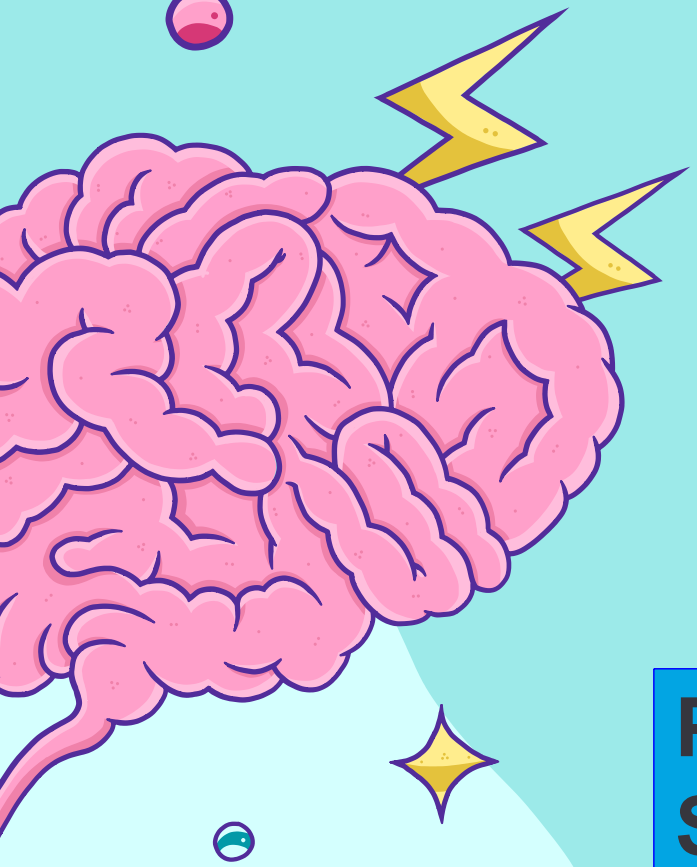


Congenital anomalies of CNS

Presented by :
Shaden Abed Al-dala'in

Craniofacial development

Normal development

Fontanelles

1. Anterior fontanelle: the largest fontanelle and Diamond shaped, 4cm (AP) × 2.5cm (transverse) at birth. (18m-24m)
Normally closes by age **2.5 yrs**.
2. Posterior fontanelle: triangular. Normally closes by age **2–3 mos**.

3. Sphenoid and mastoid fontanelles: small, irregular.
Normally, former closes by **age 2–3 mos**, latter by age **1 yr**.

Cranial vault (skull)

Growth: largely determined by growth of brain.

90% of adult head size is achieved by age 1 yr; 95% by age 6 yrs. Growth essentially ceases at age 7 yrs.

47 cm

55-56 cm

→ this is very important in the treatment of craniosynostosis [golden period for treatment].

By end of 2nd yr, bones have interlocked at sutures and further growth occurs by accretion and absorption.

Mastoid process: formation commences by age 2 yrs, air cell formation occurs during 6th yr

Skull is unilaminar at birth. (one layer of spongy bone).

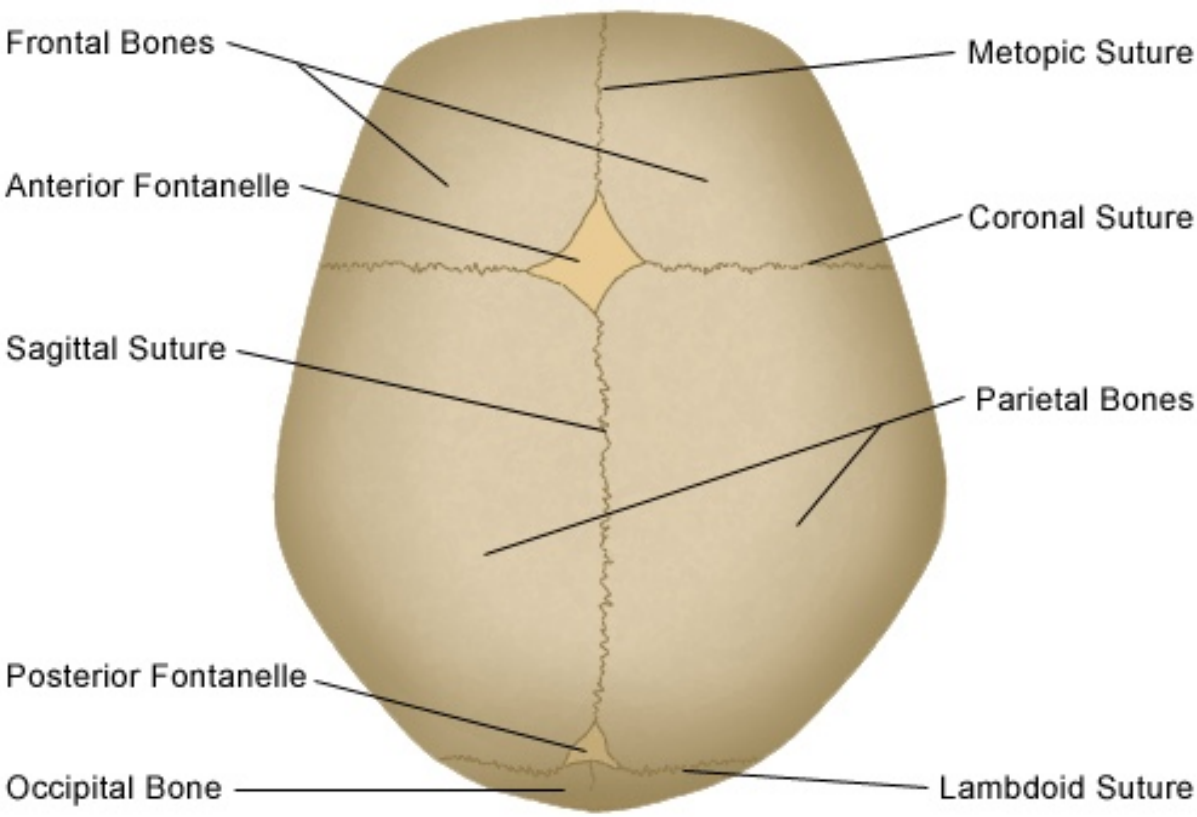
Diplöe appear by 4th yr and reach a maximum by age 35 yrs (when diploic veins form). (outer, inner tables, cortex and spongy bone in between) → not trilaminar.

.

INTRODUCTION

Brain anamolies

Normal Skull of the Newborn



Craniosynostosis (CSO

Craniosynostosis (CSO) was originally called craniostenosis, and **is the premature ossification of a cranial suture**

Epidimoilogy

Incidence: ≈ 0.6/1000 live births. It may occur in isolation, but may also be syndromic or secondary.

Once the suture ossifies, normal growth of the skull perpendicular to the suture terminates and tends to proceed parallel to the suture

مثلاً لو سكر ال coronal suture حيشكل عرفين growth ال حيبح

Primary CSO :

is usually a prenatal deformity. → congenital.

Secondary CSO

Etiologies :

or hypo?

1. metabolic (rickets,hyperthyroidism…),
2. toxic (drugs such as phenytoin, valproate, methotrexate…),
3. hematologic (sickle cell, thalassemia…)
4. structural (lack of brain growth due to e.g. microcephaly, lissencephaly,micropolygyria…).

→ inspection of a VP Shunt ? (recently proven not to be a cause.

CSO is rarely associated with hydrocephalus (HCP).

* it was previously thought that in patient with chranical malformation eg: insertion of shunt to ↓ the CSF pressure

lead to locking of intracranial pulsation → and so 2ry suture closure.

Treatment

*but new study have proven this to be wrong

usually surgical (strip craniotomy). In most case the indication for surgery is for **cosmesis** (main goal) and to prevent the severe psychological effects of having a disfiguring deformity.

However, with multisutural CSO, brain growth may be impeded by the unyielding skull. Also, ICP may be pathologically elevated, and although this is more common in multiple CSO,elevated ICP occurs in ≈ 11% of cases with a single stenotic suture.

└ HCP is ever less.

– metopic type can be ass with mental retardation (15%

Most cases of single suture involvement can be treated with linear excision of the suture.

.

Risks of surgery include blood loss, seizures, and stroke.

- Diagnosis
- Some cases of “synostosis” are really deformities caused by positional flattening (e.g. “lazy lambdoid”, If this is suspected, instruct parents **to keep head off of flattened area and recheck patient in 6–8 weeks: if it was positional, it should be improved**,
- if it was CSO then it usually declares itself. The diagnosis of CSO may be aided by:

1. palpation of a bony prominence over the suspected synostotic suture (exception: lambdoidal synostosis may produce a trough
2. gentle firm pressure with the thumbs fails to cause rela tive movement of the bones on either side of the suture

مع بجفن / او صابيحسي حركة.

plain skull X-rays:

a) **lack of normal lucency in center of suture**. Some cases with normal X-ray appearance of the suture (even on CT) may be due to focal bony spicule formation

b) **beaten copper calvaria** sutural diastasis and erosion of the sella may be seen in cases of increased ICP

skull

sign of chronic ↑ ICP

MRI:

usually reserved for cases with associated intracranial abnormalities.

Often not as helpful as

CT measurements, such as occipito-frontal-circumference may not be abnormal even in the face of a deformed skull shape

CT scan:

a) helps demonstrate cranial contour
b) may show thickening and/or ridging at the site of synostosis
c) will demonstrate hydrocephalus if present
d) may show expansion of the frontal subarachnoid space
e) three-dimensional CT may help better visualize abnormalities

questionable cases,

a technetium bone scan can be performed

a) there is little isotope uptake by any of the cranial sutures in the first weeks of life
b) in prematurely closing sutures, increased activity compared to the other (normal) sutures will be demonstrated
c) in completely closed sutures, no uptake will be demonstrated

⊛Single → cosmatic mainly (normal. قريب لـ)

⊛ head circumference [in multiple suture]
increase the size

Increased ICP

Evidence of increased ICP in the newborn with craniosynostosis include:

1. radiographic signs (on plain skull X-ray or CT, see above)
2. failure of calvarial growth (unlike the non-synostotic skull where increased ICP causes macrocrania

in the newborn, here it is the synostosis that causes the increased ICP and lack of skull growth)

3. papilledema
4. developmental delay

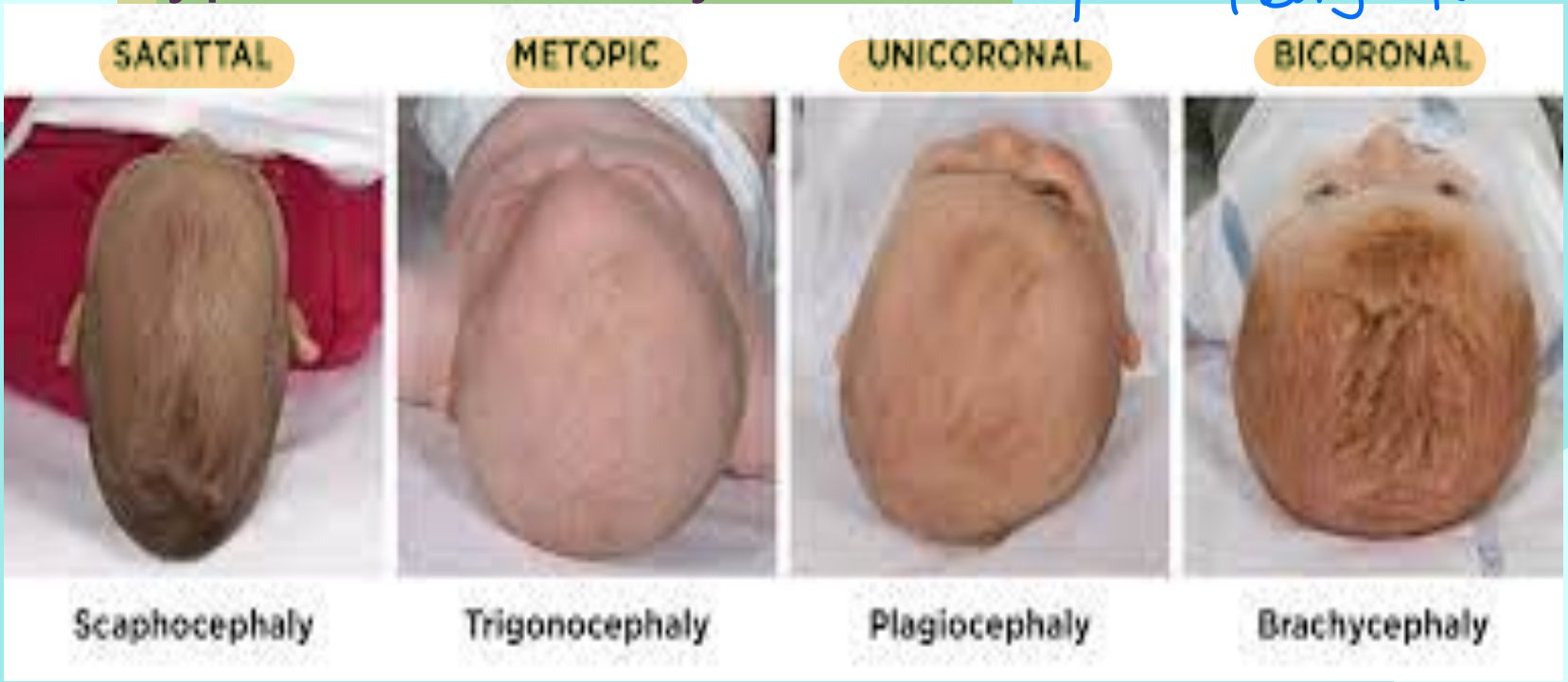
*is it normal to find papilledema in infants ?
no

*it it common to see papilledema in patient with ↑ICP ?
No, sutures is open

(not Fontanells)

