Topics
- Increased intracranial pressure (ICP)
- Head trauma
- Ischemic stroke
- Hemorrhagic stroke
- Neurologic infectious diseases
- Seizures and epilepsy
- Status epilepticus
- Brain tumors
- Hypoxic encephalopathy
- Neuromotor disorders

Increased Intracranial Pressure

Classification
- Increases in tissue volume
- Increases in blood volume
- Increases in cerebrospinal fluid (CSF) volume
Pathophysiology

- Monro-Kellie doctrine
- Compliance

![Diagram showing compliance levels and intracranial pressure](image)

Increase in contents of intracranial vault

Signs/Symptoms of Increased Intracranial Pressure

- Decreased level of conscience (LOC)
- Motor
  - Contralateral motor deficit
- Cranial nerves
  - Ipsilateral pupil abnormalities
- Headache and/or vomiting
- Cushing's triad
  - Bradycardia
  - Widening pulse pressure
  - Respiratory arrest

Intracranial Pressure Monitoring

- Indications for monitoring ICP
  - Glasgow Coma Scale (GCS) 3-8
  - CT reveals abnormality
- Types of monitoring devices
  - Intraparenchymal bolt
  - Ventriculostomy
- ICP values
  - Normal: 0–15 mmHg
  - Elevated: >20 mmHg
Cerebral blood flow (CBF) = cerebral perfusion pressure (CPP) + cerebrovascular resistance (CVR)

CPP = mean arterial pressure (MAP) - ICP

Injured brain: optimize CPP
Use target of 60-80 mmHg

Physiology: Autoregulation

- At mean arterial blood pressure (MABP) <60 mmHg, cerebral ischemia develops
- At MABP of >140 mmHg, cerebral vascular congestion can occur

Autoregulation
- Vasomotor control
  - Intact: Increase in CPP causes vasoconstriction and decrease in ICP
  - Vasomotor reactivity failure: Increase in CPP causes vasodilation and increased ICP
- Flow metabolism
  - ↑ metabolism ↑ CBF
- Metabolic substances
  - PaO₂
  - PaCO₂
  - pH (i.e., acidosis = vasodilatation)
Interventions

- Head of bed (HOB)/neck positioning
- Quiet environment
- Monitor ICP and drain CSF if ICP >20 mmHg
- Airway and breathing
  - PaO₂ and PaCO₂
  - Target PaO₂ > 80 mmHg and PaCO₂ 35-40 mmHg
  - Decrease PaCO₂ 30–35 mmHg if impending herniation
- Circulation
  - Maintain CPP >60–80 mmHg
  - Maintain euvolemia/vasopressors
  - Sedation and analgesia as indicated for control of ICP
  - Normothermia

Secondary Interventions

- Mannitol
  - 0.25–1 g/kg
  - Replace fluids lost to maintain euvolemia
- Hypertonic saline
  - 3% 200 mL over 20 minutes
  - 5% 150 mL over 20 minutes
  - 7% 100 mL over 20 minutes
  - 23.5% 15–20 mL

Tertiary Interventions

- Decompressive hemicraniectomy
  - Large
  - Early
- Temperature control
  - Hypothermia for refractory increased ICP
  - Barbiturate coma
Uncontrolled Increased Intracranial Pressure: Progression to Brain Death

- Presidential commission established the Uniform Determination of Death Act to determine brain death
  - An individual who has sustained either:
    - Irreversible cessations of circulatory and respiratory functions
    - Irreversible cessation of all functions of the entire brain

Determining brain death — 4 steps

- Establish irreversible and proximate cause of coma
  - History, examination, neuroimaging, and laboratory
  - Achieve a normal or near-normal core temperature
  - Greater than or close to 36°C
- Achieve a normal systolic BP > 100 mmHg
- Perform one neurologic exam (acceptable in most states)
  - Some states require more than one exam and some may specify a certain level of expertise (i.e., neurologist/neurosurgeon)

Perform one neurologic exam (cont)

- Coma: Lack all responsiveness (no eye opening, no eye movement, no motor response to stimuli)
- Absence of brainstem reflexes (fixed pupils, absent doll's eyes/cold caloric, absent corneal/swallow/gag, no facial)
- Apnea: Absence of spontaneous breathing (allow PaCO₂ > 60)
  - 8-10 min support with 100% FiO₂ via O₂ to ET
- Ancillary tests: EEG, cerebral angi, nuclear scan blood flow
  - Not used to confirm brain death and cannot replace the clinical exam

Prerequisites

I. PREREQUISITES

A. Clinical or neuroimaging evidence of acute CNS catastrophe that is compatible with irreversible loss of brain function.

B. Absence of complicating medical conditions:
   1. Absence of severe electrolyte, acid base or endocrine derangement or severe hyperthermia.
   2. Absence of drug intoxication, poisoning, sedatives or neuromuscular-blocking agents.
   3. Core temperature 98.9°F / 36°C or greater.

II. FIRST EXAM

A. Yes ☐ No ☐

B. Absence of apneic episodes:
   1. Prepared for intubation or tracheostomy.
   2. Poor Glasgow Coma Score.
   3. Absent cough or bronchial excitation.
   4. Absent spontaneous respiration.

Apnea Test

IV. APNEA

A. Prerequisites:
   1. Core temperature 98.9°F / 36°C or greater.
   2. Systolic BP > 100 mm Hg (with or without vasoactive agents).
   3. Arterial O2 > 100 mm Hg (known non-O2 abuser).
   4. Arterial CO2 greater than 50 or 60 mm Hg.

B. Apnea testing checklist:
   1. Preoxygenate to a PaO2 >200 mm Hg and then administer 100% O2CE during the entire test period.
   2. Disconnect the ventilator monitor with pulse oximeter throughout the test period.
   3. Deliver 100% O2 into the nares via a cannula at the level of the carina, maintaining oxygen saturation above 98%.

Note: NCS 2015 Brain Death Toolkit https://www.pathlms.com/ncs-ondemand
Apnea Test

4. Check arterial blood gases at 10 minutes and reposition the patient when either a) PaCO₂ ≤ 80 mmHg or b) PaCO₂ is greater than 30 mmHg above the patient's known baseline (within O2 saturation)

5. Abort the apnea test and immediately reposition the ventilator for any of the following reasons:
   a. Systolic BP falls below 60 mmHg or there is cardiovascular collapse
   b. Oxygen desaturation ≥ 8% for ≥ 30 seconds
   c. Significant cardiac arrhythmia
   d. Respiratory movements are observed

RESULTS of APNEA TESTING
1. APNEA CONFIRMED
   Yes ☑ No ☐
   OR
2. APNEA TESTING CONTRAINDED
   Yes ☑
   OR
3. APNEA TEST ABORTED
   Yes ☑

Uncontrolled Increased Intracranial Pressure: Progression to Brain Death

- Determining brain death
  - Time of brain death is:
    - Time the PaCO₂ reaches the target level and absent spontaneous breathing
  - If unable to do apnea, time ancillary test officially interpreted


Uncontrolled Increased ICP: Progression to Brain Death

1. IV MUST BE INSERTED TO CONFIRM DEATH BY NEUROLOGICAL CRITERIA WITHOUT THE NEED FOR ANCILLARY TESTING

V. ANCILLARY TESTING IS REQUIRED WHEN ITEMS I AND II ARE NOT MET BUT EITHER ITEM III (BRAINSTEM REFLEX TESTING) OR ITEM IV (APNEA TESTING) CANNOT BE COMPLETED OR CONVINCingly INTERPRETED

ANCILLARY Study Performed:

- Conventional Cisternal-based Cerebral Angiography
- Nuclear Medicine Cerebral Blood Flow Study (Technetium HMP AECI)
- Transcranial Doppler
- Electroencephalography
- Demonstrated Absence of Cerebral Blood Flow or Cerebral Electrical Activity

Question 1
A brain injured patient was admitted 4 hours ago after sustaining a large subdural hematoma (SDH) which was evacuated surgically. An ICP monitor with CSF drainage was placed by the MD. The ICP increases to 24 mmHg while the patient’s CPP is 64 mmHg. After draining CSF, the first intervention would be to lower the ICP by:

A. Lowering the HOB
B. Administering hypertonic saline or mannitol
C. Inducing a pentobarbital coma
D. Lowering the PaCO2 to 28 mmHg

