Intracranial Pressure

Sonya Mertz MSN, FNP-C,RN, CNRN

Quick A&P

Skull

- The skull consists of 8 bones of the cranium and 14 bones of the face
Characteristics of CSF

- **Appearance**: clear and colorless
- **Cells**: 0-5 cell/ml
- **Protein mg/dl**
  - Lumbar 15-45 mg/dl
  - Ventricular 5-15 mg/dl
- **Glucose 50-75 mg/dl**
- **Pressure**
  - Ventricular 0-15 mmHg
  - Lumbar 70-180 mm Hg

- **Bright Red** - indication of acute hemorrhage
- **Xanthochromia** - yellowish to light red discoloration due to breakdown of RBC’s; can indicate old blood
- **Cloudiness** - turbidity indicates infection due to increased WBC’s or protein.
- **Elevated Protein** - CNS tumors, viral meningitis, hemorrhage, multiple sclerosis, Guillain Barre Syndrome.
- **Elevated WBC’s**:
  - Lymphocytes: Viral or TB meningitis, MS, CNS tumors, herpes, syphilis
  - Granulocytes: bacterial meningitis
- **Decreased Glucose** - bacterial meningitis, SAH
- **Elevated lactate** - increased G. metabolism associated with bacterior fungal meningitis, TBI
Cerebrum

- **Frontal lobe**
  - Motor strip
- **Parietal lobe**
  - Sensory strip
- **Temporal lobe**
  - Sound, language, and short-term memory
- **Occipital lobe**
  - Vision, visual association area

Brain Stem

- **Diencephalon**
  - Thalamus—chief traffic relay station for directing sensory and other signals
  - Hypothalamus—controls internal body functions (i.e., temperature, blood pressure, shivering, sweating, gastrointestinal stimulation, water conservation, pituitary hormonal activity, and stress/adaptation response)
- Midbrain contains the nuclei of cranial nerves III-IV, pons V-VIII, and medulla oblongata IX-XII
- Controls basic and reflexic activities such as sleep, wakefulness, breathing, blood pressure, and heart rate
Cranial nerves

Vasculature of the Brain

ANATOMICAL TERRITORIES

MCA:
- Frontal, primary motor strip
- Parietal, primary sensory strip
- Occipital
- ACA:
  - Medial portion
  - Optic tract
  - Subcortical structures
  - ACOM
  - Connects ACAs
  - PCOM
  - Close Cir of Willis
  - Provide blood to ant vessels of Cir of Willis, if ICA occluded/stenosed
Getting blood back to the heart...

Venous circulation consists of:
- Brain's veins
- Dural venous Sinuses
- Meningeal veins
- Diploic veins

These veins are valveless.

Blood-Brain Barrier

- Provides very tight junctions responsible for selectivity to the substances that cross the neuronal membrane and gain entrance to the neuron.
- Protects the brain from toxic elements that may be circulating within the bloodstream.

Meninges
ICP Concepts & Cerebral Hemodynamics

Intracranial Pressure (ICP)

- Low pressure, dynamic number
  - 0-10 mmHg normal
  - 0-15 mmHg acceptable
  - 15-20 mmHg tolerable for a short time
- Above 20 mmHg, sustained for > 2 minutes can be damaging to cells
- Transient brief increases may occur without damage

Intracranial hypertension

- Intracranial hypertension
  - Also known as increased ICP
  - Defined as a sustained ICP elevation of > 20 mm Hg for more than 5 minutes
  - Malignant hypertension is ICP > 30 mm Hg
  - Multiple causes
  - Underlying cause needs to be identified and treated
Patient presents with signs of increased ICP. Differential diagnosis of intracranial emergencies:

- Subdural hematoma
- Epidural hematoma
- Subarachnoid hemorrhage (SAH)
- Acute hydrocephalus
- Increased ICP

- Cerebral herniation
- Status epilepticus
- Pituitary apoplexy
- Stroke
- Acute mental status changes

Learn the early signs and symptoms of increased ICP and act upon them to prevent secondary injury.

Monro-Kellie Hypothesis

- The skull's intracranial content volume is approximately 1,700 ml and includes
  - Brain 80%
  - Blood 10%
  - CSF 10%
- The fully developed skull is a rigid container that limits volume; there is no room for expansion.