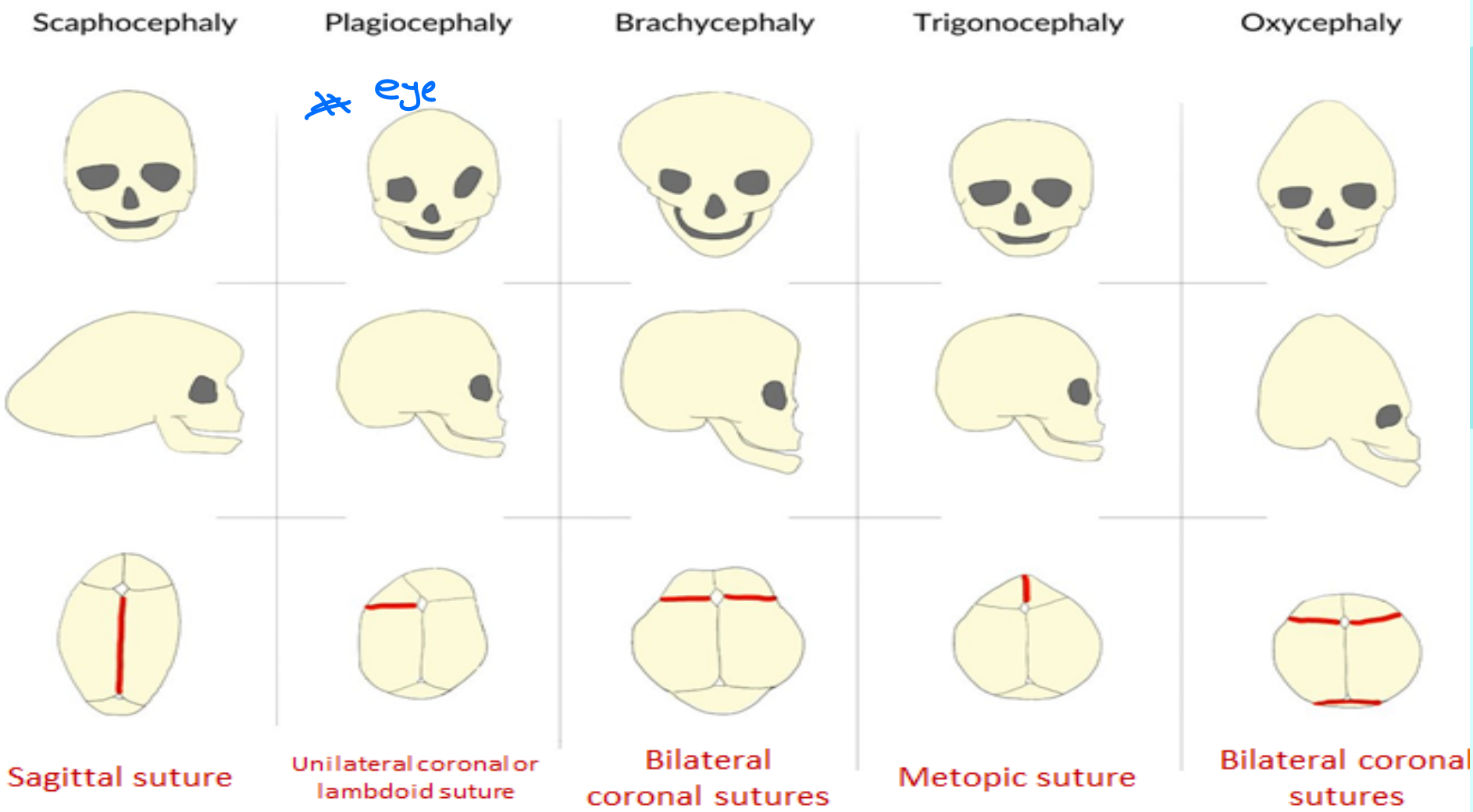
Types of craniosynostosis

eye retracted + bony prominance. ipsilateral



* coronal + lambdoid → normal growth: anterior + posterior
abnormal: elongation

Craniosynostosis



lateral groove

Sagittal synostoiss

General information

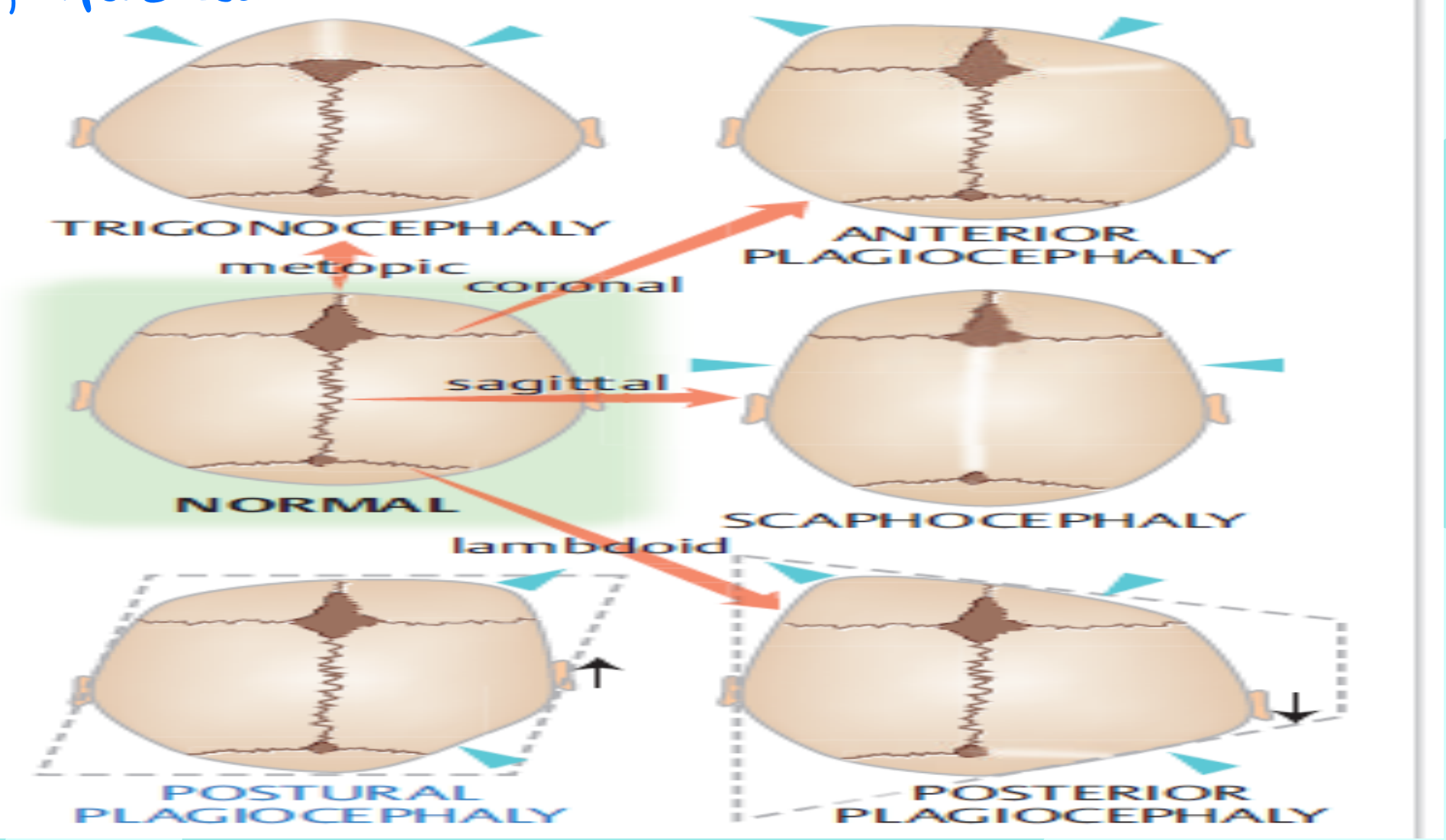
. The most common CSO affecting a single suture. 80% male. Produces a **palpable keel like sagittal ridge** and dolichocephaly (elongated skull with high forehead/frontal bossing) or scaphocephaly (“boat shaped skull” with prominent occiput). OFC remains close to normal, but the biparietal diameter is markedly reduced.

As many as 44% of patients with nonsyndromic sagittal synostosis have elevated ICp

*Sagittal suture

anterior + posterior

normally


TRIGONOCEPHALY
metopic
coronal
ANTERIOR PLAGIOCEPHALY
sagittal
NORMAL
SCAPHOCEPHALY
lambdoid
POSTURAL PLAGIOCEPHALY
POSTERIOR PLAGIOCEPHALY

Surgical treatment

Skin incision may be longitudinal or transverse. A linear "strip" craniectomy is performed, excising the sagittal suture from the coronal to the lambdoid suture, preferably within the first 3–6 months of life. The width of the strip should be at least 3 cm; ideal age to do surgery is from 6m to 1yr

. no proof exists that interposing artificial substances(e.g. silastic sheeting over the exposed edges of the parietal bone) retards the recurrence of synostosis.

Great care is taken to avoid dural laceration with potential injury to the underlying superior sagittal sinus.

The child is followed and reoperated if fusion recurs before 6 months of age.

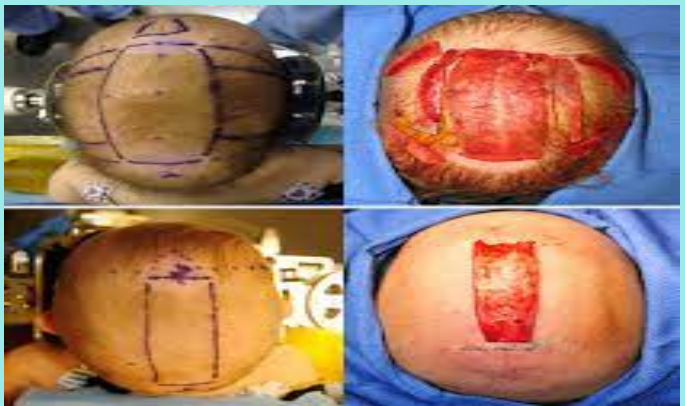
After≈ 1 yr of age, more extensive cranial remodelling is usually required.

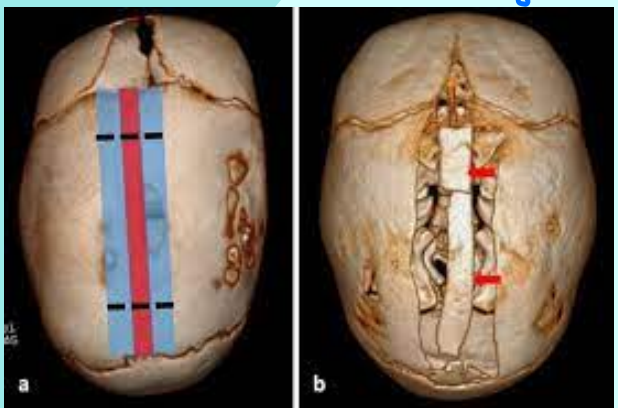
*what is the best time to do it?

6m – 12year.

less risk, and more benefits.

*risk of the surgery





. unilaminar ← الجمجمة في bone
skull → بتتزود كتير → skull have rich CT and the 1st step in hemostasis is: VC and in skull no vasospasm and no clotting.

blood loss.
وزن المريض و الـ blood volume

• 7kg : وزن تقريبًا 3months الطفل على عمر الـ *
• Shock ما يتحمل الخلايا ، وممكن يصير عند
high risk for strokes + MI. , ↑ mortality.

Coronal synostosis

General information

90% of adult head size reached by 1 year → الطفل عمره 1 سنة

Accounts for 18% of CSO, more common in females.

In **Crouzon's syndrome** this is accompanied by abnormalities of sphenoid, orbital, and facial bones (hypoplasia of midface),

and in **Apert's syndrome** is accompanied by syndactyly.

Unilateral coronal CSO → **anterior plagiocephaly** with the forehead on the affected side flattened or concave above the eye and compensatory prominence of the contralateral forehead.

X no compensatory

The supra-orbital margin is higher than the normal side, producing the **harlequin eye sign**.

The orbit rotates out on the abnormal side, and can **produce amblyopia**. Without

flattened cheeks develop and the nose deviates to the normal side (the root of the nose tends to rotate towards the deformity).

Bilateral coronal CSO (usually in craniofacial dysmorphism with multiple suture CSO, e.g. Apert's)

→ **brachycephaly with broad, flattened forehead (acrocephaly), is more common than unilateral**.

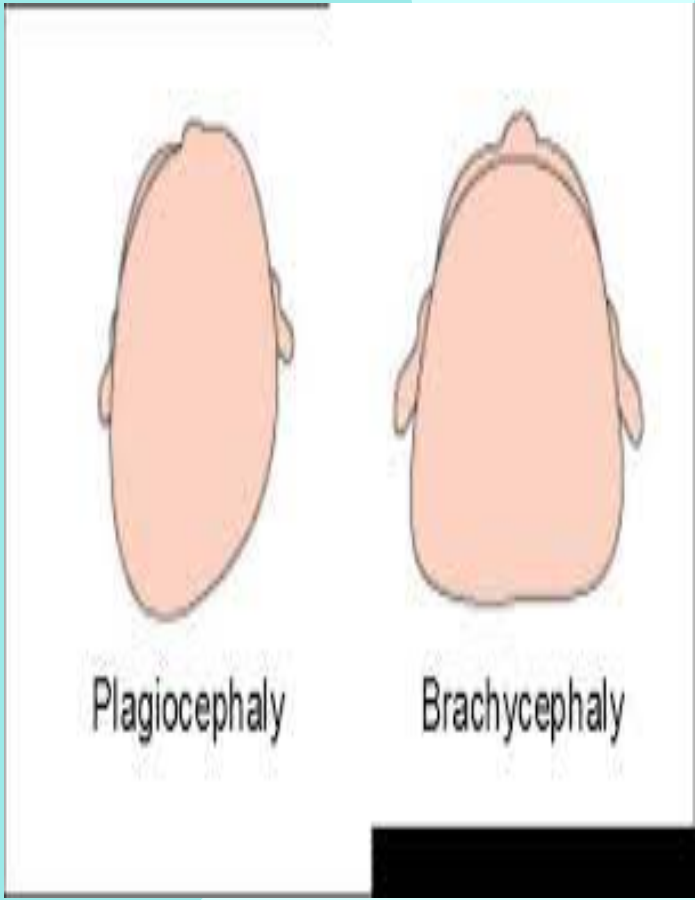
When combined **with premature closure of frontosphenoidal and frontoethmoidal sutures, it results in a foreshortened anterior fossa with maxillary hypoplasia, shallow orbits, and progressive proptosis**.

