I_  _NEUROSURGERY INITIATIVE

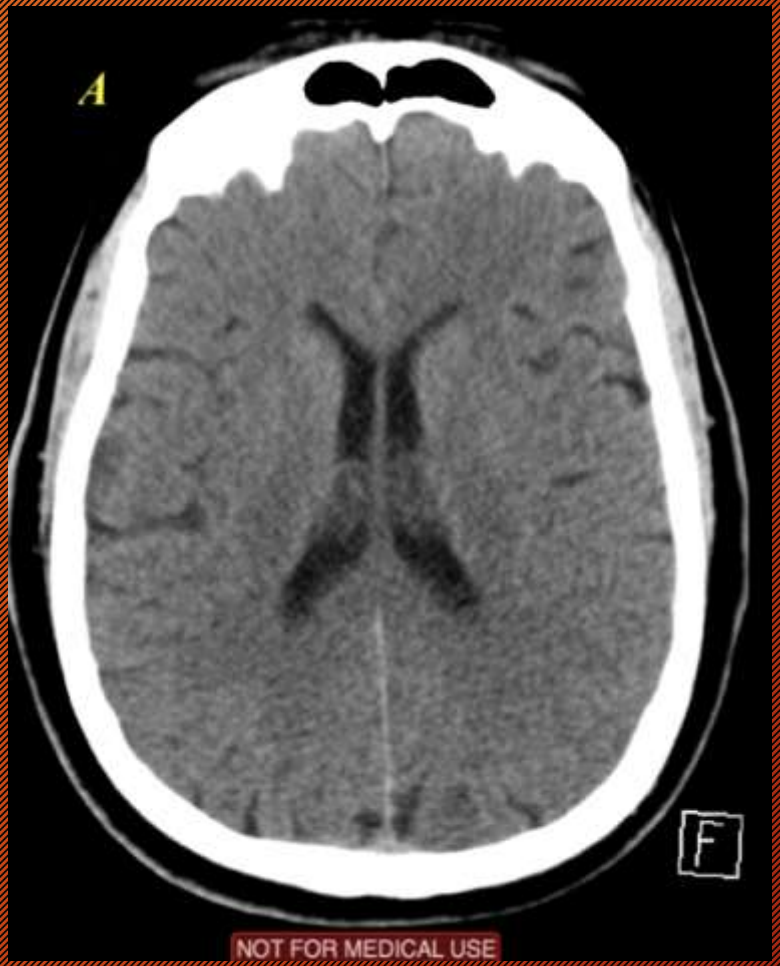
INCREASED INTRACRANIAL PRESSURE

WALID S. MAANI MD, FRCSEd.
PROFESSOR OF NEUROSURGERY
JORDAN UNIVERSITY HOSPITAL AND MEDICAL SCHOOL

PHYSIOLOGICAL PRINCIPLES

- *The skull is a rigid structure. It contains:*
  - *Brain*
  - *Blood*
  - *Cerebro spinal fluid (CSF)*

Normal Brain



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

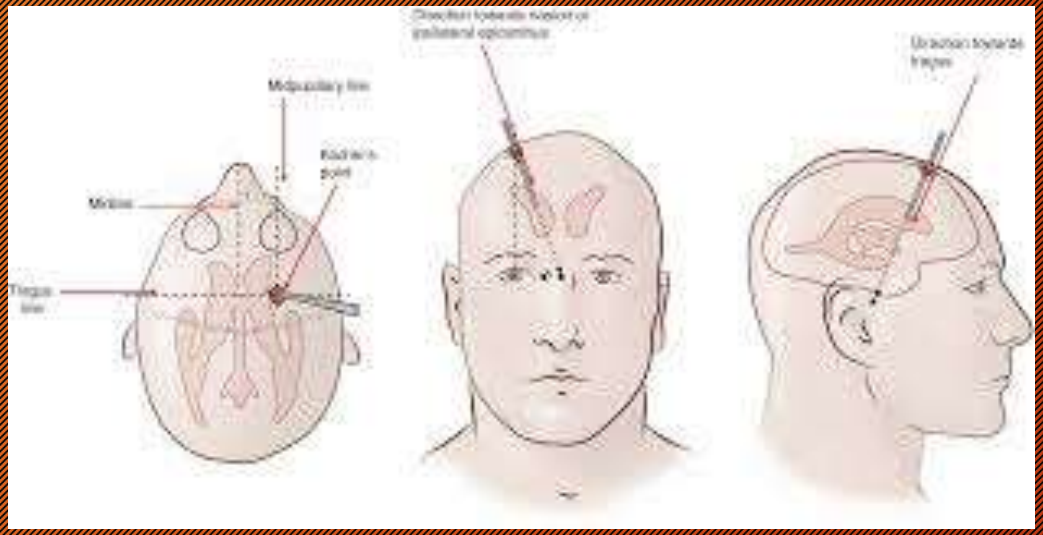
INTRACRANIAL PRESSURE FACTS

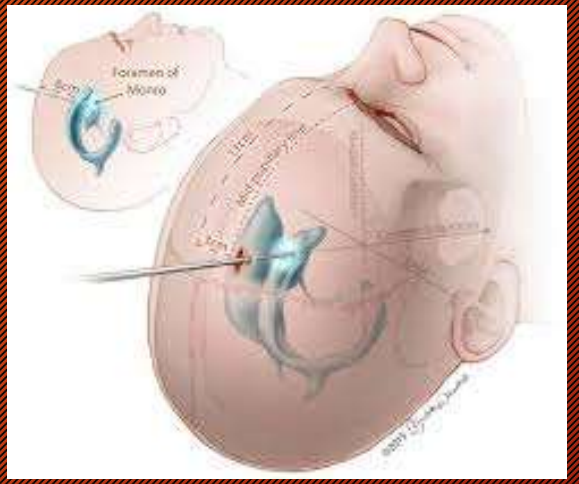
- *Normally 0-140 mm CSF (0-10 mm Hg)*
- *There are normal regular waves due to pulse and respiration*
- *With increased pressure "pressure waves" appear*
- *With continued rise of ICP the Perfusion of the brain falls*
- *When perfusion falls the blood flow is reduced*
- *Electrical cortical activity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

