment = Statin
  - Ischemic Stroke
  - Stroke + LDL>100mg/dl
  - Statin prior to admission
    - Contraindications (if any) must be documented
- Anticoagulant in presence of A. fib
  - Contraindications (if any) must be documented
Coagulation Cascade and New Anticoagulants

Intrinsic Pathway

Extrinsic Pathway

XIIa
IXa
Xa
VIIa

Tissue Factor

Warfarin
Tecarfarin
II, VII, IX, X

Prekallekrein
Kininogen

Rivaroxaban
Apixaban
Idraparinux

Inhibition of Thrombolysis

Platelet Activation

Reactivation of Coagulation Cascade

FIBRIN

Acute Cerebrovascular Syndromes

- New term introduced by AHA in 2013
  - Acute focal neurological deficit due to spontaneous disruption of the blood supply to the brain
- Encompasses all subtypes of stroke
  - Acute ischemic stroke
  - Transient Ischemic attack
  - Acute intracerebral hemorrhages
  - Acute Subarachnoid hemorrhages
- Emphasizes early recognition to facilitate timely intervention
- Treatment strategies are as varied as the different cause and depends on establishment of underlying cause
INTRACEREBRAL HEMORRHAGE

• Intracerebral hemorrhage may be:
  – Primary
  – Or secondary due to:
    • Coagulopathy:
      – Patient anticoagulated (heparin/coumadin)
      – Patient treated with thrombolytics (IV TPA)
      – Hematological disorder (decreased platelet count, etc)
INTRACEREBRAL HEMORRHAGE
MANAGEMENT

• Patients neurological status may deteriorate:
  – Mass effect
  – Hydrocephalus
  – Medical complications
    • Infections
    • Pulmonary embolism
INTRACEREBRAL HEMORRHAGE MANAGEMENT

• Add to initial labs:
  – Toxicology screen in urine.
  – Pregnancy test in women of childbearing age.

• Admit to NSICU or Stroke Unit.
INTRACEREBRAL HEMORRHAGE MANAGEMENT

- Elevate head of the bed to 30°.
- Head should be midline, and head turning to either side should be avoided.
- Endotracheal tube tape should not compress jugular veins.
Management of HTN in primary ICH

- Clinically reexamine the patient every 15 minutes
- Intravenous medications to be considered for treatment of elevated blood pressure in patients with primary ICH:
Primary ICH
Blood pressure treatment

• Treatment goal:
  – If no concern for increased ICP the treatment goal is slightly lower
  – Systolic blood pressure < 140mmHg

• Blood pressure monitoring every 5 minutes! (if outside parameters)
Primary ICH

Blood pressure treatment

- Cerebral perfusion pressure (CPP) is the difference between mean arterial pressure (MAP) and intracranial pressure (ICP)
- CPP = MAP - ICP
- MAP = (systolic pressure + 2 x diastolic pressure)
Primary ICH Acute Management

- Management of arterial hypertension (HTN) depends mostly on 2 aspects:
  - Level of blood pressure elevation
  - Presence of increased intracranial pressure (ICP)
- Signs of increased ICP are:
  - Decreased level of alertness
  - Lethargy, coma
  - Nausea, vomiting
  - Evidence of mass effect on CT
Acute Blood Pressure Treatment
AHA Guidelines

- AHA 1999: MAP 130mmHg (~190/100)
- AHA 2007: MAP 110 mmHg (160/80)
  - But 130 mmHg if increased intracranial pressure is present
- AHA 2010:
  - In patients with systolic pressure between150-220mmHg it is probably safe to lower blood pressure to 140mmHg systolic.
    » (Class II A, reasonable)
    » (New recommendation)
Intravenous medications for HTN in primary ICH

<table>
<thead>
<tr>
<th>Drug</th>
<th>Intravenous bolus dose</th>
<th>Continuous infusion rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>5 to 20 mg every 15 min.</td>
<td>2 mg/min (maximum 300 mg/d)</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>NA</td>
<td>5 to 15 mg/h</td>
</tr>
<tr>
<td>Enalapril</td>
<td>1.25 to 5 mg IV P every 6 h*</td>
<td>NA</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>NA</td>
<td>0.1 to 10 μg/kg/min</td>
</tr>
</tbody>
</table>

NA, not applicable.

