Increased intracranial pressure, hydrocephalus

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Aim of this presentation is, to elucidate

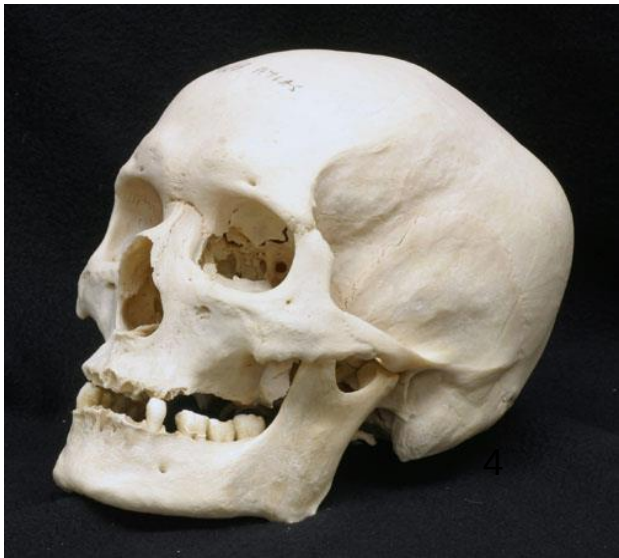
- Definition of ICP: its measurement and determinants
- Hemodynamics of cerebral circulation
- Herniation syndromes
- Diagnostic groups with increased ICP
- Disturbances of CSF circulation (hydrocephalus and treatment modalities)

Intracranial Pressure

- Definition: pressure exerted by intracranial volume of:
  - Brain
  - Blood
  - CSF
- Normal ICP: 5-15 mm Hg.
- Increased ICP: >20 mm Hg.

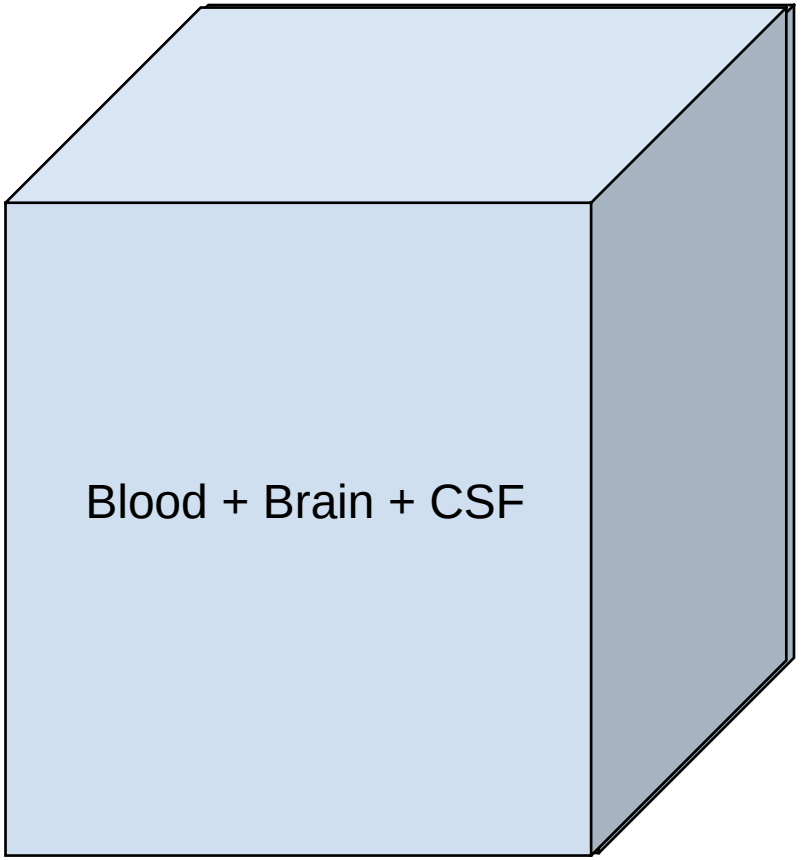
"Monro-Kellie doctrine"

- The skull is basically a rigid structure. Since its contents - brain, blood and cerebrospinal fluid (CSF) - are incompressible, an increase in one constituent or an expanding mass within the skull results in an increase in intracranial pressure (ICP).
- Skull is rigid (except in infants where ↑**ICP** causes sutural diastasis)
- Intracranial Contents
  1. Brain (80%)
  2. Blood (10%)
  3. Cerebrospinal Fluid (10%)



Monro-Kellie Hypothesis

- If the volume of one compartment increases, the volume of one or both of the other compartments decreases in order to maintain intracranial balance of volumes
- If the compensatory mechanism of volume-shift becomes exhausted, ICP inherently rises



Monro A (1783). *Observations on the structure and function of the nervous system*. Edinburgh: Creech & Johnson.
Kelly G (1824). "Appearances observed in the dissection of two individuals; death from cold and congestion of the brain". *Trans Med Chir Sci Edinb* **1**: 84–169.

Intracranial compliance

- Ability to accommodate increase in volume without a corresponding increase in pressure
- BUT at critical point: small change in volume = large change in pressure

