HEMORRHAGIC STROKE: THE GOLDEN HOUR

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DISCLOSURES: NONE

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ENTERPRISE STROKE PROGRAM MANAGER: CLEVELAND CLINIC HEALTH SYSTEM

DISCLOSURES: NONE
HEMORRHAGIC STROKE: OBJECTIVES

OBJECTIVES
• Discuss the 2 types of hemorrhagic stroke: intracerebral and subarachnoid
• Review cerebral anatomy
• Discuss acute treatment of hemorrhagic stroke
• Discuss transfers considerations

HEMORRHAGIC STROKE: TYPES

PATHOLOGICAL ACCUMULATION OF BLOOD WITHIN THE CRANIAL VAULT
• Intracranial hemorrhage
  • May occur within brain parenchyma or surrounding meningeal spaces
  • May extend into the ventricles (IVH)
• Subarachnoid hemorrhage
  • Aneurysmal rupture
  • AVM rupture
HEMORRHAGIC STROKE: LOCATIONS

INTRACEREBRAL HEMORRHAGE

MOST COMMON CAUSE: BLOOD VESSEL WALL DAMAGE DUE TO HYPERTENSION - 60% OF CASES

OTHER CAUSES:

- Autoregulatory dysfunction (re-perfusion injury, hemorrhagic transformation)
- Arteriopathy (amyloid angiopathy, moya-moya)
- Altered hemostasis (thrombolysis, anticoagulation)
- Hemorrhagic necrosis (tumor, infection)
- Venous outflow obstruction (cerebral venous thrombosis)
- Sympathomimetic drugs (cocaine, methamphetamine)
ICH: MORBIDITY/MORTALITY

20,000 DEATH ANNUALLY IN US

30 DAY OVERALL MORTALITY RATE OF 44%

• 75% at 24 hours with pontine/brainstem hemorrhages

Coronal section of the brain with a hypertensive putaminal hemorrhage associated with mass effect

Photographs courtesy of Jose Biller, MD

ICH: PATHOPHYSIOLOGY

ICH is a dynamic and complex process

ICH: EARLY DETERIORATION

EARLY DETERIORATION IS COMMON
GREATER THAN 20% WILL EXPERIENCE DECREASE IN GCS OF 2 OR MORE POINTS BETWEEN EMS ASSESSMENT AND ED INITIAL EVALUATION
15%–23% DEMONSTRATE CONTINUED DETERIORATION WITHIN THE 1ST FEW HOURS AFTER HOSPITAL ARRIVAL

ICH: HEMATOMA EXPANSION

RELATED TO ACTIVE BLEEDING THAT MAY PROCEED FOR HOURS AFTER SYMPTOM ONSET
TENDS TO OCCUR EARLY
INCREASES RISK OF POOR FUNCTIONAL OUTCOME AND DEATH
28%–38% HAVE HEMATOMA EXPANSION OF GREATER THAN 1/3 OF THE INITIAL HEMATOMA VOLUME ON FOLLOW UP CT
PATIENT PRESENTATION: HEMORRHAGIC STROKE

SUDDEN FOCAL NEUROLOGICAL DEFICIT
HEADACHE – 40%

NAUSEA AND VOMITING – 40%-50%
• Common with posterior fossa stroke

DECREASED LEVEL OF CONSCIOUSNESS – 50%
• Not as common with ischemic stroke

ELEVATED BLOOD PRESSURE – 90%

SEIZURES – 6–7%

ED MANAGEMENT: THE GOLDEN HOUR

• PERFORM A RAPID NEUROLOGICAL EX
• ABC’S/STABILIZE
• DIAGNOSE
  • Calculate the ICH score if possible
• CLASSIFY
• CORRECT COAGULAPATHY
• MANAGE BP
• COMMUNICATE WITH TEAM
• GET PATIENT TO THE RIGHT PLACE
ED MANAGEMENT: THE GOLDEN HOUR

