

INCREASED INTRACRANIAL PRESSURE

***WALID S. MAANI
PROFESSOR OF NEUROSURGERY
JORDAN UNIVERSITY AND MEDICAL SCHOOL***

PHYSIOLOGICAL PRINCIPLES

- *The skull is a rigid structure. It contains:*
 - *Brain*
 - *Blood*
 - *Cerebro spinal fluid (CSF)*
- *Any increase of these contents and/or the addition of any mass will lead to increase in the pressure, unless a similar volume is removed (the Monroe-Kellie doctrine).*

PHYSIOLOGY

- *Brain 1300-1750 mls*
 - *Tissue 300-400 mls.*
 - *Intra-cellular fluid 900-1000 mls.*
 - *Extra-cellular fluid 100-150 mls*
- *Blood 100-150 mls.*
- *CSF 100-150 mls.*

PHYSIOLOGY

- **COMPENSATORY MECHANISMS FOR EXPANDING MASSES:**

- *Immediate*

- *Decrease in CSF volume by movement of fluid to the lumbar area.*
- *Decrease in the blood volume by squeezing blood out of sinuses*

- *Delayed*

- *Decrease in the extra-cellular fluid.*

PHYSIOLOGY

- **CEREBROSPINAL FLUID**

- *Secreted at the rate of 500 mls per day*
- *Secreted by the choroid plexus in the lateral ventricles*
- *Flows through the ventricular system*
- *Exits to the subarachnoid space through the foramina of Magendie and Luschka*
- *Absorbed into the venous system via the arachnoid granulations*
- *Any obstruction to the flow will lead to **HYDROCEPHALUS** and increased pressure.*

PHYSIOLOGY

● **INTRACRANIAL PRESSURE**

- *Normally 0-140 mm CSF (0-10 mm Hg)*
- *There are normal regular waves due to pulse and respiration*
- *With increased pressure “pressure waves” appear*
- *With continued rise of ICP the PP falls*
- *When PP falls CBF is reduced*
- *Electrical cortical activity fails if CBF is 20ml/100gm/min*
- *When intracranial pressure reaches mean arterial pressure circulation to the brain stops.*

PHYSIOLOGY

● **BRAIN OEDEMA**

- *An excess of brain water may occur:*
 - *Around lesions within the brain:*
 - *Tumor*
 - *Abscess*
 - *In relation to traumatic damage (contusions)*
 - *In relation to ischemic brain insult*
- *Leads to increase in the pressure.*
- *Types of edema:*
 - *Vasogenic (extra cellular): tumors*
 - *Cytotoxic (intra cellular): metabolic states*
 - *Interstitial (extra cellular): increased IVP*

PHYSIOLOGY

- **CEREBRAL PERFUSION PRESSURE (CPP)**

Cerebral Perfusion Pressure (CPP)=

Mean Arterial pressure – intracranial pressure

MAP (120+80) / 2 = 100 mm Hg

ICP = 15 mm Hg

CPP = 100 – 15 = 85 mm Hg (60-120 is good)

PHYSIOLOGY

- **CEREBRAL BLOOD FLOW (CBF)**

$$\text{Flow} = \frac{\text{Pressure}}{\text{Resistance}}$$

$$\text{Cerebral Blood Flow} = \frac{\text{Cerebral Perfusion Pressure} \\ (\text{Mean Art. pressure} - \text{intracranial pressure})}{\text{Cerebral Vascular Resistance}}$$

PHYSIOLOGY

- *Increased ICP is defined as a sustained elevation in pressure above 20mm of Hg*

PHYSIOLOGY

- *During slow increase in volume in a continuous mode, the ICP rises to a plateau level at which the increase level of CSF absorption keeps pace with the increase in volume.*