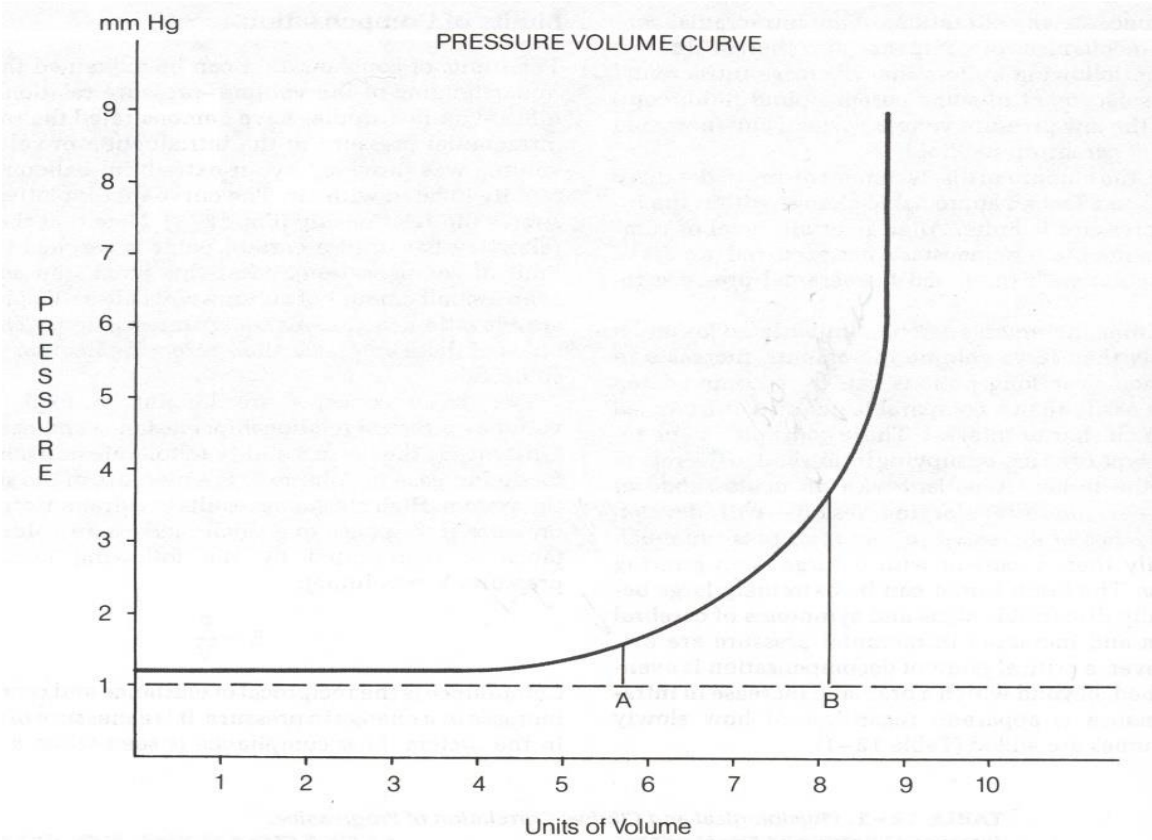
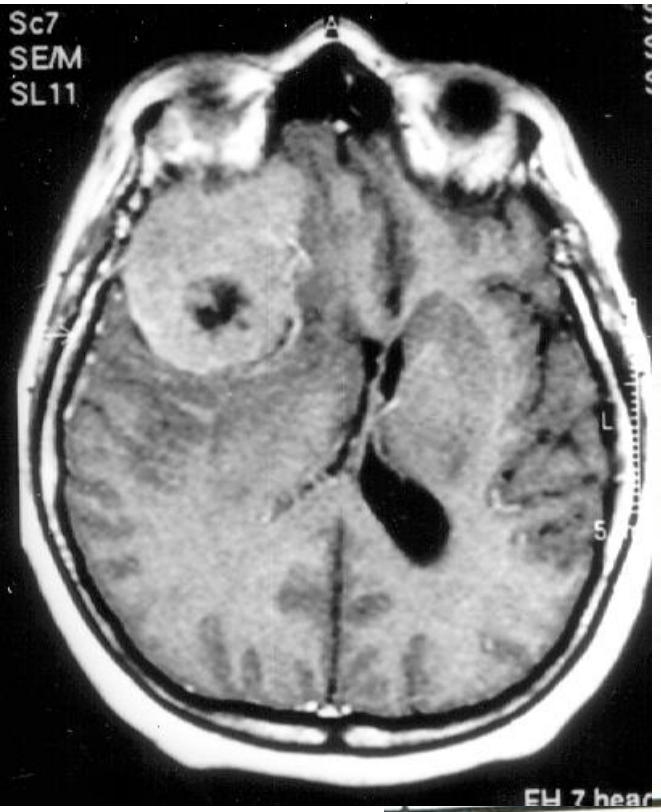
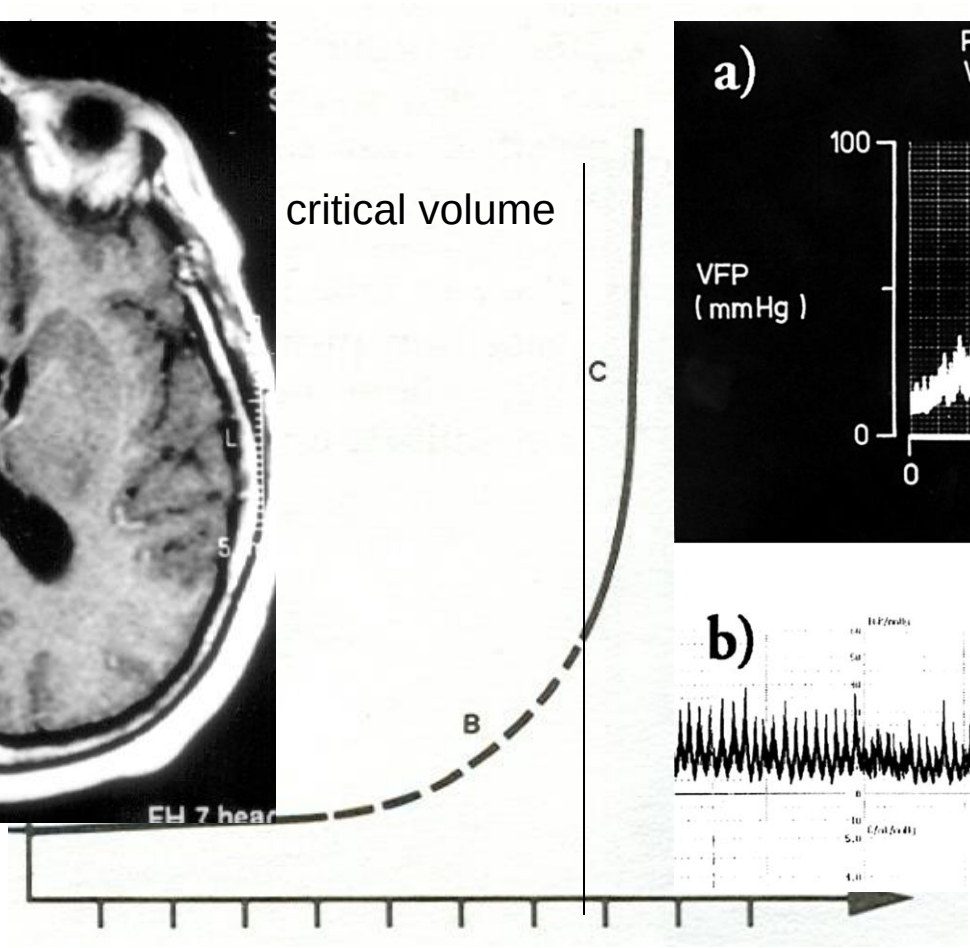
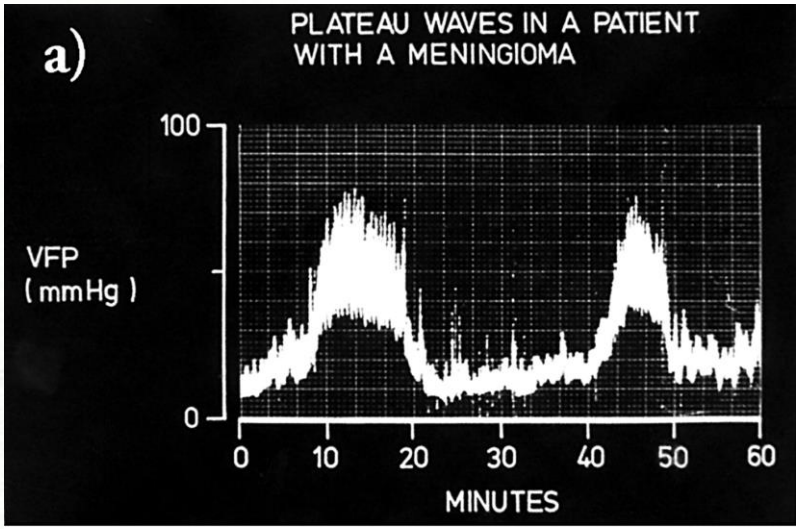


FIG. 12–1. Pressure volume curve. To point "A", addition of volume has little effect on pressure (high compliance); after that point, there is a dramatic increase in response to addition of volume, especially from point "B" onward (low compliance).

Intracranial pressure-volume relationship





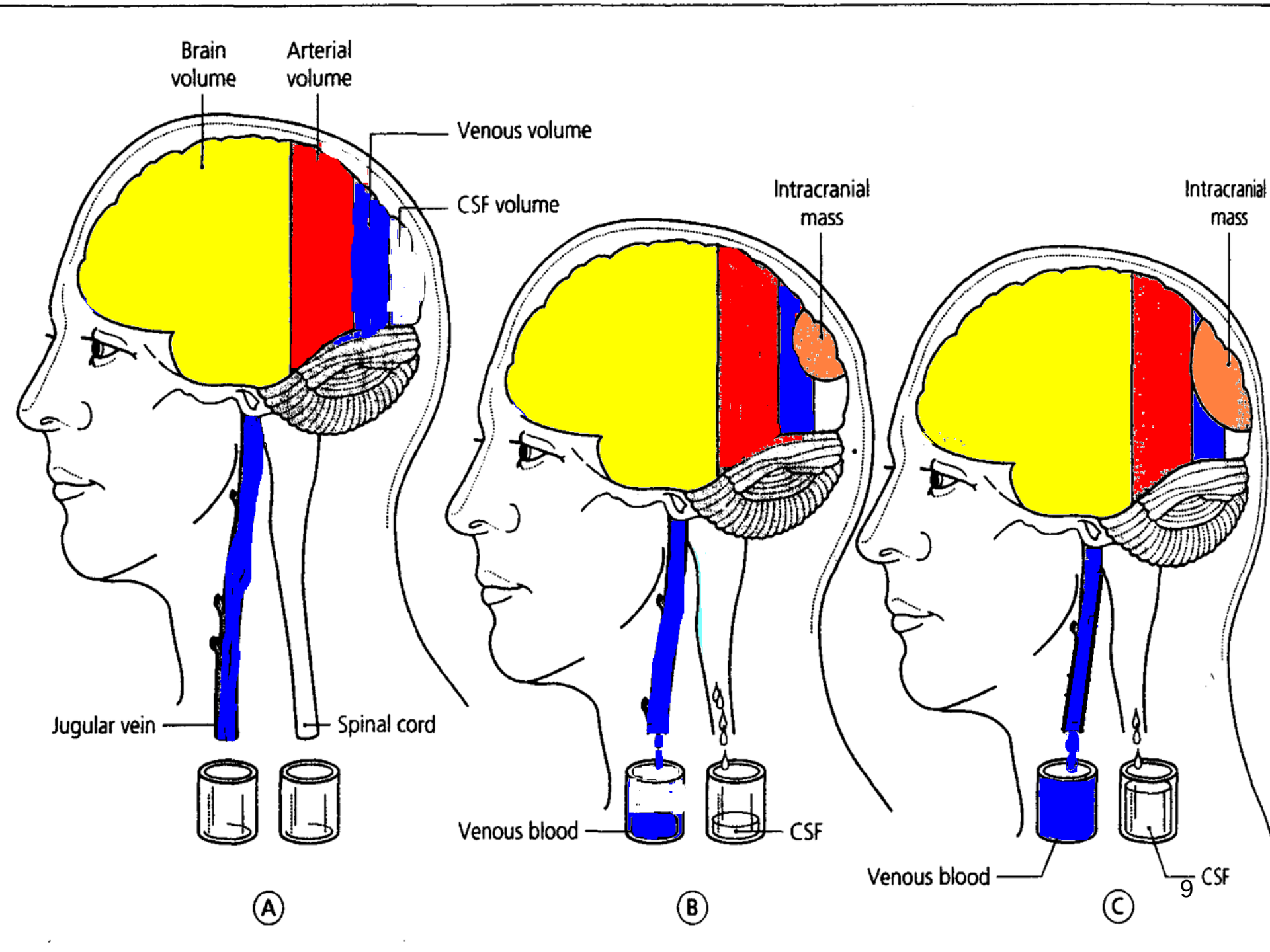


Intracranial volume V

IICP—compensatory mechanisms

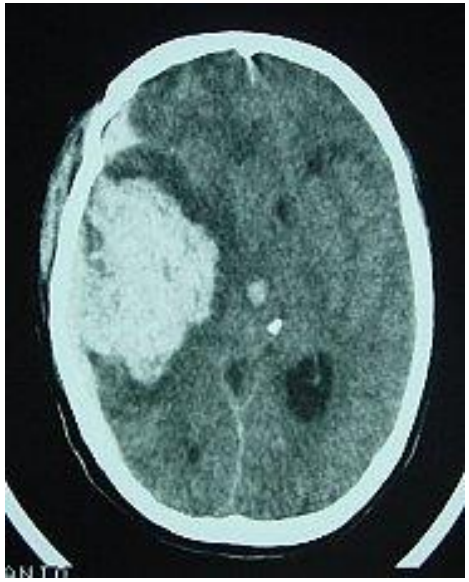
- 1. Blood displaces - (venous system)
- 2. CSF displaces - (spinal reservoir)
- 3. Brain displaces - herniation syndromes.

