Intracranial Pressure
Monitoring

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Intracranial Pressure

- Normal ICP is less than 10 to 15 mmHg.
- Intracranial hypertension
  - is defined as ICP greater than 20 mmHg
- Sustained intracranial hypertension
  - is defined as an ICP greater than 20 mmHg that persists for 5 minutes or longer

Intracranial hypertension

- Permanent neurological damage and disability
- Assessment and early aggressive resuscitation of critically ill pts. May prolong life.

Normal Values:
- CSF Clear, colorless, odorless
- ICP <20 mmHg
- CPP 60-100 mm Hg in Adult
  - >60 mmHg in Child (>1 year)
  - >50 mmHg in Infant (0-12 months)
- CPP = MAP - ICP
- MAP = (2 Diastolic + Systolic) ÷ 3

Monro-Kellie Doctrine

- Skull is a rigid compartment that contain 3 component
  - Brain tissue
  - Arterial and Venous blood
  - Cerebrospinal fluid (CSF)
- Balance in a state of Dynamic Equilibrium
Pathophysiology

- Decrease cerebral perfusion and blood flow.
- Sustained ICP elevations can also lead to herniation syndromes.
- The brain requires 50 to 55 mL of blood per 100 g of brain tissue to maintain a normal metabolic state.
- Adequate CPP is vital for supporting cerebral perfusion.

Many clinicians advocate that CPP in adults should be maintained at no lower than 70 mmHg.
CPP of 60 to 70 mm Hg is adequate after traumatic brain injury. (Robertson)

Indication for ICP Monitoring

- In Severe Head injury GCS less than 8
  - Abnormal CT Brain (Diffuse Gr.II-IV)
  - Normal CT Brain (Diffuse Gr. I)
    - Plus Two or more than Adverse Feature
      - Age more than 40 Yrs.
      - Systemic Blood Pressure less than 90 MMhg
      - Decerebrate / Decorticate Posturing

Assessment Findings

Early Findings:
- Decreased level of consciousness
- Decreased mental status
- Confusion
- Lethargy
- Findings on eye examination
- Abnormal findings on eye examination
- Decreased or abolished auditory responses
- Pupillary response: needling of optic disc, because it causes compression
- Decreased or abolished corneal reflex
- Presence of other abnormal findings
- Seizure activity
- Severe changes

Late Findings:
- Continued decrease in level of consciousness
- Stupor
- Comatose state
- Headache
- Hemiplegia
- Decerebrate (unilateral) posture
- Decorticate (unilateral) posture
- Ventilation (apneic, oronasal respirations)
- Wasting (without resuscitation)
- Brain death

Decrease or loss of protective reflexes: cough, gag, corneal reflexes
Changes in mental state, heart rate, hypertension (initial)
Respiratory changes (rate, depth, pattern)
Chalky-Statius respirations, silent respirations.

Terminal Findings:
- Unilateral blood pressure, heart rate
- Profound hypotension

Types of Diffuse Injury

Diarhetic Injury
Diffuse Injury I
Diffuse Injury II
Diffuse Injury III
Diffuse Injury IV

<table>
<thead>
<tr>
<th>Diagnose Criteria</th>
<th>Diffuse Injury I</th>
<th>Diffuse Injury II</th>
<th>Diffuse Injury III</th>
<th>Diffuse Injury IV</th>
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<tbody>
<tr>
<td>Headache</td>
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<td>Age &lt; 40 yrs.</td>
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<td>yes</td>
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<td>Coma</td>
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<td>yes</td>
<td>yes</td>
<td>yes</td>
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<tr>
<td>Pupillary response</td>
<td>yes</td>
<td>yes</td>
<td>yes</td>
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<td>Hypotension (initial)</td>
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<tr>
<td>Hypotension (final)</td>
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<tr>
<td>Systemic Blood Pressure</td>
<td>yes</td>
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</table>
Cingulate/subcallosal herniation
Detected by using neuroimaging such as head computed tomography or magnetic resonance imaging. Often an indication of brain decompensation and poor intracranial compliance. This herniation syndrome does not have a specific set of assessment findings but may lead to further injury related to localized compromise in blood flow and tissue distortion.

