سحيّات في طب الأطفال

أخماج الجملة العصبية المركزية

Central Nervous System Infections

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Member of The International Society for Low Vision Research and Rehabilitation

كل الوسائط مسموحة:
تسجيل .. تصوير .. نسخ .. نقل الخ

Serious life-threatening infections

الأخماج الكبرى في الطب عموماً وطب الأطفال خصوصاً

أخماج الجملة العصبية المركزية
انتانات الدم

التهابات السحايا
التهابات السحايا والدماغ
التهابات الدماغ
الارتكاسات السحائية
الخراجات العصبية المركزية

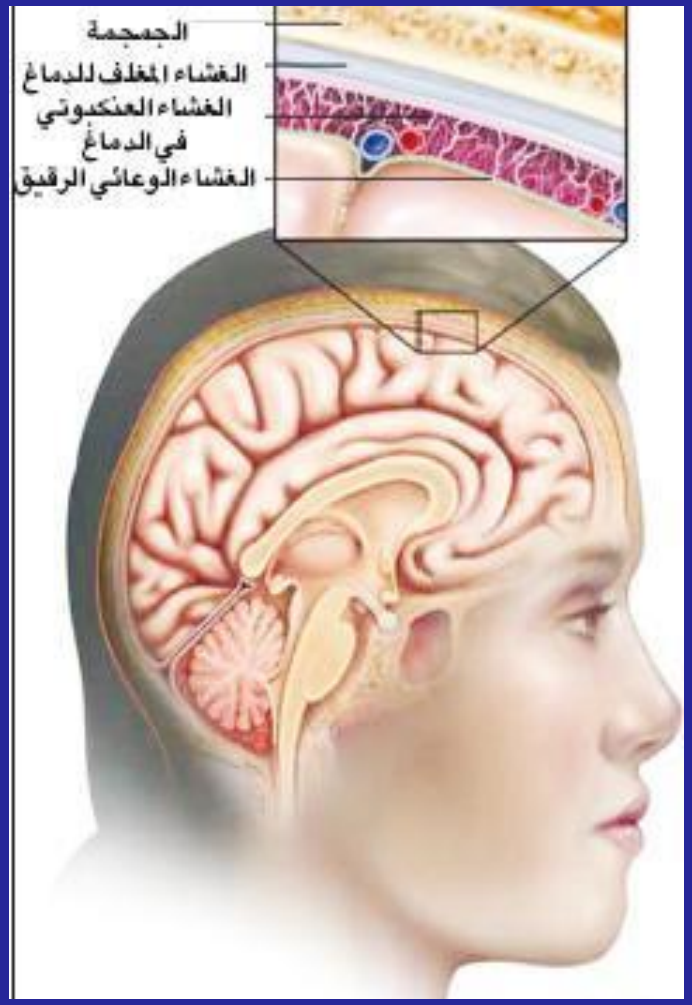
Sepsis

This is considered in Chapter 6 (Paediatric Emergencies).