Monro-Kellie Hypothesis

- If one component's volume increases, another, or both, must decrease to maintain equilibrium within the skull.
- If this compensatory mechanism fails, ICP increases.
CONDITIONS THAT INCREASE INTRACRANIAL VOLUME

**Brain:**
- Increase brain volume
- Space occupying masses
- Tumor
- Hematoma
- Abscess
- Aneurysm
- Air
- Cerebral edema

**Blood:**
- Vasodilation
  - Hypercapnia (Inc CO2)
  - Hypoxia
  - Hyperemia
  - Altered autoregulation
- Venous outflow obstruction
  - Sagittal Sinus Thrombosis
  - Jugular Vein Compression
- Increased intrathoracic or intra-abdominal pressure
  - Valsalva
  - Coughing
  - Vomiting
  - PEEP

**CSF:**
- Obstruction of flow between ventricles (Obstructive hydrocephalus; non-communicating)
- Tumor or cyst in or around ventricular passageways
- Decreased absorption (Communicating HCP)
- SAH Blockage of arachnoid villi
- RBCs
- WBCs
- Increased production
- Tumor in choroid plexus
- More common in children than adults

Compliance

- Compliance measures the ability of the brain to maintain equilibrium in the presence of physiological and external changes.
- Compliance also is called “brain stiffness.”
- Compliance represents the ratio of change in content volume to the change in pressure, ICP.
Pressure-Volume Curve

- Amount of volume increase
- Time frame of the increase
- Volume of the intracranial components
  - Small increases in volume over a long period of time are better tolerated than large increases in volume over a short period of time.

Compensatory Mechanisms

- Displacement of CSF into the spinal subarachnoid space
- Compression of the dural sinuses
- Decreased production of CSF
- Vasoconstriction of the blood vessels of the cerebral structures
- Increased venous outflow

Cerebral Hemodynamics (CBV)

- Cerebral blood volume (CBV) is the amount of blood in the cranial cavity.
  - Approximately 10% of the cranial cavity's volume
  - Affected by compensatory mechanisms
  - Increases when pressure in the venous system is increased
  - Increased ICP follows CBV increases
Cerebral Hemodynamics (CBF)

• Cerebral blood flow (CBF) = CPP/CVR
  – Required to provide oxygenation to brain tissue
  – Brain receives 20% of total cardiac output and uses 15% of oxygen consumed in the basal state
    • 750–800 ml/min to the whole brain

Normal mL/100gm/min
• Global 40–55 mL/100gm/min
• Gray matter 67–80 mL/100gm/min
• White matter 18–35 mL/100gm/min

Cerebral Blood Flow (CBF)

• Cerebral Blood Flow (CBF)
  – Extrinsic Factors:
    1. Systemic blood pressure
    2. Cardiac output
    3. Blood viscosity
    4. Vascular tone

If MAP falls below 70 mmHg, CBF will decrease affecting cerebral autoregulation.

Cerebral Blood Flow: Intracerebral Regulation

• Autoregulation: ability of an organ to maintain constant blood flow within a limited homeostatic range.
  – Independently controlled by myogenic mechanisms: arterial intraluminal stretch receptors (vasoconstrict&dilate).
    – Limited, will fail when:
      • ICP>40 mmHg
      • MAP exceeds parameters of 60-150 mmHg
      • Ischemia
      • Inflammation (Causes vasomotor paralysis/reduced cerebrovascular tone)
    – CBF&CBV passively dependent on SBP
Cerebral Blood Flow: Intracerebral regulation

<table>
<thead>
<tr>
<th>Chemical-Metabolic</th>
<th>level</th>
<th>response</th>
<th>CBF</th>
<th>notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaCO₂</td>
<td>High</td>
<td>vasodilate</td>
<td>↑</td>
<td>2-3% change per value</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>Low</td>
<td>vasoconstrict</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>PaO₂</td>
<td>Low</td>
<td>vasodilate</td>
<td>↑</td>
<td>~50mmHg</td>
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<tr>
<td>PaO₂</td>
<td>High</td>
<td>vasoconstrict</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>Low</td>
<td>vasodilate</td>
<td>↑</td>
<td>acidosis</td>
</tr>
<tr>
<td>pH</td>
<td>High</td>
<td>vasoconstrict</td>
<td>↓</td>
<td>alkalosis</td>
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</table>

Cerebral Perfusion Pressure (CPP)

- CPP = MAP – ICP
  - MAP: the force trying to drive blood, and the oxygen it transports into the brain
  - ICP: the force that resists (or regulates) “how much” blood gets through
- CPP is the blood pressure gradient across the brain.
  - Normal range is 70–100 mm Hg.
- In the patient with cerebral trauma CPP can go to 60 mmHg.
- Inadequate CPP leads to ischemia, which can lead to infarction < 40 mm Hg: vessel will collapse.