Central herniation
Early findings
- Pupils constricted and reactive to light
- Limitations on upward gaze and eye movements
- Confusion, progressive decrease in level of consciousness
- Weakness followed by paralysis contralateral to lesion
- Babinski reflex (An object such as a key is used to stroke the lateral aspect of the sole of the foot. Dorsiflexion or "turning" of the toes in response is a Babinski reflex and generally indicates a lesion in the central nervous system.)
- Decreased responses to stimulation (tactile, auditory)
- Alterations in respiratory pattern; Cheyne-Stokes respirations

Late findings
- Pupils areflexic and nonreactive progressing to pupils fixed and dilated
- Dysconjugate gaze progressing to eyes fixed
- Coma state
- Decerebrate posturing progressing to decerebration progressing to flaccidity
- Variations in body temperature (hyperthermia)
- Changes in vital signs
- Unstable vital signs progressing to cardiopulmonary arrest
- Potentially, diabetes insipidus

Uncal herniation
Early findings
- Unilateral dilated, slow-reactive pupils
- Weakness of extraocular movements
- Restlessness progressing to confusion
- Slight weakness in an affected side
- Decreased ability to respond to stimulation
- No Babinski reflex

Late findings
- Unilateral fixed, dilated pupils progressing to bilateral fixed or dilated pupils
- Paralysis of eye movements
- Rapid decrease in level of consciousness progressing to coma
- Decerebrate posturing progressing to decerebration progressing to flaccidity
- Babinski reflex progressing to flaccidity
- Altered respiratory patterns progressing to apnea
- Changes in vital signs progressing to cardiopulmonary arrest
Significant of ICP Pulse Waveform

- Consist of 3 components
  - Percussion Wave, P-1
    - Reflects pulsations of the choroid plexus as transmitted from the cardiovascular system at systole
    - Highest
  - Tidal Wave, P-2
    - Often elevated in response to a rapidly expanding mass lesion
  - Dicrotic Wave, P-3
    - Lowest

Intracranial compliance

- Ability of the brain to tolerate stimulation
- ICP that returns to normal or baseline values quickly (within 5 mins.)

Decrease of intracranial compliance

How to known???

- Poor compliance
  1. Fluid filled technique
  2. ICP take time about 20-25 mins and then return to normal
  3. ICP pulse waveform

Type of ICP Monitoring

1. fluid filled catheter-transducer system (external transducer) – Gold standard
2. catheter tip transducer system (internal transducer)
ICP monitoring techniques

How to measure?

General
- Adequate oxygenation and prevention of hypoxemia
- Head elevation
- Prevention of venous obstruction, coughing and straining
- Maintaining CPP around 70 mmHg
- Institution of invasive monitoring and goal-directed therapy

Emergency measurement

Specific
- Mannitol and other diuretics
- Sedation
- Cerebroprotective measures
- Hypothermia and prevention of pyrexia
- Steroids
- CSF drainage
- Seizure control

Treatment for Intracranial Hypertension

- General measures for reducing ICP
- Definitive treatment → Removal of the cause

Management for Intracranial Hypertension

- Maintaining adequate arterial oxygen tension and ensuring normal vascular volume and normal osmosis
- Pyrexia
- Seizures
- CSF drainage
- Head elevation
- Analgesic and sedation
- Neuromuscular blockade
- Diuretics
- Hyperventilation
Second line

- Barbiturate coma.
- Optimized hyperventilation: more aggressive hyperventilation, with adequate oxygen supply.
- Hypothermia: cooling to 35°C. There also appears to be a significant rebound in ICP when induced hypothermia is reversed.
- Decompressive craniectomy