Monroe-Kellie doctrine

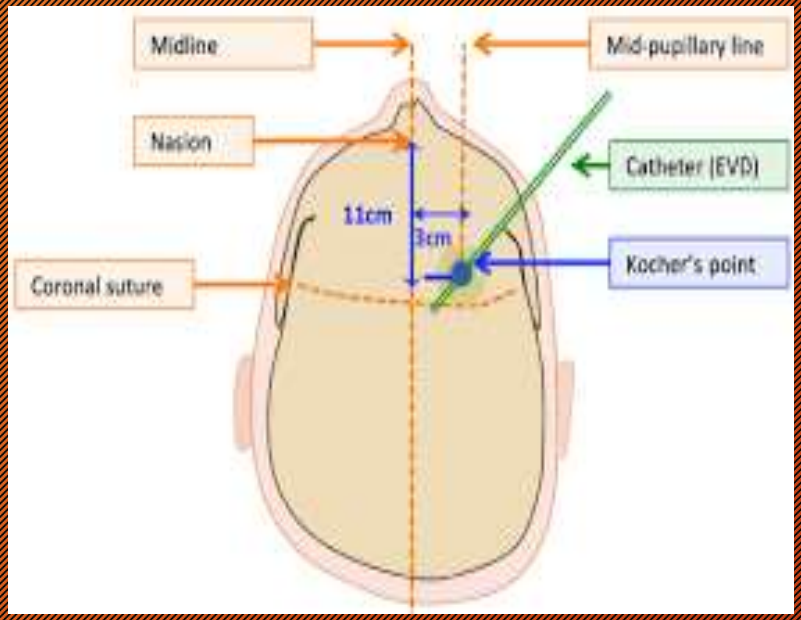
- *The intracranial pressure is the sum of the pressures exerted upon the wall of the skull by its contents.*
- *Any increase in one of these contents and/or the addition of any mass will lead to increase in the pressure, **unless** a similar volume is removed (the Monroe-Kellie doctrine).*