*Because of the risk of precipitous blood pressure lowering, the enalapril first test dose should be 0.625 mg.
ICP management

- Hyperventilation to keep pCO2 32-34 mm Hg
- Mannitol
  - Hit hard (1 to 1.5 g/kg or more for initial dose)
  - Continue 0.25-0.5g/kg q 4-6 hours
  - Try to keep serum osmolality < 320 mOsm/l
  - Maintain euvolemia
- Hypertonic saline (aim for Na 150-160)
- Place ventriculostomy if hydrocephalus
- Generally avoid steroids
Seizures

- Incidence of seizures may be 10-20%
  - Usually at the time of onset
- With continuous EEG monitoring 20-30%
- Continuous EEG monitoring for those with unexplained depressed mental status
- Clinical seizures should be treated with antiepileptic drugs

(Class I; A)
Seizures

- Prophylactic anticonvulsant medication should not be used.
  (Class III, B)
  (New recommendation)

- This may be harmful
  - Data mostly from patients on phenytoin
INTRACEREBRAL HEMORRHAGE MANAGEMENT

• If patient presents seizures:
  – IV diazepam (5-10 mg IV q 10-15 min. as indicated by neurologist)
  – IV phenytoin (Initial dose: 15-20 mg PE/kg IV x 1; then 4-6 mg PE/kg IV TID, as indicated by neurologist)
Prevent further Injury

- Fever:
  - High incidence of 80-90%.
  - May be infectious or due to presence of hematoma itself
  - Is independently associated with a poor outcome.
Prevent further Injury

- Hyperglycemia:
  - Associated with poor outcome
  - Diabetics also have worsened outcome
  - May exacerbate peri-hematoma edema
  - Avoid glucose containing IV fluids
To Have in Mind

- DVT prophylaxis indispensable
  - Avoid SQ heparin early in the course (&lt; 48 h)
  - Use Intermittent Pneumatic Compression devices
Increasing Mass Effect
Surgical Treatment

- For the majority of ICH no clear surgical indications
- A good number of trials have assessed surgical evacuation with no clear beneficial result
Surgical Treatment

- Few clear indications:
  - Cerebellar hematoma > 3cm or 40ml
  - Superficial expanding ICH in a deterioration young patient.

- No role in brainstem or deep thalamic ICH

- No role for ultra-early surgery
INTRACEREBRAL HEMORRHAGE MANAGEMENT

• On call neurosurgery pager:
  – (305) 585-2255 ext 0311

• Neurovascular attending physicians:
  – Dr. Roberto Heros, (Contact pager operator (305) 585-5400, for cell phone connection)
  – Dr. Jacques Morcos, Pager number: (305) 736-4916
  – Dr. Ali Sultan, Pager number: (305) 277-1702
Large Superficial Clots

Before Surgery

After Surgery
Cerebellar Hematoma

Before Surgery

After Surgery

Treatment with ventricular drainage alone rather than surgical evacuation is not recommended

(Class III; C)
(New recommendation)
Intracerebral Hemorrhage
Warfarin Associated

Acute Management
Warfarin associated ICH

- 10-20% of all Intracranial Cerebral Hemorrhage
- Carries worse prognosis
- Greater initial hemorrhage size
- Greater hematoma growth
- High INR also adversely affects mortality in ICH
  - 62% vs. 17%

(CHANT Trial, Cucchiara 2008)
INTRACEREBRAL HEMORRHAGE SECONDARY TO COAGULOPATHY

• If patient is anti-coagulated with heparin:
  – Treat with Protamine sulfate (1mg per 100 Units of heparin patient is receiving)
    – Note: Protamine sulfate dose needs to be adjusted according to time elapsed since the last heparin dose
INTRACEREBRAL HEMORRHAGE SECONDARY TO COAGULOPATHY

- If patient is anticoagulant with warfarin (Coumadin):
  - Use Vitamin K (10 mg IV x 1 and subsequent doses based on INR)
  - Consider activated factor VII, and Pro-thrombin Complex Concentrates
  - Also fresh frozen plasma (FFP) (15-20 mL/Kg)
Prothrombin Complex Concentrates

- Primarily to treat Factor IX deficiency
- Contain II, VII, IX, X
- Increasingly recommended for warfarin reversal
- Reversal within minutes
- Factor VII does not provide all the clotting factors
  - Recommendations against routine use of Factor VII only for reversal of oral anticoagulation associated hemorrhage [American Society of Hematology and AHA guidelines 2010]
Warfarin Associated ICH

- PCCs have not shown improved outcome compared with FFP but may have fewer complications compared with FFP and are reasonable to consider as an alternative to FFP  
  (Class IIa; B)