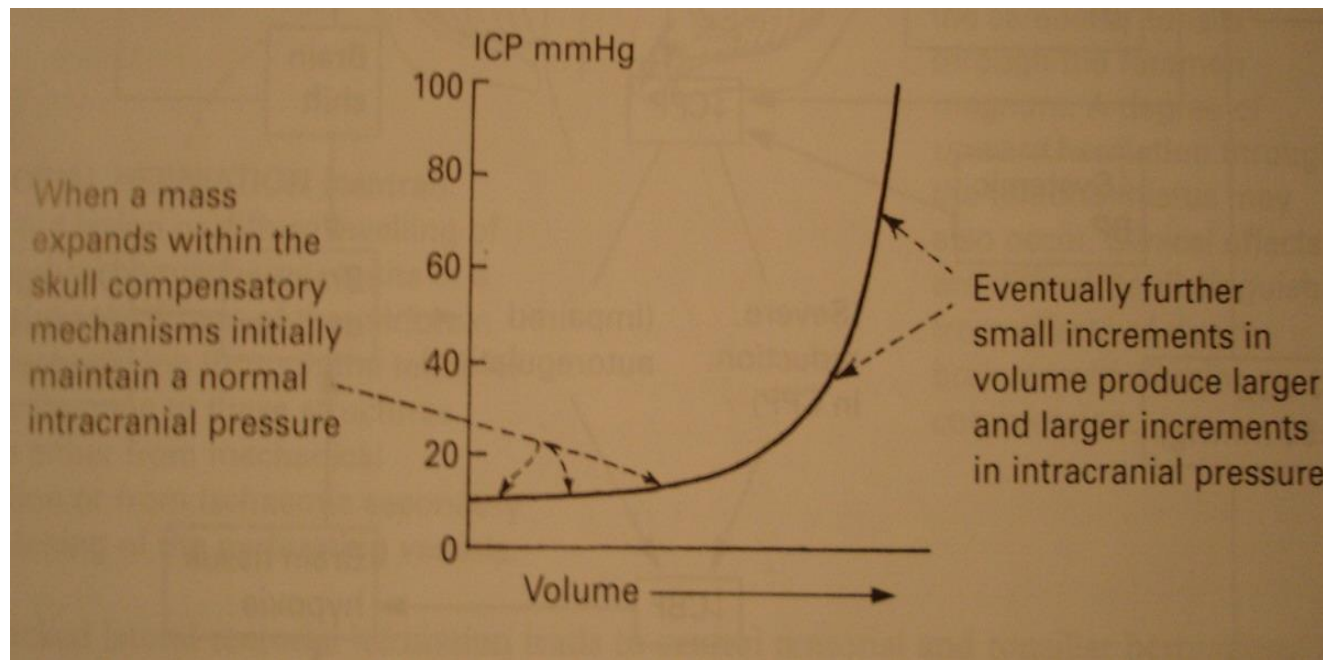
PHYSIOLOGY

- *Intermittent expansion causes only a transient rise in ICP at first. When sufficient CSF has been absorbed to accommodate the volume the ICP returns to normal.*

PHYSIOLOGY

- *Expansion to a critical volume does however cause persistent rise in ICP which thereafter increases logarithmically with increasing volume (volume - pressure relationship).*

THE VOLUME PRESSURE RELATIONSHIP



PHYSIOLOGY

- *The ICP finally rises to the level of arterial pressure which it self begins to increase, accompanied by bradycardia or other disturbances of heart rhythm (Cushing response). This is accompanied by dilatation of small pial arteries and some slowing of venous flow which is followed by pulsatile venous flow.*

PHYSIOLOGY

- **COMPENSATORY MECHANISMS**
 - *Push CSF out to spinal theca > expands*
 - *Compress venous sinuses and push blood out*
- **WHEN COMPENSATORY MECHANISMS FAIL; BRAIN HERNIATION OCCURS**
 - *Push cerebral hemisphere to other side (midline shift and Cingulate (subfalcine herniation))*
 - *Push brain down*
 - *Uncal 'L' and Central 'M' (transtentorial herniation)*
Occulomotor (dilated pupil), pons, PCA, crus cerebri (contra hemiplegia), reticular formation (loss of conc),
 - *Tonsillar (transforaminal herniation)*

CLINICAL PICTURE

- *The rise in ICP disturbs brain function by:*
 - *(1) Reduction in CBF*
 - *(2) Transtentorial or foramen magnum herniation resulting in selective compression and ischaemia in the brain stem.*

CLINICAL PICTURE

- *Early:*
 - *Headache, projectile vomiting and papilloedema.*
- *Late:*
 - *Change in the level of consciousness,*
 - *Loss of motor and sensory functions, pupillary changes)compression of Cranial Nerve III(*
 - *Vital sign changes including widening pulse pressure, bradycardia and irregular respirations,*
 - *Posturing :decorticate)flexion(, decerebrate)extension(or mixed)intermittent(*
 - *Coma*
 - *Changing and irregular respiratory patterns*

CLINICAL PICTURE



PAPILLEDEMA

CLINICAL PICTURE

Herniation of intracerebral contents

- **Supratentorial herniation**

*Uncal: most frequently noted herniation
Results in ipsilateral pupil dilatation, decreased level of consciousness, changes in respiratory patterns, respiratory arrest, and contralateral hemiplegia*

Subfalcine which results in affection of the ACA leading to contralateral leg weakness

- **Central/Transtentorial herniation**

Results in loss of consciousness, small reactive pupils advancing to fixed/dilated pupils, respiratory changes leading to respiratory arrest and decorticate posturing advancing to flaccidity.

- **Infratentorial herniation**

Tonsillar: As a result of a downward herniation the medula oblongata is compressed and displaced causing respiratory and cardiac arrest

MANAGEMENT

- **REDUCE THE INTRACRANIAL PRESSURE**
 - **REMOVE THE CAUSE OF INCREASED PRESSURE**
 - **AND/ OR:**
 - *1- raise bed 30 degrees to increase venous drainage*
 - *Hyperventilation to induce vasoconstriction by keeping the PaCO₂ below 25 mm Hg*
 - *Osmotic diuresis like mannitol*
 - *Barbiturates*
 - *Surgical decompression*
 - *Shunts or drains*
 - **INTRACRANIAL PRESSURE MONITORING IS REQUIRED**

MANAGEMENT

INTRACRANIAL PRESSURE MONITORING



- 1) Subdural***
- 2) Subarachnoid***
- 3) Interparenchymal***
- 4) Intraventricular***