**Suspicion of Intracerebral Hemorrhage**

- Adequate Airway
  - Yes
  - Non-Contrast Head CT
    - No ICH
      - ICH, Coma, or Other Appropriate ENLS Protocol
    - ICH
      - BP Control 140-180 mm Hg Systolic
      - Hemoctasis
        - INR < 1.4
        - Reverse anti-coagulants
        - Consider platelets or fibrinogen if surgery planned
      - Consider Surgery
        - Life-threatening deterioration
        - Cerebellar > 3 cm diameter
      - Consider EVD
        - Suspended T ICP and GCS < 9
        - Goal ICP = 20 mm Hg
        - Goal CPP = 60 mm Hg

**Characterize ICH**
- ICH Score
- Volume
- Location

**Transfer**
- Plan to communicate transfer to NCCU or capable care site

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**Checklist for the 1st hour**

- Complete blood count with platelet count, PT, PTT, INR
- Head imaging results: hematoma size, location, presence of intraventricular hemorrhage
- Glasgow Coma Scale (GCS) score
- Calculate ICH Score

**Interventions**

- Coagulopathy reversal (goal INR < 1.4)
- Blood pressure lowering (goal SBP 140-180)
- Surgical hematoma evacuation (if indicated)
- Airway/ventilation management
ED MANAGEMENT: THE GOLDEN HOUR

INITIAL FOCUS ON ARRIVAL:
• Life support (A-B-C)
• Make sure patient is safe to go to CT
  • Can they protect their airway?
  • Hypoxia?
  • Avoid hyperventilation

GOLD STANDARD FOR DIAGNOSIS OF ICH:
NON-CONTRAST HEAD CT
ICH: SPOT SIGN

CALCULATING ICH SCORE-

Supratentorial Origin L Temporal Lobe Mild IVH

Volume = \( \frac{ABC}{2} \)

\[ A = \text{largest diameter} = 4.2 \text{ cm} \]
\[ B = \text{perpendicular} = 2.8 \text{ cm} \]
\[ C = \text{clot thickness} = 2.5 \text{ cm} \]

\[ 0.25 \text{ cm slices x 10 slices (9 full and 2 half)} \]

\[ \frac{4.2 \times 2.8 \times 2.5}{2} = 14.7 \text{ cc} \]

ICH score is required for CSCs-
Not required for PSC/TSC or ASR
### CALCULATING ICH SCORE

<table>
<thead>
<tr>
<th>Component</th>
<th>ICH Score Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>GCS Score</td>
<td></td>
</tr>
<tr>
<td>3-4</td>
<td>2</td>
</tr>
<tr>
<td>5-12</td>
<td>1</td>
</tr>
<tr>
<td>13-15</td>
<td>0</td>
</tr>
<tr>
<td>ICH Volume (cc)</td>
<td></td>
</tr>
<tr>
<td>&lt; 30</td>
<td>1</td>
</tr>
<tr>
<td>&gt; 30</td>
<td>0</td>
</tr>
<tr>
<td>Intraventricular Hemorrhage</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Infratentorial Origin</td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Age (years)</td>
<td></td>
</tr>
<tr>
<td>&gt; 80</td>
<td>1</td>
</tr>
<tr>
<td>&lt; 80</td>
<td>0</td>
</tr>
<tr>
<td><strong>Total ICH Score</strong></td>
<td><strong>1</strong></td>
</tr>
</tbody>
</table>

### ICH SCORE - WHY?

- Each point increase in the ICH Score is associated with an increased risk of mortality and a decreased likelihood of good functional outcome.
- It should not be used for prognosis; use it as a method for communicating disease severity.