- JMH protocol
- PCC and FFP
Dabigatran and Rivaroxaban and ICH

- PTT may be elevated, INR is not changed
  - Ecarin Clotting Time [ECT]
- Overdose if ingested within 1-2 hours
  - Activated charcoal
  - Hemodyalisis [due to low protein binding]
- Vitamin K and protamine do not help
Dabigatran and Rivaroxaban and ICH

- Consider PCC and FFP

- Short half life of Dabigatran/Rivaroxaban
  - ~12 hours in absence of renal failure
INTRACEREBRAL HEMORRHAGE SECONDARY TO COAGULATIONPATHY

- If patient was treated with IV thrombolysis (IV TPA) and had a hemorrhagic complication:
  - Infuse platelets (6 to 8 Units) and cryoprecipitates that contain factor VIII

- If there is a hematological disorder
  - Treat accordingly depending on the cause (platelet transfusion, steroids, etc)
Case examples
Case 1

- 72 y/o right-handed woman with h/o HTN
- Presents to ER with left-sided weakness for 2 days.
- Has not been taking her BP medications for 6 months
Case 1

- Vital signs:
  - BP: 200/110
  - HR: 88/min
  - RR: 16/min

- Examination
  - Awake and oriented to person, place and time
  - Mild left facial palsy
  - Left hemiparesis (arm/leg drift)
Brain CT scan: Intracerebral hemorrhage (ICH)

- Intracerebral hemorrhage:
  - Blood is “white” in plain (non-contrasted) brain CT scan (black arrow)
  - This hematoma is located in the right basal ganglia, one of the most common places for hypertensive ICH

- Important:
  - No significant “mass effect”: there is no shifting of brain tissue across the midline
  - No extension of the ICH into the ventricles (cavities inside the brain, filled with cerebrospinal fluid) (white arrows)
Case 1

- Patient was treated with Cardene drip, starting at 5 mg/hour, titrated to keep SBP <180 and DBP <110
- After 48 hours, treatment was switched to oral medications (Lisinopril/hydrochlorothiazide)
- Her blood pressure was well controlled and after a week, patient was DC to home
Follow-up, case 1

- CT head, 3 months later:
  
  Resolution of ICH
Case 2
Different outcome
Case 2

- 40-year-old, right-handed man with history of severe hypertension
- Non compliant with medications
- 2 previous episodes of intracerebral hemorrhages in the last 3 years
- Seizure disorder
Case 2

- Found by a neighbor at 11 AM, unresponsive on the ground.
- Last seen well: previous night at 10 PM.
- Brought to ER by EMS. Intubated on the field
- No convulsive movements seen
Case 2

- Vital signs:
  - BP: 240/140
  - HR: 110/min
  - RR: 24/min
  - O2 Sat: 95%

- On exam:
  - Comatose
  - Pupils are equal, round and sluggishly reactive to light
  - Withdraws to painful stimuli on right arm/leg
  - No movements seen on left hemibody.
Case 2: Initial CT scan

- Large right ICH (red arrow):
  - Mass effect with shifting of tissue across the midline
  - Intraventricular extension (white arrow)
  - Hydrocephalus (dilated ventricles) [arrow head]
Case 2

• Treatment:
  – HTN: Cardene drip
  – Hydrocephalus: External ventricular drainage (Ventriculostomy)
Case 2

- After prolonged hospitalization, patient required tracheostomy (for mechanical ventilation) and percutaneous gastro-jejunostomy placement (for feeding)
- He remained with severe disability: Left hemiplegia and profound cognitive impairment
- Discharged to a nursing home
Case 2

- Follow-up CT scan
- Resolution of the hematoma
- Enlarged ventriles and cerebral atrophy
Acute Cerebrovascular Syndromes

- New term introduced by AHA in 2013
  - Acute focal neurological deficit due to spontaneous disruption of the blood supply to the brain
- Encompasses all subtypes of stroke
  - Acute ischemic stroke
  - Transient Ischemic attack
  - Acute intracerebral hemorrhages
  - Acute Subarachnoid hemorrhages
- Emphasizes early recognition to facilitate timely intervention.
- Treatment strategies are as varied as the different cause and depends on establishment of underlying cause.
Causes of Subarachnoid Hemorrhage (SAH)

- Common causes:
  - Trauma
  - Aneurysm (aSAH)
- Other
  - Cerebral venous thrombosis
  - Perimesencephalic SAH
  - Reversible vasoconstriction syndrome
  - Others
Aneurysmal Subarachnoid Hemorrhage
aSAH
Epidemiology aSAH