Surgical treatment

Simple strip craniectomy of the involved suture has been used, often with excellent cosmetic result.

However, some argument that this may not be adequate has been presented.

Therefore, a more current recommendation is to do frontal craniotomy (uni- or bilateral) with lateral canthal advancement by taking o ff orbital bar



between 2 Frontal.

Metopic synostosis

At birth, the frontal bone consists of two halves separated by the frontal or metopic suture.

Abnormal closure produces trigonocephaly (pointed or triangular shaped) forehead with a midline ridge and hypotelorism.

Incidence: 1/15,000 live births. 75% are male.

Many of these have a **19p chromosome** abnormality and are **mentally retarded**

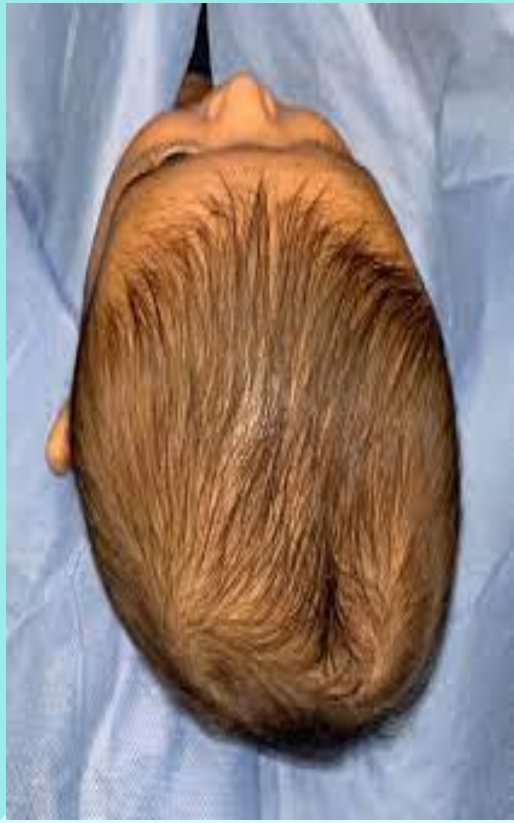
→ between parietal and occipital

Lambdoid synostosis

Epidemiology

Rare, with a reported incidence of 1–9% of CSO More common in males (male:female = 4:1).

Involves the right side in 70% of cases. **Usually presents between 3–18** months of age, but may be seen as early as 1–2 months of age.



Clinical findings

Flattening of the occiput. May be unilateral or bilateral.

If unilateral, it produces posterior plagiocephaly.

Bilateral lambdoid synostosis produces brachycephaly with both ears displaced anteriorly and Inferiorly

Unlike the palpable ridge of sagittal or coronal synostosis, an **indentation may be palpated along the synostotic lambdoid suture** (although a perisutural ridge may be found in some).

Mimic: **“lazy lamboid” (positional flattening).**

Lambdoid synostosis must be distinguished from positional flattening, the so-called “lazy lambdoid.” Flattening of the occiput here is associated with anterior displacement of the ipsilateral ear. there is bulging of the ipsilateral cheek and forehead.

Positional flattening (or molding) may be produced by:

1. decreased mobility: patients who constantly lie supine with the head to the same side, e.g. cerebral palsy, mental retardation, prematurity, chronic illness
2. abnormal postures: congenital torticollis ,congenital disorders of the cervical spine
3. intentional positioning: due to the recommendation in 1992 to place newborns in a supine sleeping position to reduce the risk of sudden infant death syndrome (SIDS) sometimes with a foam wedge to tilt the child to one side to reduce the risk of aspiration
4. intrauterine etiologies: intrauterine crowding (e.g. from multiparous births or large fetal size), uterine anomalies

Diagnostic evaluation

The physical exam is the most important aspect of diagnosis.

Skull X-ray may help differentiate.

If the skull X-ray is equivocal, **prevent the infant from laying on the affected side for several weeks**. A bone scan should be obtained if no improvement occurs In definite cases of synostosis, and for some cases of refractory positional flattening (which usually corrects with time, but may take up to 2 years), surgical treatment may be indicated.

Skull X-ray: Shows **a sclerotic margin along one edge of the lambdoid suture** in 70% of cases. Local **"beaten copper cranium"** (BCC) occasionally may be seen due to indentations in the bone from underlying gyri, which may be due to locally increased ICP. BCC produces a characteristic mottled appearance of the bone with lucencies of varying depth having round and poorly marginated edges. BCC correlates with generalized ↑ ICP only when it is seen with sellar erosion and sutural diastasis.

CT scan: Bone windows **may show eroded or thinned inner table in the occipital region** in 15–20% of cases,23 > 95% are on the side of the involvement. The suture may appear closed. Brain windows **show parenchymal brain abnormalities** in < 2%: **heterotopias, hydrocephalus, agenesis of the corpus callosum**; but ≈ 70% will have **significant expansion of the frontal subarachnoid space** (may be seen in synostosis of other sutures

Bone scan: **Isotope uptake in the lambdoid suture increases during the first year**, with a peak at 3 months of age 24 (following the usual inactivity of the first weeks of life).

The findings with synostosis are those typical for CSO

Treatment

Early surgical treatment is indicated in cases with severe craniofacial disfigurement or those with evidence of increased ICP.

Otherwise, children may be **managed nonsurgically for 3–6 months**.

The majority of cases will remain static or will improve with time and simple nonsurgical intervention.

Approximately 15% will continue to develop a significant cosmetic deformity.

Nonsurgical management

Although improvement can usually be attained, some degree of permanent disfigurement is frequent.

Repositioning will be effective in ≈ 85% of cases.

Patients are placed on the unaffected side or on the abdomen.

Infants with occipital flattening from torticollis should have aggressive physical therapy and **resolution should be observed within 3–6 months**.

More severe involvement may be treated with a trial of molding helmets(however, no controlled study has proven the efficacy).

Surgical treatment:
Required in only ≈ 20% of cases.
The ideal age for surgery is **between 6 and 18 months**.

Surgical options range from **simple unilateral craniectomy of the suture** to elaborate reconstruction by a craniofacial team.
Linear craniectomy is often adequate for patients ≤ 12 weeks of age without severe disfigurement.

Better results are obtained with earlier surgery, more radical surgery may be necessary after the age of 6 months.

Multiple synostoses

Fusion of many or all cranial sutures → oxycephaly (tower skull with undeveloped sinuses and shallow orbits). These patients have elevated ICP.

Craniofacial dysmorphic syndromes

Over 50 syndromes have been described number of craniosynostosis syndromes are due to mutations in the FGFR (fibroblast growth factor receptor) genes.

FGFR gene-related craniosynostosis syndromes include some classic syndromes (Apert, Crouzon, Pfeiffer…) as well as several newer entities (Beare-Stevenson, Muenke, Jackson-Weiss syndromes). All exhibit autosomal dominant inheritance.

*single / multi → treat cause

syndromatic / non

unilateral / bilateral mc

more indication for surgery

DDx

lambdoid suture → lazy lambdoid : by physical examination

adult ear foreward → positional
posteriorly → CSinostosis.

* Radiological:

x-ray / CT / bone scan → equivocal diagnose

not used

* 3D reconstruction. [bone]

* Sensetive / Diagnostic

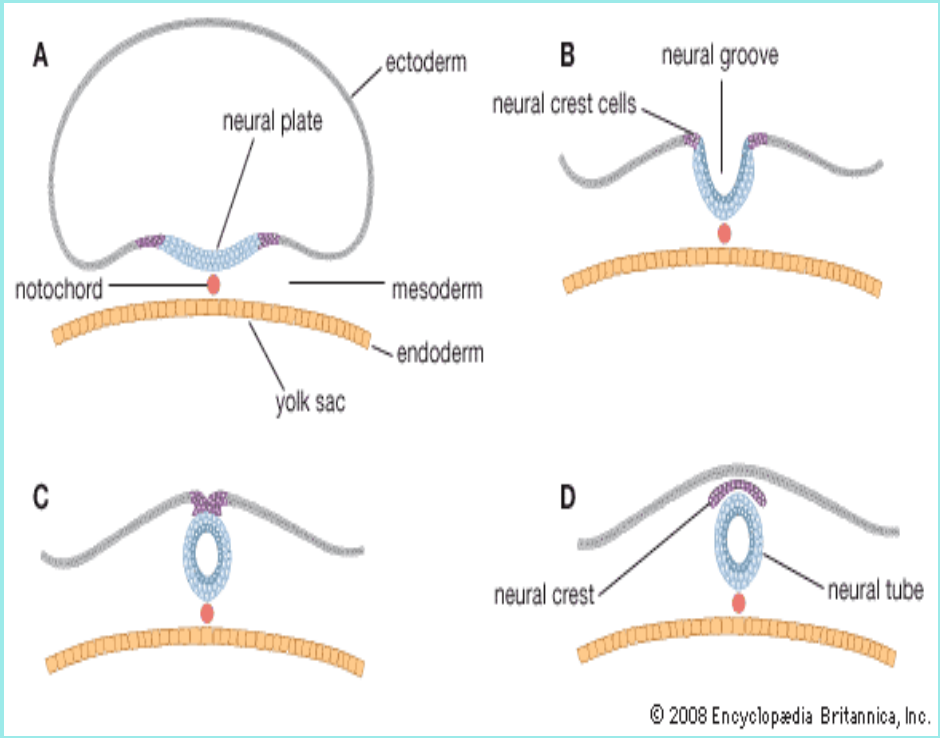
مش قادر اميز
[most sensetive]

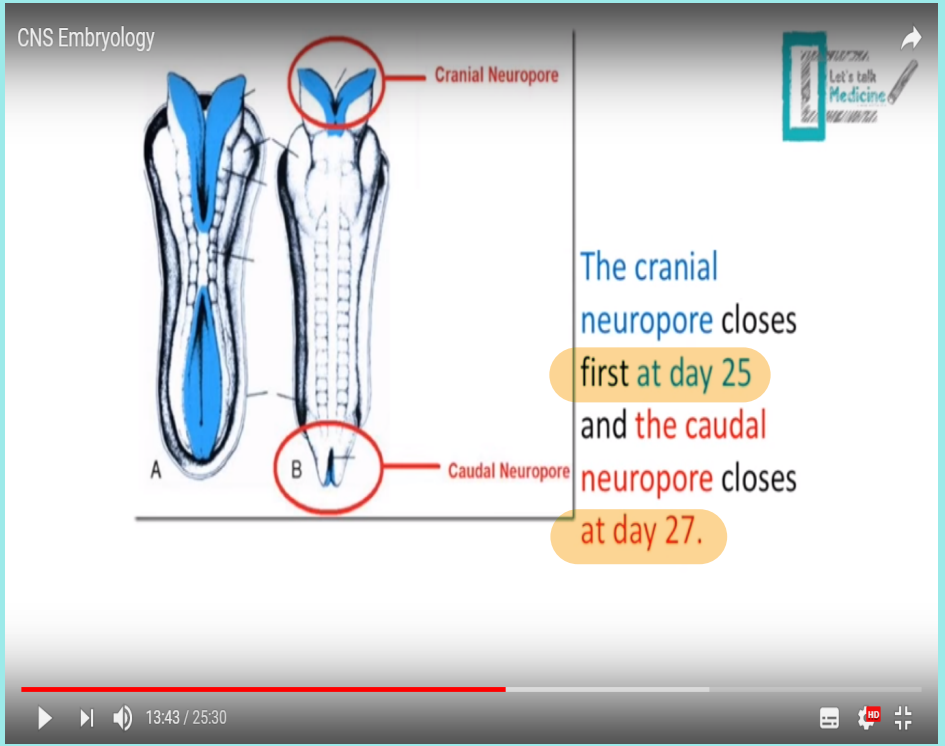
* metabolism
↓ uptake normal suture

metobic type 8—

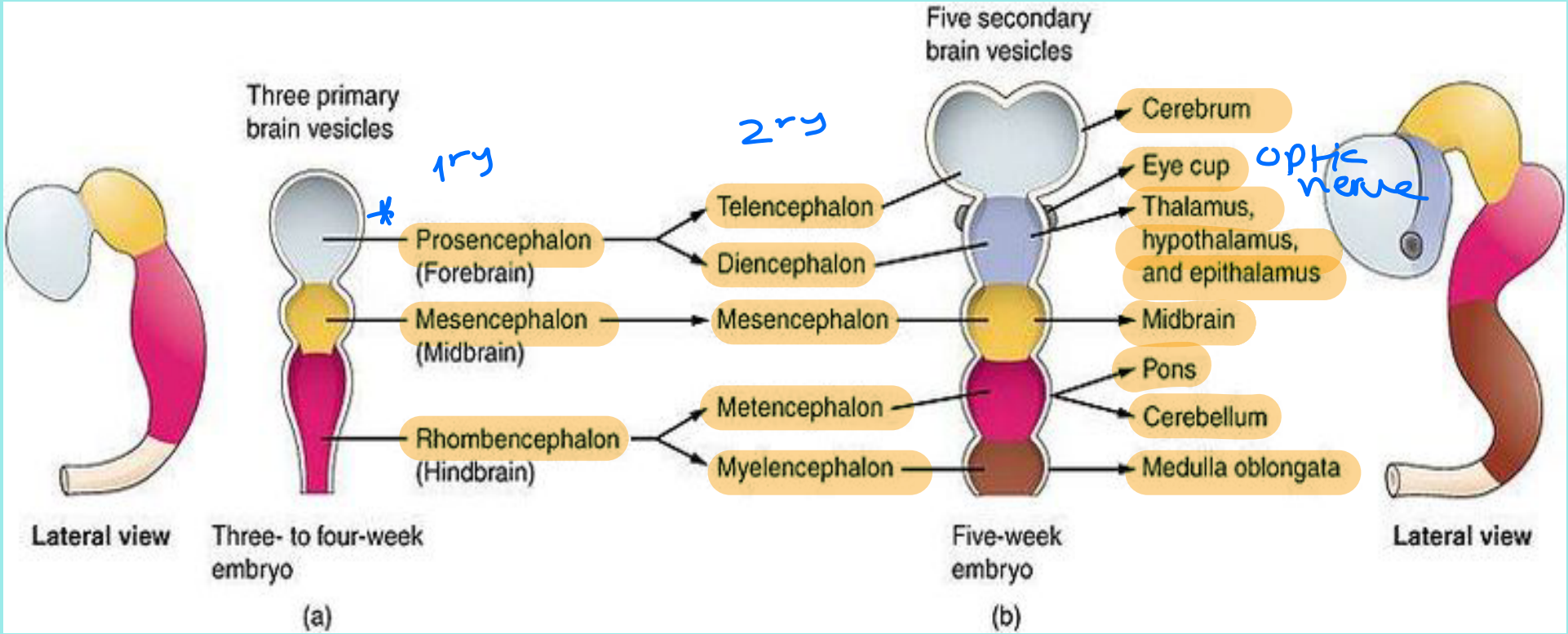
* 10% Hydroceph

Development of the neural tube





Development of the brain



Anencephaly

no brain

Anencephaly

- Serious NTD (failure of closure of cranial neuropore) in which :
  - The brain (cerebral hemispheres) is absent.
  - Cranial vault are grossly malformed.
  - The cerebellum are reduced or absent .
  - The hindbrain is present.