- *Our aim is to augment compliance before brain displaces.*


Brain volume
Arterial volume
Venous volume
CSF volume
Jugular vein
Spinal cord
A
Intracranial mass
Venous blood
CSF
B
Intracranial mass
Venous blood
CSF
C

IICP: Etiologies

1. Increase in “brain” volume
    i. Space-occupying lesion (eg. Hematomas, tumours, abscesses)
    ii. Cerebral edema (eg. Tumor, stroke)

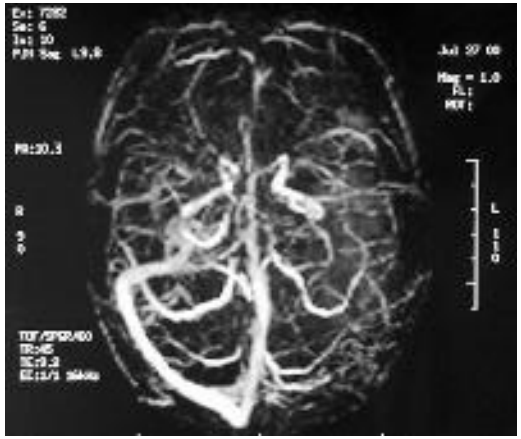


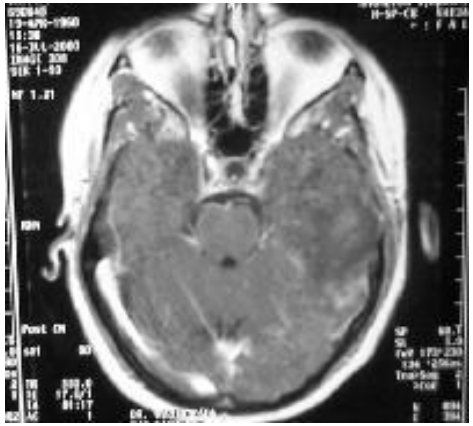
Hematoma

Tumour/ Edema

IICP: Etiologies (space occupying lesions)

2. Increase in blood volume
    i. Obstruction of venous outflow→edema, hemorrhage
    ii. Hyperemia (status epilepticus, post AVM surgery)
    iii. Hypercapnia (↑CO2)
    iv. Hypoxia

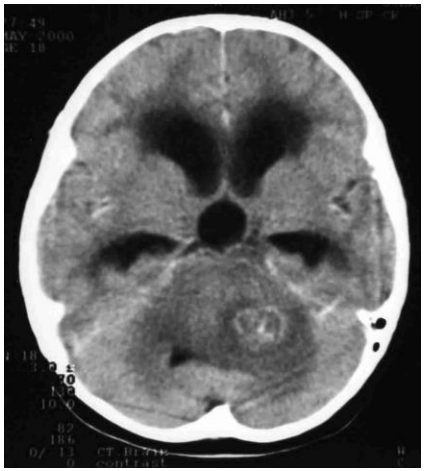




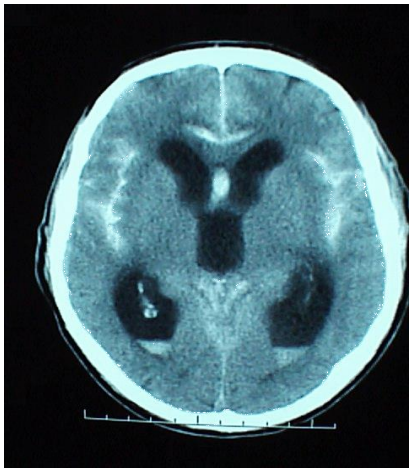
Venous Sinus Thrombosis

IICP: Etiologies (space occupying lesions)

3. Increase in CSF
    i. Increase CSF production (eg. Choroid plexus papilloma)
    ii. Decreased CSF absorption (Communicating hydrocephalus)
    iii. Obstruction of CSF flow (Obstructive hydrocephalus)