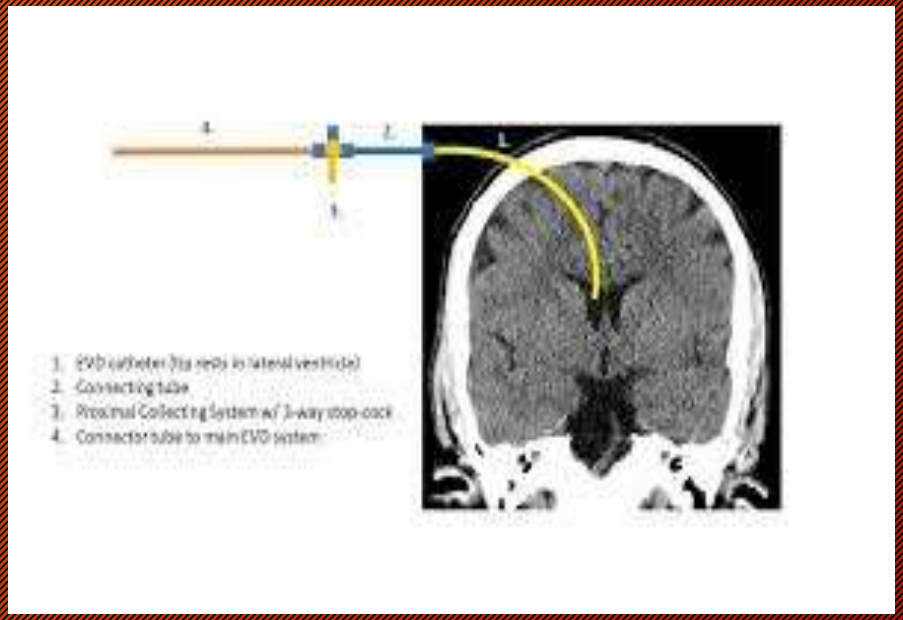
Monroe-Kellie doctrine

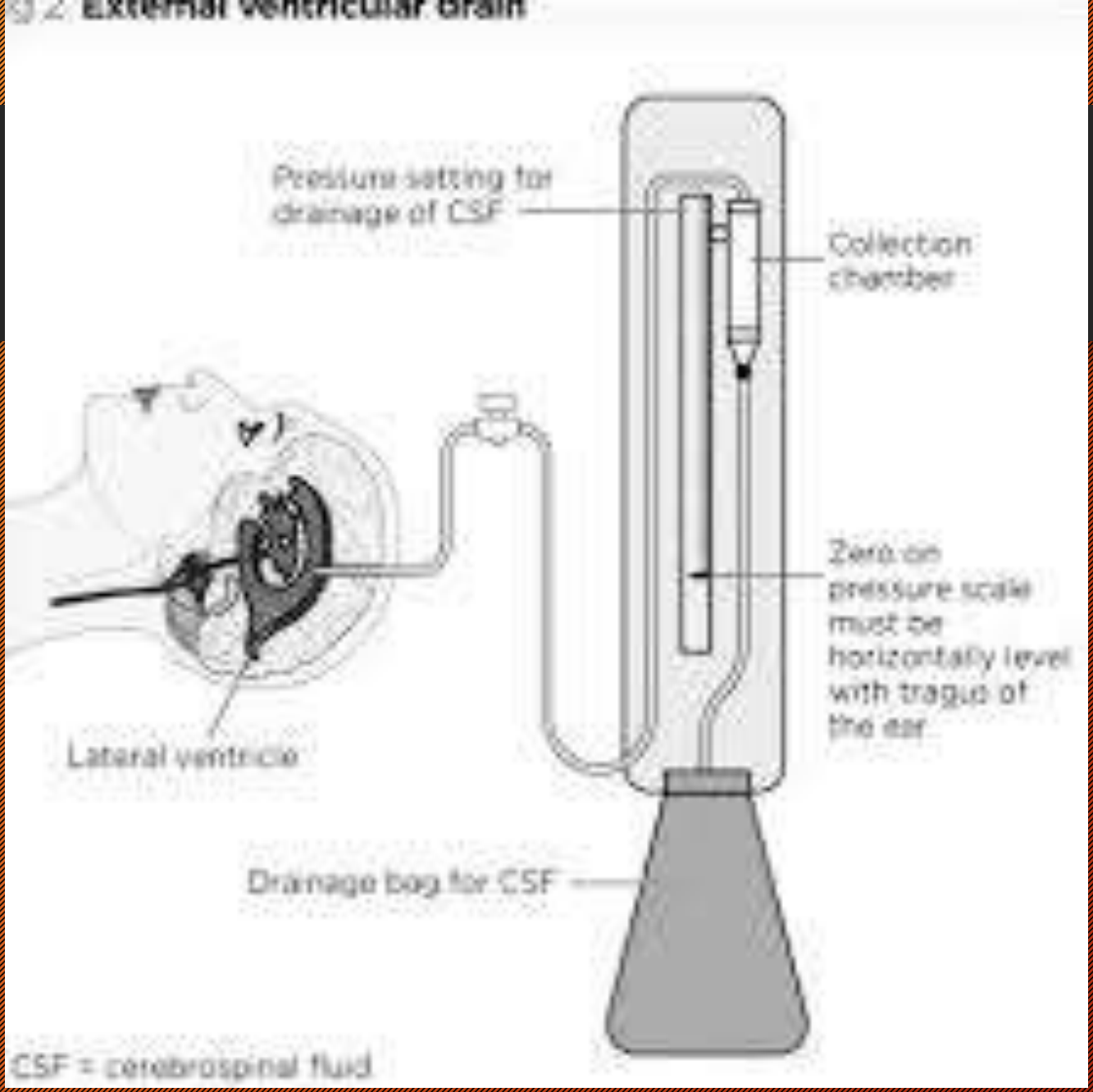




Monroe-Kellie doctrine

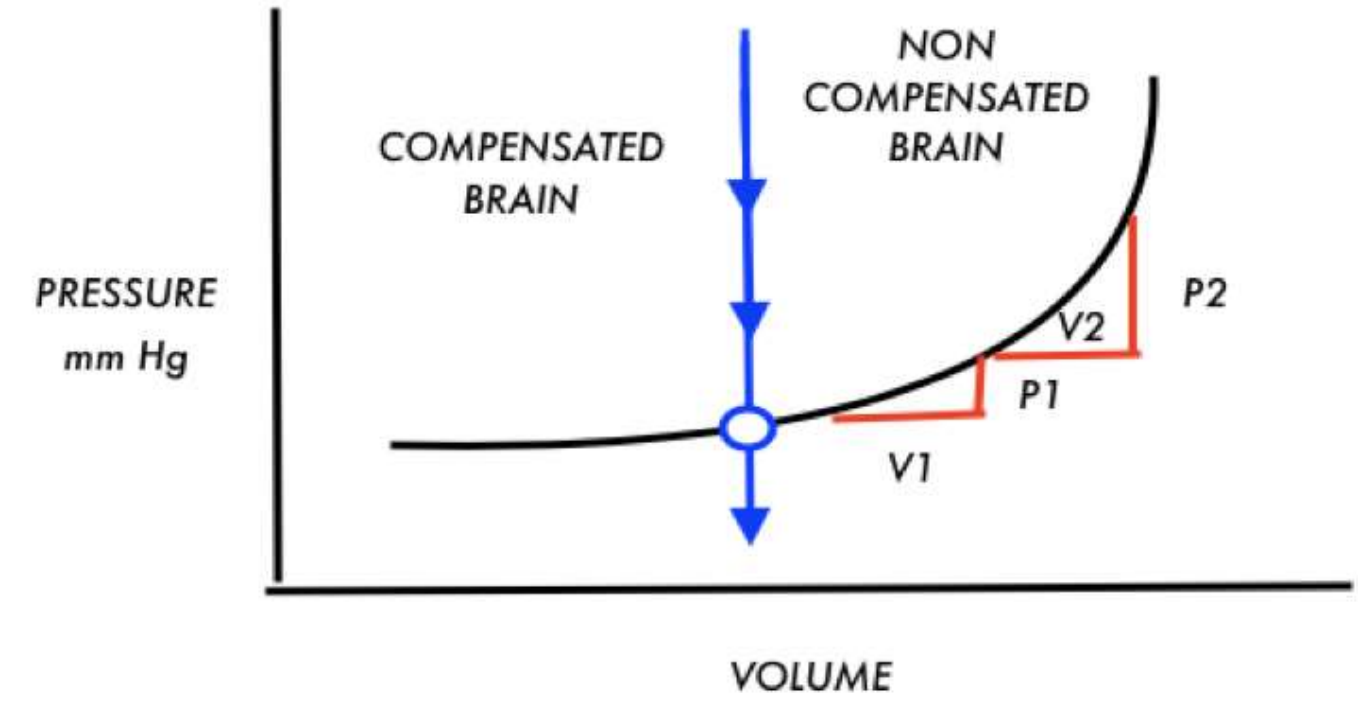





Pressure setting for drainage of CSF
Collection chamber
Zero on pressure scale must be horizontally level with tragus of the ear
Lateral ventricle
Drainage bag for CSF
CSF = cerebrospinal fluid