- leading to **early fetal loss, stillbirth, or neonatal death after few hours/ days**.
- "if a baby born with anencephaly is usually blind, deaf, unaware of its surroundings and unable to feel pain".

- Survival period for such a patient is 5 months

Birth Prevalence : 1/10,000 Pregnancy P: 1/1000
Females > Males .

Associated anomalies :
- cleft lip/palate , omphalocele

Etiology

* عشان هيك بنتخي النساء قبل الحمل Folic acid

neural tube defects

1. **Inadequate Folic acid or Antagonists such as :**
Valproic acid , carbamazepine ,phenobarbital , methotrexate trimethoprim

Main ,cause

2- Maternal type 1 diabetes mellitus

3-Maternal Hyperthermia

4-Genetics Family history

5- Amniotic band syndrome .

6-alcohol consumption

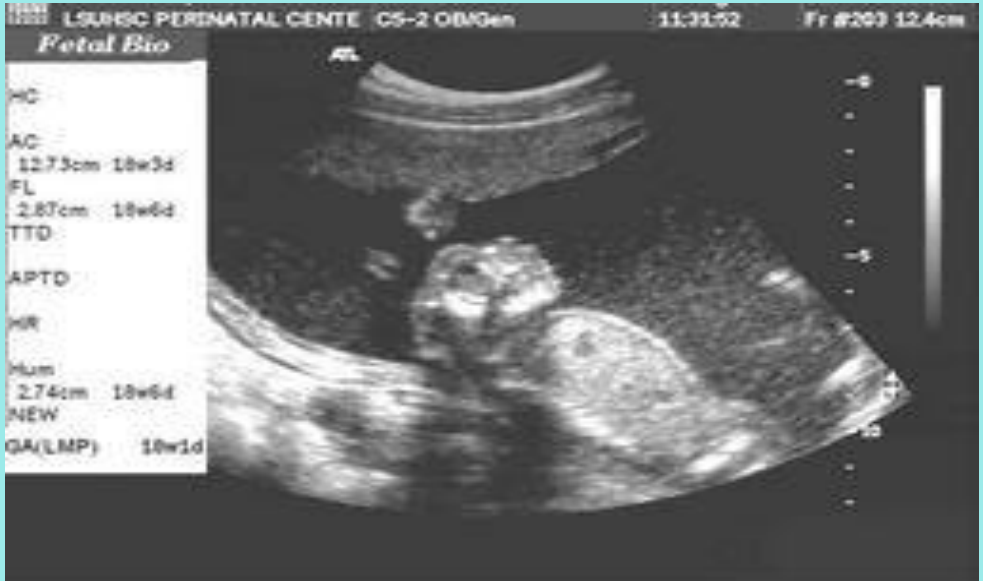
Screening

Lab studies : mainly during second trimester :

- maternal serum AFP , AF AFP ↑ .

Imaging studies :

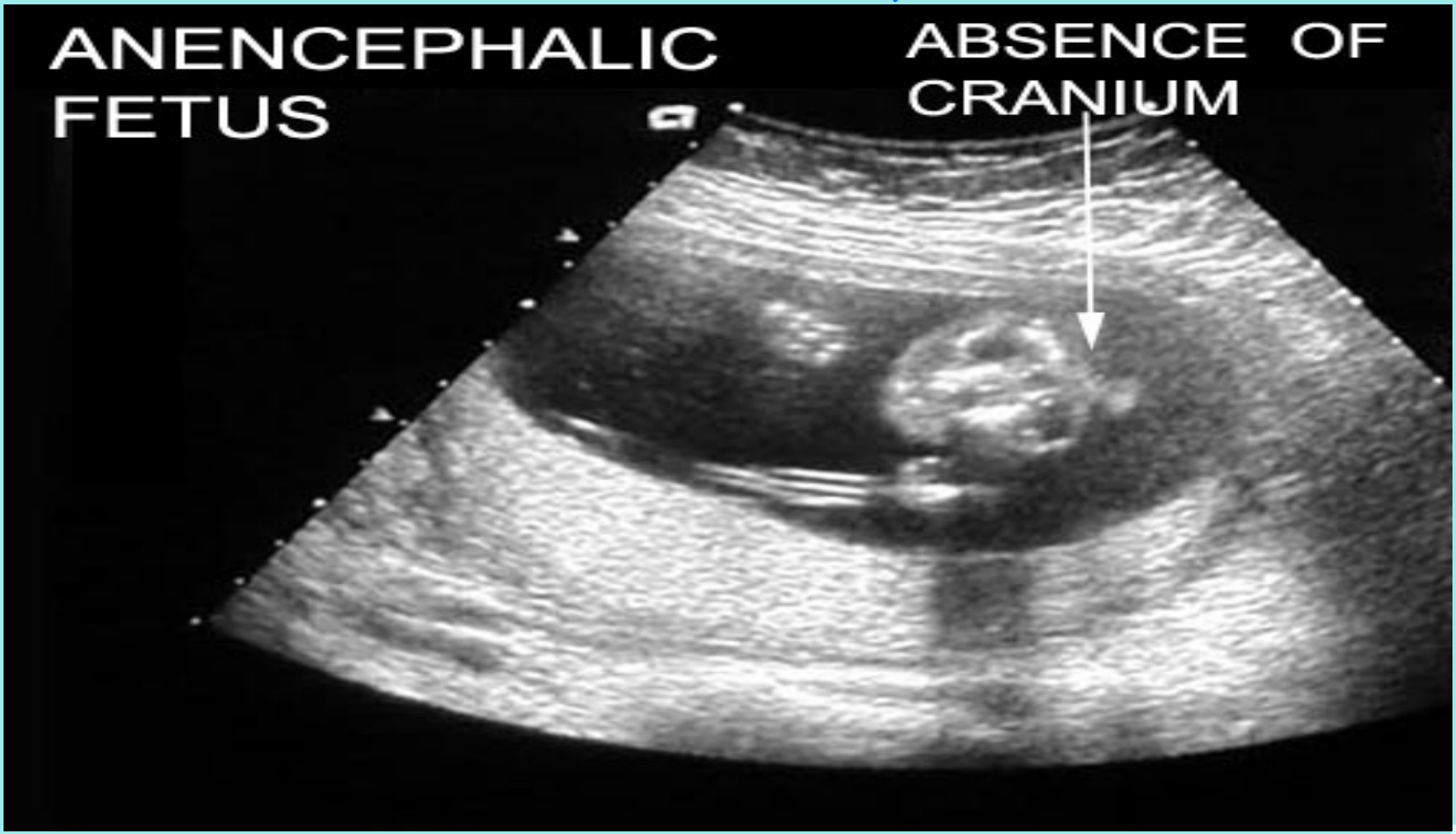
- **US** (identified from 13 week gestation)



- **US** (not diagnostic before 12th week of gestation) :
  1. absence of brain and calvaria superior to the orbits on coronal views of the fetal head
2. reduced crown-rump length
3. Later on : polyhydramnios in 50% .

(neurological problems

swallowing
* defect
* esophageal atresia



- **Prevention :** previous / family history

.**4 mg of folic acid** daily beginning at least 3 months prior to conception is indicated for women who desire pregnancy and have had a child with an NTD or taking anticonvulsants.

- **(0.4mg/day)** is recommended for all women who are pregnant or who may become pregnant.

- Stop the folic acid antagonist at least in the first trimester.

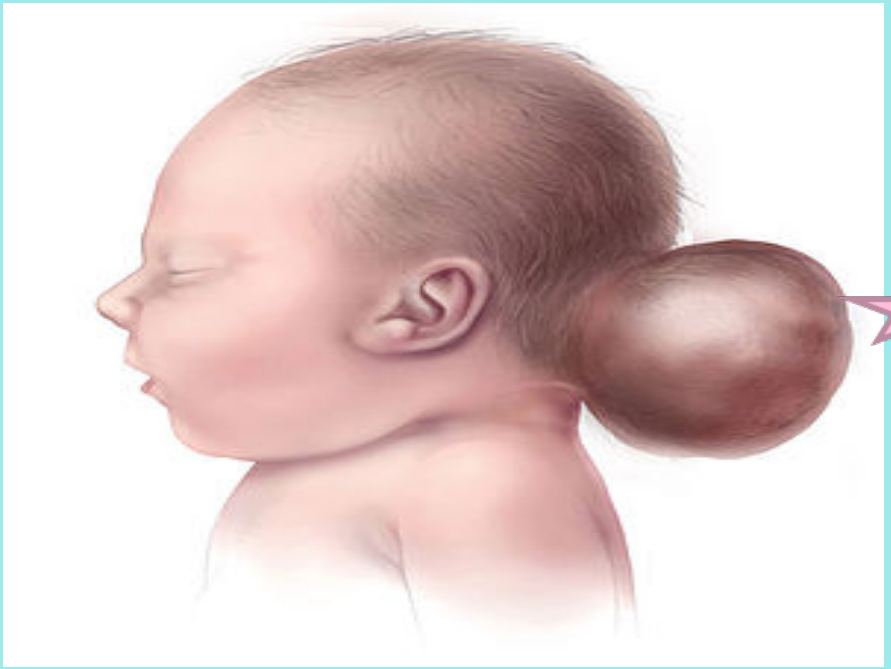
- Control glucose level in diabetic patients.

* Cephalocele

brain

cyst

Cephalocele



* الـ neurological symptoms
تختلف حسب وين مكانها .

refers to the outward herniation of CNS contents through a defect in the cranium.

(failure of rostral / **cranial** end closure)

Most commonly **occipital** then **Parietal**.

Mostly in the midline

- Comment on :

1-Location **Occipital , parietal ,ethmoidal bone ..etc**

2-Contents

3-Associated anomalies

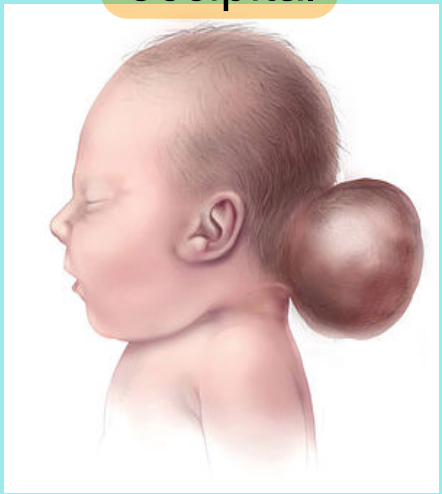
4-Relation to vascular structure

* sphenoidal → normal shape → but by PE: * recurrent meningitis

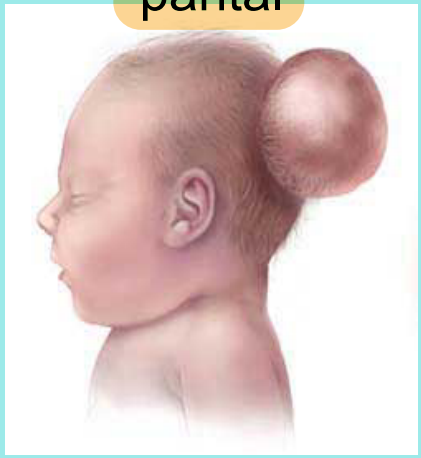
+ CSF leak

1-Location

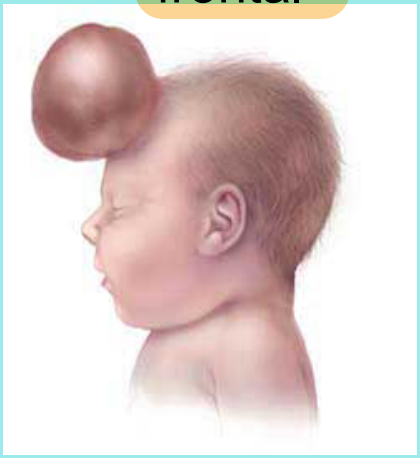
occipital



parital



frontal



ethmoidal

*small in size

*content → low

↳ dysplastic brain tissue

Content

Cephaloceles

Pathological classification (based on the contents of the sac)

- **Meningo-cele**
  - CSF
- **Meningo-encephalocele**
  - CSF
  - BRAIN (dysgentic & nonfunctioning)
- **Meningo-encephalo-cystocele**
  - CSF
  - BRAIN
  - VENTRICLES
- **Atretic cephalocele**
  - Fibro-fatty tissue
- **Gliocele**
  - CSF
  - Glial tissue
    ↳ Fibrosis = gliosis.

* اكثر اشي نخاف امنه في ال content:
ventricles
+ venous.

*venus bleeding of

the sinus is fatal.