Cerebral Perfusion Pressure (CPP)

- An acutely injured brain has higher metabolic rate, therefore requires higher CPP.
- CPP should be maintained at a minimum of 60mmHg up to 80mmHg.
- When ICP is elevated, monitor and maintain MAP >80 mmHg by using fluids and vasopressors.
Cerebral metabolism

- Brain has High dependence on Glucose and Oxygen to support function and Energy production.
  - Unable to store.
- Aerobic: 1 mole G. yields 38 moles ATP
- Anaerobic: 1 mole G. yields 2 moles ATP
  - less available ATP to fuel ATP dependent sodium-potassium pump in cell membrane.

Ischemic Penumbra
Pathophysiology: cerebral edema

**Vasogenic:** extracellular edema of white matter.

- Increase capillary permeability with protein & filtrate
- Local: brain tumor, cerebral infarct
- Generalized: cerebral trauma, meningitis
- Treatment:
  - dexamethasone (corticosteroid) effective with brain tumors.
  - Mannitol (osmotic diuretic) acute phase.

**Interstitial:** acute/subacute HCP, benign intracranial HTN (pseudotumor cerebri)

- Periventricular of white matter
- High intraventricular pressure forces CSF across ependymal cells
- Treatment:
  - Acetazolamide (Diamox)
  - Temporary drainage of CSF until self-corrects
  - Placement of shunt
  - Corticosteroids, osmotic diuretics are ineffective

**Cytotoxic:** intracellular space swelling (neurons, glia, endothelial cells)

- Affects gray & white matter (diffuse)
- Associated with hypoxic & anoxic events, hyponatremia, SIADH
- Results in: ATP-dependent sodium-potassium pump failure (~potassium leaves cell, sodium remains water follows).
  - treatment: corticosteroids not effective
    Osmotic diuretics with acute stage

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Pathophysiology: cerebral edema

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- Affects gray & white matter (diffuse)
- Associated with hypoxic & anoxic events, hyponatremia, SIADH
- Results in: ATP-dependent sodium-potassium pump failure (~potassium leaves cell, sodium remains inside) follows.
- Treatment: corticosteroids not effective
  Osmotic diuretics with acute stage
**COMPLIANCE AND COMPENSATORY MECHANISM EXHAUSTION**

commonly known as a “tight brain” and pt. at risk for herniation

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**Herniation Syndromes**

- **Cushings Response:**
  - Late sign, ischemic response
- **Goal for the body to maintain CBF in presence of Increased ICP**
- **Cushings TRIAD:**
  - Reflects IICP, Pressure on medullary center
- **S/S:**
  - **HTN:** Vasomotor center
  - Widening pulse pressure
  - **Bradycardia:** Cardiac Center
  - **IR RR:** Respiratory Center
SIGNS AND SYMPTOMS OF INCREASED ICP (IICP)

• General signs
  • Headache
  • Vomiting: w/ or without nausea; May be projectile

• EARLY: Decreased level of consciousness
  • Restlessness, agitation
  • More stimuli required to arouse
  • Confusion
  • Less able to follow commands
  • Less purposeful response to pain
  • Pronator drift

• Infants
  • Failure to thrive, poor feeding, irritability, vomiting, seizures, bulging fontanels, split crani sutures

As brain compression increases, there is pupillary dysfunction, motor weakness, headache, seizures, vomiting, that leads to posturing and coma. Time is Brain.

SIGNS AND SYMPTOMS OF INCREASED ICP (IICP)

• Pupillary changes
  • Anisocoria (unequal pupils)
  • Sluggish reaction to light
  • Hippus
  • No reaction to light

• Motor changes
  • Asymmetrical weakness
  • Bilateral weakness
  • Posturing
  • Flaccidity

SIGNS AND SYMPTOMS OF INCREASED ICP (IICP)

Vital sign changes

• Heart rate:
  • Dysrhythmia
  • Tachycardia; may progress to bradycardia

• Blood pressure
  • Increasing SBP
  • Increasing Pulse Pressure

• Respiration
  • Cheyne-Stokes
  • Central neurogenic hyperventilation
  • Apneustic
  • Cluster
  • Ataxic
Management of ICP