<table>
<thead>
<tr>
<th>Component</th>
<th>ICH Score Points</th>
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</thead>
<tbody>
<tr>
<td><strong>GCS</strong></td>
<td></td>
</tr>
<tr>
<td>3-4</td>
<td>2 pts</td>
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<tr>
<td>5-12</td>
<td>1 pt</td>
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<tr>
<td>13-15</td>
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<tr>
<td><strong>ICH volume</strong></td>
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<tr>
<td>&gt; 30 cm³</td>
<td>1 pt</td>
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<tr>
<td>&lt; 30 cm³</td>
<td>0 pts</td>
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<tr>
<td><strong>IVH</strong></td>
<td></td>
</tr>
<tr>
<td>Yes</td>
<td>1 pt</td>
</tr>
<tr>
<td>No</td>
<td>0 pts</td>
</tr>
<tr>
<td><strong>Location</strong></td>
<td></td>
</tr>
<tr>
<td>Infratentorial</td>
<td>1 pt</td>
</tr>
<tr>
<td>Supratentorial</td>
<td>0 pts</td>
</tr>
<tr>
<td><strong>Age</strong></td>
<td></td>
</tr>
<tr>
<td>&gt; 80 yrs</td>
<td>1 pt</td>
</tr>
<tr>
<td>&lt; 80 yrs</td>
<td>0 pts</td>
</tr>
</tbody>
</table>
SITES OF SPONTANEOUS ICH

- **Lobar Subcortical Hemorrhage** (24%)
- **Putaminal Hemorrhage** (34%)
- **Thalamic Hemorrhage** (20%)
- **Pontine Hemorrhage** (6%)
- **Cerebellar Hemorrhage** (7%)


CASE STUDY: PT PRESENTATION

45 YR OLD MALE

PRESENTS TO ED VIA AMBULANCE AT 2153 WITH C/O:
• Disorientation, slurred speech, facial droop, right sided weakness

STROKE TRIAGE
• Last normal/Onset time: 1830-1900
• Exam: drowsy, follows commands, weak on right, speech slurred, right facial droop, confused, GCS 14
• Finger stick glucose: 109

CASE STUDY: ICH PRESENTATION

VS: BP 198/100; HR 83; RR 18; SAO2 98%
IV START
LAB DRAW
• CBC, PT, INR, aPTT, BMP, type & screen
BEGIN COLLECTING FOCUSED HISTORY
• HTN, hyperlipidemia, obesity, sleep apnea, recently started on coumadin for AF; no trauma, surgeries, stroke, ICH
CT SCAN
EKG AT SOME POINT
CHEST XRAY IF INDICATED *Don’t delay CT for EKG/Chest X-ray; Hold off on labs if difficult stick*
CASE STUDY: ICH- DIAGNOSE AND CLASSIFY

LG ACUTE INTRAPARENCHYMAL HEMORRHAGE
LEFT BASAL GANGLIA
3.2 X 1.5 CM’S
RUPTURED INTO LEFT LATERAL VENTRICLE
SMALL AMOUNT OF BLOOD IN RIGHT LATERAL VENTRICLE AND 3RD VENTRICLE

CASE STUDY: ICH- LABS

<table>
<thead>
<tr>
<th>WBC</th>
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<tbody>
<tr>
<td>HGB</td>
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<td>HCT</td>
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<td>PLAT</td>
<td>198</td>
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<td><strong>INR</strong></td>
<td><strong>2.1</strong></td>
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<tr>
<td>APTT</td>
<td>35</td>
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<tr>
<td>GLU</td>
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<td>BUN</td>
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<td>CR</td>
<td>.96</td>
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<tr>
<td>NA</td>
<td>141</td>
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<table>
<thead>
<tr>
<th>K</th>
<th>3.3</th>
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</thead>
<tbody>
<tr>
<td>CL</td>
<td>119</td>
</tr>
<tr>
<td>CO2</td>
<td>17</td>
</tr>
<tr>
<td>CA</td>
<td>8.3</td>
</tr>
<tr>
<td>ALB</td>
<td>2.1</td>
</tr>
<tr>
<td>ALT</td>
<td>40</td>
</tr>
<tr>
<td>AST</td>
<td>38</td>
</tr>
<tr>
<td>ALK PHOS</td>
<td>92</td>
</tr>
<tr>
<td>T BILI</td>
<td>0.2</td>
</tr>
</tbody>
</table>
ICH: THE GOLDEN HOUR: WHAT CAN GO WRONG???