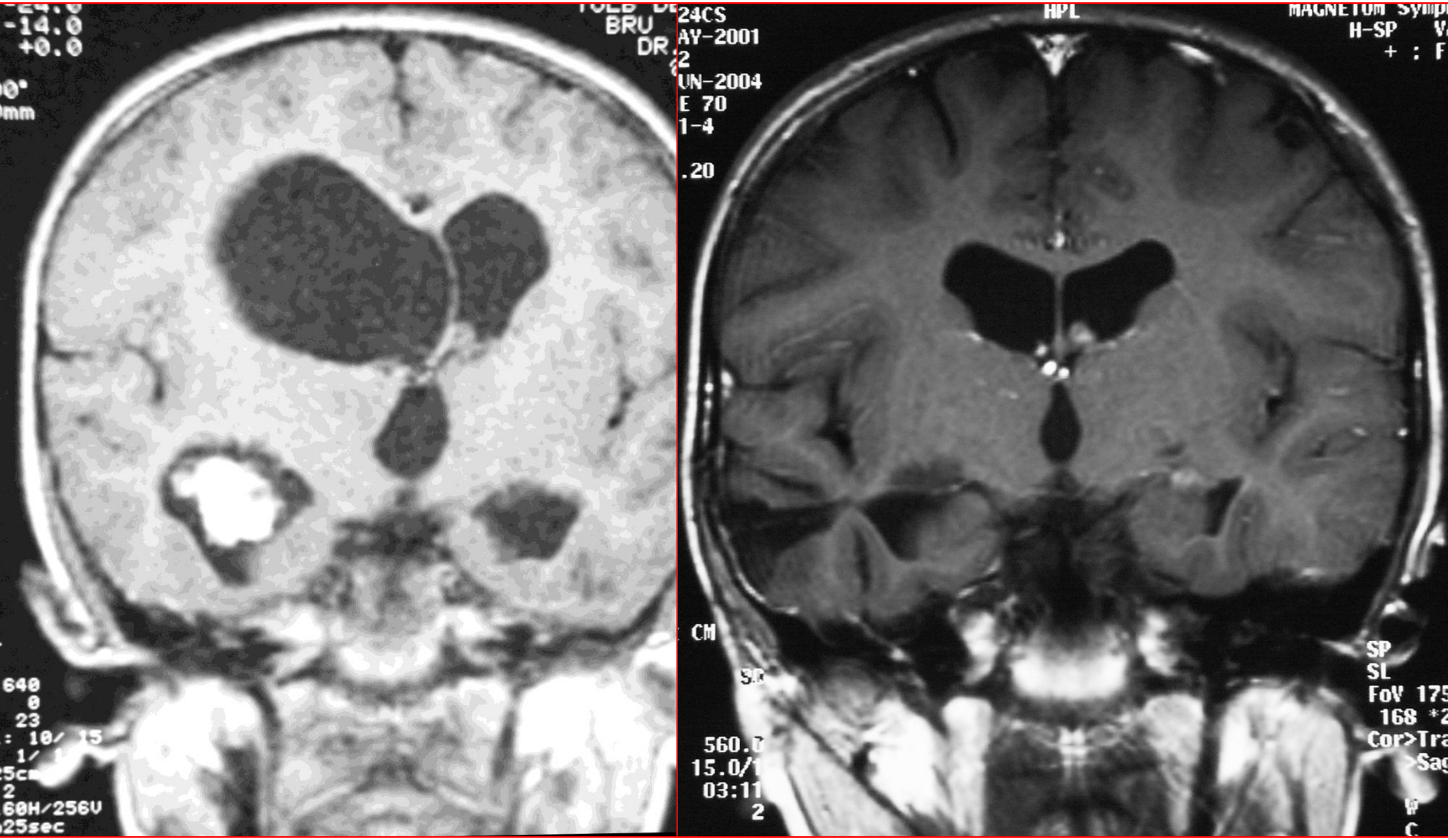
Obstructive

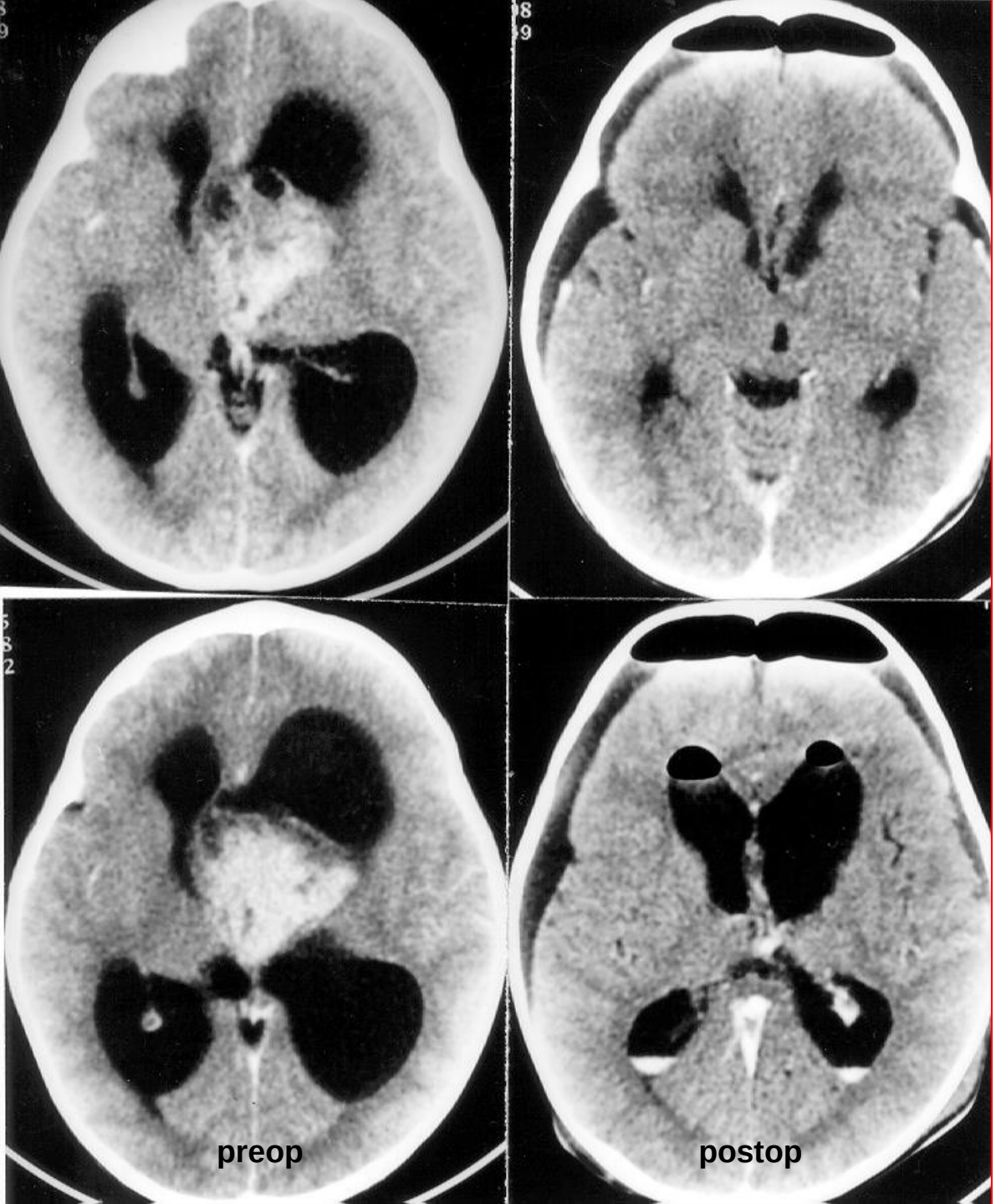


Communicating

1yM Choroid plexus papilloma

3 years after surgery





Intraventricular A1

with blocade of the foramen of Monro

M 32y

- **Brain Water/Edema:**

cerebral oedema = an excess of brain water

Types of brain edema:

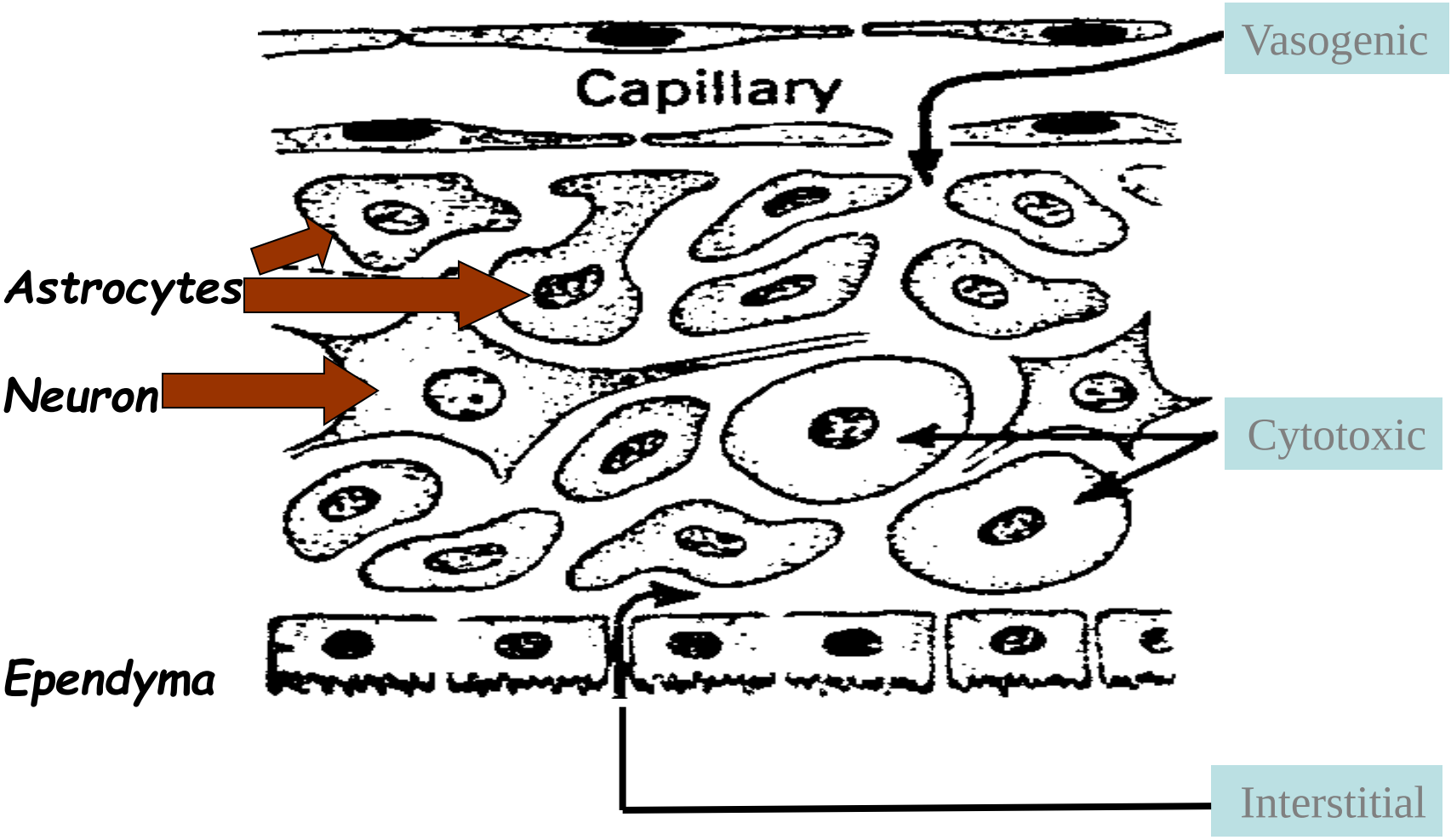
1-*Vasogenic*: *excess fluid (protein rich) passes through damaged vessel walls to the extracellular space - especially in the white matter. The extracellular fluid gradually infiltrates throughout normal brain tissue towards the ventricular CSF and this drainage route may aid clearance. E.g. adjacent to* ***tumor, abscess***

2- Cytotoxic: *fluid accumulates within cells – neurons and glia i.e. intracellular. e.g.* ***toxic or metabolic states.***

3- Interstitial: *when obstructive hydrocephalus develops, CSF is forced through to the extracellular space especially in the periventricular white matter.*

* ***With ischemic damage,*** as cell metabolism fails, intracellular Na^{+} and Ca^{++} increase and the cells swell i.e. ***cytotoxic edema.*** *Capillary damage follows &****vasogenic edema***

Types of brain edema:



Measuring ICP

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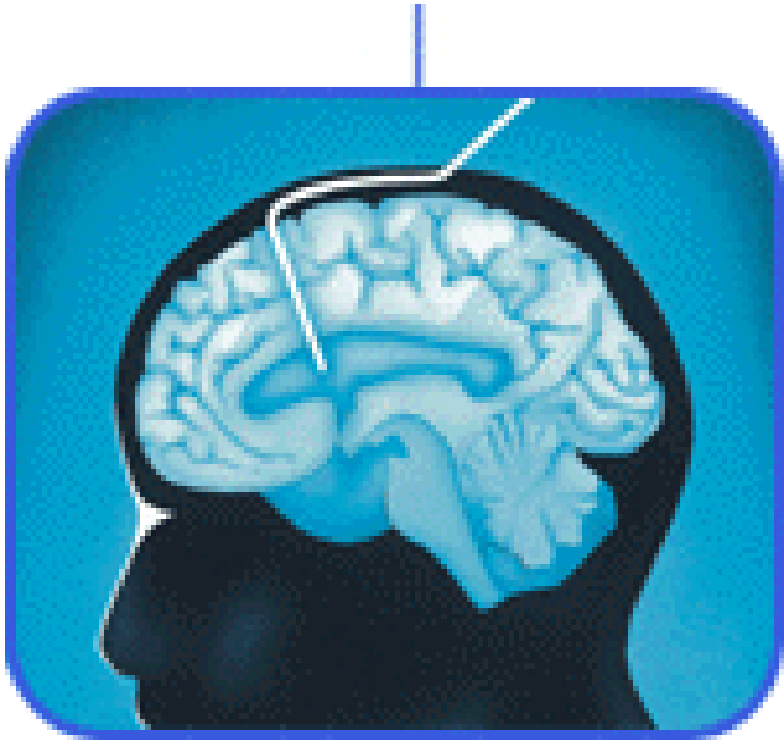
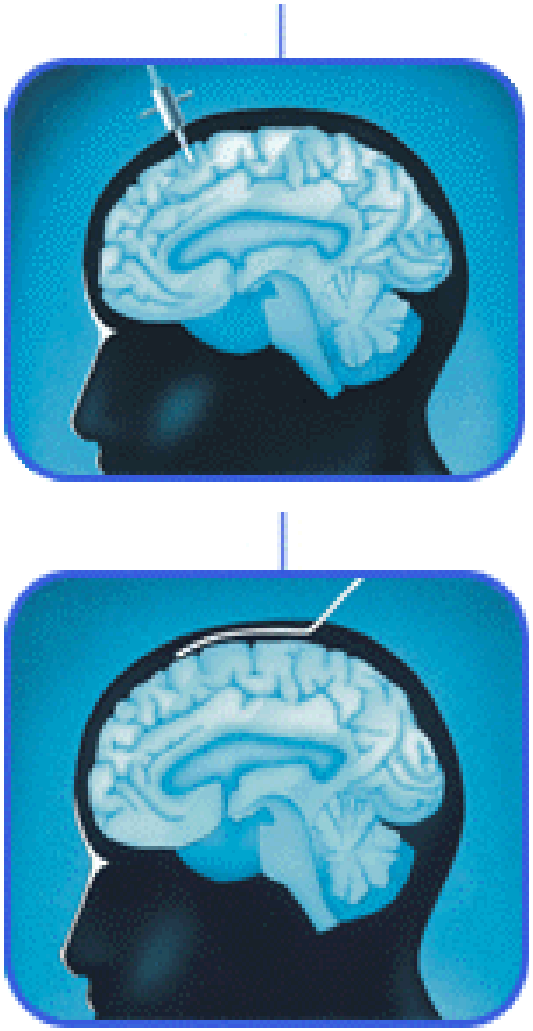
Measuring ICP 1

1. Ventricular catheter (EVD)
   - accurate readings
   - therapeutic option to drain CSF
   - can measure compliance
   - *need external transducer, frequent leveling



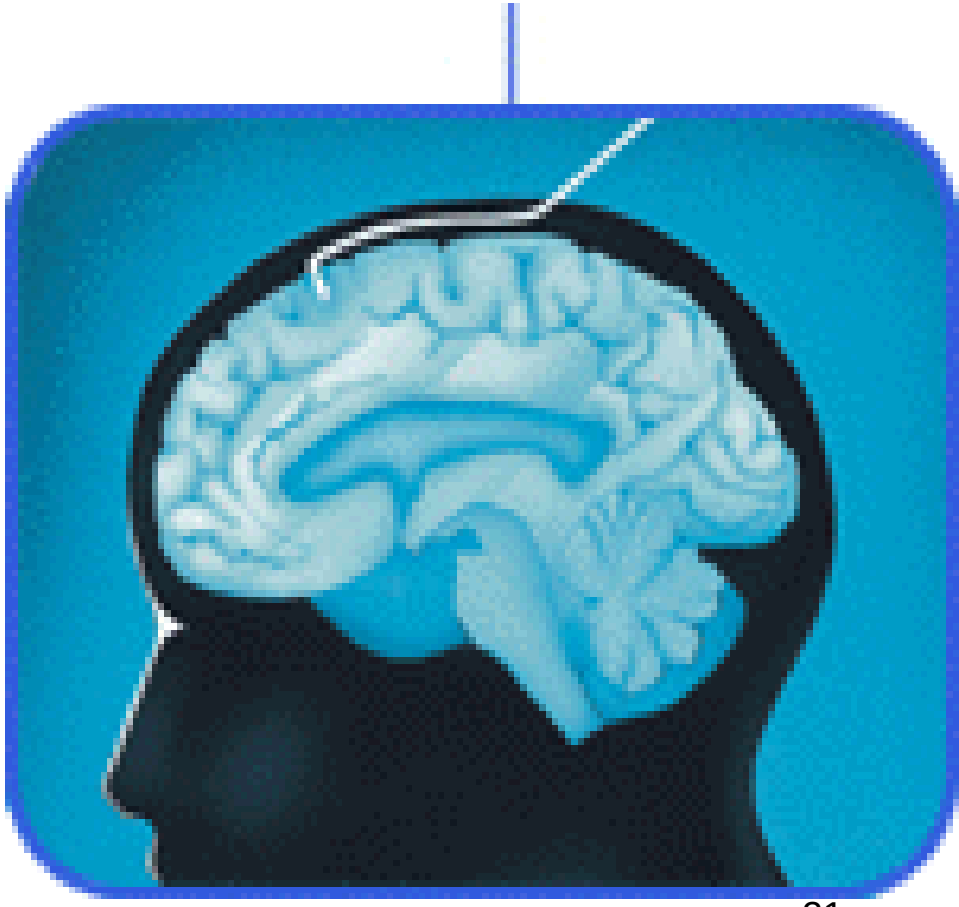
Measuring ICP 2



2. Epidural, subdural or subarachnoid catheter or bolt
   - Can place if ventricles collapsed, no penetration of brain required
   - *Tend to dampen, fail to transduce
   - *No access to drain CSF

Measuring ICP 3

3. Parenchymal catheter
   - Transducer on fibreoptic catheter
   - eliminates need to level a transducer
   - *Unable to drain CSF, need to penetrate brain
   - *concern re “drift”: cannot rezero while in situ



ICP monitoring in the ICU

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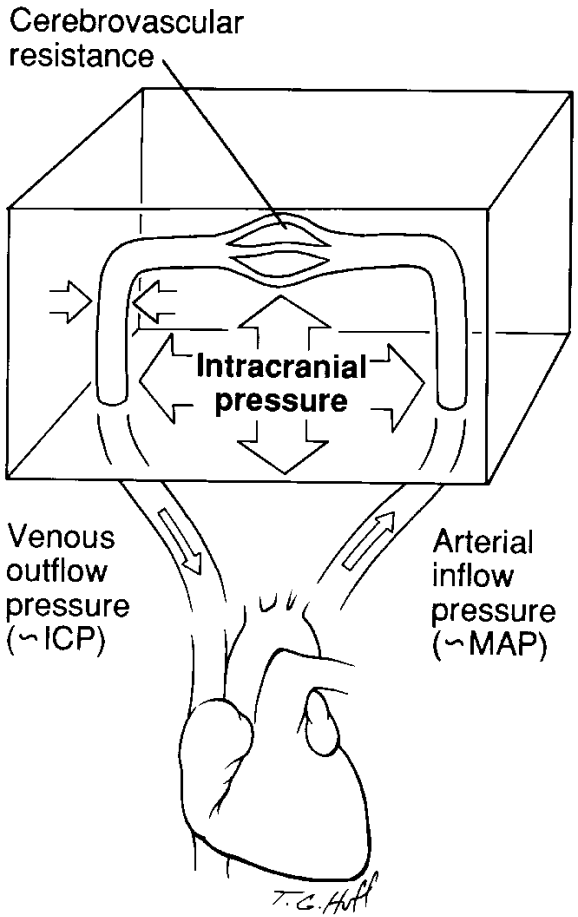
Cerebral Blood Flow (CBF)