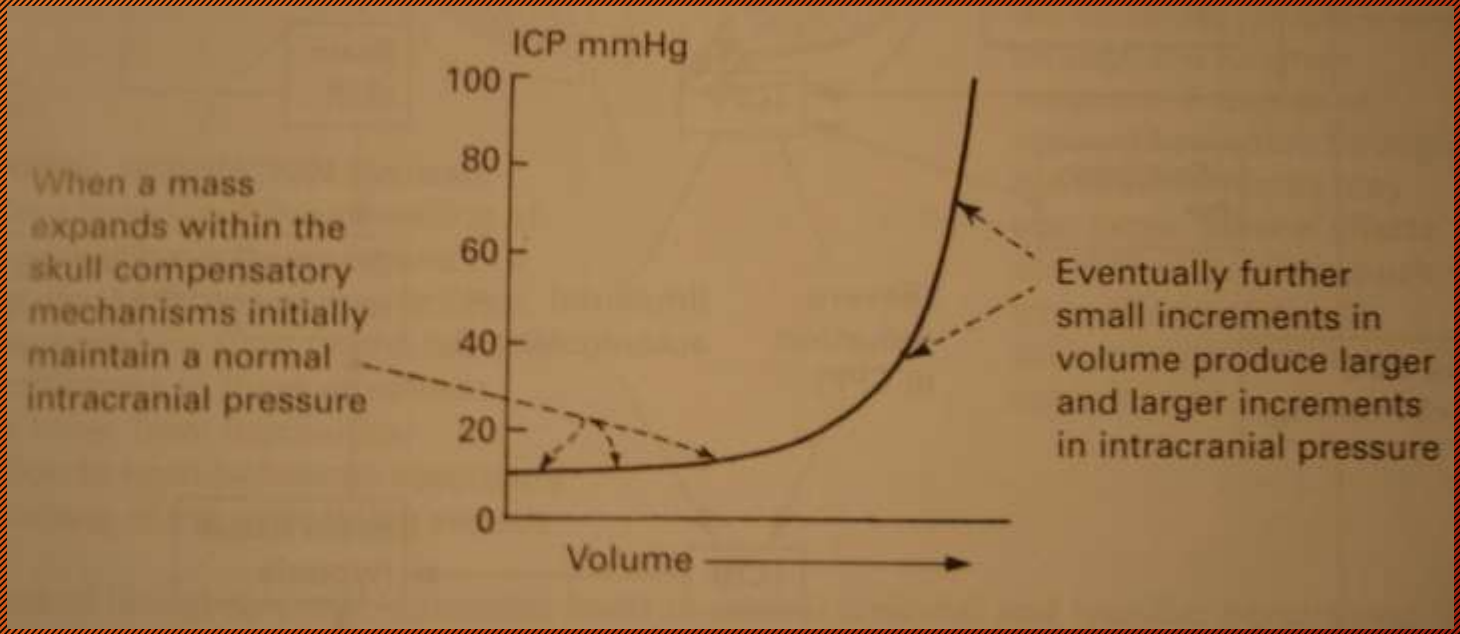
PHYSIOLOGY

THE PRESSURE VOLUME RELATIONSHIP



PHYSIOLOGY

THE PRESSURE VOLUME RELATIONSHIP



PHYSIOLOGY

- COMPENSATORY MECHANISMS
  - Push CSF out to spinal theca
  - Compress venous sinuses and push blood out
- ONCE COMPENSATORY MECHANISMS FAIL then brain herniations occur.
  - Push cerebral hemisphere to other side (midline shift and Cingulate (subfalcine herniation)
  - Push brain down
    - Uncal and Central (transtentorial herniation)
    - Tonsillar (transforaminal herniation)

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

- *TO **MAINTAIN PERFUSION AND FLOW** there are two **AUTOREGULATORY MECHANISMS***

1. Elevate blood pressure by peripheral vasoconstriction.,

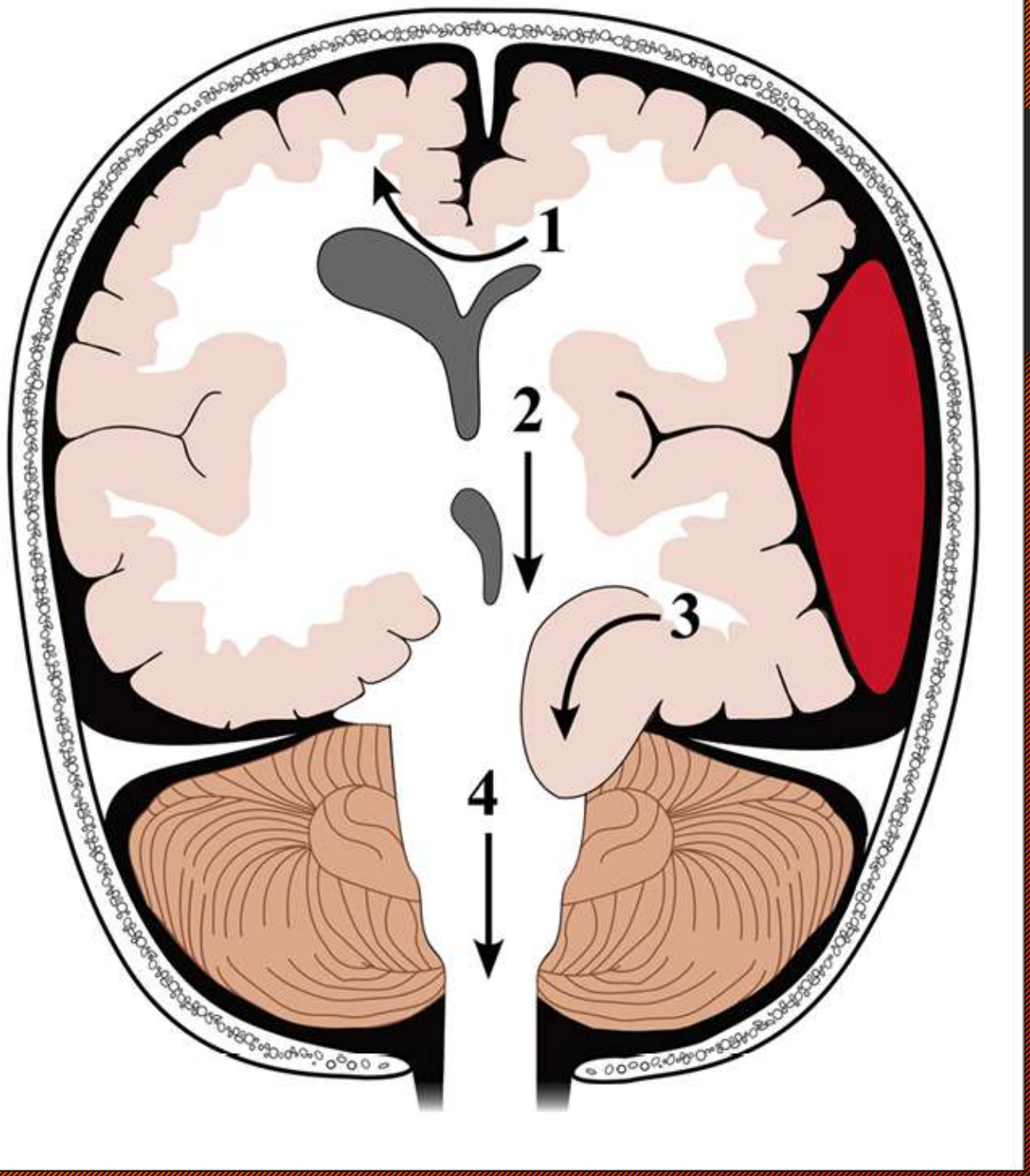
2. Dilate cerebral vessels by CO2 retention.

PHYSIOLOGY

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

PHYSIOLOGY

*The **ICP finally rises to the level of the mean arterial pressure** which it self begins to increase, accompanied by bradycardia or other disturbances of respiratory 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.*


1
2
3
4

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 ischemia in the brain stem.*

CAUSES

Increases in the ICP could be due to:

a) Increase in volume of the normal intracranial constituents.

b) Any added volume due to an abnormal condition like a space occupying lesion.