• What Can Go Wrong (or Is Going Wrong)?
  • Herniation and brain(stem) compression
  • Airway compromise
  • Hematoma expansion
  • Elevated intracranial pressure
  • Secondary brain injury
    » Seizures
    » Fever
    » Hyperglycemia

ICH: THE GOLDEN HOUR: FOCUS

1. Stabilization and reassessment of the patient’s airway, breathing, circulation
2. Rapid and accurate diagnosis using neuroimaging
3. Concise clinical assessment regarding ICH characteristics and patient condition
4. Targeted assessment for potential early interventions:
   Control of elevated blood pressure
   Correction of coagulopathy
   Need for early surgical intervention
5. Anticipation of specific patient care needs such as:
   Specific treatment related to underlying ICH cause
   Risk for early clinical deterioration and hematoma expansion
   Need for intracranial pressure (ICP) or other monitoring
   Patient disposition from the emergency department
CASE STUDY: ICH- BP MANAGEMENT

• FOR ICH PATIENTS PRESENTING WITH SBP BETWEEN 150 AND 220 MM HG AND W/O CONTRAINDICATIONS TO ACUTE BP TREATMENT, ACUTE LOWERING OF SBP TO 140 MMHG IS SAFE (CLASS 1, LEVEL A) AND CAN BE EFFECTIVE FOR IMPROVING FUNCTIONAL OUTCOME (CLASS IIA; LEVEL B)

• FOR ICH PATIENTS PRESENTING WITH SBP >220 MMHG, IT MAY BE REASONABLE TO CONSIDER AGGRESSIVE REDUCTION OF BP WITH A CONTINUOUS IV INFUSION AND FREQUENT BP MONITORING (CLASS ILB, LEVEL C)


CASE STUDY: ICH – BP CONTROL

COMMON MEDICATIONS

LABETALOL (NORMODYNE)
• Labetalol 10-20 mg IV over 1-2 mins, may repeat or double every 10 mins for max of 300mg

NICARDIPENE (CARDENE)
• Nicardipine 5mg/hr IV infusion as initial dose; titrate to desired effect by increasing 2.5mg/hr every 5 mins to max of 15mg/hr
CORRECTION OF COAGS: BASED ON MEDICATION

WARFARIN (COUMADIN) ASSOCIATED
- K Centra: 4 factor Prothrombin complex concentrate (PCC)
- Profil 9: 3 factor PCC – use instead of K Centra if heparin allergy
- Vitamin K 5-1-mg IV

PRADAXA (DABIGATRAN)
- Reversal agent – Praxbind (Idarucizumab)
  - Dose = 5 grams
  - 2 vials, 2.5 grams/50 mls each
  - Draw up and administer IV push, one after the other (no more than 15 mins apart)

FACTOR XA INHIBITORS (HOT OFF THE PRESS...)
- Andexanet alfa (Andexxa)
- Only approved for Rivaroxiban and Apixaban

DIRECT ORAL ANTICOAGULANTS: AKA NOACS

Direct thrombin inhibitor
- Pradaxa (Dabigatran)

FACTOR XA INHIBITORS
- Xarelto (Rivaroxiban)
- Eliquis (Apixaban)
- Savaysa (Edoxaban)
CORRECTION OF COAGULOPATHY

USEFULNESS OF PLATELET TRANSFUSIONS ICH PATIENTS WITH HISTORY OF ANTIPLATELET USE IS UNCERTAIN (CLASS IIB, LEVEL C)