Question 1—Rationale
A brain injured patient was admitted 4 hours ago after sustaining a large subdural hematoma (SDH) which was evacuated surgically. An ICP monitor with CSF drainage was placed by the MD. The ICP increases to 24 mmHg while the patient’s CPP is 64 mmHg. After draining CSF, the first intervention would be to lower the ICP by:

B. Administering hypertonic saline or mannitol
   - Lowering the HOB—Lowering the HOB would increase the ICP
   - Inducing a pentobarbital coma—Instituting a pentobarbital coma would be a tertiary intervention
   - Lower the PaCO2 to 28 mmHg—Since lowering the PaCO2 to 28 mmHg would decrease CBF and create ischemia, especially on day 1 of a traumatic brain injury (TBI) when cerebral blood flow is often critically reduced, lowering PaCO2 <30 mmHg should be avoided
Question 2

It is day 4 of FZ's hospitalization following a severe TBI. He has experienced an increase in urine output (1000 mL) over the past 3 hours. His MAP has decreased to 75 mmHg and his ICP has increased to 30 mmHg, resulting in a CPP of 45 mmHg. If FZ has intact autoregulation, the most appropriate intervention would be to:

A. Administer 500 mL fluid bolus to increase MAP and decrease ICP
B. Administer 50 g mannitol to reduce ICP
C. Begin nicardipine to lower MAP and ICP
D. Do nothing since MAP/ICP/CPP are within normal ranges

Question 2—Rationale

It is day 4 of FZ's hospitalization following a severe TBI. He has experienced an increase in urine output (1000 mL) over the past 3 hours. His MAP has decreased to 75 mmHg and his ICP has increased to 30 mmHg, resulting in a CPP of 45 mmHg. If FZ has intact autoregulation, the most appropriate intervention would be to:

A. Administer 500 mL fluid bolus to increase MAP and decrease ICP
   - Administer 50 g mannitol to reduce the ICP—Administering 50 g mannitol to reduce the ICP may significantly lower the BP, resulting in a further increase in ICP
   - Begin nicardipine to lower the MAP and ICP—Beginning nicardipine to lower the MAP and ICP would actually increase the ICP
   - Do nothing, since MAP/ICP/CPP are within normal ranges—ICP is elevated (>20) and the CPP is not in the ideal range of 50–70 mmHg
Etiology of Brain Injury

- Mechanisms of injury
  - Trauma
  - Blunt
  - Penetrating
  - Blast

Pathophysiology of Brain Injury

- Primary injury
  - Trauma
    - Skull integrity
    - Brain integrity
      - Focal injuries
      - Diffuse injuries

Classification

- Secondary injury r/t event
  - Cerebral edema
  - Changes in CBF
  - Cellular

- Secondary injury
  - Hypotension
  - Hypoxia
  - Hypocapnia
  - Anemia
  - Fever
Primary Injuries

- Cerebral injuries
  - Diffuse
  - Focal

Diffuse Injury: Concussion

- Stretch injury of axons causing temporary disturbance in neurologic function
- Focus on prevention and recognition
- Interventions
  - Monitor for neurologic deterioration
  - Cognitive and physical rest
  - No TV, phone, iPod, computer
  - Return to school, work and play
  - Educate on postconcussive syndrome (PCS)

Diffuse Axonal Injury

- Shearing
  - Severe disruption of axons
  - CT—small punctate bleeds
- Degrees of coma
  - Varies
- Control of ICP
- Prolonged recovery
- Prognosis varies
Neurologic Epidural Hematoma

- Associated with linear skull fracture
  - Children may not have skull fracture with epidural
  - Rare in older adults
- Location: 75% temporal region
- CT—biconvex
- Exam
- Treatment
  - Removal of hematoma
  - Post-op monitoring of neurologic status and determining any cognitive deficits

Epidural Hematoma

- Associated with high-velocity deceleration
  - Timing and cause
    - Acute within 24–48 hours
    - Rupture of bridging cortical veins
    - Increased ICP/contusions
    - Subacute 48 hours to 10 days
    - Chronic: 10 days to 6 weeks
    - Rupture of bridging veins across parasagittal space
- CT
- Clinical presentation

Subdural Hematoma
**Acute Subdural Hematoma**

- Usually bilateral isodensity
- 2–6 weeks after event

**Subacute Subdural Hematoma**

- Usually unilateral isodensity
- 2–10 days after event

**Chronic Subdural**

- Usually bilateral isodensity
- 2–6 weeks after event

**Management of Subdural Hematoma**

- Acute
  - Acute surgical decompression
  - Synchronous with increased ICP management
- Subacute and chronic
  - Burr holes for removal of fluid
  - Flat for 1–2 days
  - Gradual elevation of HOB
  - Evaluation for deficits
Cerebral Contusions

- Types
  - Fracture
  - Coup
  - Contrecoup
  - Herniation
  - Surface
  - Gliding (focal hemorrhage in cortex/subjacent white matter found in diffuse axonal injuries)
- Frequently frontal or temporal regions
- Vasogenic edema and central necrosis
- Diagnosis: CT and exam

Focal Injury: Cerebral Contusions

- Care priorities
  - Monitor neurologic status
  - Manage ICP if monitor placed
  - Manage life-support systems
  - Assessment of deficits and consultation with PT/OT/ST
  - Education of patient/family

Head Injury Assessment

- History of event
- Severity of injury based on GCS
  - Mild 13-15
  - Moderate 9-12
  - Severe 3-8
- Signs of increased ICP and evidence of injury
- Diagnostics
Neurologic

Airway
- Oxygenation: airway and oxygen
  - Ventilation:
    - Initial: PaCO₂ 35–45 mmHg

BP and volume
- CPP 60–80 mmHg
- Fluids/vasopressors
- Sedation and analgesia
- Mannitol or hypertonic saline
- Positioning
  - HOB 30° with neck midline

Interventions

Brain Trauma Foundation. J Neurotrauma. 2007;24(Supple 1).

Interventions

- Draining CSF for ICP >20 mmHg
- Temperature regulation—normothermia at 37°C
- Refractory increased ICP
  - Decompressive hemicraniectomy
  - Mild hypothermia
- Nutrition
- System support
- Rehab

Review Questions
Question 3

JR, a 34-year-old male, sustained a severe TBI falling from a ladder. His GCS on arrival was 1-3-1 with pupils 2 and minimally reactive to light. CT scan of the brain shows multiple punctate hemorrhages, along with an acute EDH. The neurosurgeon evacuated the bleed and placed an ICP/brain oxygen monitor. When JR is admitted to ICU: GCS 1-4-1; MAP 80; ICP 10; CPP 70; PbtO2 12 (normal 20–40 mmHg); PaCO2 30; CVP 10. What is your first intervention?

A. Give 250 mL NS to increase CVP
B. Administer mannitol 25 g IVP
C. Decrease the tidal volume or rate to increase the PaCO2
D. Do nothing, since the patient’s parameters are within the normal zone

Question 3—Rationale

C. Decrease the tidal volume or rate to increase the PaCO2
- Give 250 mL NS to increase CVP—Incorrect, since the CVP and CPP are normal
- Administer mannitol 25 g IVP—Incorrect, since the ICP is normal
- Do nothing, since the patient’s parameters are within the normal zone—Incorrect, because the oxygen level in the brain is critically low

Question 4

EG, an 80-year-old male, was previously independent and able to care for himself. His daughter stated he is now unable to walk and is confused/disoriented. A CT scan of the brain reveals bilateral large chronic SDH. The neurosurgeon evacuated both SDH and has placed a Jackson-Pratt drain to gravity. The patient was extubated post-op. Where should the HOB be maintained?

A. HOB flat for 24–48 hours
B. HOB at 15° for 10 days
C. HOB at 30° to reduce ICP
D. HOB at 45° to reduce ICP
Question 4—Rationale

EG, an 80-year-old male, was previously independent and able to care for himself. His daughter stated he is now unable to walk and is confused/disoriented. A CT scan of the brain reveals bilateral large chronic SDH. The neurosurgeon evacuated both SDH and has placed a Jackson-Pratt drain to gravity. The patient was extubated post-op. Where should the HOB be maintained?