CPP = Arterial inflow – Venous inflow

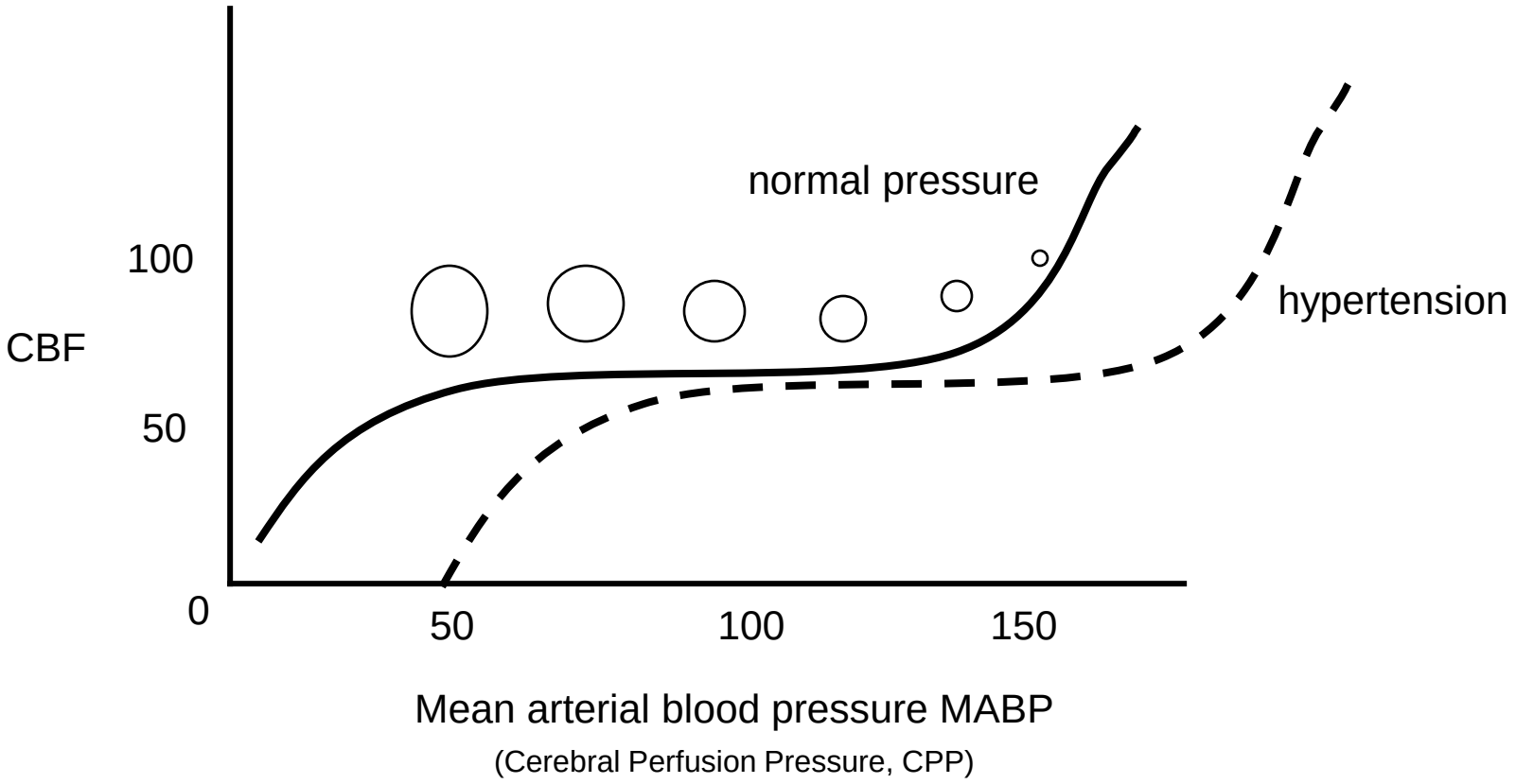
= MAP - ICP

CBF * CVR = MAP - ICP

CBF = (MAP – ICP) / CVR

- **normal = 40-60 ml/100gm/min**
- **Symptoms = 20-30 ml/100gm/min**
- **EEG = 16-20 ml/100gm/min**
- **Na-K = 10-12 ml/100gm/min**
- **Complete = <10 ml/100gm/min**

Regulation of CBF 1
Autoregulation



Regulation of cerebral blood flow 2 Chemical regulation

- CO_2 is the most effective determinant of CBF ($PaCO_2$)
- O_2 PaO_2 (<60mmHg) results in vasodilation, PaO_2 (>400mmHg) causes vasoconstriction
- pH ↓ (acidosis) elicits vasodilation, pH(alkalosis) elicits vasoconstriction

Signs & Symptoms of ↑ICP

SYMPTOMS

- Headache
- Nausea
- Vomiting (morning)
- Blurred vision
- Diplopia

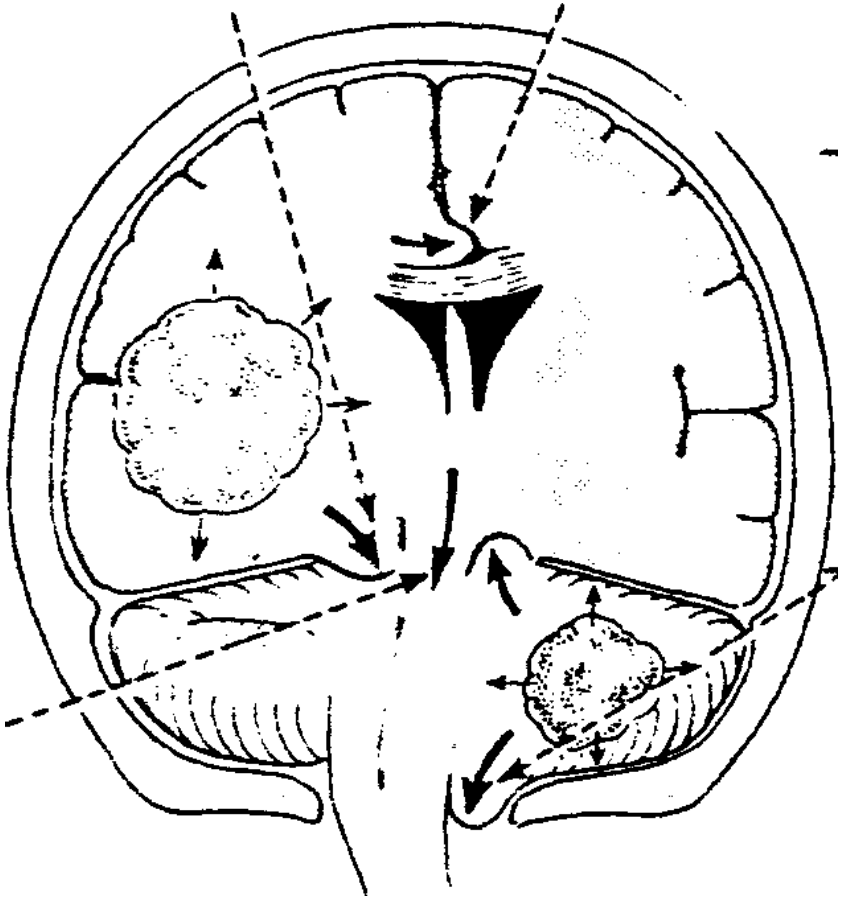
SIGNS

- Progressive focal deficit
- Deterioration in LOC
- VI Nerve palsy
- Papilledema
- Pupillary changes-size, reactivity
- Abnormal posturing
- Changes in vital signs
  - Bradycardia
  - Arterial HTN

Types of Brain Shift

Tentorial herniation (lateral).

Subfalcine herniation



Tonsillar herniation.

Tentorial herniation (central).

NB: Unchecked lateral tentorial herniation leads to central tentorial and tonsillar herniation, associated with progressive brain stem dysfunction from midbrain to medulla.

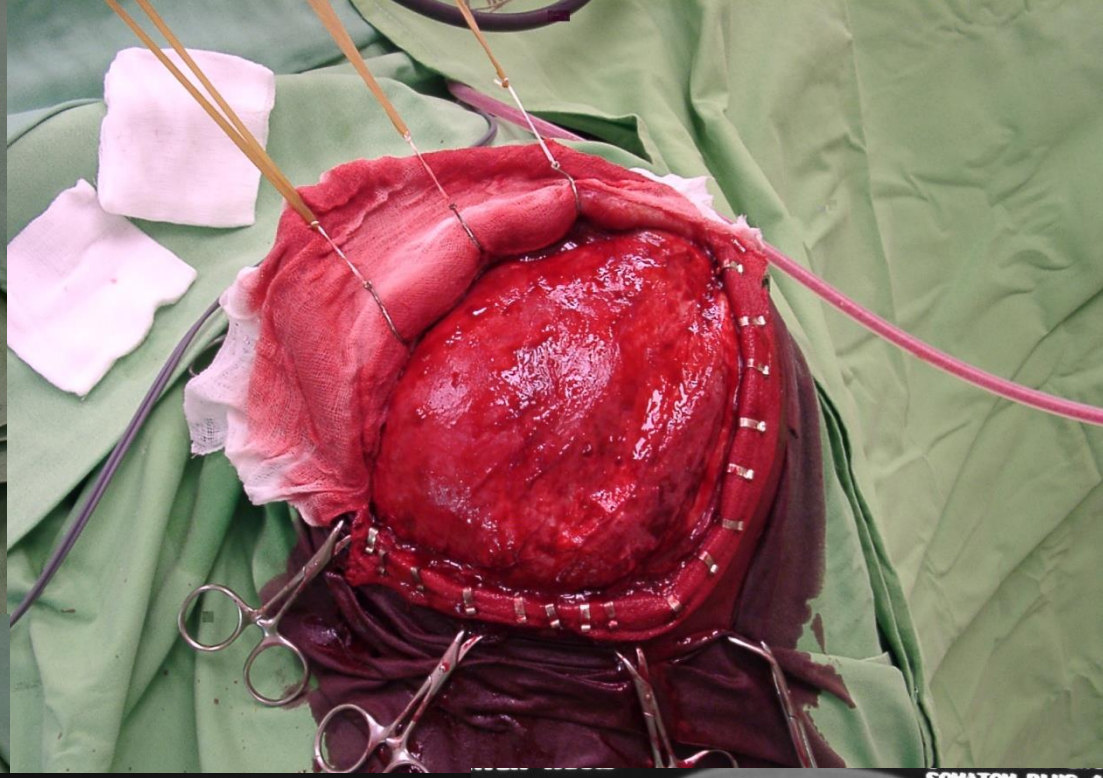
Management of ↑ ICP (brain)