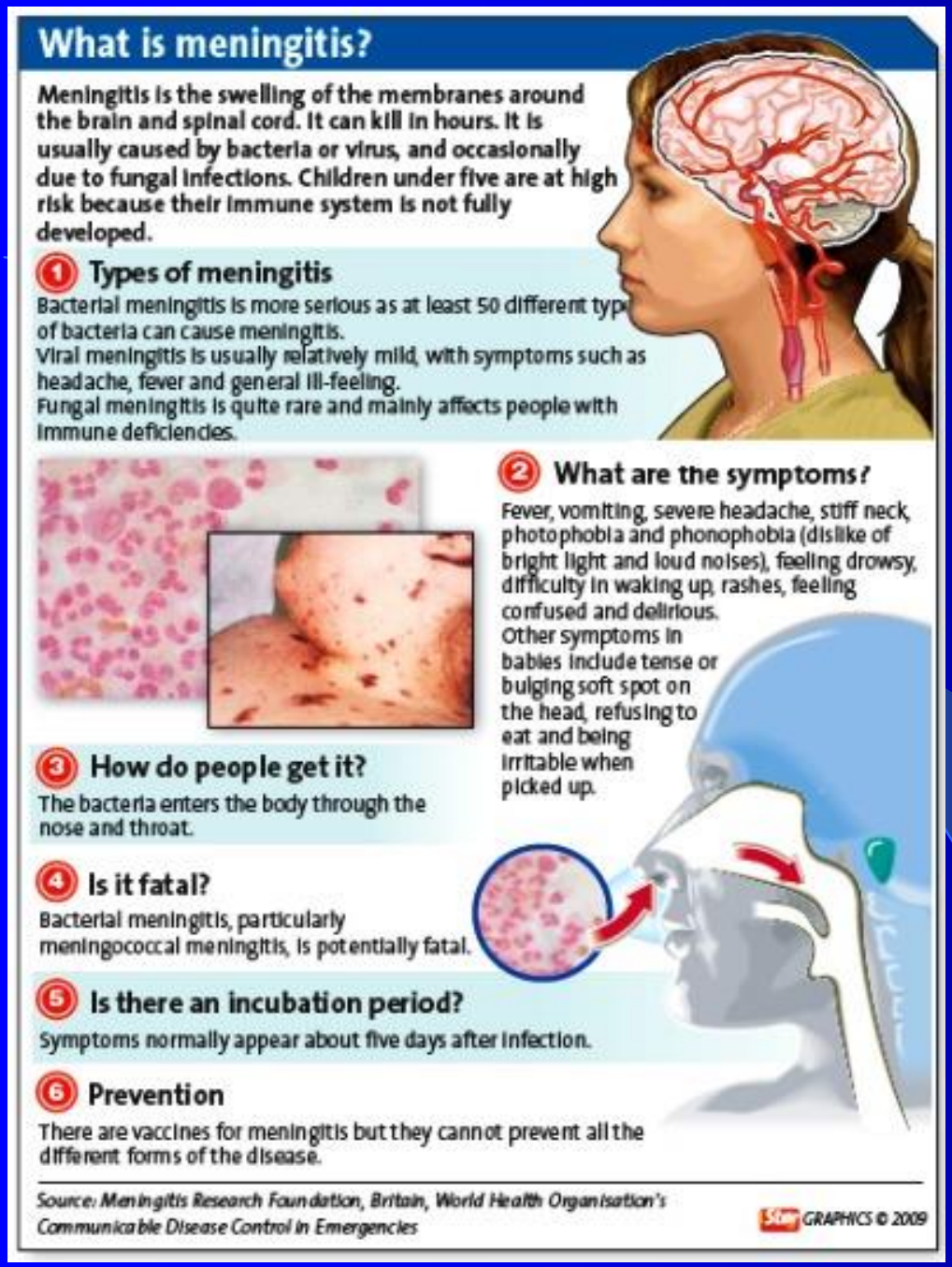
Meningitis

Meningitis occurs when there is inflammation of the meninges covering the brain. This can be confirmed by finding white blood cells in the cerebrospinal fluid (CSF). Viral infections are the most common cause of meningitis, and most are self-resolving. Bacterial meningitis may have severe consequences. Tuberculous meningitis is rare in countries with low TB prevalence. TB meningitis mainly affects children under 5 years of age. Fungal and parasitic meningitis are rare in children and predominately affect immunocompromised individuals. Causes of noninfectious meningitis include malignancy and autoimmune diseases.

التهاب السحايا ..