A. HOB flat for 24–48 hours
   - HOB at 15° for 10 days—B is incorrect due to the position and length of time
   - HOB at 30° to reduce ICP; HOB at 45° to reduce ICP—C and D are both incorrect, since ICP is usually normal and the brain needs to reexpand into the space occupied by the blood clot. Typically, elderly individuals undergo significant brain atrophy, and thus have more space in the cranial vault.

Ischemic Stroke

- Risk factors
- Signs/symptoms
  - FAST
    - Face, arm, speech, time
  - Motor weakness
  - Asymmetrical smile
  - Difficulty speaking
  - Numbness
  - Visual changes
  - Difficulty swallowing
  - Use NIH Stroke Scale/Score (NIHSS) to assess
    - Full
    - Abbreviated
Ischemic Stroke

- **Etiology**
  - Thrombotic 20%–25%
  - Embolic 20%
  - Lacunar 25%
  - Cryptogenic 30%

- **Pathophysiology**
  - Reduced blood flow
  - Importance of collateral flow
  - BP management
  - Impact of glucose and temperature

Pathophysiology of Ischemic Stroke

- Three factors affecting outcome
  - Time-dependent
  - Degree of ischemia
  - Collateral circulation

Pathophysiologic Issues Related to Stroke

- Edema and increased ICP
  - Occurs as natural evolution of insult
  - Minimized if perfusion restored
  - Assess for change in neurologic status
  - Do not medicate with sedation agents unless monitoring for increased ICP
  - Prepare for CT
Ischemic Stroke

- Intervention
  - ABC
  - Supplemental O₂ for O₂ Sat <94%
  - BP management
    - No tissue plasminogen activator (tPA): SBP >220 mmHg, DBP >120 mmHg, or MAP >130 mmHg
    - tPA: SBP >185 mmHg or DBP >110 mmHg
    - Only use labetalol or nicardipine
  - Monitoring
    - Neuro exam every 15 minutes x 4 until treatment decision made
    - IV and labs

Diagnoses

- CT scan
  - No hemorrhage
  - No edema
  - 12-lead: NSR

Ischemic Stroke

- Time window: Candidates for tPA
  - Intravenous tPA

Time window was <3 hours

It has now moved to 4½ hours per American Stroke Association (not FDA-approved)
Scientific Advisory: rtPA

- rtPA should be administered to eligible patients who can be treated within 3–4.5 hours after stroke

Intravenous tPA: Indications

- Patient symptoms <4.5 hours from symptom onset
  - CT scan excludes hemorrhage
  - NIH stroke scale ≥4
  - Isolated aphasia
  - Age >18
- Note exclusions for 3–4.5 hour IV tPA
  - Age >80 years
  - Taking oral anticoagulants
  - NIHSS >25
  - Combination of history of prior stroke + diabetes

General Exclusions for tPA 2015

- Current intracranial hemorrhage
- Subarachnoid hemorrhage
- Active internal bleeding
- Recent (within 3 months) intracranial or intraspinal surgery or serious head trauma
- Presence of intracranial conditions that may increase the risk of bleeding (ie, AVM, aneurysm, certain neoplasms)
- Bleeding diathesis
  - (Platelet count <100,000; currently using anticoagulant with an INR <1.7 or PT >15 seconds). Current severe uncontrolled hypertension (SBP >185 or DBP >110), current use of novel anticoagulants (within 48 hours)
Ischemic Stroke

- Decision point
  - Thrombolytic and embolectomy procedures <6 hours
  - IV tPA 3–4.5 hours from symptom onset
    - Achieve door-to-needle time in under 60 minutes
    - Joint Commission requirement to achieve 60-minute window in >50% of tPA administered
  - IA <6 hours (off label)
  - Thrombectomy devices
    - Four studies: EXTEND IA, ESCAPE, MR. CLEAN, and SWIFT PRIME 2015 results
      - Improved outcomes when patients receive IV tPA and embolectomy
      - Time to groin <120 minutes
    - Supportive care—no tPA/embolectomy procedure
      - Aspirin 325 mg rectally within 48 hours

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tPA Infusion Guidelines

- Preparation of IV tPA drip
  - 0.9 mg/kg
  - 10% IV bolus over 1–2 minutes
  - 90% IV over 60 minutes
- Administration of tPA
  - Monitor VS: Q 15 min x 2 hrs, Q 30 min x 6 hrs, then Q 1 hour x 16 hours
  - Treat BP accordingly

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Ischemic Stroke

- tPA
  - BP considerations: treatment
    - SBP >185/DBP >110
  - Administration: IV
    - Postinfusion care: Tx SBP >180/DBP >105
- Supportive care
  - Do not drop BP unless:
    - Systolic BP >220/diastolic BP >120
    - Treat with labetalol or nicardipine

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Neurology 22
Ischemic Stroke

- V5/neuro checks 1–2 hours
- NIHSS
- Support airway/O₂/pulse ox
- Cardiac telemetry
- HOB flat vs 30°
  - Flat if no signs of increased ICP—supports improved flow
  - HOB 30° if signs of increased ICP
- NPO until swallow assessment

Ischemic Stroke

- Temperature management
  - Treat temperature >98.6°F
  - Hydrate/control serum glucose <180
- Observe neurologic status
- Medications: statins and antiplatelet agents
  - Begin nutrition
  - Early mobility
- Prevent complications
  - Aspiration: NPO until swallow assessment
  - DVT
  - UTI
- Educate family
- Secondary stroke prevention

Review Questions
Question 5

LL, a 65-year-old female, developed acute onset of left sided weakness with neglect to the left side at 12:35 PM. Her speech became slurred and she didn’t know where she was. LL’s son brought her to the ED, where a CT scan of the brain revealed no hemorrhage. A CTA demonstrated an occlusion of the right middle cerebral artery. The ED physician/neurologist has ordered IV tPA. It is 2 hours, 50 minutes since symptom onset. The patient’s last BP was 190/100 mmHg. Your first action is to:

A. Administer tPA 0.9 mg/kg IV dose because the treatment window is only 10 minutes
B. Administer tPA 0.6 mg/kg IV dose and labetalol 10 mg IVP at the same time
C. Start IV nitroprusside to reduce BP
D. Administer 10–20 mg IV labetalol to reduce BP prior to administering tPA

Question 5—Rationale

LL, a 65-year-old female, developed acute onset of left-sided weakness with neglect to the left side at 12:35 PM. Her speech became slurred and she didn’t know where she was. LL’s son brought her to the ED, where a CT scan of the brain revealed no hemorrhage. A CTA demonstrated an occlusion of the right middle cerebral artery. The ED physician/neurologist has ordered IV tPA. It is 2 hours, 50 minutes since symptom onset. The patient’s last BP was 190/100 mmHg. Your first action is to:

D. Administer 10–20 mg IV labetalol to reduce BP prior to administering tPA
  • Administer tPA 0.9 mg/kg IV dose because the treatment window is only 10 minutes—A is incorrect because controlling the BP must be done prior to administering tPA
  • Administer tPA 0.6 mg/kg IV dose and labetalol 10 mg IVP at the same time—B is incorrect because the dose of tPA is incorrect and BP must be lowered prior to administration of tPA
  • Start IV nitroprusside to reduce the BP—C is incorrect. Labetalol and nicardipine are the first two drugs of choice when lowering BP in acute ischemic stroke

Question 6

BP, a 90-year-old female, presents with acute onset of aphasia and right-sided hemiparesis. She was last seen normal 12 hours ago. Her BP is 195/105. Which medication do you anticipate the physician team will order first?

A. Labetalol 10 mg IV to lower BP
B. tPA 0.9 mg IV (10% bolus/90% drip over 60 minutes)
C. Aspirin 325 mg PO
D. Aspirin 325 mg per rectum
Question 6—Rationale

BP, a 90-year-old female, presents with acute onset of aphasia and right-sided hemiparesis. She was last seen normal 12 hours ago. Her BP is 195/105. Which medication do you anticipate the physician team will order first?