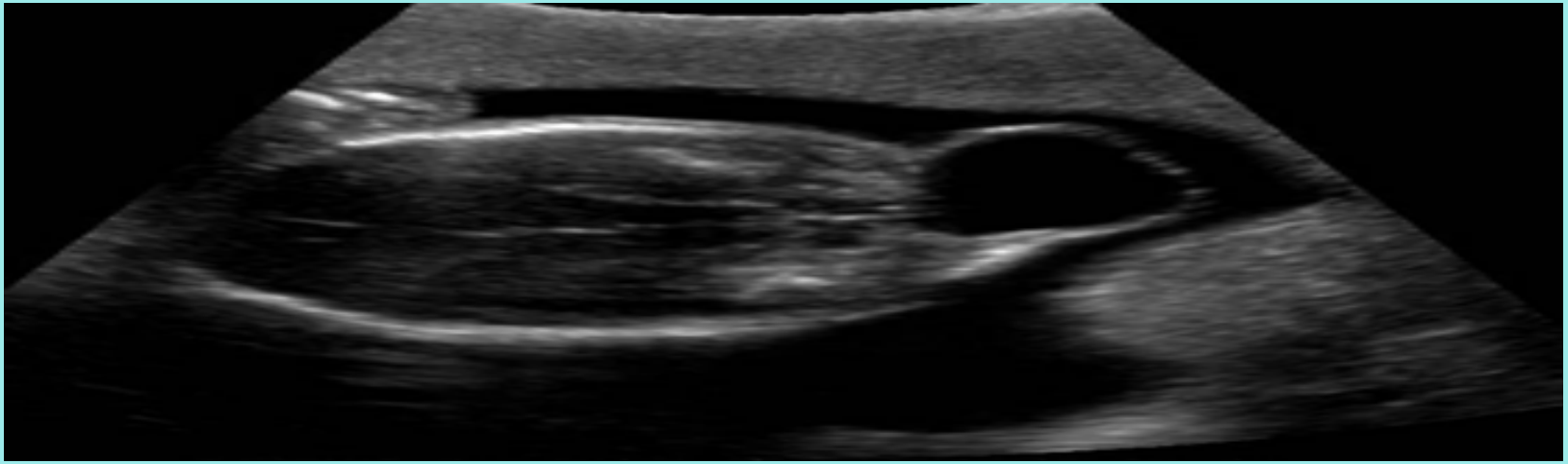
- **Associations** :
  - Trisomy 13 , 18 , hydrocephalus , microceph , Meckle Gruber syndrome ,triad of(occipital encephalocele with multiple renal cysts , postaxial polydactyly).
  - and chiari 3 malformation

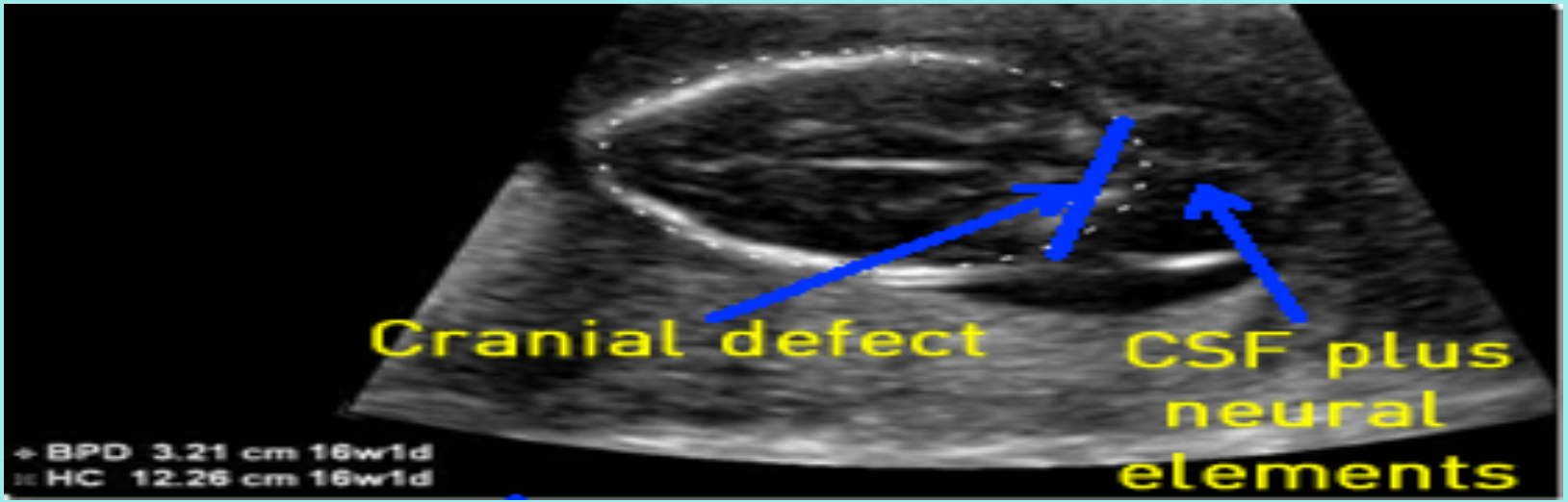
- **Markers** : MS AFP ↑ .
- **Imaging** : **US , MRI** .
- **Management** : **Surgery** to remove the herniated sac (tissue should be excised) and repair the opening in the skull .
- **Excision of herenated sac followed by crainorephy**

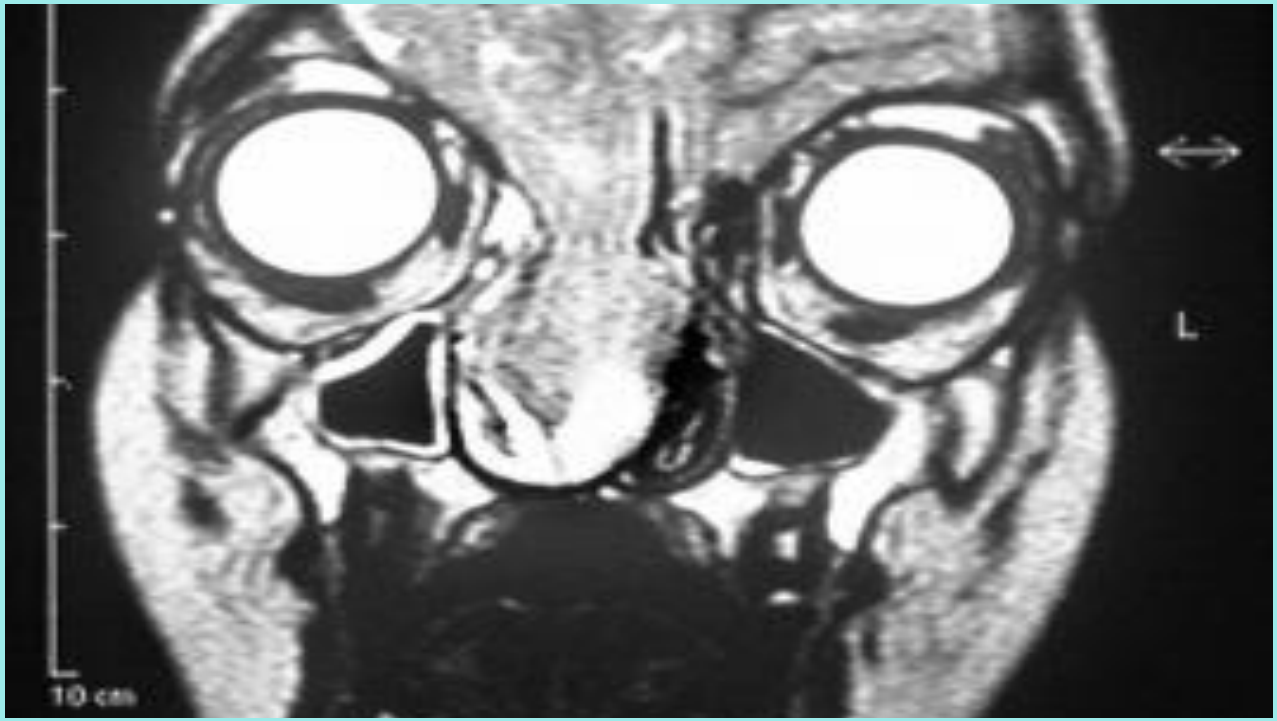
*dysplastic tissue

*اي nasal polyp ماجسئلها الا لما اتأكد مل هي cephalocele و لا polyp عادية

اي اي دماغ ال encephalocele




Cranial defect
CSF plus neural elements
BPD 3.21 cm 16w1d
HC 12.26 cm 16w1d

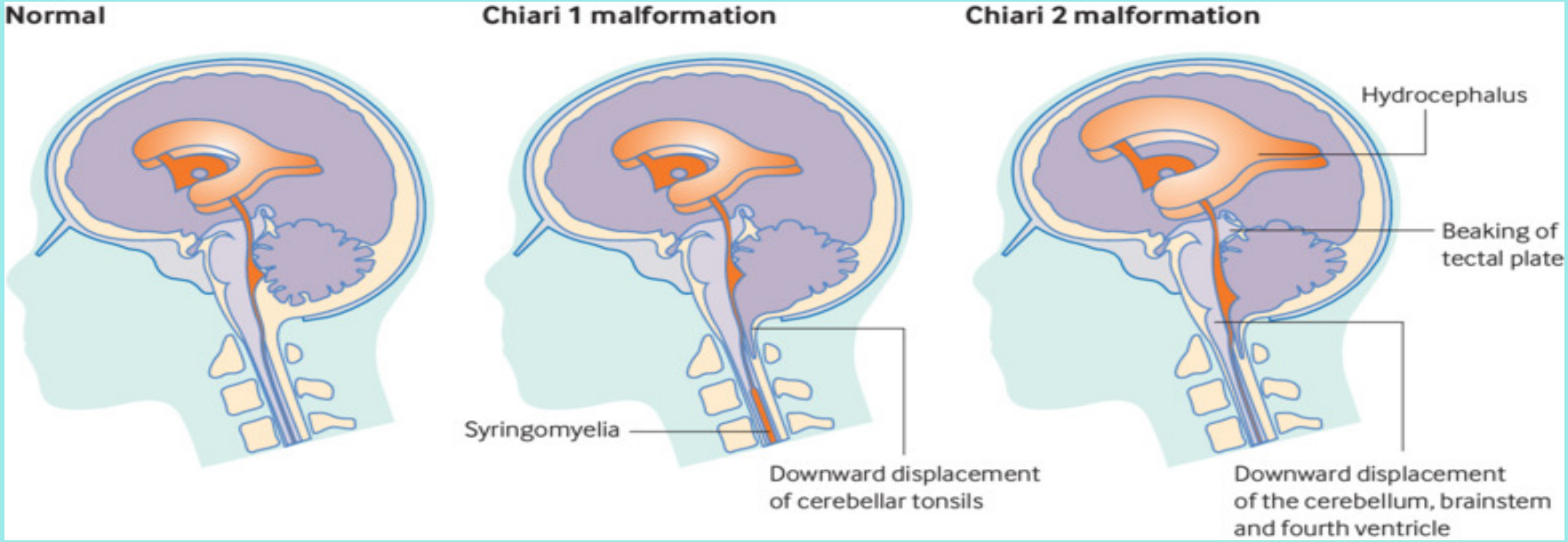


Sphenoidal encephalocele

Chiari Malformation

Chiari Malformation

- Caudal protrusion of “peg-shaped” cerebellar tonsils below foramen magnum



Chiari Malformation

- **congenital caudal 'displacement' of the cerebellum and lower brainstem (hindbrain).**

*4 types :

Type 1 : Most common , least severe.
CD of the **cerebellar tonsils** below the foramen magnum >5mm into the upper cervical canal , usually in **the young adulthood** .

- may be the result of formation of a **small posterior fossa**
  ? overcrowding ? herniation .

بتروح chiari لما تعالج ؟

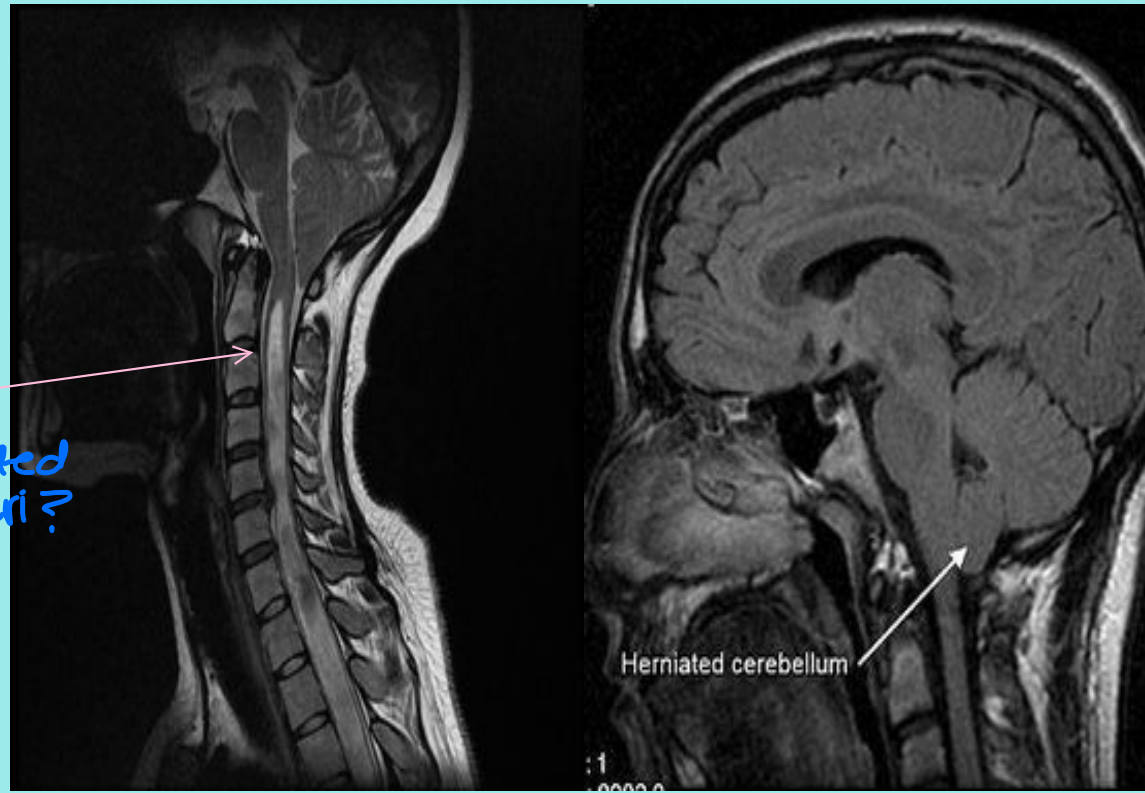
. Syringomyelia ؟ ممكن تعالجها بـ Shunt.