CAUSES

Increase in Normal Constituents

A) Brain

1) Cerebral edema

2) Idiopathic increased ICP

B) CSF

Hydrocephalus

C) Blood

Vasodilatation due to hypercapnia

CAUSES

***Abnormal* Constituents**

Brain

1. *Neoplasm*
2. *Infection*
3. *Hemorrhage*

CLINICAL PICTURE

The clinical picture is due to one of the following or a combination.

- ***Due to the ICP itself***
- ***Due to the herniations***
- ***Due to the causative lesion***

CLINICAL PICTURE

- *Early:*
  - *Headache, projectile vomiting and visual manifestations.*
- *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 rigidity*
  - *Changing and irregular respiratory patterns*

THE HEADACHE

- *Morning headache.*
- *Ordinary with no specific character.*
- *Responds to simple analgesia.*

D.D.

Migraine

THE VOMITING

- *Morning vomiting.*
- *Projectile (not preceded by nausea).*

D.D.

Pregnancy

Migraine

THE VISUAL MANIFESTATIONS

Called also amaurosis fugax or obscurations of vision.

- *Diminished acuity of vision*
- *Scotomas*

D.D.

Migraine

THE VISUAL MANIFESTATIONS

PAPILLEDEMA

D.D

Optic neuritis

CLINICAL PICTURE

Herniation of intracerebral contents

Supratentorial herniation

- *Subfalcine: which results in affection of the ACA leading to contralateral leg weakness*
- *Uncal (lateral transtentorial): and central transtentorial are the most frequently noted.*
  Result in ipsilateral pupil dilatation, decreased level of consciousness, changes in respiratory patterns, respiratory arrest, and contralateral hemiplegia. 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 medulla oblongata is compressed and displaced causing respiratory and cardiac arrest*

CLINICAL PICTURE

- SYMPTOMS AND SIGNS DUE TO THE CAUSATIVE LESION

MANAGEMENT

We are guided by how high is the pressure and what effects did it produce on the health of the patient.

- *So it is necessary to examine the patient*
- *Estimate his Glasgow Coma Score (GCS) on the Glasgow coma scale. AND ALSO GTS*
- *Find what the pressure is directly or indirectly.*
- *Do the required imaging.*

MANAGEMENT

The goals of any management should aim at:

1) Normalization of ICP by keeping ICP below 20 mmHg.

2) Maintaining normal CBF, by keeping CPP above 65 mmHg.

3) Relieving herniation.

4) Treatment of the cause.

IMAGING

Evidence of increased pressure could be seen in different types of imaging

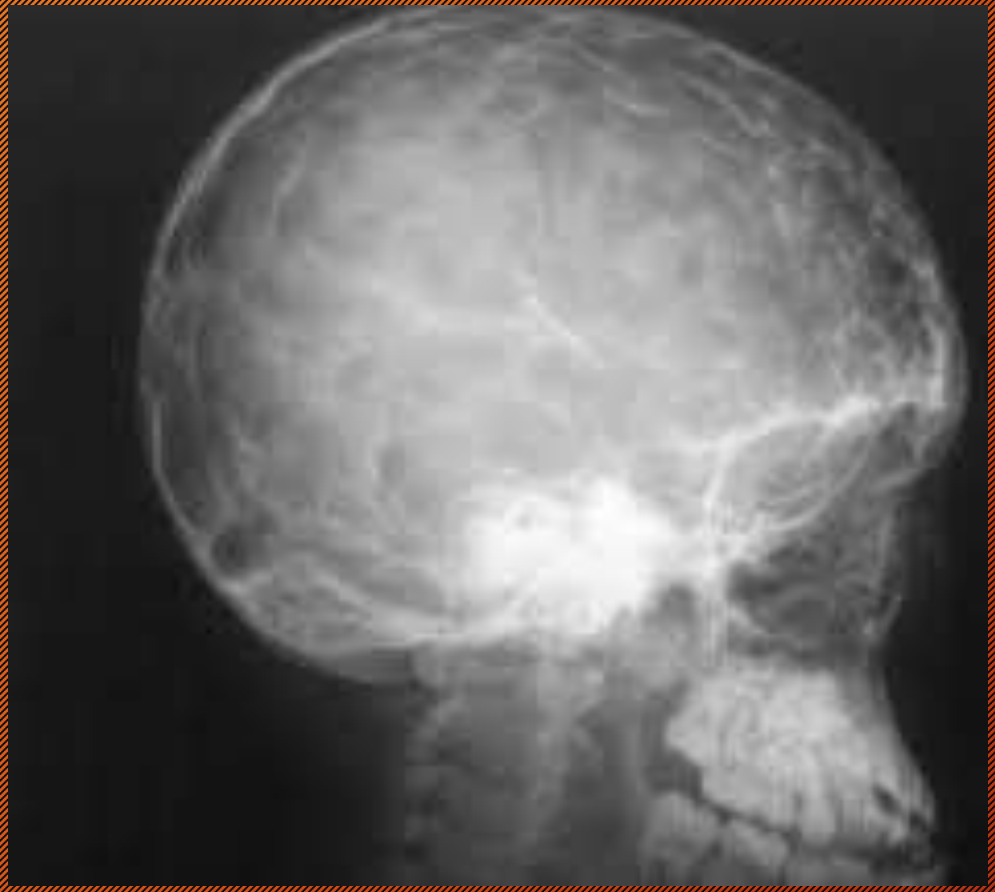
A) Plain skull X-rays:

- *Thumb impressions (beaten silver or beaten copper appearance) in the inner bone table of the skull.*
- *Erosion of the posterior clinoid processes.*
- *Widening of the skull sutures.*

B) MRI and CT:

- *Effacement of the cortical sulci.*
- *Compression of the ventricles.*
- *Shifts.*
- *Herniations.*