D. Aspirin 325 mg per rectum
   • Labetalol 10 mg IV to lower BP—A is incorrect; BP threshold in the non-tPA patient is 220/120
   • tPA 0.9 mg IV (10% bolus/90% drip over 60 minutes)—B is incorrect because the symptoms are >3 hours
   • Aspirin 325 mg po—C is incorrect because the patient may have difficulty swallowing and needs to have her swallow evaluation prior to receiving any po medications

Hemorrhagic Stroke

Intracerebral Hemorrhage

- Usually a result of hypertension
- Releases toxins that lead to vasospastic activity
- Local decrease in perfusion
- Global decrease in perfusion
- Cellular changes
Subarachnoid Hemorrhage: Aneurysm

- Aneurysms
- Occur at bifurcation
- Defect in artery
- Rupture point
- Bleeds into subarachnoid space (SAS), brain tissue, or ventricles
- Danger
  - Rebleeding
  - Vasospasm

Hemorrhagic Stroke

- Assessment: ICH
  - Headache, altered LOC, and N/V
  - Motor weakness and sensory changes
  - Cranial nerve deficits
  - Signs of increased ICP

Hemorrhagic Stroke

- Assessment: SAH
  - "Worst headache of my life"
  - Meningeal: photophobia and nuchal rigidity
  - Nausea/vomiting and dizziness
  - Focal deficits, ie, third nerve
Neurologic Aneurysms—assessment grading: Hunt and Hess scale

- I: Alert, no deficit, and minimal HA
- II: Awake, CN palsies, mild-to-severe HA
- III: Drowsy, confusion, mild focal deficit
- IV: Unresponsive and hemiplegia
- V: Comatose, moribund, extensor posturing

Hemorrhagic Stroke

Diagnostic work-up
- Lumbar puncture
- Presence of RBCs/WBCs
- Elevated protein
- CT scan
- Presence of SAH
- Angiogram
- Spiral CT angiogram
- MRI/MRA

Medical management of ICH
- Surgery vs medical management vs palliative care
- Ventriculostomy and ICP monitoring

Team management
- ABC
  - Airway/ventilation
  - BP control
  - Circulation
    - Normovolemia and VTE prophylaxis
  - ICP control: CSF drainage and mannitol vs hypertonic saline bolus
  - System support

System support
Hemorrhagic Stroke

- Management of SAH
  - Pre-op
    - BP control (SBP <140–150 mmHg)
    - Frequent assessments to monitor for neurologic deterioration due to hydrocephalus or rebleeding
  - Surgical clipping vs coiling

- ABC
  - Airway: PaO₂ >80 mmHg and PaCO₂ >35 mmHg
  - Watch for myocardial stunning
  - Target BP 120-150 mmHg unless vasospasm present
  - ICP control: CSF drainage
  - Vasospasm: Most likely days 4-14
    - BP management
      - Keep BP normalized until vasospasm begins
      - If vasospasm, elevate BP and Hct to target of 30
      - Nimodipine
      - Watch for change in exam and/or sodium drop and diuresis

Interventions

- Nasogastric tube
- IV fluids
- Maintain anticonvulsants
- Examine patient for injury
- Treat hyperthermia
- Begin nutrition
- Early mobility
- Psychological support
Question 7
SS, a 50-year-old female, presents with the worst headache of her life and photophobia. Her neuro exam is otherwise normal. CT scan of the brain shows a large SAH from a possible cerebral aneurysm. Her BP is 170/90. Initially, the physician will want the BP treated if:

A. Systolic BP >90 mmHg
B. Systolic BP >150 mmHg
C. Systolic BP >180 mmHg
D. Systolic BP >220 mmHg

Question 7—Rationale
SS, a 50-year-old female, presents with the worst headache of her life and photophobia. Her neuro exam is otherwise normal. CT scan of the brain shows a large SAH from a possible cerebral aneurysm. Her BP is 170/90. Initially, the physician will want the BP treated if:

B. Systolic BP >150 mmHg
   • Systolic BP >90 mmHg—SBP <90 would not be beneficial to cerebral perfusion
   • Systolic BP >180 mmHg—These ranges are too high and may contribute to rebleeding of the aneurysm
   • Systolic BP >220 mmHg—These ranges are too high and may contribute to rebleeding of the aneurysm
Question 8

BP, a 52-year-old female, was admitted to the ICU following rupture of a cerebral aneurysm and large SAH. CT scan of her brain revealed acute hydrocephalus. The most likely cause of BP’s hydrocephalus is:

A. Cerebral aneurysm exerting pressure on the choroid plexus in the ventricles, limiting the CSF absorption
B. Blood in SAS mixing with CSF and occluding the arachnoid villi, which helps reabsorb CSF in the brain
C. Overproduction of CSF from the aneurysm rupture
D. Reduced absorption of CSF by the choroid plexus

Question 8—Rationale

BP, a 52-year-old female, was admitted to the ICU following rupture of a cerebral aneurysm and large SAH. CT scan of her brain revealed acute hydrocephalus. The most likely cause of BP’s hydrocephalus is:

B. Blood in SAS mixing with CSF and occluding the arachnoid villi which helps reabsorb CSF in the brain
- Cerebral aneurysm exerting pressure on the choroid plexus in the ventricles limiting the CSF absorption—Choroid plexus makes CSF, it does not absorb CSF
- Overproduction of CSF from the aneurysm rupture—Aneurysmal rupture does not increase CSF production by the choroid plexus
- Reduced absorption of CSF by the choroid plexus—CSF is absorbed by the arachnoid villi

Question 9

SS is now 10 days post aneurysm rupture. The aneurysm was surgically clipped on day 2. SS began complaining of a headache this morning. Her motor exam has changed in the last 2 hours, with her right arm motor strength diminished from 5/5 to 3/5. She is having word-finding problems with her speech. The most likely explanation for this change in status is:

A. Increased ICP
B. Rebleeding
C. Ischemic stroke
D. Vasospasm
**Question 9—Rationale**

SS is now 10 days post aneurysm rupture. The aneurysm was surgically clipped on day 2. SS began complaining of a headache this morning. Her motor exam has changed in the last 2 hours, with her right arm motor strength diminished from 5/5 to 3/5. She is having word-finding problems with her speech. The most likely explanation for this change in status is:

D. Vasospasm
- Increased ICP—Although cerebral edema is possible after SAH, a change in LOC should occur
- Rebleeding—Since the aneurysm has been surgically repaired, this complication is unlikely
- Ischemic stroke—If vasospasm is not treated, the end result could be an ischemic stroke

**Neurologic Infectious Diseases**

- An inflammation of the meninges
- Primary causes
  - Bacterial
  - Viral
  - Fungal
- Other causes include parasites and cancer

Neurology
# Meningitis: Causative Agents

<table>
<thead>
<tr>
<th>Bacterial</th>
<th>Viral</th>
<th>Fungal</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pneumonia</em></td>
<td>Enteroviruses:</td>
<td>Cryptococcus neoformans</td>
</tr>
<tr>
<td><em>Neisseria meningitidis</em></td>
<td><em>Coxsackieviruses</em></td>
<td><em>Coccidioides immitis</em></td>
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<td><em>Neurospora fumigates</em></td>
<td><em>Echoviruses</em></td>
<td><em>Candida albicans</em></td>
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<td><em>Group B streptococcus</em></td>
<td><em>Polioviruses</em></td>
<td><em>Blastomyces dermatitidis</em></td>
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<td><em>Escherichia coli</em></td>
<td><em>Arboviruses</em> (eg, St. Louis</td>
<td><em>Histoplasma capsulatum</em></td>
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<tr>
<td><em>Streptococcus</em></td>
<td>meningitis)</td>
<td><em>Paracoccidioides brasiliensis</em></td>
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<tr>
<td><em>Treponemia pallidum</em></td>
<td>*Transmitted by arthropod</td>
<td><em>Sporothrix schenckii</em></td>
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<tr>
<td><em>Mycobacterium tuberculosis</em></td>
<td>vectors (eg, mosquitoes or</td>
<td><em>Pseudallescheria boydii</em></td>
</tr>
<tr>
<td></td>
<td>ticks)</td>
<td><em>Aspergillus fumigates</em></td>
</tr>
<tr>
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<td></td>
</tr>
</tbody>
</table>

# Meningitis Pathophysiology
- **Access routes:** open wound, mucous membrane, or infected tissue
  - Direct:
    - Penetrating wounds, skull fractures, neuro-op procedures, LP, IOP, otitis media, sinusitis, or osteomyelitis
  - Hematogenous:
    - Septicemia/bacteremia, septic emboli, bacterial endocarditis, URI, pelvic abscesses
- CSF leak: rhinorrhea/otorrhea