2018 SYSTEMATIC REVIEW:

• No benefit, may be harmful


CASE STUDY: ICH- WHAT NEXT

22:17 - CHANGE IN NEURO STATUS IS NOTED: SPEECH INCOMPREHENSIBLE, INCREASED DROWSINESS

22:33 - RAPID SEQUENCE INTUBATION TO PROTECT AIRWAY

23:00 – REPEAT CT

GROWN CONSIDERABLY IN SIZE OVER 1 HOUR

3.2 X 1.5 → 4.5 X 3.6 CM’S

• Hematoma volume = ~45 cc’s

BLOOD IN ALL VENTRICLES

VENTRICLES ALREADY ENLARGING

8MM MIDLINE SHIFT

VASOGENIC EDEMA
### CASE STUDY: ICH- CONSIDER SURGERY

<table>
<thead>
<tr>
<th>Location</th>
<th>Surgery urgently:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebellum</td>
<td>• Declining neuro exam&lt;br&gt;• Size &gt; 3 cm, or&lt;br&gt;• Compressive effects brainstem, or&lt;br&gt;• Hydrocephalus</td>
</tr>
<tr>
<td>Lobar</td>
<td>ICH causing mass effect/herniation in severely affected but salvageable patient and as a life-saving measure</td>
</tr>
</tbody>
</table>

Location: Surgery urgently:
- Life-threatening deterioration
- Cerebellar size > 3 cm diameter

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### CASE STUDY: ICH- CONSIDER EVD

ICP MAY BE ELEVATED

PATIENTS WITH IVH ARE AT RISK FOR HYDROCEPHALUS AND ELEVATED ICP

**EVD RECOMMENDED IN:**
- GCS < 9
- LARGE MASS EFFECT
- HYDROCEPHALUS

Consider EVD

- Suspected ↑ ICP and GCS < 9
- Goal ICP < 22 mm Hg
- Goal CPP > 60 mm Hg
OTHER CONSIDERATIONS: SEIZURES

CLINICAL SEIZURES SHOULD BE TREATED WITH AED’S

DEPRESSED MENTAL STATUS OUT OF PROPORTION TO DEGREE BRAIN INJURY IS AN INDICATION FOR EEG MONITORING

ELECTROGRAPHIC SEIZURES ON EEG SHOULD BE TREATED WITH AED’S

PROPHYLACTIC ANTICONVULSANT MEDICATION SHOULD NOT BE USED (CLASS III)

SEIZURE TREATMENT: ANTICONVULSANTS

LEVITERACITAM (KEPPRA)
FOSPHENYTOIN (CEREBYX)
PHENYTOIN (DILANTIN)

Ativan is usually given IV for emergency treatment – followed by loading dose of any of the above AED’s
OTHER CONSIDERATIONS: NAUSEA

ZOFRAN

PHENERGAN
• Not a preferred agent
• Causes drowsiness or confusion – compromises neuro exam

OTHER CONSIDERATIONS: CEREBRAL EDEMA

• MANNITOL – OSMOTIC DIURETIC
  – Typically, Mannitol 20% IV 0.25g/kg to 1g/kg over 2-10 minutes
  • Calculation:
    • # grams ordered X pt’s wt in kg’s = desired dose
    • Desired dose X 100 = #cc’s to give
    20
  – Increases intravascular osmotic pressure by drawing water from the extracellular space, thus decreasing brain mass

• Hypertonic Saline – Osmotic Diuretic
  – Given as small bolus or continuous infusion
  – Watch sodium levels!
**TREATMENT**

INCLUDES MOSTLY MEDICAL MANAGEMENT/SUPPORTIVE CARE

NEURO-ICU

SURGERY INDICATED ONLY FOR PLACEMENT OF EVD, CEREBELLAR HEMORRHAGE OR RELIEF OF ICP WITH HEMICRANIECTOMY

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**RESEARCH**

**MISTIE III**

- Minimally invasive surgery plus Rt-PA for ICH Evacuation Phase III
- Missed primary endpoint- but there were encouraging results
- For those that had their hematoma reduced to a volume of 15mL or less, there was a 10.5% difference in the likelihood of achieving a good functional outcome.