Type 1

Most common type

- Herniation of cerebellar tonsils through foramen magnum
- no herniation of brain stem
- most common form
- usually diagnosed in adolescence or adulthood
- cause syringomyelia → infected chiari ?

(in which a cyst or cavity forms within the spinal cord. This cyst, called a syrinx, can expand and elongate over time)



Type 1 clinical presentation

- Compression of upper cervical cord resulting in myelopathy .
- Compression of cerebellum may result in ataxia, dysmetria, intentional tremor , nystagmus.
- Disruption of CSF flow through foramen magnum : ↑ ICP , suboccipital headache . Neck pain, vomiting , visual defects , hydrocephalus < 10% of cases
- Syringomyelia related symptoms: cape-like loss of pain and temp sense , weakness and wasting of the small muscle of the hand , and progressive motor deficit of the lower and upper limbs

- Chiari 1 : lead to distruption of CSF flow that is caused by cerebellar tonsils herniation جَلك الدماغ بجنبة

. 30% only

Treatment for chiari 1 malformation:

1)Posterior fossa decompression with or without tonsillectomy "
tonsillectomy is done specially if they reach C5 C6 level "

2)If the initial treatment failed we do secondary shunt

Duro-rrhaphy X

so, T; posterior Fossa ← craniotomy.

* Chiari 1 → abnormal → due to narrowing of posterior Fossa. CSF circulation and the level of Foramen magnum. → usually associated with tonsiller descent

Type 1

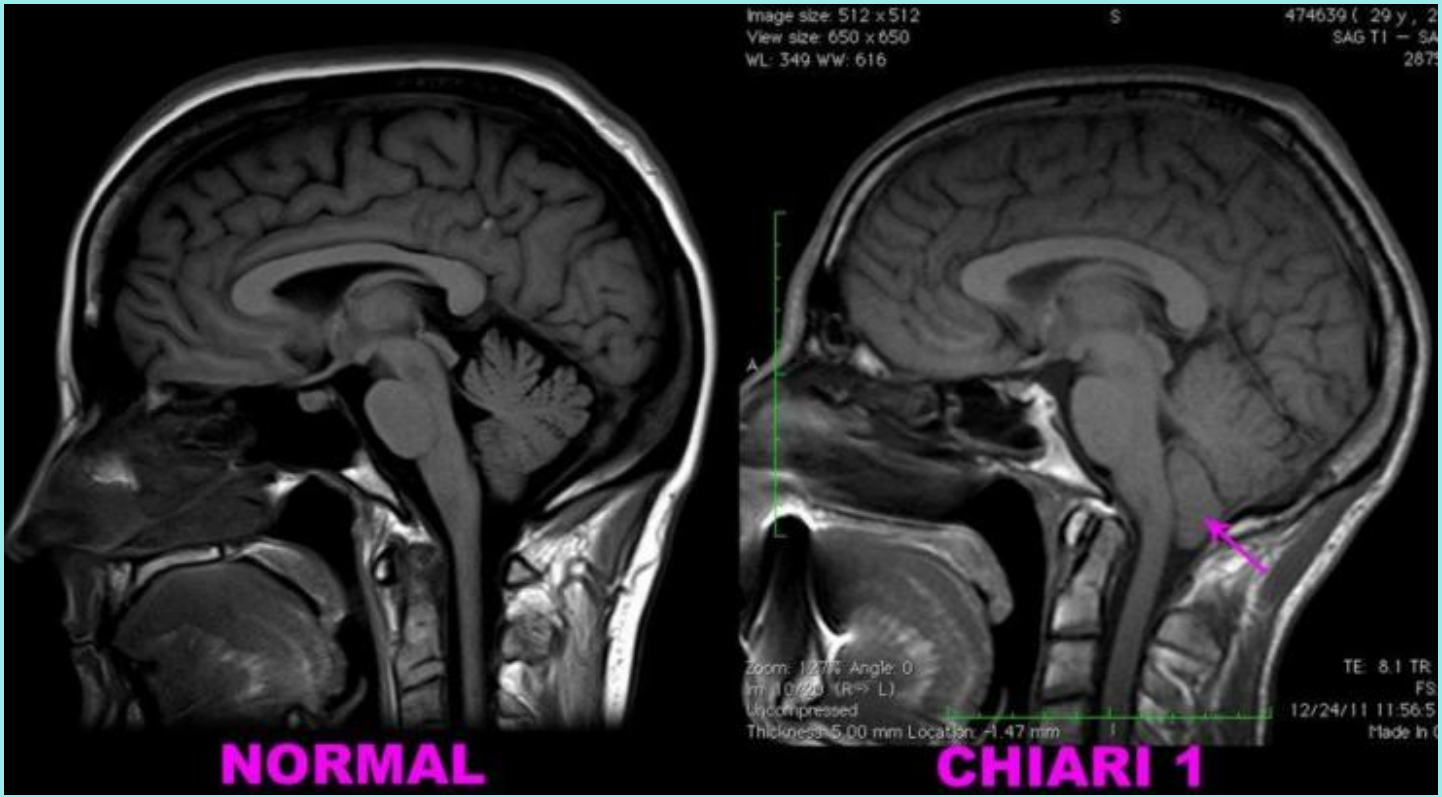


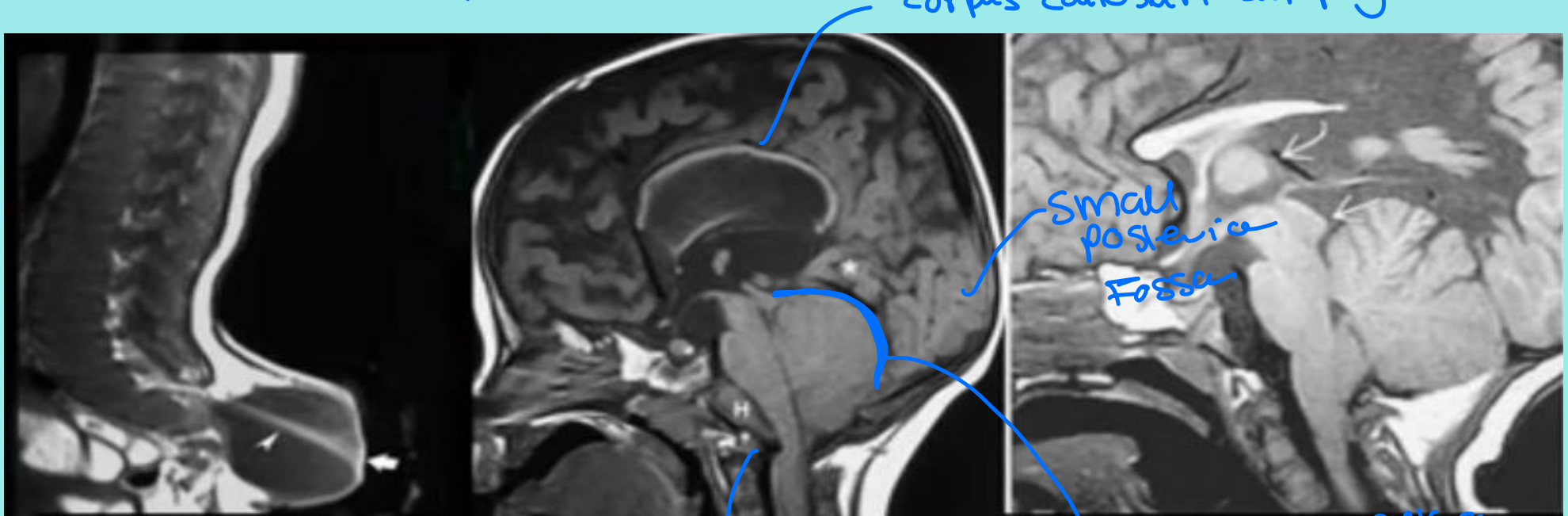
Image size: 512 x 512
View size: 650 x 650
WL: 349 WW: 616
NORMAL
CHIARI 1

Type 2

- caudal displacement of the **cerebellar vermis , 4th ventricle and medulla oblongata** below foramen magnum , present **in infancy** , almost always association with **myelomeningocele .**

-- signs of brainstem dysfunction predominate:
swallowing/feeding difficulties, stridor, apnea, respiratory depression .nystagmus .
Hydroceph. And syringomyelia are more common than type 1 .

97%

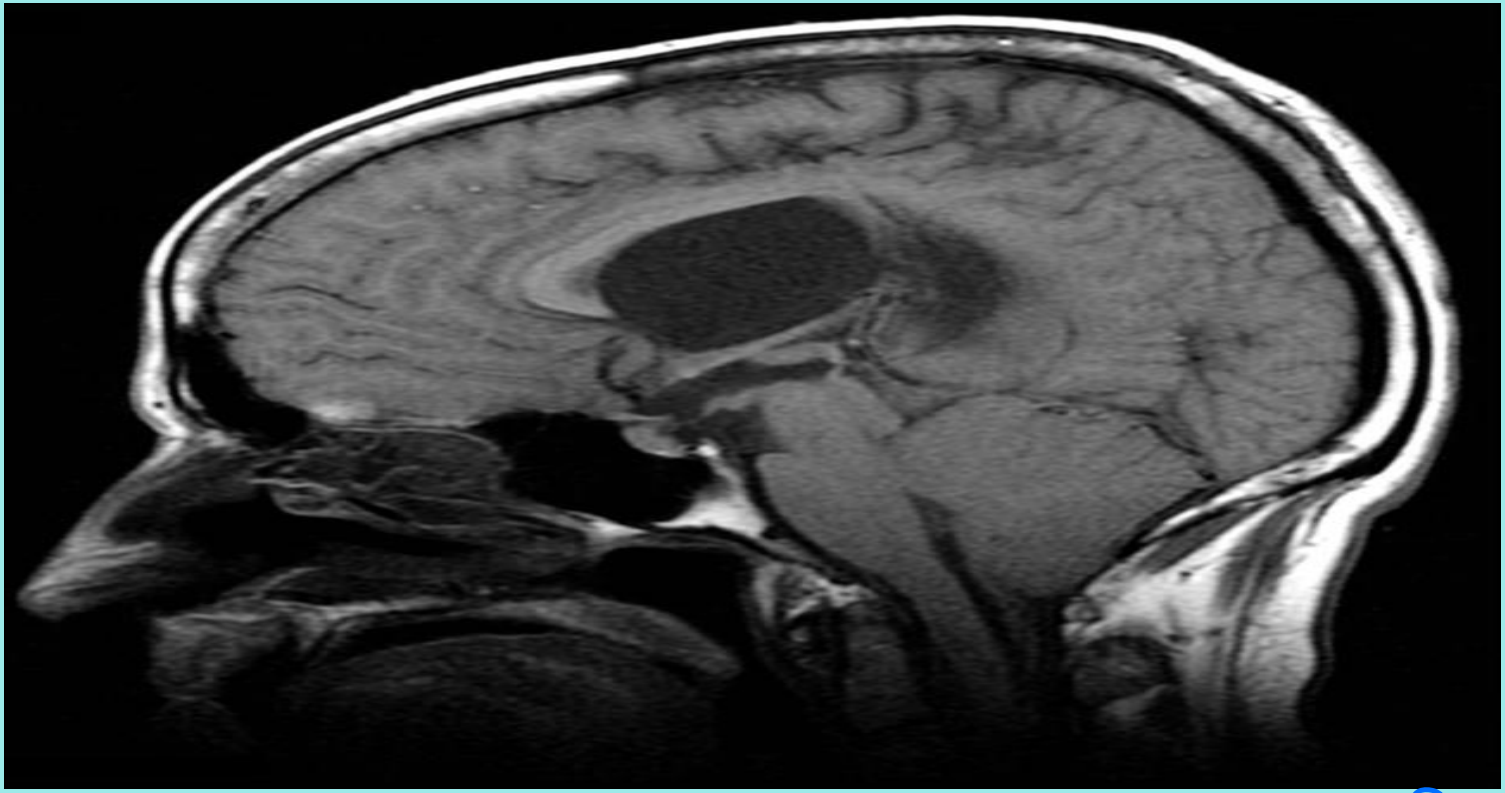


kink (peaking of the tectum)

Type 2

*birds peak.