- قيحي أم عقيم
  Septic vs Asptic



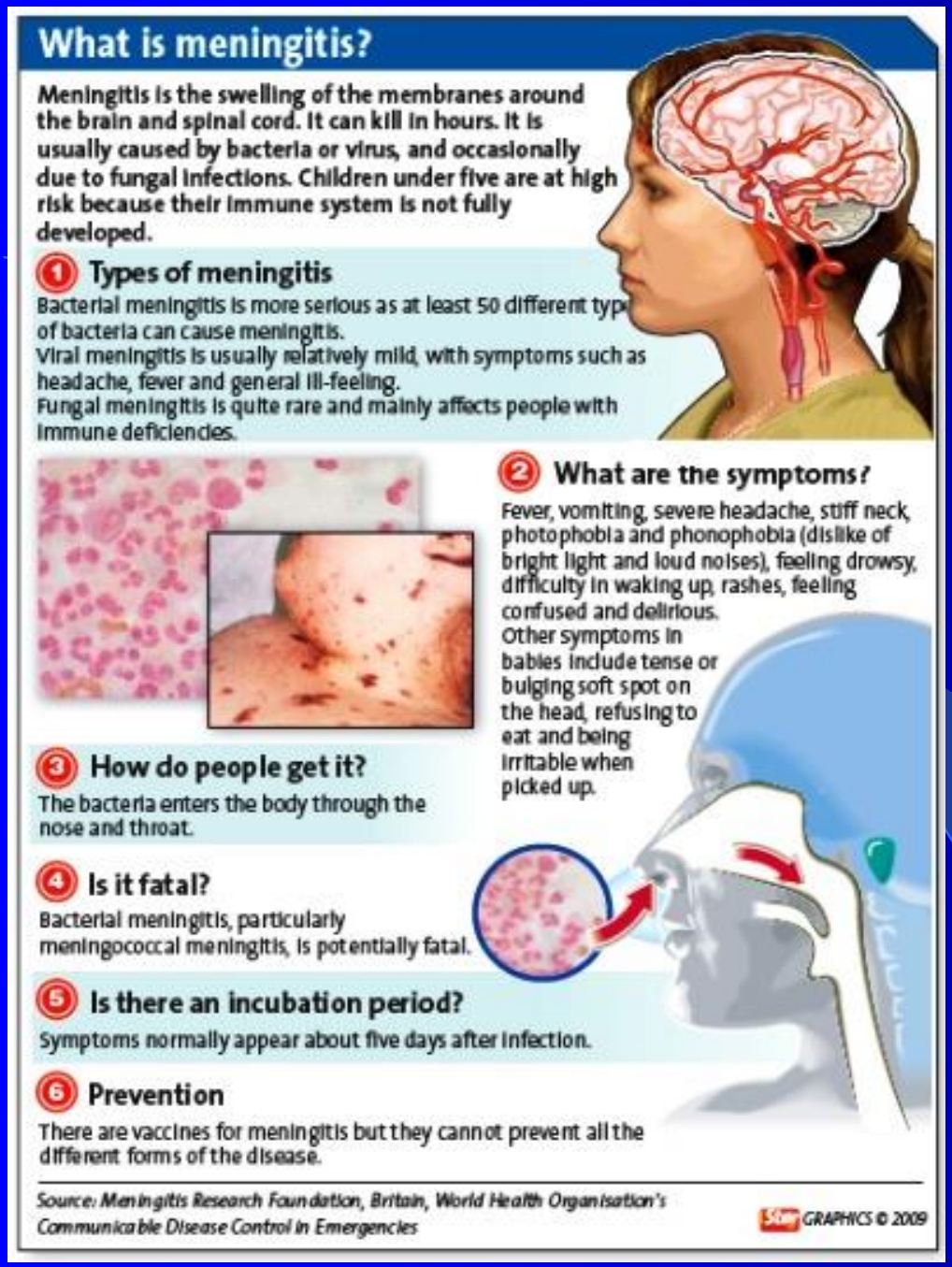
التهاب السحايا القيحي

Septic Meningitis

التهاب السحايا الجرثومي
Bacterial Meningitis

* الجرثومي أشمل.

* القيحي تحت عباءة الجرثومي.



التهاب السحايا العقيم

Aseptic Meningitis

التهاب السحايا الفيروسي
Viral Meningitis

* الفيروسي جزء كبير من العقيم.

* العقيم أشمل.

Bacterial meningitis

Over 80% of patients with bacterial meningitis in the UK are younger than 16 years of age. Bacterial meningitis remains a serious infection in children, with 5% to 10% mortality. Over 10% of survivors are left with long-term neurological impairment.