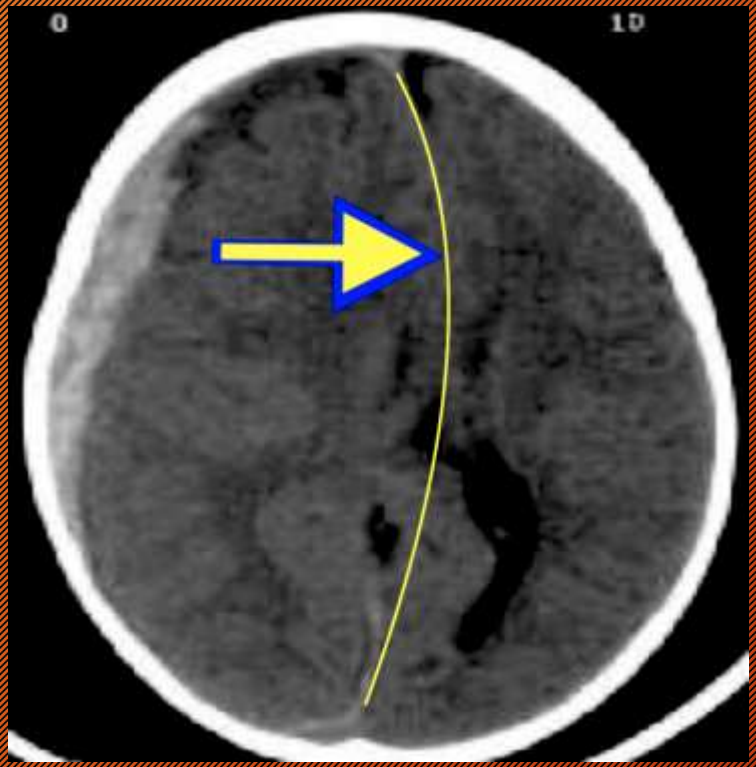
IMAGING: Skull X-rays



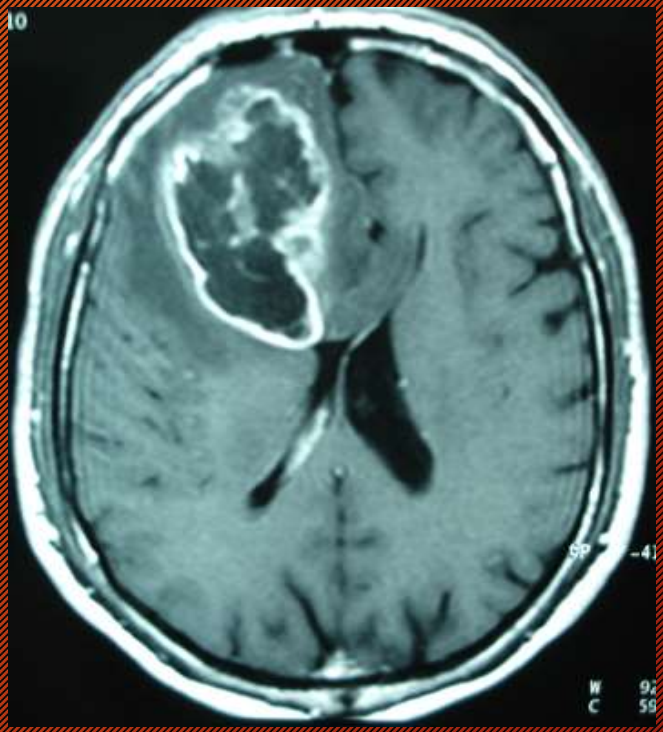
- Beaten Silver or Beaten Copper appearance

IMAGING: CT and MRI

CT: Effacement and midline shift



MRI: Compressed ventricles and shift



ICP MEASUREMENT

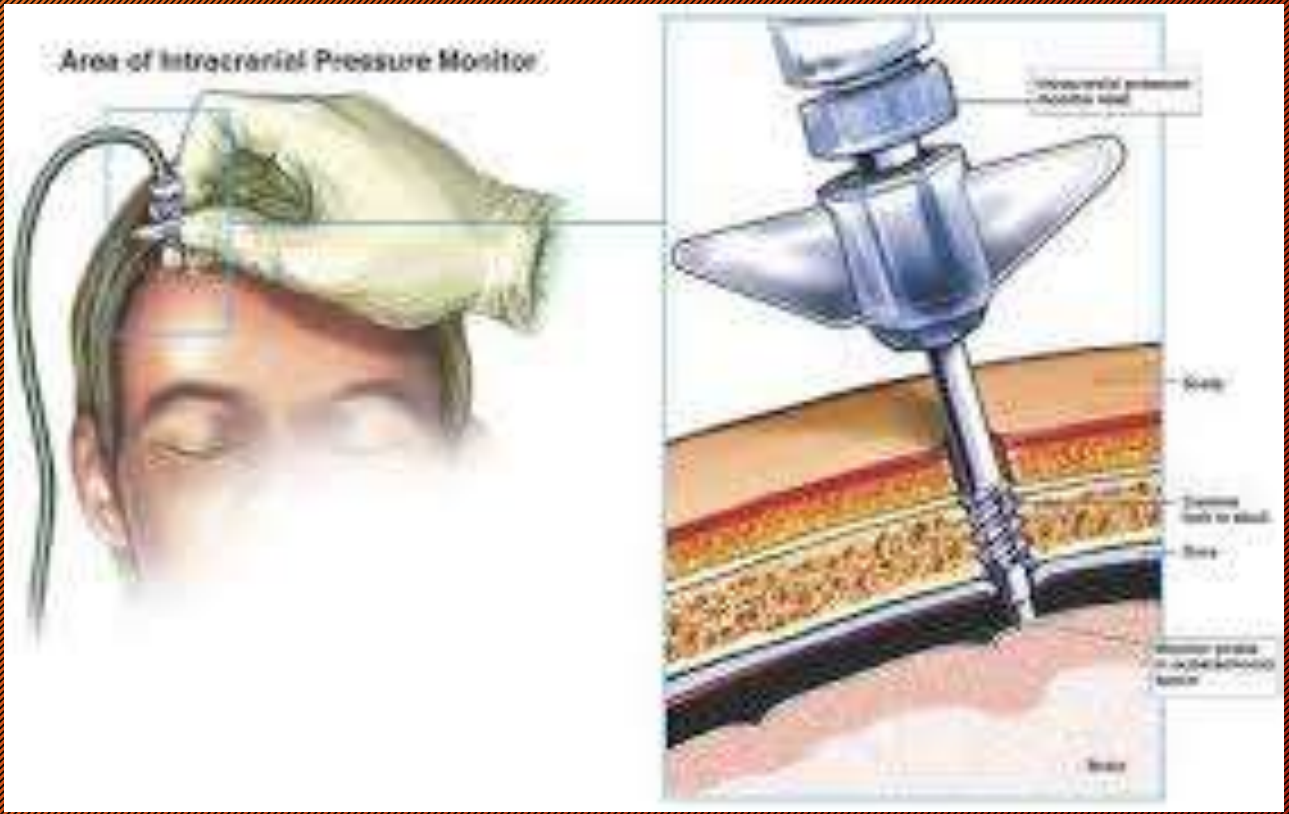
Intracranial pressure monitoring:

The intracranial pressure could be measured by a variety of methods, for diagnosis and to help in determining the need for, or the efficacy of treatment. These methods could be invasive or non-invasive.