- **Bacterial process**
  - Colonization: invades tissue/gains access to blood
  - Crosses blood-brain barrier
  - No host immune defense in CSF
  - Rapid replication of bacteria
  - Rapid increase in neutrophil
- Lysis of bacteria
  - Produces exudate and inflammation of meninges
  - Cerebral edema, vasculitis, infections, hydrocephalus, and increased ICP
### Bacterial Meningitis: Assessment

- **Subjective data**
  - Varies depending upon the pathogen
  - Headache, neck or back pain, photophobia, and malaise

- **Objective data**
  - Clinical triad: fever, nuchal rigidity, altered mental status
  - Meningeal irritation: headache, nuchal rigidity, photophobia, Kernig's sign, and Brudzinski's sign

- **Kernig's sign**
  - Patient supine
  - Flex hip 90°
  - Straighten leg = pain in hamstring

- **Brudzinski's sign**
  - Patient supine
  - Flex neck passively = involuntary flexion of knees and hips

---

### Bacterial Meningitis: Objective Data

- **Focal neurological deficits**: cranial nerve palsies (CNII-CN VIII), diplopia, seizures, hemiparesis, and altered mental status

- **Petechial rash**
  - Occurs in 50% of meningococcal cases
  - Tiny red or purple pinprick rash that progresses to purple blotches, located on trunk, lower extremities, mucous membranes, and conjunctiva
  - Poor outcome with rapidly evolving rash; requires emergent care
  - Tumbler test
  - Similar rashes observed in pneumococcal, H influenzae, and enteroviral meningitis

- **Nausea; vomiting; chills; malaise; pain in the back, abdomen, and extremities**

---

### Bacterial Meningitis: Diagnostics

- **Medical history and clinical exam**

- **Laboratory studies**
  - Chemistry and coagulation profile, cultures, CSF studies, serology

- **CT/MRI of the head**

- **Lumbar puncture**
  - Contraindications: ↑ ICP; CNS mass lesion, focal neurological deficits, papilledema, coagulation disorders, meningococcal septicemia, or septic shock

- **Radiography**: Chest and sinus

- **EEG**
Meningitis

- Data assessment—diagnostics
  - Cultures, cells, glucose, protein

<table>
<thead>
<tr>
<th>Normal CSF</th>
<th>Bacterial</th>
<th>Viral</th>
<th>Fungal</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC cells/µL</td>
<td>0-5 cells</td>
<td>1000-5000</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Cell diff (ie, neutrophils)</td>
<td>&lt;80%</td>
<td>&gt;80% (PAN)</td>
<td>&lt;40% early neutrophils</td>
</tr>
<tr>
<td>Protein mg/dL</td>
<td>18-45</td>
<td>100-500</td>
<td>&lt;200</td>
</tr>
<tr>
<td>Glucose mg/dL</td>
<td>45-80 (0.6xBS)</td>
<td>5-40 (&lt;0.3xBS)</td>
<td>&gt;40</td>
</tr>
</tbody>
</table>

Bacterial Meningitis: Patient Problems

1. Infection of meninges
2. Elevated body temperature r/t CNS inflammation
3. Acute pain r/t inflammation of meninges, headache, nuchal rigidity, irritation of pain receptors, or ↑ ICP secondary to CNS inflammation
4. Seizures: risk for injury r/t seizure activity secondary to cerebral irritation
5. Increased ICP r/t CNS infections
6. Hydrocephalus r/t CNS inflammation
7. Respiratory complications: risk for ineffective respiratory function r/t immobility and pain, ineffective airway clearance, risk for aspiration r/t seizure activity

Bacterial Meningitis: Infection of Meninges

Characteristics
- Pain
- meningeal irritation
- petechial rash

Medical interventions
- ABX therapy
  - Strep pneumonia: pen G, ceftriaxone, cefotaxime
  - Resistant to pen—vancomycin
  - Neisseria: pen G or ampicillin
  - H flu: cefotaxime or ceftriaxone
  - Corticosteroid therapy

Surgical interventions
- Treat complications (eg, drainage of CNS abscesses or insertion of ventricular-peritoneal shunt for communicating hydrocephalus)

Nursing interventions
- Droplet precautions
- Vital signs
- Neuro checks
- Monitor lab results
- Administer ABX, antipyretics, analgesics, and IV fluids
- Provide quiet, darkened room and encourage rest
- Bed rest, neutral alignment
- Promote nutritional intake and fluids
- Provide skin care

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Bacterial Meningitis: Infection of Meninges

**Expected outcomes**
- Infection source identified and infection successfully treated
- Return to prior baseline status
- No residual neurological deficits
- Remains seizure-free

**Potential complications**
- Waterhouse-Friderichsen syndrome
- Disseminated intravascular coagulation
- Sensorineural hearing or vision loss
- Encephalitis or hydrocephalus
- Brain abscess or subdural effusions
- Increased ICP → cerebral herniation → death
- Permanent neurological deficits
- Paralysis; ARDS
- Seizures

---

**Expected outcomes**
- ICP remains <15 mmHg and CPP >60 mmHg or as ordered
- Neurological status improves or baseline is maintained
- Complications of ICP are resolved
- No complications of ICP monitoring develop

**Potential complications**
- Complications of ICP monitoring; fever, infection, seizures, excess fluid volume, herniation, and death
Bacterial Meningitis:
Elevated Body Temperature r/t CNS Inflammation

Characteristics
- Fever
- Warm, flushed skin
- Diaphoresis
- Tachycardia
- Tachypnea
- Seizures

Medical interventions
- ABX
- Antipyretics
- Cooling devices

Nursing interventions
- Administer antipyretics as necessary
- Administer IV fluids as ordered
- Monitor I&O
- Institute cooling measures
- Monitor vital signs
- Monitor LOC
- Monitor lab values: WBC and electrolytes

Expected outcomes
- Patient remains afebrile at 37°C
- BP, RR, and HR are WNL
- Patient remains seizure-free

Potential complications
- Seizures
- Activity intolerance r/t fatigue and malaise secondary to infection
- Delayed growth and development r/t brain damage secondary to infectious process, ↑ ICP
- Disturbed sensory perception: impaired auditory, kinesthetic, visual acuity r/t CNS infection

Bacterial Meningitis:
Acute pain r/t inflammation of meninges, headache, nuchal rigidity, irritation of pain receptors, or ↑ ICP secondary to CNS inflammation

Characteristics
- C/o headache, neck and back pain, or nuchal rigidity
- Guarding or protective behavior
- Withdrawal from social contact
- Facial mask of pain
- Moaning, crying, irritability
- Increase in vital signs, restlessness

Medical interventions
- Analgesic medications as necessary
- Supportive therapy

Nursing interventions
- Assess location, quality, severity of pain
- Assess patient behavior and physiological signs secondary to pain
- Provide quiet, darkened room with minimal disturbances
- Implement comfort measures to promote relaxation
- Institute nonpharmacological measures for pain control
- Administer analgesics as prescribed
- Assess patient’s pain and effectiveness of interventions and SE
- Administer pain medication prior to activities

Expected outcomes
- Patient remains seizure-free
- BP, RR, and HR are WNL
- Patient remains afebrile at 37°C

Potential complications
- Seizures
- Activity intolerance r/t fatigue and malaise secondary to infection
- Delayed growth and development r/t brain damage secondary to infectious process, ↑ ICP
- Disturbed sensory perception: impaired auditory, kinesthetic, visual acuity r/t CNS infection

Neurology
**Bacterial Meningitis: Acute pain r/t inflammation of meninges, headache, nuchal rigidity, irritation of pain receptors, or ↑ ICP secondary to CNS inflammation**

**Expected outcomes**
- Patient reports adequate pain control
- Vital signs are WNL

**Potential complications**
- Risk for injury r/t restlessness and disorientation secondary to pain

**Bacterial Meningitis: Seizures**
Risk for injury r/t seizure activity secondary to cerebral irritation

**Characteristics**
- Loss of consciousness
- Incontinence of bowel and bladder
- Profuse salivation
- Apathy and cyanosis
- Respiratory and metabolic acidosis

**Medical interventions**
- Antiepileptic drugs
- Sedation

**Expected outcomes**
- Patient remains seizure free
- No injuries occur during seizure
- No toxic SE results from AEDs

**Nursing interventions**
- Implement seizure precautions
- Protect patient from injury
- Document seizure activity
- Monitor neurological status and vital signs after seizure
- Administer antiepileptic drugs as ordered