**ENRICH**

- Early Minimally-invasive Removal of IntraCerebral Hemorrhage
- No preliminary data, however, they have started excluding anterior basal ganglia hemorrhages
ANEURYSMAL SUBARACHNOID HEMORRHAGE

BLEEDING INTO THE SPACE BETWEEN THE ARACHNOID MEMBRANE AND THE PIA MATER

ACCOUNTS FOR 6%-8% OF ALL STROKES

ASAH

SAH: MORBIDITY AND MORTALITY

LOW INCIDENCE: 10/100,000 PEOPLE PER YEAR

- 11% DIE BEFORE REACHING MEDICAL ATTENTION
- 40% DIE WITHIN 4 WEEKS AFTER ADMISSION TO HOSPITAL
- 30% OF SURVIVORS HAVE SIGNIFICANT MORBIDITY AND ARE DEPENDENT FOR ADLS
- NEARLY 50% OF SURVIVORS DEVELOP COGNITIVE DYSFUNCTIONS

PRESENTATION: SAH

- SUDDEN, SEVERE, GENERALIZED HEADACHE
  - “Thunder-clap headache”
  - “The worst headache of my life” 97%

- 30-60%- SENTINEL HEMORRHAGE OR WARNING HEADACHES IN THE WEEKS BEFORE SAH

- TRANSIENT LOSS OF CONSCIOUSNESS

- NAUSEA/VOMITING, BLURRED VISION

- PHOTOPHOBIA

- SEIZURES

PRESENTATION: SAH

CLASSIC | NOT-SO-CLASSIC
---|---
Abrupt onset of severe headache (HA), i.e. thunderclap | HA is not reported as abrupt (patient may not remember event well)
NEW, QUALITATIVELY DIFFERENT HA | HA responds well to non-narcotic analgesics
May have nausea, vomiting and neck pain | HA resolves on its own in few hours
May transiently lose consciousness, present in coma, or have focal deficits | 40% patients with aneurysmal SAH will have normal neuro exam without meningismus
Nature of HA onset distinguishes from other forms of stroke | Do not necessarily appear acutely ill
ED MANAGEMENT: SAH THE GOLDEN HOUR

- ABC’S/STABILIZE
- DIAGNOSE
- CLASSIFY
- CORRECT COAGULAPATHY
- MANAGE BP
- COMMUNICATE WITH TEAM
- GET PATIENT TO THE RIGHT PLACE
SAH CHECKLIST

Checklist

- Brain Imaging
- Labs: PT/PT, CBC, electrolytes, BUN, Cr, troponin, toxicology screen
- 12 lead ECG
- Blood pressure goal established
- Consult neurosurgery
- Address hydrocephalus

ED MANAGEMENT: THE GOLDEN HOUR

INITIAL FOCUS ON ARRIVAL:
- Life support (A-B-C)
- Make sure patient is safe to go to CT
  - Can they protect their airway?
  - Hypoxia?
  - Avoid hyperventilation
**CLINICAL SEVERITY OF SAH**

### Hunt & Hess Clinical Grading Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Asymptomatic, mild headache, slight nuchal rigidity</td>
</tr>
<tr>
<td>2</td>
<td>Moderate to severe headache, nuchal rigidity, no neurologic deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>3</td>
<td>Drowsiness / confusion, mild focal neurologic deficit</td>
</tr>
<tr>
<td>4</td>
<td>Stupor, moderate-severe hemiparesis</td>
</tr>
<tr>
<td>5</td>
<td>Coma, decerebrate posturing</td>
</tr>
</tbody>
</table>