• 1/3 of all hemorrhagic strokes
  – 12% initially misdiagnosed
• 30,000 aneurysmal SAH each year in US
• Considerable worldwide variation: incidence 2/100,000 China vs. 22.5/100,000 Finland, US 9-14.5/100,000
• Typical age onset in 40-60, mean >50 years
• Women at greater risk: incidence 1.24 times vs. men
Risk Factors for aneurysm formation and rupture

**Modifiable**
- Hypertension
- Tobacco
- Excessive alcohol
- Cocaine, other sympathomimetics

**Non-modifiable**
- Female sex
- Presence of unruptured aneurysm
- First degree relative with aSAH
- Genetic syndromes: PCKD, ED IV
Prevention aSAH

- Hypertension should be treated; treatment will prevent ischemic stroke, intracerebral hemorrhage, cardiac and renal disease (I-A), and may reduce risk of aSAH (I-B)
- Tobacco use and alcohol misuse should be avoided to reduce risk of aSAH (I-B)
- Diet rich in vegetables may lower risk of aSAH (IIb-B)
- Reasonable to screen in those with familial aSAH: at least 1 first degree relative (IIb-B)
Symptoms of SAH

Meningeal irritation

- Increased intracranial pressure
- Severe “worse” headache
  - Sentinel HA in 10-43%
- Photophobia
- Nuchal rigidity, back pain
- Seizures

- Vomiting
- Altered mental status
- Sudden loss of consciousness
- Double vision
SAH Signs: Third Nerve Palsy

Severe HA + double vision: always consider aneurysm!

Compressive CN III palsy by a P-com aneurysm
Outcomes aSAH

- Mortality 32% US, 44% Europe, 27% Japan
- Does not account for pre-hospital death: 12-15%
- 50% fatality at 30 days
- 20% major disability/dependent
- Another 20% “independent” with significant cognitive impairment
- 50%
- Lifetime cost $228,000
Outcome after aSAH

- Severity of hemorrhage is the most important predictor of outcome
- Severity graded by clinical status and amount of blood on CT
- Clinical scales: Hunt and Hess, World Federation of Neurological Surgeons
  - Initial clinical severity should be determined by validated scales as an outcome indicator (I-B)
- Radiological scales: Fisher
SAH Assessment: Clinical

- Hunt & Hess scale classifies severity SAH
  Grade 1 - Asymptomatic, mild headache, slight nuchal rigidity
  Grade 2 - Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
  Grade 3 - Drowsiness / confusion, mild focal neurological deficit
  Grade 4 - Stupor, moderate-severe hemiparesis
  Grade 5 - Coma, decerebrate posturing
SAH Assessment: CT

- Fisher scale: CT scale useful in predicting vasospasm:
  Group 1 - No blood detected
  Group 2 - SAH layer <1 mm
  Group 3 - Localized clots, layer >1 mm
  Group 4 – ICH or IVH
Clinical Case

• 18 y/o previously healthy man
• Soon after consuming cocaine in a party, he developed sudden-onset, severe headache and then lost consciousness
• Brought to the hospital by EMS
Clinical Case

- Vital signs:
  - BP: 190/95
  - HR: 65/min
  - RR: 12/min
  - T: 98°F
  - O2 Sat: 98%

- On exam:
  - Comatose
  - Right pupil was dilated and sluggishly reactive to light
  - Stiff neck
Plain brain CT scan (case 1)

Abnormal hyper-density in subarachnoid space, consistent with bleeding (red arrows). Compare to normal CT head, below:
SAH Assessment: CSF

Xanthochromia

- CSF done if CT without SAH
- Xanthochromia: yellow CSF
  - It is the result of presence of blood products in the CSF
- Xanthochromia in a patient with severe headaches is very suggestive of SAH.
Diagnosis of aSAH

- High level of suspicion in acute severe headache (I-B)
- Urgent evaluation to reduce risk of re-bleeding (I-B)
- Initially CT, followed by LP if CT non-diagnostic (I-B)
- CTA and MRI (FLAIR, GRE) may be considered if prior non-diagnostic (IIb-C)
- Catheter angiography required to detect aneurysm and plan treatment (I-B)
SAH Complications

- Most common complications:
  - Rebleeding
  - Hydrocephalus
  - Seizures
  - Hyponatremia
  - Vasospasm
SAH: Initial ICP & BP management

• If patient is alert: BP control with titratable agent (I-B); goal SBP < 160 mmHg (IIa-C) but at JMH recommend <140 mmHg
  – Labetalol 10 to 20 mg IV over 1 to 2 minutes, may repeat x1;
  – Nicardipine infusion, 5 mg/h, titrate up by 0.25 mg/h at 5 to 15-minute intervals, maximum dose 15 mg/h; when desired blood pressure attained, reduce to 3 mg/h.