→ low lying tentorium



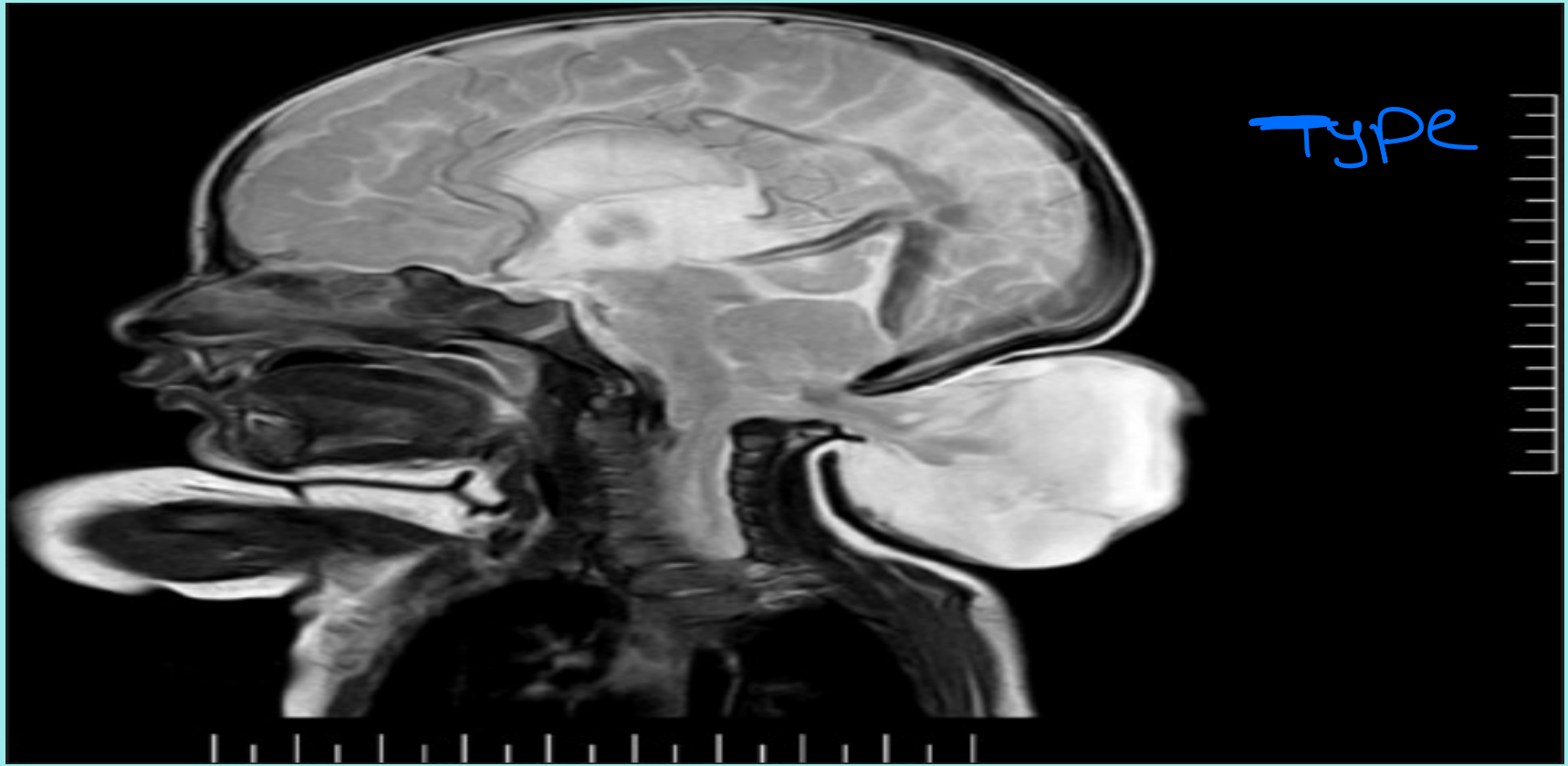
*brain stem dysfunction

*hydrocephalus

* VP shunt in occipital

*need shunt.

bone



All patients with myelomeningocele have chiari 2 malformation

Presentation

-

- Number of them have breathing problems such as difficulties in breathing , o2 sat levels below 80% on chiari 2

Type 3 : similar to Chiari II but with an **occipital and/or high cervical encephalocoele .**

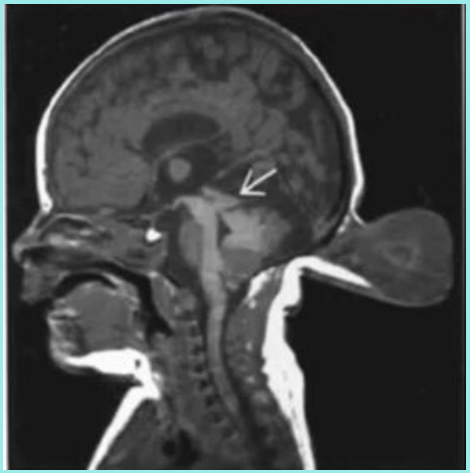
type 2 + encephalocele

Type 4 : cerebellar hypoplasia or agenesis

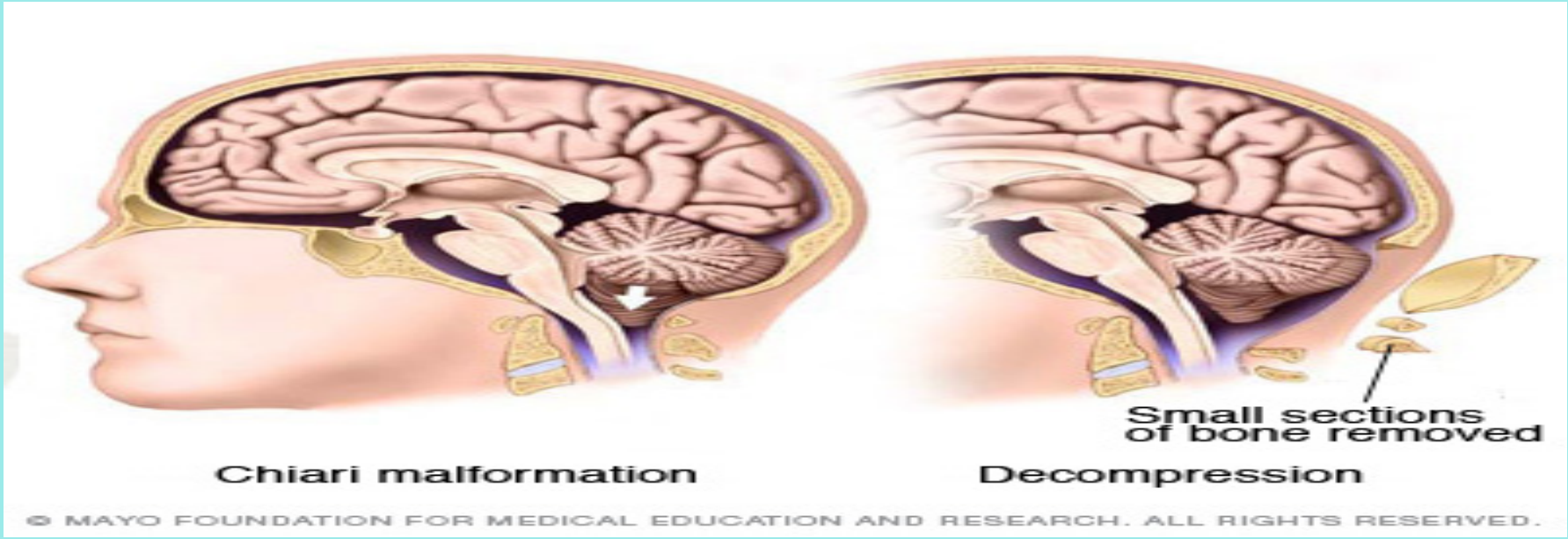
.**Management :**

posterior fossa decompression: your surgeon removes a small section of bone in the back of your skull, relieving pressure by giving your brain more room.

If you have a syrinx or hydrocephalus, you may need a tube (shunt) to drain the excess fluid.



- **Imaging studies** : most useful is MRI .
- **Management** : Post.fossa decompression (mainly for type 1) , VP shunt for hydroceph .



observation

✓ Arachnoid Cysts

- Arachnoid cysts are the most common type of brain cyst. They are often benign congenital, or present at birth (primary arachnoid cysts). Head injury or trauma can also result in a secondary arachnoid cyst.

- An arachnoid cyst forms when the layers of the arachnoid membrane split apart and become filled with cerebrospinal fluid.

- The cysts are fluid-filled sacs, not tumors

mostly asymptomatic

-

*most common presentation of arachnoid cyst. ?
incedental finding [asymptomatic]
SAH

→ ± cysto peri-oneal shunt
→ Fenestration.

Locations :

Sylvian fissure 50% most common.

Cerebellopontine angle 10%

Quadrigeminal 10%

✓ Suprasellar 10% can cause endocrine dysfunction → hypothalamus + pituitary

Vermian 8%

Cerebral convexity 5%

Other 7%

- Arachnoid cyst is mostly presented as incidental findings .

Sylvian fissure

- Most common

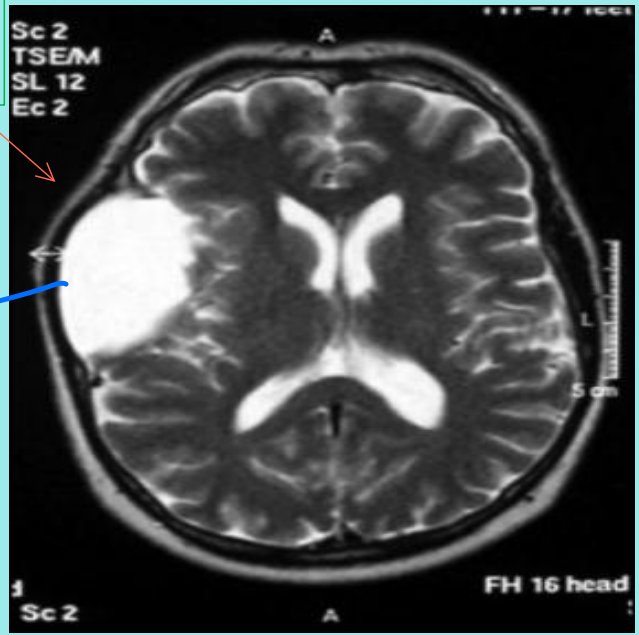
- **Clinical features :**

  1- ↑ ICP : headache , Nausea ,Vomiting

  2- Seizures

  3- with minor head trauma > hrg > acute presentation

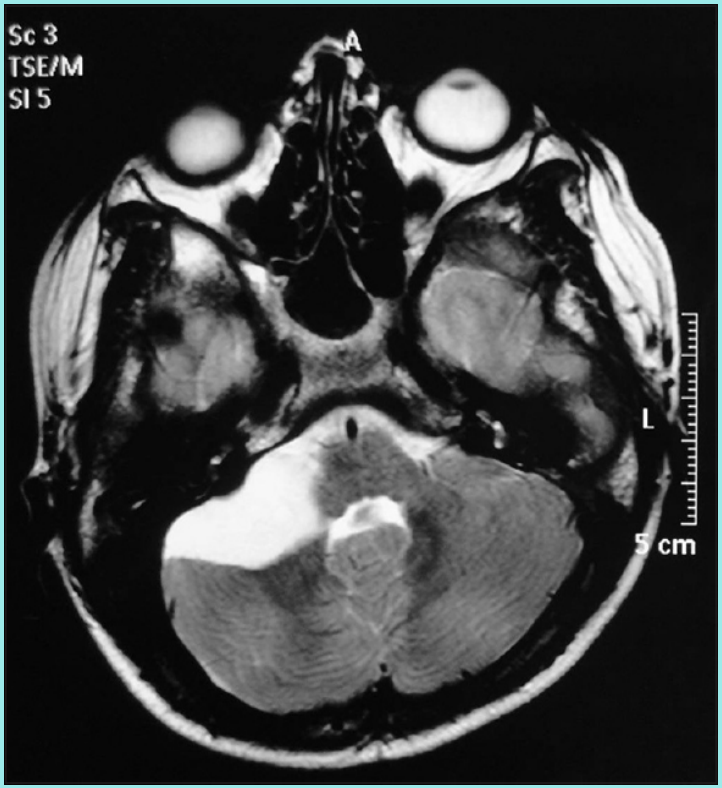
Bone remodeling



Cerebellopontine angle

- Clinical features as in acoustic neuroma :
  - SN hearing loss
  - Tinnitus
  - Vertigo

- May cause compression on 5th CN .



Suprasellar (the only extradural one)

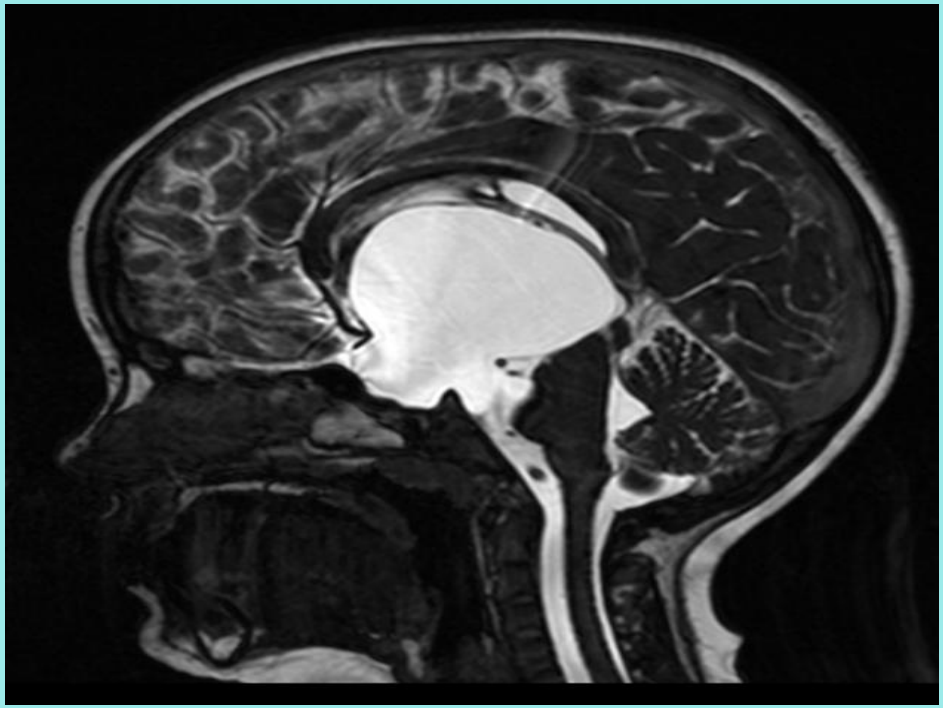
- In children and adolescents .