### World Federation Neurological Scale

<table>
<thead>
<tr>
<th>Grade</th>
<th>Criteria</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>GCS 15</td>
</tr>
<tr>
<td>2</td>
<td>GCS 13-14, without neurological deficit</td>
</tr>
<tr>
<td>3</td>
<td>GCS 13-14, with neurological deficit</td>
</tr>
<tr>
<td>4</td>
<td>GCS 7-12</td>
</tr>
<tr>
<td>5</td>
<td>GCS 3-6</td>
</tr>
</tbody>
</table>

**DIAGNOSIS OF SAH**

- **HISTORY AND NEURO EXAM RESULTS**
- **CT SCAN W/O CONTRAST**
  - Within 48 hrs blood appears white
  - Will detect in 95% or more of cases
- **NEGATIVE CT → LUMBAR PUNCTURE USED IN SELECTED CASES (CONTRAINDICATED IF INCREASED ICP IS SUSPECTED)**
  - Need cell count on all tubes sent to lab
- **CEREBRAL ANGIOGRAPHY – GOLD STANDARD**
  - Prepare for trip to OR

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ED MANAGEMENT: THE GOLDEN HOUR- IMAGING

- Non-contrast CT imaging of the brain is the gold-standard for identifying SAH with sensitivity of 95-100% if:
  - Classic presentation with thunderclap HA
  - CT completed within six hours of HA onset
  - The patient is completely neurologically intact
  - The CT is read by an attending radiologist
- Sensitivity of CT decreases with time
- Falsely negative CT: time, anemia (HCT <30) low volume SAH, and a technically poor scan

ED MANAGEMENT: THE GOLDEN HOUR- LUMBAR PUNCTURE

- Must perform LP if CT is negative and history suggests SAH
- Rationale for LP is to confirm xanthochromia- staining of CSF by heme breakdown products
- Presence of xanthochromia is time dependent- takes several hours to develop
### SAH- GOLDEN HOUR- LUMBAR PUNCTURE

<table>
<thead>
<tr>
<th>Typical LP Findings</th>
<th>Atypical or Inconclusive</th>
<th>Not suggestive of SAH</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ RBCs, No clearing from tube 1→4</td>
<td>Clearing of RBCs from tube 1→4</td>
<td>CSF clear of RBCs</td>
</tr>
<tr>
<td>&lt; 5 WBC, WBC:RBC ratio 1:700</td>
<td>↑ WBC:RBC ratio suggest another process, meningitis or encephalitis</td>
<td>Occasionally, rapidly expanding unruptured aneurysm may present with HA, recommend urgent consultation</td>
</tr>
<tr>
<td>Xanthochromia present (However if CSF Protein &gt;100mg/dL may be false positive)</td>
<td>Xanthochromia absent (Assuming LP is done more than 12 hours following headache onset).</td>
<td>Xanthochromia absent</td>
</tr>
<tr>
<td>Opening pressure elevated (~2/3 patients)</td>
<td>OP normal</td>
<td>OP normal</td>
</tr>
</tbody>
</table>

### SAH: INITIAL MANAGEMENT

Once SAH is diagnosed, take these first steps:
- Bed rest
- Obtain pre-intervention labs: CBC, Platelets, PT/PTT, INR, electrolytes, BUN, Cr, cardiac enzymes
- 12-lead ECG
- Cardiac telemetry
- Nimodipine 60 mg po/ng (watch for hypotension)
- AED until aneurysm secured
- Consult Neurosurgery
BP MANAGEMENT: ACUTE SAH

BETWEEN SYMPTOM ONSET AND ANEURYSM OBLITERATION BP SHOULD BE CONTROLLED WITH A TITRATABLE AGENT TO BALANCE RISK OF STROKE, HTN RELATED REBLEEDING AND MAINTENANCE OF CPP (CLASS I; LEVEL B).