**Potential complications**
- Status epilepticus
- Injury
- Respiratory compromise secondary to ineffective airway clearance

**Bacterial Meningitis: Hydrocephalus r/t CNS Inflammation**

**Characteristics**
- Headache, N/V
- Lethargy
- Visual disturbances
- Ataxia
- Incontinence

**Surgical interventions**
- EVD
- Ventricular peritoneal shunt

**Expected outcomes**
- Hydrocephalus is relieved
- Shunt insertion post-op recovery is unremarkable
- Shunt functions properly
- ↑ ICP leading to cerebral herniation and death
- Shunt malfunction

**Nursing interventions**
- Monitor neurological status and vital signs
- Maintain EVD
- Provide ventricular shunt post-op care
- Monitor for signs of shunt malfunction

**Potential complications**
- ↑ ICP leading to cerebral herniation and death
- Shunt malfunction

**Expected outcomes**
- Hydrocephalus is relieved
- Shunt insertion post-op recovery is unremarkable
- Shunt functions properly
- ↑ ICP leading to cerebral herniation and death
- Shunt malfunction
Viral Meningitis

- **Pathophysiology**
  - Viral transmission: fecal-oral contamination or respiratory droplets
  - Virus replicates at site of entry
  - Primary viremia is followed by viral replication in blood and spread to CSF
  - Patient is infectious from 3 days after infection to 10 days after symptoms develop
    - Incubation period 3-7 days
  - Data assessment: neurologic exam
    - Viral
      - Milder than bacterial
      - Resembles influenza
      - Headache, fever, photophobia, malaise, and nausea

Viral Meningitis: Patient Problems

1. Infection of meninges
2. Elevated body temperature r/t CNS inflammation
3. Acute pain r/t CNS inflammation
4. Hydrocephalus r/t meningitis
5. GI complications r/t inadequate nutrition
6. Impaired mobility r/t CNS infection
7. Psychosocial anxiety r/t CNS infection

Viral Meningitis: Infection of the Meninges

- Characteristics
  - Headache
  - Neck pain
  - Back pain

- Medical interventions
  - Empiric ABX therapy initiated until bacterial cultures negative
  - Acyclovir (Zovirax) used to treat herpes virus types 1 & 3 and varicella-zoster virus
  - Supportive therapy

- Nursing team interventions same as bacterial meningitis

- Expected outcomes
  - Patient recovers without neurological sequelae

- Potential complications
  - Encephalitis
  - Hydrocephalus
  - Cerebral edema with ↑ ICP
Fungal Meningitis

- All major fungal agents can produce meningitis
- Cryptococcus neoformans most common
- High-risk groups include immunocompromised persons, especially with HIV infection
- Medical: C neoformans meningitis is treated with amphotericin B and flucytosine

Pathophysiology same as bacterial meningitis

Fungal Meningitis: Assessment

**Clinical manifestations**
- Clinical symptoms often nonspecific, but include:
  - Headache
  - Fever
  - Photophobia
  - Malaise
  - Nausea

Different symptoms in patient with/without HIV infection

**Diagnostics**
- Laboratory studies
- CSF studies
- Serology (antigen tests)
- India ink examination
- Latex agglutination tests
- ELISA to isolate fungi in CSF
- CT or MRI
- Identify focal lesions, especially in C neoformans

Fungal Meningitis: Patient Problems

1. Infection of the meninges
2. Elevated body temperature r/t CNS inflammation
3. Acute pain r/t CNS inflammation
4. Hydrocephalus r/t meningitis
5. GI complications r/t inadequate nutrition
6. Impaired mobility r/t CNS infection
7. Psychosocial anxiety r/t CNS infection
Fungal Meningitis: Infection of Meninges

Characteristics
- Pain: headache, neck, and back
- Fever, malaise, nausea
- Lethargy, personality changes
- Cranial nerve palsies, papilledema
- Meningeal headache, nuchal rigidity, photophobia, Kernig’s and Brudzinski’s signs

Medical interventions
- Antifungal therapy
  - *C. neoformans* treated with amphotericin B and flucytosine
  - Supportive therapy

Nursing interventions same as bacterial meningitis

Expected outcomes
- Infection source is identified and infection successfully treated
- Patient does not experience any permanent neurological deficits
- Patient remains seizure-free

Patient complications
- Encephalitis
- Hydrocephalus
- Cerebral edema with ↑ ICP

Encephalitis

Definition
- Inflammation of the brain parenchyma caused by virus, bacterium, fungus, or parasite

Etiology
- Virus is most common cause
  - Respiratory system: mumps, measles, varicella virus
  - Oral: enteroviruses/polio
  - Oral or genital: herpes simplex
- Bites
  - Animal: rabies
  - Insect: arbovirus (mosquito), Lyme disease (tick)

Pathophysiology
- Arbovirus: Includes St. Louis/eastern/western equine and West Nile virus
- Humans bitten by the vector are asymptomatic or develop vague flu-like symptoms
- Produces diffuse disintegration of single nerve cells, inflammation, and necrosis of both white and gray matter (spares the brainstem)
Encephalitis

- Pathophysiology
  - Virus
  - Enters body and colonizes
  - Penetrates cell
  - Transcribes virus-coated proteins and replicates the viral nucleic acid
  - Blood-brain barrier prevents virus from entering CNS, but it can enter through cerebral capillaries or choroid plexus and/or along peripheral nerves
  - Virus attacks susceptible neurons and causes cell lysis

Encephalitis: Assessment

Subjective data
- Symptoms vary with organism and area involved
  - Headache is usually present

Objective data
- Signs
  - Fever
  - Nuchal rigidity
  - Photophobia
  - Altered LOC
  - Focal neurological deficits
  - Aphasias
  - Babinski’s reflex
  - Involuntary movements
  - Cranial nerve deficits
  - New psychiatric symptoms
  - Cognitive deficits
  - Seizures

Encephalitis: Diagnostics

Medical history and clinical exam
Laboratory studies
- Cultures: blood, urine, stool, nasopharynx, or sputum
- CSF studies
  - Cell count, cytological characteristics, culture
  - Protein level slightly elevated
  - Glucose level normal
- PCR
- Serology: EUISA and serological assays for antiviral IgM and IgG

EEG
- MRI—If not possible, CT of head with and without contrast

Surgery—Tissue biopsy in some cases
Encephalitis: Patient Problems

1. Infection of the CNS
2. Elevated body temperature r/t CNS inflammation
3. Acute pain r/t CNS inflammation
4. Seizures r/t CNS irritation
5. Increased ICP r/t CNS inflammation
6. Respiratory complications
7. Cardiovascular complications
8. GI complications r/t inadequate nutrition
9. Impaired mobility r/t CNS infection
10. Psychosocial anxiety r/t CNS infection

Encephalitis: Herpes Simplex Virus

- Most common and most severe form
- Herpes simplex virus-1 (HSV-1)
- 2,000 cases annually in US
- 30%-70% mortality rate, neurological deficits
- HSV accounts for >50% of encephalitis cases in patients over the age of 50 years and patients with HIV
- No geographical or seasonal pattern

HSV Encephalitis: Pathophysiology

- HSV lies dormant within dorsal ganglia of trigeminal nerve in 90% of people in US
- Virus is activated in trigeminal ganglia, follows fibers, and attacks frontal and temporal lobes, causing bilateral hemorrhagic necrosis
Neurologic HSV Encephalitis: Assessment

Subjective data
- Headache
- Confusion
- Hallucinations

Objective data
- Change in LOC
- Personality and behavioral changes
- Memory loss
- Aphasia
- Hemiparesis
- Temporal-lobe seizures
- Progression to deep coma as cerebral edema increases; herniation may occur, leading to death

Neurologic HSV Encephalitis: Diagnostics

Lumbar puncture
- Elevated open pressure
- CSF studies: Presence of lymphocytes, increased protein level, normal glucose level.
- PCR: Positive for HSV-1 antigen in 98% cases

EEG
- High-voltage, sharp waves in temporal areas
- Slow-wave complexes at regular 2- to 3-second intervals

CT
- Normal initially
- Later scans show gyral enhancement, hypodensity in temporal areas, and mass effect in 40-50% of patients

MRI
- Edema and hemorrhage over inferior portion of frontal and temporal lobes
- Blood-brain barrier abnormalities seen with contrast