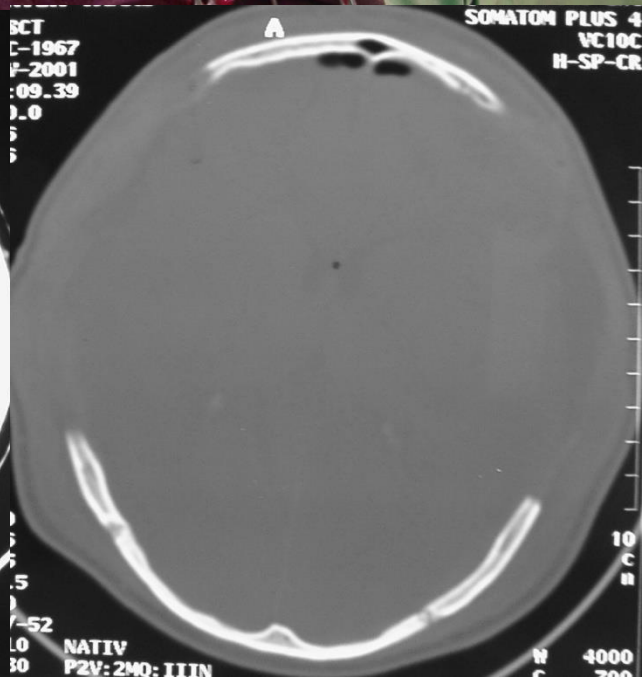
- Decrease edema
  - Mannitol (0.5-1G/kg), hypertonic saline
    - Monitor electrolytes, osmolality (aim <320)
    - Aware of “rebound”
  - Dexamethasone (tumors, abscess, NOT stroke)
  - Furosemide (Lasix)
- Surgical decompression/evacuation
- Decompresssive craniectomy
- CSF drainage procedure

Decompressive craniectomy

- **Cushing H**: The establishment of cerebral hernia as a decompressive measure for inaccessible brain tumors: with the description of intermuscular methods of making the bone defect in temporal and occipital regions.

  Surg Gynecol Obstet 1:297314, **1905**

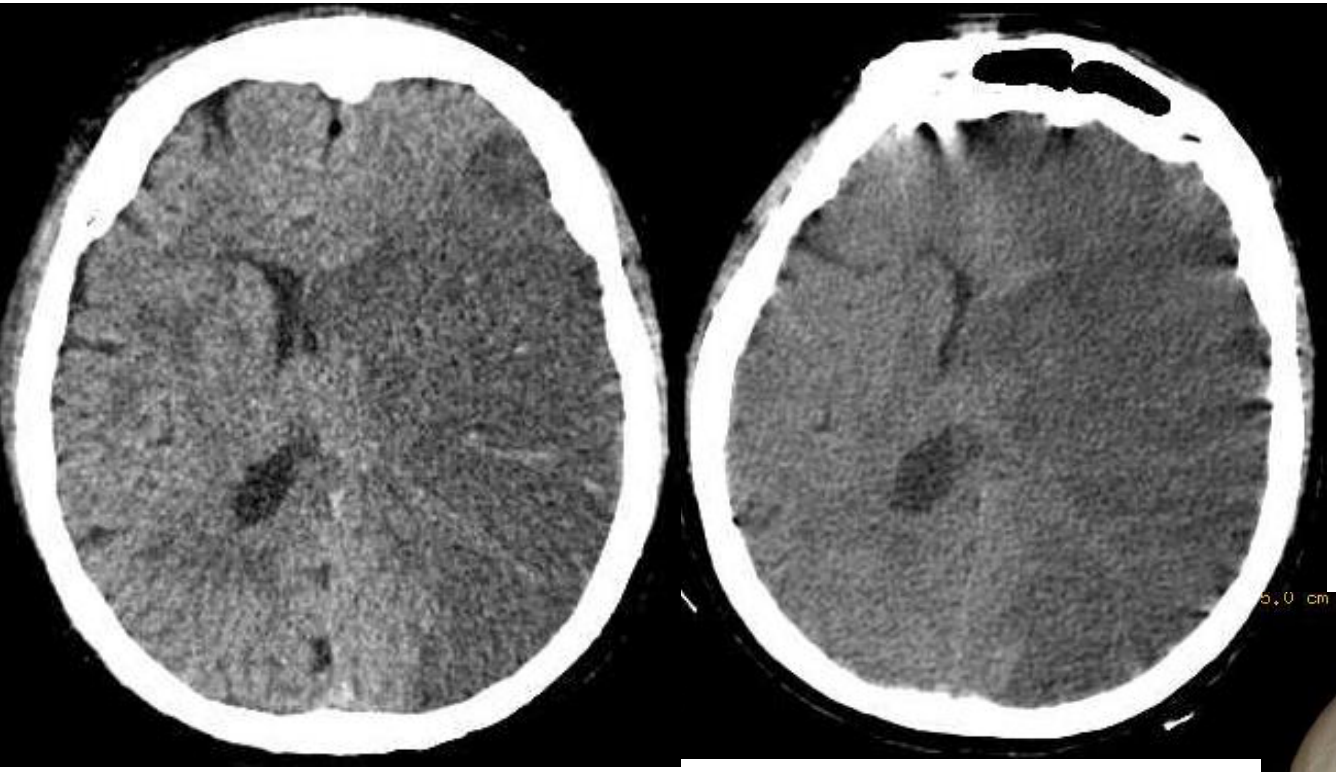




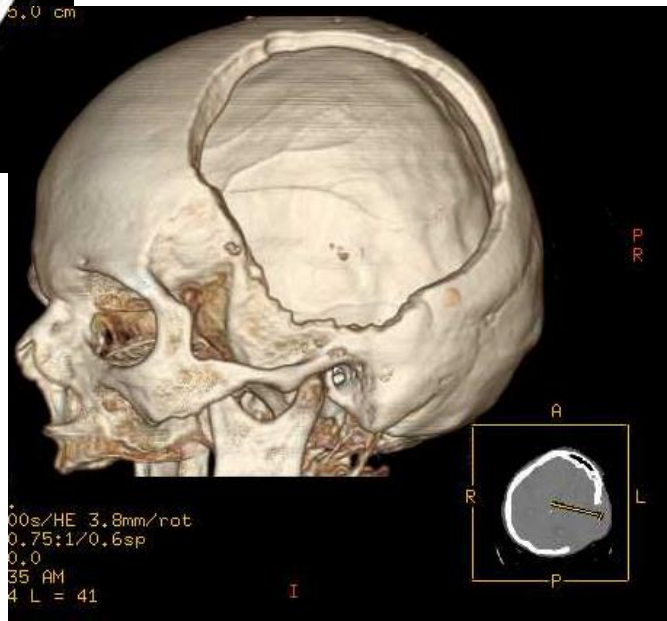
Decompressive craniectomy

Protection after bilateral decompressive craniectomy

66y F acute stroke (total occlusion of left MCA)



Jan 27, 2015



Jan 29, 2015

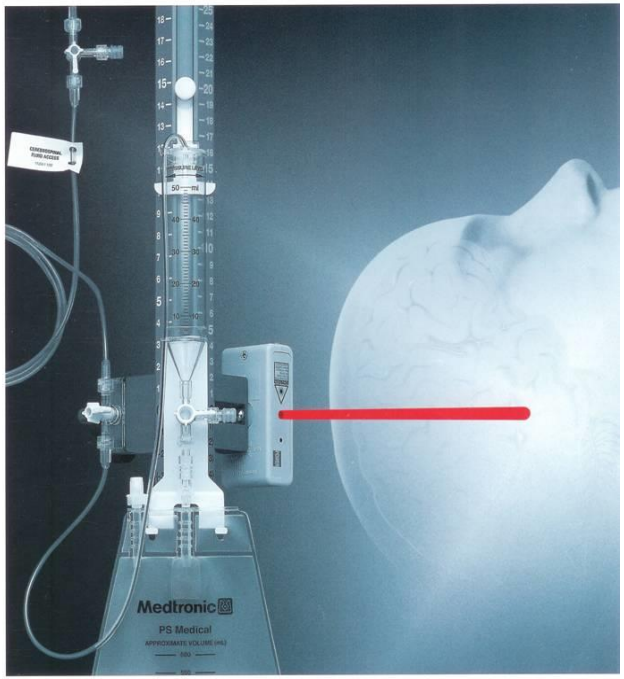
66y F acute stroke (total occlusion of left MCA) cont.