MAGNITUDE OF BP CONTROL NOT ESTABLISHED, BUT DECREASE IN SBP TO <160 MMHG IS REASONABLE (CLASS LLA; LEVEL C).

- Precise guidelines for BP management in SAH unfortunately do not exist
- Retrospective data suggest higher rates of re-bleeding with SBP > 160 mmHg
- Over treatment of BP can potentially lead to brain ischemia - especially if hydrocephalus or vasospasm is present.
- Pre-morbid BP should be taken into considerations
- **Experts recommend to aim for SBP < 160 mmHg, or MAP < 110 mmHg**, keeping principles above in mind
- Use short acting, titratable intravenous medications such as beta blockers or nicardipine.
- Avoid long-term nitroprusside due to concern of raising ICP

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EARLY COMPLICATIONS: HYDROCEPHALUS

COMMUNICATING

- Problem with absorption of CSF; blood in CSF plugs the arachnoid villi

DIAGNOSED BY CT – DILATED VENTRICLES

SEVERITY RELATED TO SIZE OF BLEED

- Arachnoid villi unable to reabsorb CSF, laden with byproducts of blood breakdown

MAY REQUIRE EMERGENT INSERTION OF EXTRA-VENTRICULAR DRAIN

ASTUTE NEUROLOGICAL ASSESSMENTS

- WILL BECOME SLEEPIER...SLOWER TO RESPOND
- OVERALL DECREASE IN LOC
EARLY COMPLICATIONS: HYDROCEPHALUS

The following CT scan shows hydrocephalus. Note the enlargement of the ventricles with CSF, as denoted in black.


OTHER CONSIDERATIONS: SEIZURES

- **DIFFERENCE FOR ASAH:**
- **ROUTINE USE OF PHENYTOIN NOT RECOMMENDED** (LOW QUALITY EVIDENCE; STRONG RECOMMENDATION)
- **ROUTINE USE OF OTHER ANTICONVULSANTS FOR PROPHYLAXIS MAY BE CONSIDERED** (VERY LOW QUALITY EVIDENCE; WEAK RECOMMENDATION)
- **IF ANTICONVULSANT PROPHYLAXIS USED, A SHORT COURSE IS RECOMMENDED** (3-7-DAYS) (LOW QUALITY EVIDENCE; WEAK RECOMMENDATION)

ANEURYSM TREATMENT

- Neuro ICU care
- Stay generally around 21 days depending on complications
- Specific management to treat complications (vasospasm, hydrocephalus, electrolyte imbalances

http://surgicalunits.com/aneurysm-clip-311.html

STROKE OUTPATIENT MEASURES

STK-OP-1 DOOR TO TRANSFER TO ANOTHER HOSPITAL

- Hemorrhagic stroke
  - Will need to track door in door out times when transferring all hemorrhagic stroke patients
  - No benchmark at this time

- Transfer consideration
  - Hospice- keep the patient locally if able
PACKAGING FOR TRANSFER

ADEQUATE AIRWAY PROTECTION?
• If not intubated – is this patient going to be able to manage their airway for transport?

BLOOD PRESSURE MANAGEMENT
• Is the blood pressure within recommended guideline or per MD recommendations from receiving center?

DISTANCE (CRITICAL CARE TRANSPORT)
• Air vs ground transport
• ACLS with paramedic

IMAGING RESULTS
• Do you have some kind of cloud service or sharing capability with receiving center?
• If not, will need a disc to go with patient

ANY NECESSARY CHART COPIES

PACKAGING FOR TRANSFER

COMMUNICATION
• Obtain cell phone number for family
  • Provide them with information about transfer facility if available
• Obtain contact number to call report to receiving facility ICU
  • History, any treatments done at your ED/facility
    • BP meds, seizure meds, nausea meds, etc...
    • Imaging done
  • Last neuro exam at your facility (be specific – terms like obtunded, stuporous, or unresponsive are not helpful)
QUESTIONS

CONTACT INFORMATION

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