Surgery to obtain cultures of cerebral tissue

Neurologic HSV Encephalitis: Patient Problems

1. Infection of brain parenchyma
2. Acute pain r/t CNS inflammation
3. Seizures r/t CNS irritation
4. Increased ICP r/t CNS inflammation
5. Respiratory complications
6. Cardiovascular complications
7. GI complications r/t inadequate nutrition
8. Impaired mobility r/t CNS infection
9. Psychosocial anxiety r/t CNS infection
**Question 10**

SP, a 20-year-old college student, presents to the ED complaining of photophobia, stiff neck, temp of 103°F, malaise, and purple blotches located primarily on the trunk area. He is given antibiotics in the ED and admitted to the ICU with a BP of 85/50, HR 140, and ventilated. Cultures have been done, but there are no results. SP’s likely infectious illness is:

A. Lyme disease
B. Herpes simplex encephalitis
C. Neisseria meningitides
D. Guillain-Barré syndrome

**Question 10—Rationale**

SP, a 20-year-old college student, presents to the ED complaining of photophobia, stiff neck, temp of 103°F, malaise, and purple blotches located primarily on the trunk area. He is given antibiotics in the ED and admitted to the ICU with a BP of 85/50, HR 140, and ventilated. Cultures have been done, but there are no results. SP’s likely infectious illness is:

C. Neisseria meningitides
   - Lyme disease—Lyme disease is a viral encephalitis that presents with bullseye rash and malaise. It is not accompanied by purple blotches
   - Herpes simplex encephalitis—The patient may experience HA, fever, and malaise but also has strange behavior, personality changes, and seizures
   - Guillain-Barré syndrome—Presentation includes ascending weakness accompanied by sensory loss and cranial nerve dysfunction
Neurologic Question 11

Patients with herpes simplex encephalitis commonly experience headache, confusion, hallucinations, decreases in level of consciousness, personality and behavioral changes, and possibly aphasia due to the virus attacking which parts of the brain parenchyma?

A. Occipital lobe
B. Frontal and temporal lobes
C. Diencephalon
D. Brainstem and cerebellum

Question 11—Rationale

B. Frontal and temporal lobes
- Occipital lobe is responsible for vision
- Diencephalon regulates homeostasis and smoothness of movement
- Brainstem and cerebellum is responsible for coordination, balance, and respirations/heart rate
Seizures and Epilepsy

- Abnormal firing in the brain
- Epileptogenesis occurs during an imbalance between cerebral excitation and inhibition
  - Brain cells become abnormally linked together, leading to abnormal electrical firing
  - Excitation (glutamate/NMDA) vs inhibition (GABA facilitates activity of CI channels)—opens K channels or closes Ca channels
- Classification
  - Partial seizure: onset of synchronous cortical discharges involving a focal brain region
  - Generalized seizure: sudden onset involving both hemispheres

Seizures and Epilepsy

- Partial seizures
  - Simple partial: focal brain region activity without alteration in consciousness
    - An aura can be indicative of simple partial seizures (SPS)
    - Motor events such as face twitching/hand jerking
    - Somatosensory events such as unusual taste in mouth
    - Psychic events—illusions/hallucinations/déjà vu
  - Complex partial seizures: brain region affected with alteration in consciousness
    - Often preceded by SPS, with progressive impairment in conscious
    - Automatisms (lip smacking/blinking/picking at clothes)
    - Motor phenomena—wandering, running, arm jerking
    - Partial seizure that generalize secondarily

Seizures and Epilepsy

- Generalized seizure—both hemispheres
  - Absence seizure: impaired responsiveness, minimal motor involvement, lasts <30 seconds
  - Myoclonic: sudden shock-like muscle contraction
  - Atonic: drop attacks (brief loss of consciousness/loss of muscle tone)
  - Tonic: increased tone in extensor muscles
  - Clonic: start with loss of consciousness/sudden hypotonia, then limb jerking
  - Tonic clonic: loss of consciousness, increased tone, rhythmic muscle jerks
Seizures and Epilepsy

- Subjective
  - Obtain description of event, including time
  - Precipitating factors?
  - Postictal behavior
- Objective
  - Assess mental status and cognition
  - Evidence of trauma/infection
  - Focal findings
  - Asymmetries
- Diagnostics
  - Lab tests
  - EEG
  - CT/MRI to rule out mass lesions

Nursing Assessment

- History
- Type of seizure activity
  - Absence (petit mal)
  - Focal
  - Tonic-clonic
  - Electrical status
  - Status

Diagnostics

- Lab
  - Electrolytes
  - Hypoglycemia
  - Hypoxemia
  - Prolactin level
  - Myoglobin
  - Anticonvulsant drug levels
- EEG
Seizures and Epilepsy

<table>
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<tr>
<th>Treatment: pharmacologic</th>
<th>Drug level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenobarbital 60-250 mg/day</td>
<td>15-40 mcg/mL</td>
</tr>
<tr>
<td>Phenytoin (Dilantin) 300-600 mg/day</td>
<td>10-20 mcg/mL</td>
</tr>
<tr>
<td>Carbamazepine (Tegretol) 600-1200 mg/day</td>
<td>4-12 mcg/mL</td>
</tr>
<tr>
<td>Primidone (Mysoline) 25 mg tid</td>
<td>5-12 mcg/mL</td>
</tr>
<tr>
<td>Valproic acid (Depakote) 15-60 mg/kg/day</td>
<td>50-100 mcg/mL</td>
</tr>
<tr>
<td>Lamotrigine (Lamictal) 100-300 mg/day</td>
<td>2-4.5 mcg/mL</td>
</tr>
<tr>
<td>Gabapentin (Neurontin) 900-3600 mg/day</td>
<td>2-20 mcg/mL</td>
</tr>
<tr>
<td>Levetiracetam (Keppra) 1000-3000 mg/day</td>
<td>3-37 mcg/mL</td>
</tr>
<tr>
<td>Lacosamide (Vimpat) 10-400 mg/day</td>
<td>3.8-9.3 mcg/mL</td>
</tr>
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</table>

Seizures/Epilepsy

- **Side-effects profile**
  - Stevens-Johnson syndrome: erythema multiforme
  - Aplastic anemia
  - Allergic dermatitis
  - Hepatic failure
  - Most common: somnolence, dizziness, memory impairment, cognitive slowing, ataxia

- **Nursing interventions**
  - Education
  - Explain drug-drug interactions
  - Other: ketogenic diet (high in fat/low carbs/restrict calories); vagal nerve stimulator; surgery (temporal lobectomy or corpus callosotomy)

Status Epilepticus
Pathophysiology
- Tonic-clonic seizures
- Increase cerebral metabolic rate/oxygen use
- CBF increases 3-5 x normal
- Cellular swelling
- Systemic metabolic acidosis

Etiology/Precipitating Factors
- Withdrawal from anticonvulsants, alcohol, or drugs
- CNS infections
- Brain tumors
- Metabolic disorders—uremia, hypoglycemia, or hyponatremia
- Craniocerebral trauma
- Cerebral edema
- Stroke

Interventions
- Airway and ventilations
  - Supplemental oxygen and pulse oximetry
  - Intubate if prolonged seizure
  - Check blood glucose
- Normal saline IV
  - Thiamine 100 mg diluted with 8 mL NS slow IV push over 5 minutes, then:
  - 50% dextrose 50 mL slow IV push (if bedside glucose <70)
- Stop seizure
  - Lorazepam 2 mg IV push q 1 minute up to 8 mg
  - Fosphenytoin (Cerebyx) 20 PE mg/kg
- Protect from injury
Interventions

- If still seizing:
  - Fosphenytoin 5 PE mg/kg IV; may repeat with another dose to a loading dose of 30 mg/kg
  - Phenobarbital 20 mg/kg IV; infuse at 50-100 mg/min; may repeat with additional 5-10 mg/kg to a total dose of 30 mg/kg (must be intubated and mechanically ventilated)
  - Lorazepam 2 mg/kg IV push every 1 minute until a 20 mg total loading dose is given

