INTRACRANIAL PRESSURE -

Significance

- raised ICP main cause of death in severe head injury
- main cause of morbidity in moderate and mild head injury
- main target and prognostic indicator in the ITU setting

Definition

- normal ICP - 0 - 10 mm Hg or 0 - 13.3 mm H_20 in adult in recumbent posture
- physiological rise in coughing, straining, head down positioning

- sustained mean of 20 mm Hg increased
- 20 - 30 mm Hg - moderately increased
- > 30 mm Hg severely increased

Brain Swelling	Space - occupying lesion	Increased CSF volume
increase in blood volume 1. Arteriolar dilatation • hypoxia • hypercapnia • hypertension • loss of vasomotor tone (cascular injury) • seizures • hyperpyrexia • pain • anaesthetic agents	1.Intracranial haematoma • Extradural • subdural • subarachnoid • intracerbral	1. Hydrocephalus 2. midline shift causing blocking of drainage 3. Lateral horn entrapment of lateral ventricle
2. Venous dilatation • posture (head low) • valsalva (cough , strain) • circumferential neck constriction • venous obstruction (brain swelling / shift)	2. Cerebral contusion	
Brain Oedema 1. cytotoxic oedema • evolving primary injury • ischaemia 2. Osmotic Oedema • hyponatraemia 3. Vasogenic oedema • oedema in peripheral zone of contusion		

Pathophysiology of increased ICP in acute head injury

Monro - kelly doctrine

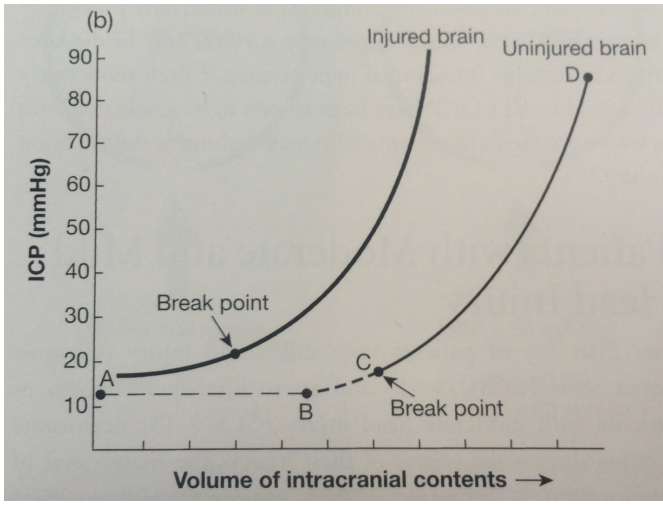
- is a pressure - volume relationship between ICP, volume of CSF, blood, brain tissue and cerebral perfusion pressure
- the hypothesis states that ‘ the cranial compartment is incompressible, and the volume inside the cranium is a fixed volume. Any increase in volume of one of the cranial constituents must be compensated by a decrease in volume of another’

CPP = MAP - ICP

v.intracranial = v.brain + v CSF + v blood + v.mass lesion

anything which increases the right hand side of the equation will raise ICP thus ultimately reducing the CPP

- uninjured brain can auto regulate
- manages:-
    - by displacement of CSF
    - displacement of venous blood
    - viscoelastic compression of brain
- following loss of compensation the intracranial pressure rises exponentially



- injured brain - auto regulation fails
    - rapidity of ICP rise goes up exceeding speed of compensation mechanisms
    - compliance of brain to autoregulate goes down

In other words an injured brain has reduced capacity to autoregulate

Incidence of Increased ICP

- **severe ICP**
    - GCS < 8
    - 50% pts
    - increased if abnormal CT
    - more likely if 2 or more of
        - age > 40
        - systolic < 90
        - motor posturing

    - 10 -15 % will be medically or surgically refractory to ICP management
    - 84 -100 % of these will die

- **Mild head injury**
    - GCS 14 -15
    - 3% raised ICP

- **Moderate head injury**
    - GCS 9 - 13
    - 10 -20 % raised ICP

- **Post evacuation of Intracranial mass lesions**
    - 66 % of post evacuation pts will show raised ICP
    - a third of these ICP will be refractory
    - predictable by CT
    - may be delayed up to 48 hrs

Consequences of raised ICP

- effects are caused by reduced cerebral perfusion and cerebral ishaemia
- BUT also brain shifts with herniation and brain stem compression
- CPP can autoregulate between 50 -150 mm Hg
- important to get MAP to 90 mm Hg

1. intracranial pressure level < 20 mm Hg
2. CPP > 60 mm Hg
3. MAP above 90 mmHG
4. do NOT increase MAP to increase CPP

Brain shifts

1. Midline shift

- laterally based supratentorial lesion
- compression and displacement of lateral and third ventricles
- compression of thalamus and hypothalamus
- ischaemia - stretch deep arteries of brain
- occluding third ventricle and dilating opposite ventricle

2. Subfalcine herniation

3. Transtentorial herniation

- herniation of medial temporal lobe
- compression of IPSI lateral Oculomotor IPSI lateral blown pupil
- further deterioration of level of consciousness
- increasing contralateral hemiparesis
- compression of posterior cerebral artery - occipital infarct

Cushing reflex

Triad of signs indicating impending brain stem failure

1. Increase in systemic blood pressure
2. Bradycardia
3. irregular respiratory effort

Indications for ICP monitoring	Risk of raised ICP
Severe Head Injury (GCS 3-8)	
• Abnormal CT scan	50-60%
• Normal CT Scan Age > 40 or BP < 90mmHg or abnormal motor posturing	50-60%
• Normal CT scan No risk factors	13%
Moderate Head Injury (GCS 9-12)	
• If anaesthetised/sedated • Abnormal CT scan	approx. 10-20% will deteriorate to severe head injury
Mild Head Injury (GCS 13-15)	
• few indications for ICP measurement	Only around 3% will deteriorate

Why monitor ?

1. it determines management
2. CT and clinical assessment is unreliable and delayed
3. CT normal patients may still have raised ICP
4. assess response to treatment
5. to indicate need for CT

Indications for ICP monitoring

1. Patients with GCS < 8 after CPR with an abnormal scan
    - haematoma
    - contusion
    - oedema
    - compressed cisterns

2. Patients GCS < 8 with :-
    - age > 40
    - SBP < 90 mmHg
    - unilateral or bilateral posturing

3. After evacuation of haematoma

4. Patients with moderate or mild head injury requiring prolonged sedation

Contraindications for ICP monitoring

- Coagulopathy
- Extensive scalp wounds

Interpreting ICP data

This records ICP, CPP and shows waves.

1. Threshold for therapy
    - sustained ncrease in ICP of > 20 mm Hg for more than 5 mins in absence of extraneous stimuli
    - > 40 mm Hg indicates real trouble
    - consider with CT

2. Correlate ICP with clinical findings

3. Trends of ICP and CPP

4. Intracranial compliance

- watch for appropriate rises in ICP to suctioning, moving, etc
- no rise in ICP to stimuli is a bad thing

ALSO do not rely on findings, machines go wrong, catheter tips block

Intra cranial pressure waves - three types :-

Type A -

- rise to up to 50 mm Hg lasting 5 - 20 mins
- fall abruptly
- caused by failure of intracranial compliance
- caused by cerebral vasodilation in response to reduced CPP

Type B -

- rhythmic rises to 10 -20 mm Hg
- fall abruptly
- 0.5 - 2 waves per min
- respiratory excursion led
- more frequent in reduced compliance
- not as significant as A waves

Type C -

- small rhythmic oscillations of 20 mm Hg
- 4 -8 per min
- prob low significance