• If patient is lethargic or comatose: avoid excessive BP reduction, consider ICP monitor in place to manage CPP

• After aneurysm secure: avoid hypotension, hypo-voleemia
SAH: Prevention of rebleeding

**Rebleeding**
- 4% first day
- 1-2% /day x 14 days
- 20% at 2 weeks

**Initial measures**
- BP control: avoid hypertension
- Avoid antithrombotics: SCDs
- Avoid Valsalva: stool softeners, antiemetics, analgesia

**Secure aneurysm**
- Surgical Clipping
- Endovascular Coiling

**After aneurysm secure**
- Avoid hypotension, hypovolemia
- OK SQ heparin
Prevention of Rebleeding: Correct Coagulopathy

- Warfarin, prolonged INR: Vit K, PCC, FVII, FFP
  - Post rtPA: Platelets, Cryoprecipitate
  - Thrombocytopenia: Platelets
  - Stop antiplatelet agents
  - Antifibrinolytic treatment in non-coagulopathic cases only if unavoidable delay in securing aneurysm (IIa-B)
Secure aneurysm: prevent re-bleeding
Clipping and Coiling

- Should be performed as early as possible (I-B).
- Decision by expert team (I-C)
- Clipping: less likely to rebleed, complete occlusion, but craniotomy, greater disability.
  - MCA aneurysms, large ICH (IIb-C)
- Coiling: less disability in 1 study, more likely to have incomplete obliteration with risk of rebleeding
  - Elderly, poor clinical grade, BA aneurysms (Iib-C)
SAH Complications: Hydrocephalus

• Hydrocephalus
  – Early: Obstructive Hydrocephalus in 20-30%, life-threatening
  – Late: Communicating Hydrocephalus, disabling

• Diagnosis
  – Clinical suspicion!
  – CT confirmation

• Treatment
  – External Ventricular drain (I-B)
  – Ventricular shunt if needed (I-C)
SAH Complications: Hyponatremia

- Salt wasting syndrome different from syndrome of inappropriate antidiuretic hormone (SIADH): excessive sodium loss vs. water retention
- Water restriction is not recommended
- Replace sodium loss with hypertonic saline or fludrocortisone (IIa-B)
SAH Complications: Seizures

- Seizures increase BP, may lead to rebleeding if aneurysm unsecured
- Status epilepticus very dangerous
- Short-term seizure prophylaxis often indicated (IIb-B)
- Antiepileptic drug may not be necessary in good clinical grade patients (at intensivist’s discretion)
- EEG monitoring considered in poor grade
- If seizures occur: benzodiazepines, phenytoin
Vasospasm and Ischemia

- Angiographic vasospasm: 70%
- Clinical ischemia: 30-35%
- Radiological ischemia:
  - FLAIR 58%, diffusion 71%
- Autopsy:
  - 19% @ 3 days
  - 48% @ 4-14 d
  - 70% @ >14 d

SAH → Vasospasm → Cerebral Ischemia
Vasospasm: Diagnosis

- Early:
  - TCD, DSA
  - Cerebral blood flow
- Late:
  - Clinical findings
  - Parenchymal ischemic injury
Vasospasm Treatment

Prevention

• Monitor daily clinical exam
• Monitor daily TCD
• Nimodipine
• Avoid hypotension
• Avoid hypovolemia

Treatment

• Triple H therapy:
  – Hypertension
  – Hypervolemia
  – Hemodilution
    • Albumin
    • Vasopressors

• Endovascular treatment
  – Vasodilators
  – Angioplasty
Vasospasm and delayed cerebral ischemia

- Nimodipine to improve outcomes (I-A)
- Eu-volemia to prevent delayed cerebral ischemia (I-B)
- TCD to monitor for vasospasm (IIa-B)
- Induction of hypertension if DCI ensues (I-B)
- Angioplasty or IA vasodilators if no response (IIa-B)
- However, prophylactic HHH or angioplasty not useful (III-B)
Conclusions

- aSAH is an emergency with high fatality and poor outcomes.
- Diagnosis requires high level of suspicion.
- Rapid diagnosis leads to rapid treatment, which prevents re-bleeding.
- Securing the aneurysm should be done early.
- Awareness of pathophysiology leads to recognition and treatment of complications.
- Outcome depends on patient, aneurysm and institutional factors.
- Effective treatment is done by an expert multidisciplinary team.
- There are modifiable risk factors that prevent aneurysmal formation and rupture.
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JMH Stroke Education 2013

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