Mortality/Morbidity

- **Despite advances in care for patients with bacterial meningitis, the overall case fatality remains steady at approximately 5-30% worldwide.**

Pathophysiology

- Bacteria reach the subarachnoid space by a hematogenous route and may directly reach the meninges in patients with a parameningeal focus of infection.

Pathophysiology

Bacterial infection of the meninges usually follows bacteraemia. Much of the damage caused by meningeal infection results from the host response to infection and not from the organism itself. The release of inflammatory mediators and activated leucocytes, together with endothelial damage, leads to cerebral oedema, raised intracranial pressure, and decreased cerebral blood flow. The inflammatory response below the meninges causes a vasculopathy resulting in cerebral cortical infarction, and fibrin deposits may block the resorption of CSF by the arachnoid villi, resulting in hydrocephalus.

Organisms

The organisms that commonly cause bacterial meningitis vary according to the child's age (Table 15.1). These have changed over time with the introduction of conjugate vaccines [against *H. influenzae* type b (Hib), meningococcal group C (and recently A, C, Y, and W), and multiple pneumococcal serotypes]. The effect of the introduction of a new recombinant group B meningococcal vaccine remains to be seen on both the individual and at the population level.

Table 15.1 Organisms causing bacterial meningitis according to age

Neonatal to 3 months	Group B streptococcus
	Escherichia coli and other coliforms
	Listeria monocytogenes
1 month to 6 years	*Neisseria meningitides*
	Streptococcus pneumoniae
	Haemophilus influenza
>6 years	*Neisseria meningitides*
	Streptococcus pneumoniae

Presentation

The clinical features are listed in Fig. 15.4. The early signs and symptoms of meningitis are nonspecific, especially in infants and young children. Only children old enough to talk are likely to describe the classical meningitis symptoms of headache, neck stiffness, and photophobia. However, neck stiffness may also be seen in some children with tonsillitis and cervical lymphadenopathy. As children with meningitis may also have sepsis, signs of shock, such as tachycardia, tachypnoea, prolonged capillary refill time and hypotension, should be sought. Purpura in a febrile child of any age should be assumed to be due to meningococcal sepsis, even if the child does not appear unduly ill at the time; meningitis may or may not be present in this situation.

Assessment & investigation of meningitis/encephalitis

History	Examination	Investigations
Fever	Fever	Full blood count and differential count
Headache	Purpuric rash (meningococcal disease)	Blood glucose and blood gas (for acidosis)
Photophobia	Neck stiffness (not always present in infants)	Coagulation screen, C-reactive protein
Lethargy	Bulging fontanelle in infants	Urea and electrolytes, liver function tests
Poor feeding/vomiting	Opisthotonus (arching of back)	Culture of blood, throat swab, urine, stool for bacteria
Irritability	Positive Brudzinski/Kernig signs	Rapid antigen test for meningitis organisms (can be done on blood, CSF, or urine)
Hypotonia	Signs of shock	Samples for viral PCRs (e.g. throat swab, nasopharyngeal aspirate, conjunctival swab, stool sample)
Drowsiness	Focal neurological signs	Lumbar puncture for CSF unless contraindicated (see below for tests on CSF)
Loss of consciousness	Altered conscious level	Serum for comparison of convalescent titres
Seizures	Papilloedema (rare)	PCR of blood and CSF for possible organisms
		If TB suspected: chest X-ray, Mantoux test and/or interferon-gamma release assay, gastric aspirates or sputum for microscopy and culture (and PCR if available)
		Consider CT/MRI brain scan and EEG

History	Examination
Fever	Fever
Headache	Purpuric rash (meningococcal disease)
Photophobia	Neck stiffness (not always present in infants)
Lethargy	Bulging fontanelle in infants
Poor feeding/vomiting	Opisthotonus (arching of back)
Irritability	Positive Brudzinski/Kernig signs
Hypotonia	Signs of shock
Drowsiness	Focal neurological signs
Loss of consciousness	Altered conscious level
Seizures	Papilloedema (rare)