- Elevate HOB at 30-45°
- Maintain head alignment
- Protect Airway
- Control pain and agitation with extreme caution
- Control environment
- Space nursing activities to avoid overstimulation
- Monitor fluid and electrolyte status
- Maintain normal temperature
- Prevent and/or treat seizure activity
- Prevent infection and complications
- Medications – hyperosmotic fluids, diuretics, anti-seizure, stool softeners

When the patient presents with symptoms of Increased ICP

- Catch these symptoms in the early phase!
- URGENT situation
  - Interventions:
    1. Non-selective osmotic diuretic: Mannitol
    2. Hypertonic saline: 23.4%
    3. Placement of intracranial monitoring device
       1. EVD: External ventricular device
       2. To monitor Intracranial Pressure (ICP)
       3. To manage High values of Intracranial pressure via drainage of Cerebral Spinal Fluid (CSF)
    4. Possible hemicraniectomy
    5. Temperature Management
    6. Barbiturates
       Monitor for patient response-reversal of symptoms if caught in time
ICP MONITORING

INDICATIONS

• GCS < 8
• Moderate to severe acquired brain injury
  • Traumatic Brain Injury
  • Subarachnoid Hemorrhage
• Diffuse cerebral edema
• Acute hydrocephalus
• Post-operative
• Encephalopathic states (i.e. acute hepatic failure)
• Pseudotumor Cerebri
• Meningitis
• Status Epilepticus

ICP Monitors

• Ventricular catheter
  • Connected to external transducer via fluid-filled system
• Transducer tipped device into ventricle, parenchyma, epidural or subdural space
  • Strain gauge or fiberoptic transmission of pressure
• Blended System
• Subarachnoid bolt or screw

External Ventricular Drains (EVD)

• Used to quickly reduce the amount of CSF in ventricles and intracranial pressure
• Most widely used devices – Most Accurate
• Allows treatment and monitoring simultaneously
• A catheter is actually placed inside one of the ventricles (a fluid-filled cavity in the brain where CSF is produced)
• Fluid can be easily removed for specimen collection.
• It is usually temporary, dependency on the EVD usually results in a V-P shunt placement.
INTRAPARENCHYMAL MONITOR

Advantages
• Quick to insert
• Useful when unable to obtain ventricular access
• Accurate and reliable
• Good waveform

Disadvantages
• Reflects regional pressure rather than whole brain pressure
• Fiberoptics can be broken easily

INTRACRANIAL PRESSURE MONITORING INDICATIONS: BTF GUIDELINES

No prospective randomized controlled trial….

Level II
• ICP should be monitored in all salvageable patients with TBI
• GCS 3-8
• Abnormal CTH (hematomas, contusions, swelling, hemiation, or compressed basal cisterns

Level III
• Initiate with normal CTH if 2 or more of the following:
• Age over 40
• Unilateral or bilateral motor posturing
• SBP <90mmHg
ICP Thresholds

- Level I
  - Insufficient Data
- Level II
  - Initiate treatment with ICP > 20 mm Hg
- Level III
  - Utilize a combination of ICP values and clinical and brain CT findings to determine need for treatment
  - Beyond age and neuro exam, amount of time ICP > 20 mm Hg is most predictive of outcome

CLINICAL EVIDENCE:

- Temporary or permanent CSF diversion is recommended in symptomatic patients with chronic hydrocephalus after SAH (Class I, B)
- Ventriculostomy can be beneficial in patients with ventriculomegaly and diminished level of consciousness after acute SAH (Class II a, B)


CPP AND AUTOREGULATION: SEVERE TBI GUIDELINES

- Level I
  - Insufficient data to support
- Level II
  - Aggressive attempts to maintain CPP > 70 mm Hg with fluids and pressers should be avoided because of risk of Adult/Acute Respiratory Distress Syndrome (ARDS)
- Level III
  - Avoid CPP < 50 mm Hg
  - The target CPP is 50-70 mm Hg.
  - Can use higher CPP if autoregulation intact

*** "Ancillary monitoring of cerebral parameters that include blood flow, oxygenation, or metabolism facilitates CPP management"
Patient Assessments

- Hourly neuro checks, with comparison to baseline, along with vital signs
- Hourly ICP, MAP, CPP values
- Observe for changes in waveform
- Observe for P1, P2, P3 waveforms and correlate with the arterial pulse waveforms
- Notify MD for changes in any of the above
- Maintain the integrity of the drainage system
- Monitor dressing site