Feb 5, 2015

Feb 11, 2015 at discharge

Management of ↑ ICP (CSF)

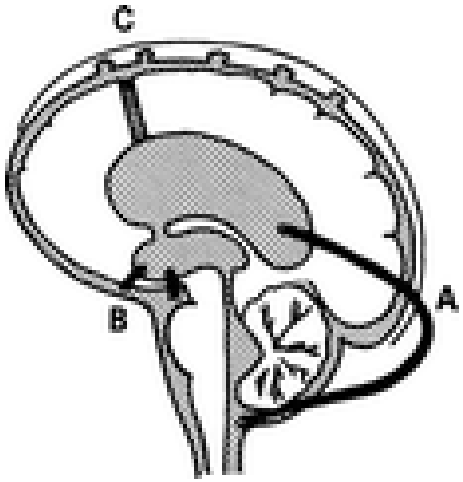
- Divert CSF to lumbar spine…HOB elevated
- *Divert CSF* out of body with ventricular drain
- Acetazolamide to decrease production

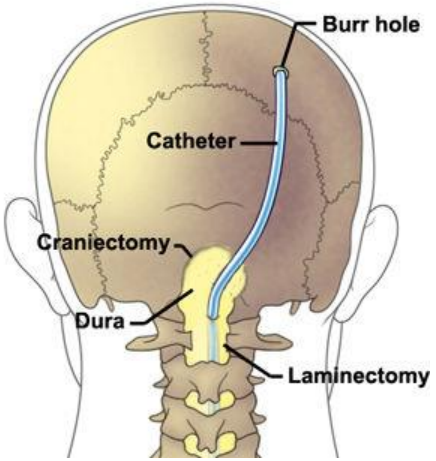


EFFICIENCY, SIMPLICITY, AND ACCURATE CONTROL

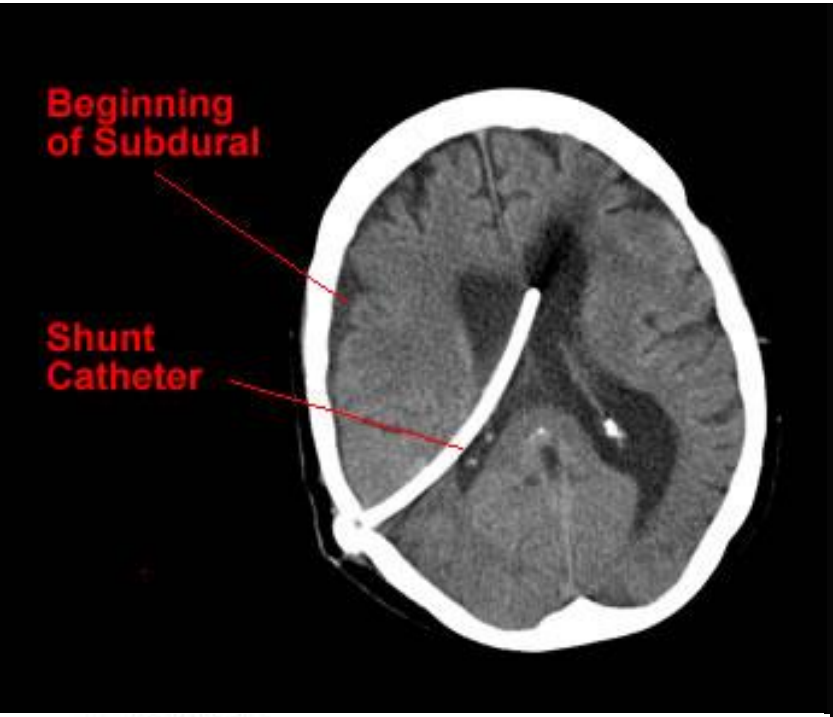
CSF diverting methods

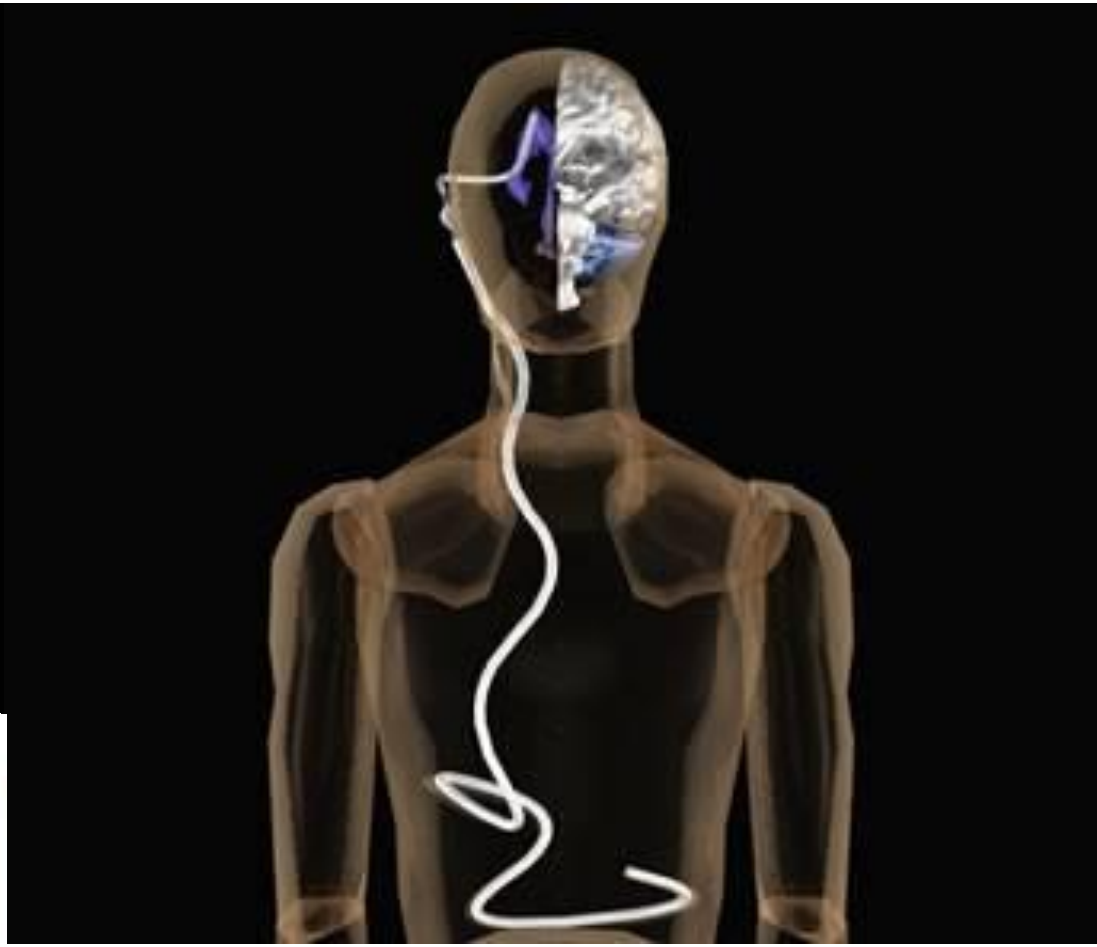
- Arne Torkildsen 1899-1968
  - Norwegian neurosurgeon (Oslo)
    - Ventriculo-cisternostomy
    - 1937- first 4 operations
    - 1939- published
- 1949- first in Hungary (Zoltán László OH)





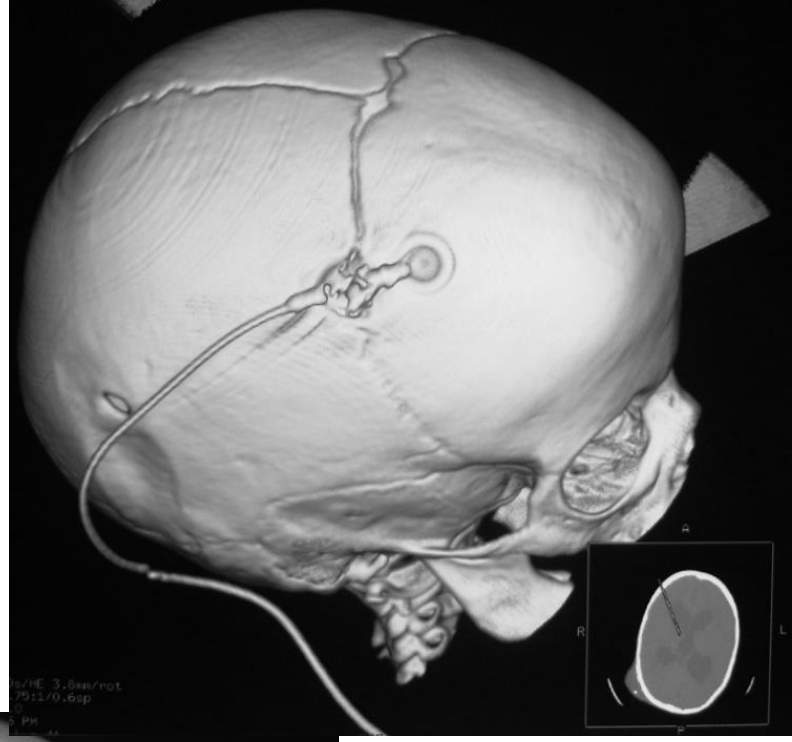
Intracranial shunts





MEDTRONIC STRATA VALVE

3D CT imaging of shunts

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