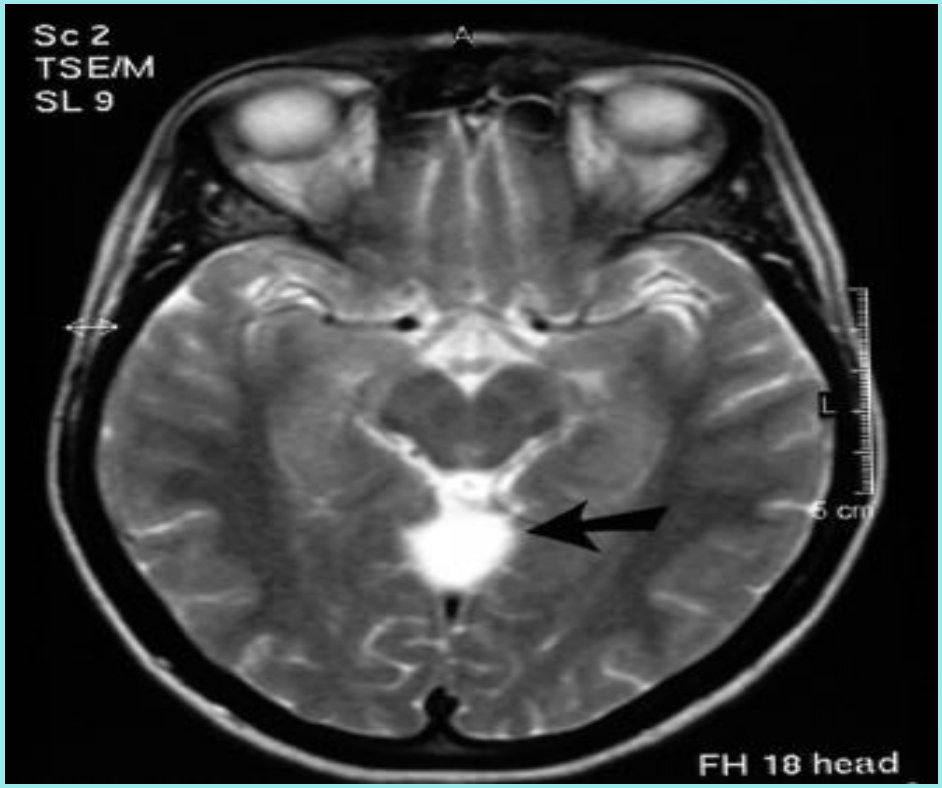
- **Clinical features :**

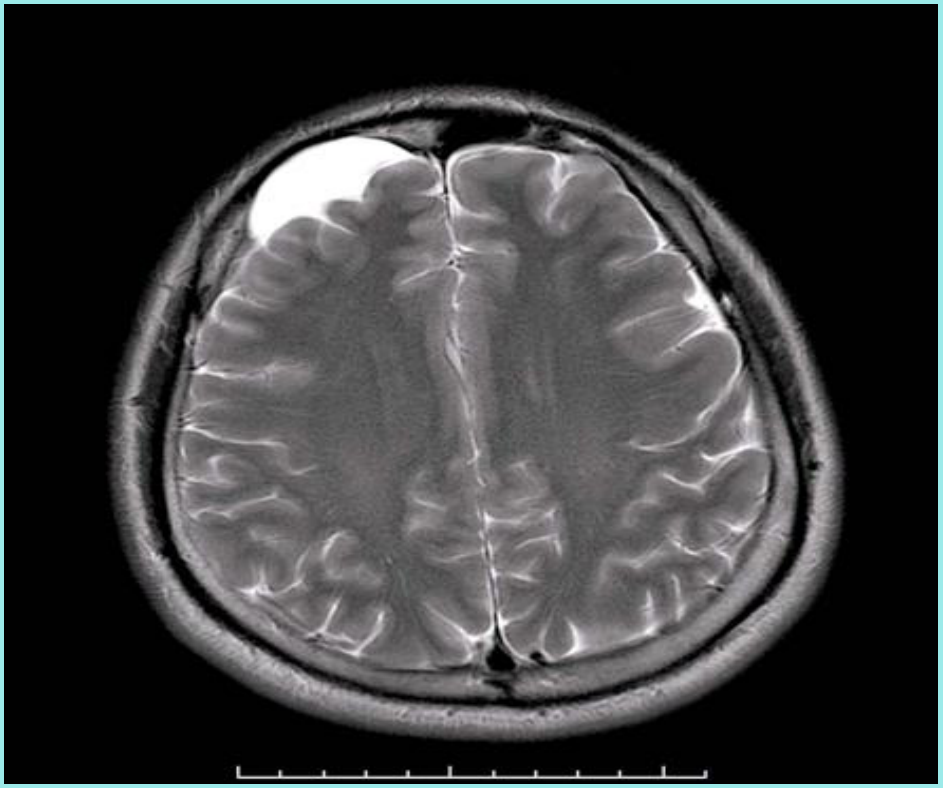
  1- hydrocephalus .

  2- visual impairment.

  3- Endoc. Dysfunction as Precocious puberty .




Sc 2
TSE/M
SL 9
A
5 cm
FH 18 head



- **Management** :

- asymptomatic , no ventricular distortion , enlargment : follow up at regular intervals.

- otherwise :

1- Craniotomy, excision of the cyst wall and opening of the membranes to allow drainage into the basal cisterns.

2- Shunt : cyst – peritoneum

3. endoscopic surgery (endoscopic fenestration)
Flush with ringer lactate.

Dandy-Walker Malformation

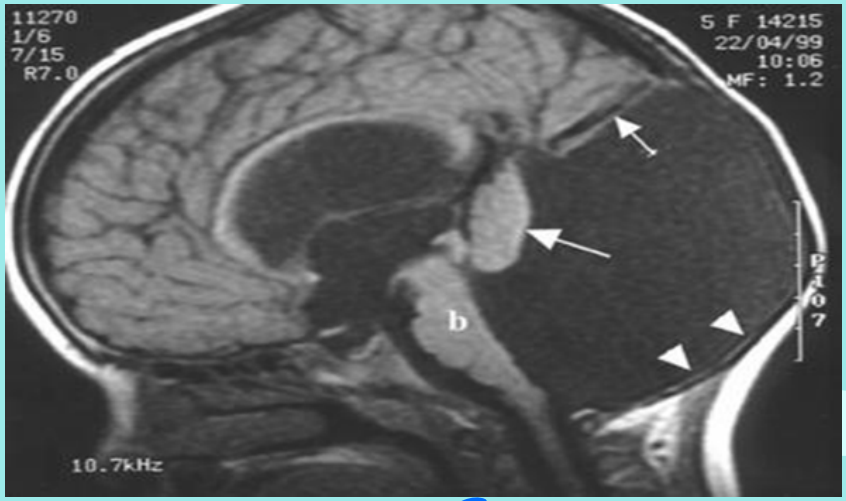
vermis agenesis.

Characterised by :

1 - Complete or partial agenesis of the **cerebellar vermis** .

2- Cystic dilatation of the **4th ventricle** (failure of foraminal outlets to open)

3- An enlarged posterior fossa with upward displacement of **tentorium , lateral sinuses** .



Chiari 2

- symptoms:

1- **90%**of patients have hydrocephalus (present as increasing head size, vomiting, excessive sleepiness, irritability)

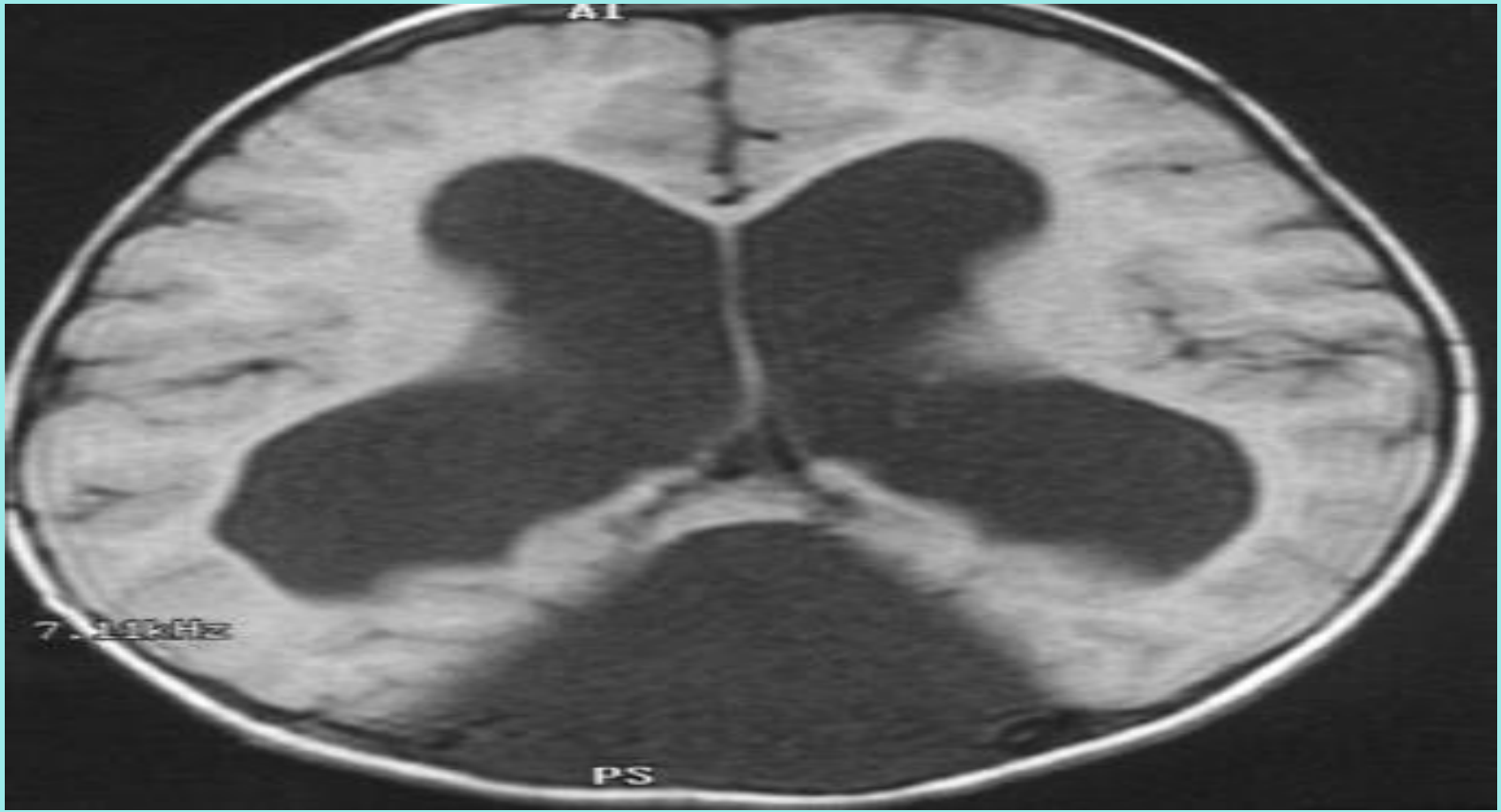
If not diagnosed postnatally : in childhood the major presenting features are ataxia , and delayed motor development

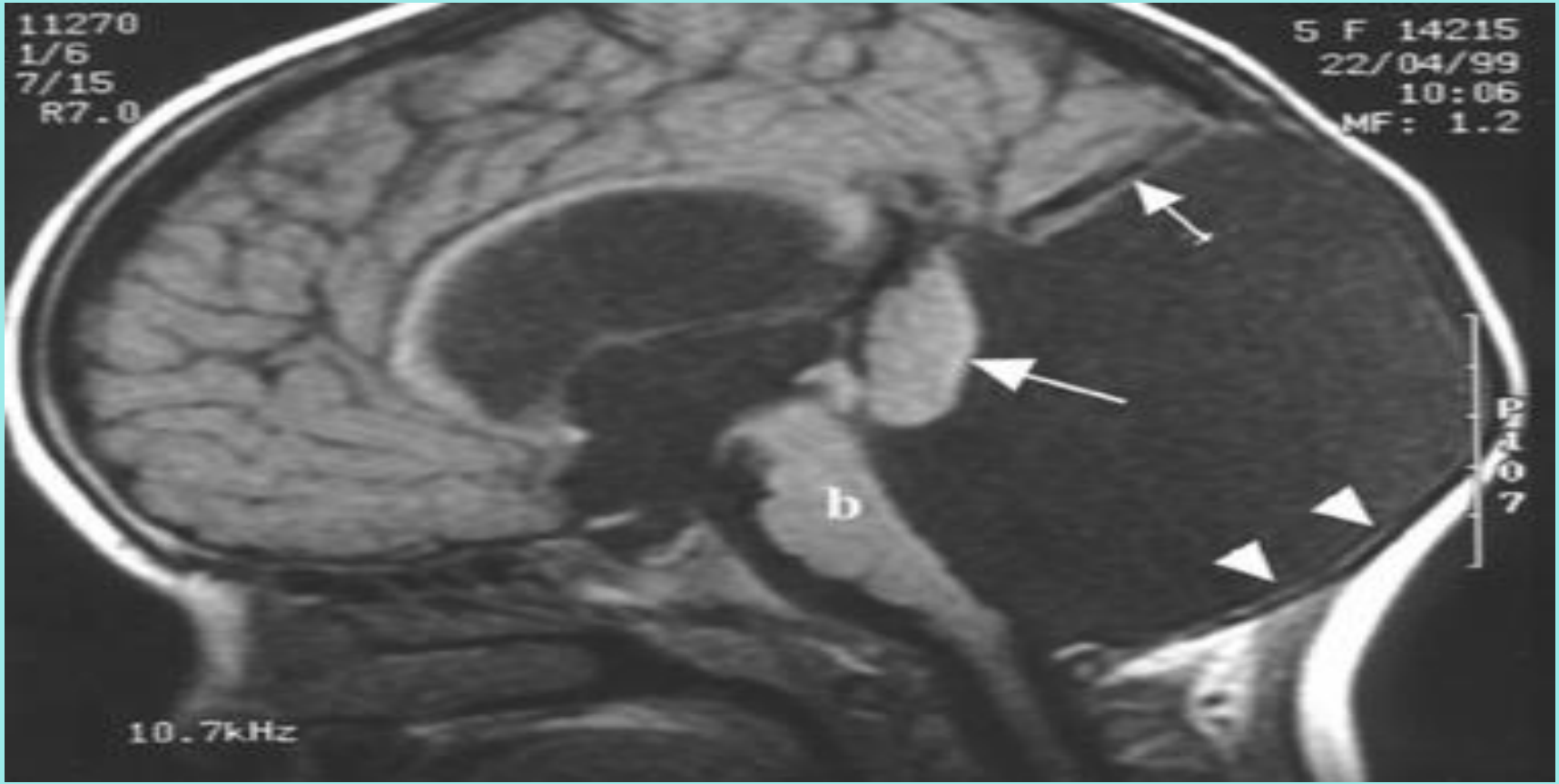
.

3-Associated anomalies : agenesis of the corpus callosum, occipital encephalocele, spina bifida, syringomyelia , cleft palate , cardiac and renal anomalies.

managment

cyst-peritoneal shunt / VP shunt. .


A1
PS


11270
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5 F 14215
22/04/99
10:06
MF: 1.2
b
10.7kHz

THANKS!

Do you have any questions?