- ***Invasive methods:***

1) Lumbar puncture.
2) Intraventricular catheters.
3) Subarachnoid screws.
4) Subdural sensors.
5) Intraparenchymal sensors.
6) Extradural sensors.

ICP MEASUREMENT: Insertion of subarachnoid bolt



ICP MEASUREMENT

INTRACRANIAL PRESSURE MONITORING

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

ICP MEASUREMENT

- ***Non-invasive methods:***

1) Ophthalmodynamometry or retinal Venous Outflow Pressure (VOP).

2) Optic nerve sheath diameter.

3) Tympanic membrane displacement.

4) Transcranial doppler.

The lumbar puncture (LP) is the simplest, but one must be careful to make sure that there are no expanding lesions inside the skull, otherwise herniation may occur.

MANAGEMENT

Measures to reduce high ICP

Steps to be taken to reduce or normalize ICP fall into three categories: general, specific and definitive.

1) General

1) Elevate head of bed 30 degrees.

2) Avoid bending the neck or compressing the jugular veins.

3) Guard against airway obstruction.

4) Maintain a normal blood pressure.

5) Control pain.

6) Drain the bladder.

7) Control headache (codeine phosphate 30-60 mg.)

MANAGEMENT

2) Specific

*1) Infuse **Mannitol**. Be careful to monitor blood pressure. Loop diuretics could be used to the same effect. Careful watch must be kept over the serum osmolarity to avoid elevation.*

*2) **Hyperventilate** the patient with the aim of regulating CO2 in the inspired air by maintaining PaCO2 between 35-40 mmHg..*

*3) **Drainage of CSF**.*

*4) **Sedation** using propofol or barbiturates with the aim of reducing the metabolic demand of the brain cells.*

*5) **Hypothermia** to reduce the metabolic rate.*

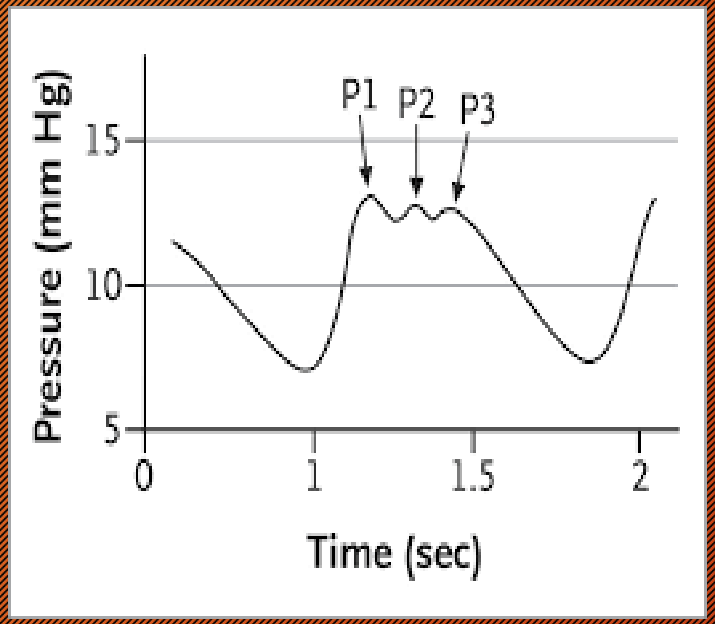
*6) **Decompressive craniectomy, craniotomy** should be done if all measures fail.*

MANAGEMENT

3) Definitive

The above mentioned general and specific measures could be the only possible lines of treatment in many cases in which the cause of the rise in ICP is general as in cases of closed head injury. But if there were lesions responsible for the elevation of the ICP, then in addition to the above measures, surgeons resort to removal of the causative lesion; be it a tumor, contusion or a hematoma. Once the lesion is removed, the ICP will settle down and all measures will not be needed anymore.

ICP MEASUREMENT: The waves



- ***There are 3 waves for ICP***

1) A wave

2) B wave

3) C wave

The A wave is the most important and is called the plateau wave, it has a sustained ICP of 50 mm of Hg for 20 minutes.

IDIOPATHIC INTRACRANIAL HYPERTENSION

It is a disease which affects obese women in the childbearing period, around 40 years of age. There is usually a history of taking medications like:

1) *OCPs,*
2) *Tetracycline,*
3) *Nalidixic acid or*
4) *Vitamins like A.*

Sometimes it accompanies other diseases like polycystic kidney, Bahcet disease, obstructive sleep apnea and hypothyroidism.

IDIOPATHIC INTRACRANIAL HYPERTENSION

- *The patients complain of:*

1) *Headache and*
2) *Visual manifestations like scotomas and transient visual loss, and their visual acuity starts to fail.*
3) *One may find a sixth nerve palsy.*
4) *But the striking feature will be the papilledema and in advanced cases, optic atrophy.*

IDIOPATHIC INTRACRANIAL HYPERTENSION

Diagnosis:

Is usually by exclusion; however, diagnosis depends on the modified Dandy Criteria (introduced in 1985 after the original in 1937, which include:

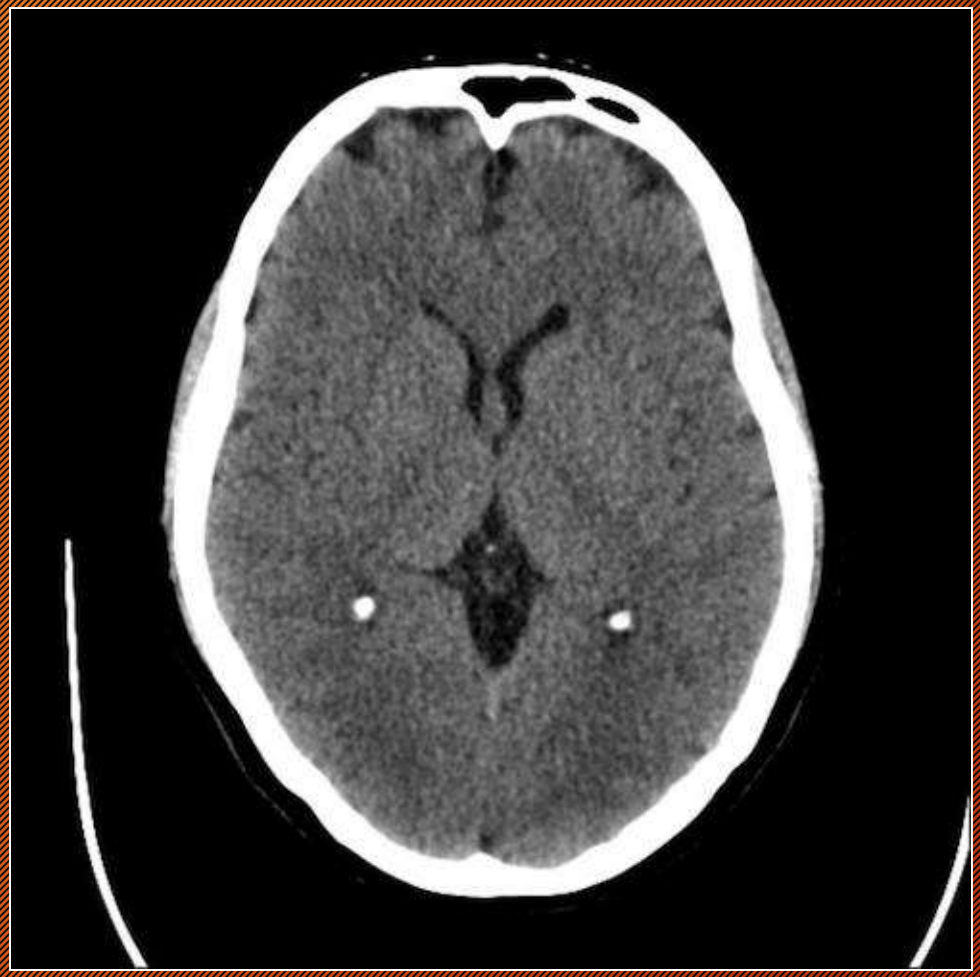
1) *Signs and symptoms of high ICP.*
2) *No neurological signs except for 6th. Nerve palsy.*
3) *Normal CT scan, with normal or small ventricles*
4) *Increased opening CSF pressure.*
5) *Normal CSF composition.*

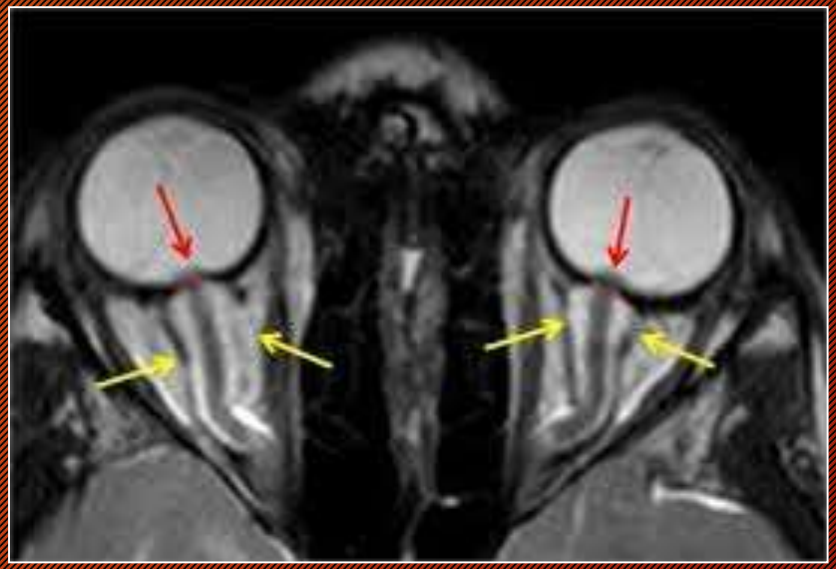
IDIOPATHIC INTRACRANIAL HYPERTENSION

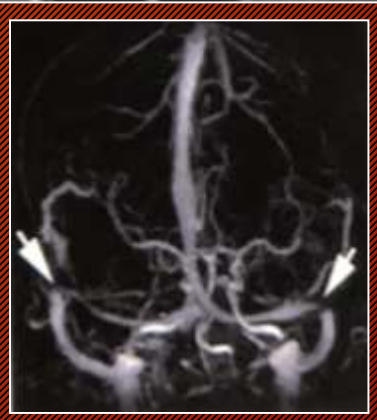
Diagnostic procedures:

1) *Visual acuity measurement, visual field charting and fundoscopic examination.*
2) *CT to exclude any other pathology.*
3) *MRV to exclude venous sinus thrombosis.*
4) *MRI orbital to look at width and shape of optic nerve sheath and optic nerve respectively.*
5) *LP to measure pressure and examine CSF composition. During the performance of the LP and after collecting the specimen the ICP should be reduced to half of the reading or to the normal level whichever is higher.*

IMAGING: *Normal CT, Tortious optic nerve and wide sheath, stenosed dural venous sinuses*







IDIOPATHIC INTRACRANIAL HYPERTENSION

Management:

The type of management proposed depends on the seriousness of the disease as measured by the visual status.

A) The Lumbar puncture itself:

On many occasions, the LP itself will result in dramatic improvement of the symptoms, which if sustained (the improvement and the normal pressure reading judged by a second LP, then nothing more is required.

IDIOPATHIC INTRACRANIAL HYPERTENSION

B) Medical conservative:

In situations when there is mild to moderate symptoms with no impending visual loss, the following measures could be used:

Reduction of weight, and maintenance of reduction.

Stopping the OCP, and any other medications thought to be part of the cause.

Giving medication to reduce CSF production like Acetazolamide (Diamox).

IDIOPATHIC INTRACRANIAL HYPERTENSION

C) Surgery

If there is impending visual loss, expanding field loss or development of a new one, or failure of medical conservative treatment, then the patient will be offered one of the following to reduce the pressure inside the cranium and lessen the effects on the optic head, and consequently preserve vision.

1) *Optic nerve sheath fenestration. The CSF will pour inside the orbit and the lymphatics will absorb it.*
2) *Lumbo-peritoneal (theco-peritoneal) shunting. The CSF will be absorbed via the wide peritoneal surface. This shunt has many complications; like slippage and malfunctioning which may require revision.*
3) *Stenting of the stenosed venous sinus.*