Assessing Compliance: Using the ICP Waveform

- ICP waveform has three peaks:
  - P1—the percussion wave (arterial)
  - P2—the tidal wave (rebound)
  - P3—the dicrotic wave (venous)
**BEDSIDE ICP WAVEFORM ANALYSIS**

Observe for dampening of waveform

- Observe P1, P2 & their relationship
- P2 > P1 may indicate decreased compliance

### Assessing Compliance: Using the ICP Waveform

<table>
<thead>
<tr>
<th>Condition</th>
<th>ICP waveform</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rapidly expanding lesion</td>
<td>Increase mean ICP, increase ICP waveform amplitude</td>
</tr>
<tr>
<td>Increased/Decrease CSF volume</td>
<td>Increase/Decrease mean ICP, increase/decrease ICP waveform amplitude, little change in ICP waveform configuration</td>
</tr>
<tr>
<td>Severe arterial hypertension</td>
<td>Increase mean ICP, increase ICP waveform amplitude</td>
</tr>
<tr>
<td>Severe arterial hypertension</td>
<td>Decrease mean ICP, decrease ICP waveform amplitude, especially P1</td>
</tr>
<tr>
<td>Severe hypercapnia and hypoxia</td>
<td>Increase mean ICP, increase ICP waveform amplitude, rounding of ICP waveform due to increase in later waveform components</td>
</tr>
<tr>
<td>Jugular vein compression</td>
<td>Increase mean ICP, increase ICP waveform amplitude, mainly P2 and P3</td>
</tr>
<tr>
<td>Hyperventilation</td>
<td>Decrease mean ICP, decrease ICP waveform amplitude P2, and to a lesser degree P3, with little change to P1</td>
</tr>
</tbody>
</table>
Case # 1

- 68 yr RH Caucasian female found down at home.
  Hx: HTN, DMII, hyperlipidemia, obesity

  LKW >9hrs per daughter, since talking on phone and finding her mother

  Exam: awakens to voice, following simple commands on right side, incomprehensible sounds, PERRL (4+), right gaze preference, unable to cross midline to left, left facial, left sided weakness and decreased sensation. GCS 14, NIHSS 20

  CT of brain: acute RMCA territory infarct associated with sulcal effacement and mild mass effect. No midline shift or hemorrhagic conversion

Case # 1

- Admitted to ICU for frequent monitoring

  Day 2

  Exam: difficult to arouse needs stimulus, not following commands, non-verbal, Pupils round (4) and sluggish (R>L), right gaze preference, unable to cross midline to left, left facial, left side flaccid upper & lower extremities. GCS 8, NIHSS 28.

  VS then became irregular w/ changes in HR 40's bpm and BP 150-200/60-80 mmHg

  CT of brain: RMCA full territory edema with effacement of R lateral ventricle

Case # 1

- Treatment:
  - Mannitol; HTS
  - Hemicraniectomy

Large MCA strokes cause swelling and mass effect which can further injure the patient or cause death
Case #2

- 38yr AA RH male
- History: HTN
- Awoke @ 0430 am with H/A, moaning, slurred speech and said " I have a H/A" to wife. EMS notified, pt. intubated.
- CT of brain: SAH (HH4/mF3) with diffuse cerebral edema Basilar tip aneurysm successfully coiled, Acom unsecured
- EVD placed with ICP>30mmHg
- Licox placement PBtO2 <20
- Follow up cerebral angiogram:
  - No signs of vasospasm

Case #2

- ICP elevation
  - Sit upright
  - Hypertonic boluses as needed
  - EVD set at 0 mmHg, draining slowly, good ICP waveform
  - Maintain SBP for MAP>75 mmHg, goal CI>2.5, goal PbtO2>15 mmHg
  - Seizure prophylaxis
- Despite interventions: ICP > 30(+) mmHg
  - Cooling protocol initiated

Temperature and Neuronal Injury

FEVER
Accelerates Injury

Temperature Management
Preserves Tissue

Normothermia vs. Hypothermia
Goal: Neuroprotection

- Decreases cerebral metabolic demand
- Inhibition of neurotransmitter release
- Inhibition of ischemic induced free radical production
- Suppression of ischemic depolarizations
- Preservation of BBB integrity
- Suppression of inflammatory cells and factors

Thank You for your Attention

Sonya Mertz MSN, FNP-C,RN, CNRN
mertzs@ccf.org