- Additional antiepileptic drug options
  - Valproate sodium (Depacon) 25 mg/kg IVB loading dose over 60 min x 1 and maintenance 10-15 mg/kg/day every 6 hours
  - Propofol 1-2 mg/kg x 1 IV (administered by MD), then drip 20-250 mcg/kg/min (must be intubated and mechanically ventilated)
  - Pentobarbital 5-15 mg/kg IV x 1 over 1 hour, then 0.5-3mg/kg/hr (must be intubated and mechanically ventilated)
  - Midazolam 10 mg IVP x 1 then 0.05-0.4 mg/kg/hr
  - Diazepam 0.15 mg/kg up to 10 mg IV push at 5 mg/min
  - Lorazepam 2 mg IV push every 2 hours
  - No paralytics unless continuous EEG in place

Review Questions
Question 12

A 10-year-old male ignores repeated requests to answer questions and often stares for short periods of time, as reported by his teacher. This may be indicative of a:

A. Partial seizure
B. Complex partial seizure
C. Myoclonic seizure
D. Generalized seizure, known as an absence seizure

Question 12—Rationale

A 10-year-old male ignores repeated requests to answer questions and often stares for short periods of time, as reported by his teacher. This may be indicative of a:

D. Generalized seizure, known as an absence seizure
  - Partial seizure—Partial seizures are localized to one area of the brain and do not impair consciousness
  - Complex partial seizure—These are manifested by automatisms and motor phenomena such as wandering, running, or arm jerking
  - Myoclonic seizure—These are generalized seizures that are sudden shock-like muscle contractions

Question 13

A patient in status epilepticus must receive medication immediately to halt the seizure activity. The first-line medication for status epilepticus is:

A. Valium 10 mg po
B. Midazolam 1 mg IV
C. Phenobarbital 30 mg IV
D. Lorazepam 2 mg IV
**Question 13—Rationale**

A patient in status epilepticus must receive medication immediately to halt the seizure activity. The first-line medication for status epilepticus is:

- **D. Lorazepam 2 mg IV**
  - Valium 10 mg po—A patient in status epilepticus will be unable to take any medication by mouth.
  - Midazolam 1 mg IV—Although midazolam is used as a secondary drug in status epilepticus, the dose would be much higher (i.e., 10 mg).
  - Phenobarbital 30 mg IV—This drug is considered a second-line drug and the dosing is 20 mg/kg IV given slowly 50-100 mg/minute.

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**Space-Occupying Lesions**

**Brain Tumors**

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**Facts/Stats**

- Incidence:
  - Primary—36,000/yr
  - Secondary—18,000/yr
- Males > females
- Age: 40–60 age group
Facts/Stats

- Classification
  - Benign: regular cell structure
  - Malignant: infiltrates brain tissue, poor boundaries, rapid mitotic activity, necrosis
  - Location: supra vs infratentorial
  - Primary vs secondary
  - Intraaxial (glial cells) vs extraaxial (meninges, CN, pituitary, and cysts)
  - Histological origin

Intrinsic Tumors

- Astrocytoma and GBM
  - Grades I-IV
- Oligodendroglioma
- White matter
- Frontal and parietal lobes
- Ependymoma
  - Ependymal cells in ventricles
- Medulloblastoma
  - Highly malignant in young children

Extrinsic Tumors

- Meningiomas
  - Benign, slow-growing
- Neuromas
  - Acoustic tumor (VIII CN)
  - Schwannoma
- Neurofibromatosis
  - Type I: café au lait spots
  - Type II: bilateral hearing loss
Congenital Tumors

- Hemangioblastoma
  - Slow-growing, vascular tumor
  - Common in cerebellum
- Craniopharyngioma
  - Embryonic Rathke’s pouch/suprasellar
  - Arise from pituitary hypophysis
  - ↑ ICP, pituitary/hypothalamic dysfunction
  - Visual disturbances

Pituitary Tumors

- Secreting
  - ACTH (Cushing) or GH (gigantism)
- Nonsecreting (90% space occupying)
  - Compresses pituitary: visual/hypopituitary

Pathophysiology

- Increased ICP
- Tumor growth
- Cerebral edema
  - Tissue surrounding the vicinity of tumor
  - Endothelial cells of white matter
  - Increase permeability of plasma and vasogenic edema
Signs/Symptoms
- Headache
- Seizures
- Vomiting
- Alterations in consciousness
- Localizing signs

Signs/Symptoms
- Tumor area
  - Frontal: affect, motor, speech, behavior
  - Parietal: numbness/sensory
  - Temporal: psychomotor seizures/receptive aphasia
  - Occipital: vision
  - Pituitary: visual, headaches, Cushing's syndrome, acromegaly
  - Ventricles: hydrocephalus, HA, changes in LOC
  - Cerebellum: ataxia, incoordination, dysmetria
  - Brainstem: CN defects, vomiting, respiratory

Complications of Tumor Growth
- Edema
- Increased ICP
- Seizures
- Hydrocephalus
- Hormonal changes
- Focal deficits
Treatment
- Stereotactic therapy
- Craniotomy
- Gamma knife
- Conventional radiation
- Brachytherapy
- Chemotherapy
- Gene therapy/virus therapy

Nursing Interventions
- Pre-op: steroids, anticonvulsants, body image
- Post-op
  - ABC
  - Monitor neurologic status
  - Watch for seizures
  - Dilantin: dosing/levels
  - System support
  - Family/patient support
  - Pituitary tumors
    - Watch I/O, Na+, specific gravity

Review Questions
Question 14

HH, a 45-year-old right-handed male, presents to the ED with headache, right arm weakness with right facial droop, and difficulty expressing speech. A CT scan reveals diffuse cerebral edema surrounded by a ring-enhancing lesion. The most likely location of the mass is:

A. Right parietal lobe of cerebral hemisphere
B. Left temporal lobe of cerebral hemisphere
C. Right frontal lobe of cerebral hemisphere
D. Left frontal lobe of cerebral hemisphere

Question 14—Rationale

HH, a 45-year-old right-handed male, presents to the ED with headache, right arm weakness with right facial droop, and difficulty expressing speech. A CT scan reveals diffuse cerebral edema surrounded by a ring-enhancing lesion. The most likely location of the mass is:

D. Left frontal lobe of cerebral hemisphere

- Right parietal lobe of cerebral hemisphere—The right parietal lobe’s main functions are sensory perception, body awareness, and sensory interpretation of input affect the left side of the body
- Left temporal lobe of cerebral hemisphere—The left temporal lobe’s main functions are interpretation of hearing, auditory perception, short-term memory, and comprehension of the spoken word
- Right frontal lobe of cerebral hemisphere—The right frontal lobe is responsible for higher mental functions and movement of the left arm/leg/face. The right frontal lobe in right-handed individuals is considered to be the nondominant hemisphere. Expressing speech is facilitated in the dominant hemisphere in the frontal lobe

Question 15

TP, a 53-year-old male, has been diagnosed with a brain mass. His symptoms include contralateral sensory loss, neglect to the left side, and inability to draw with loss of spatial orientation. The area of the brain most likely affected is:

A. Parietal lobe
B. Frontal lobe
C. Temporal lobe
D. Occipital lobe
Question 15—Rationale

TP, a 53-year-old male, has been diagnosed with a brain mass. His symptoms include contralateral sensory loss, neglect to the left side, and inability to draw with loss of spatial orientation. The area of the brain most likely affected is:

A. Parietal lobe
   - Frontal lobe—Tumors in this area affect motor to the opposite side, speech (if dominant hemisphere), judgment, personality, initiation, and continence
   - Temporal lobe—Tumors in this area present with receptive aphasia (dominant hemisphere only) and some visual field cuts
   - Occipital lobe—Tumors in this area affect visual perception and field of vision

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Question 16

TP undergoes a craniotomy for debulking of a large tumor. Postoperatively, he is extubated and admitted to the ICU. After 1 hour, TP sustains a generalized seizure lasting 45 seconds. What is your initial intervention?

A. Check the pupillary reaction
B. Administer IV midazolam
C. Assess airway and breathing and prepare for possible intubation
D. Get a stat CT scan of the brain

Question 16—Rationale

TP undergoes a craniotomy for debulking of a large tumor. Postoperatively, he is extubated and admitted to the ICU. After 1 hour, TP sustains a generalized seizure lasting 45 seconds. What is your initial intervention?

C. Assess airway and breathing and prepare for possible intubation
   - Check the pupillary reaction—Although the pupils need to be checked, this would not precede assessing the airway
   - Administer IV midazolam—Airway stabilization is first. Lorazepam IV would be the drug of choice to reduce recurrence
   - Get a stat CT scan of the brain—This intervention would be done after C, B, and A