BABIES & TODDLERS

Fever – cold hands & feet

Refusing food or vomiting

Fretful, dislike of being handled

Pale blotchy skin

Blank, staring

Drowsy,

Stiff neck,

High pitched

CHILDREN/ADULTS

Stiff neck

Headache

Fever

Vomiting

Light Sensitivity

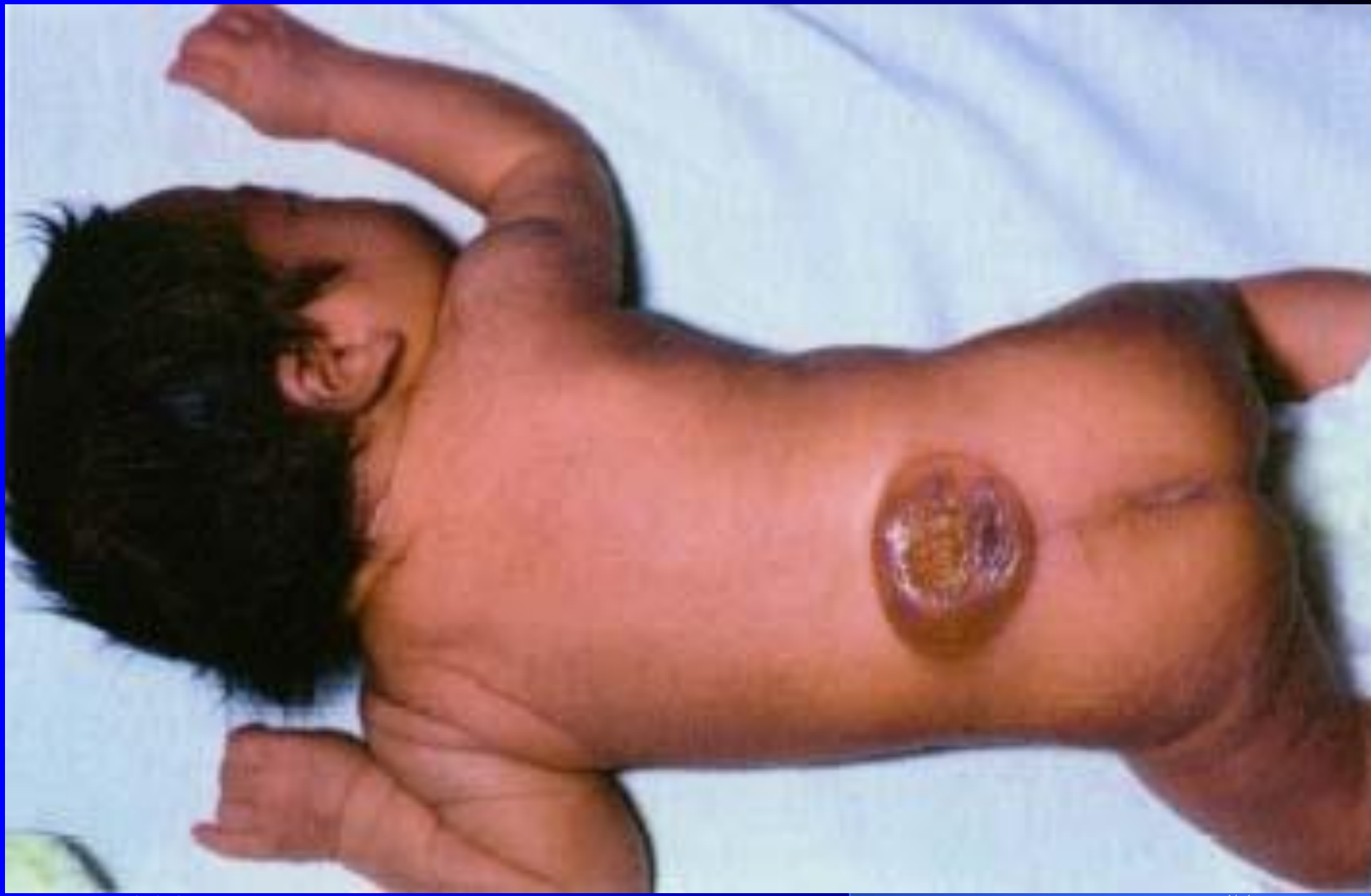
Drowsiness or confusion

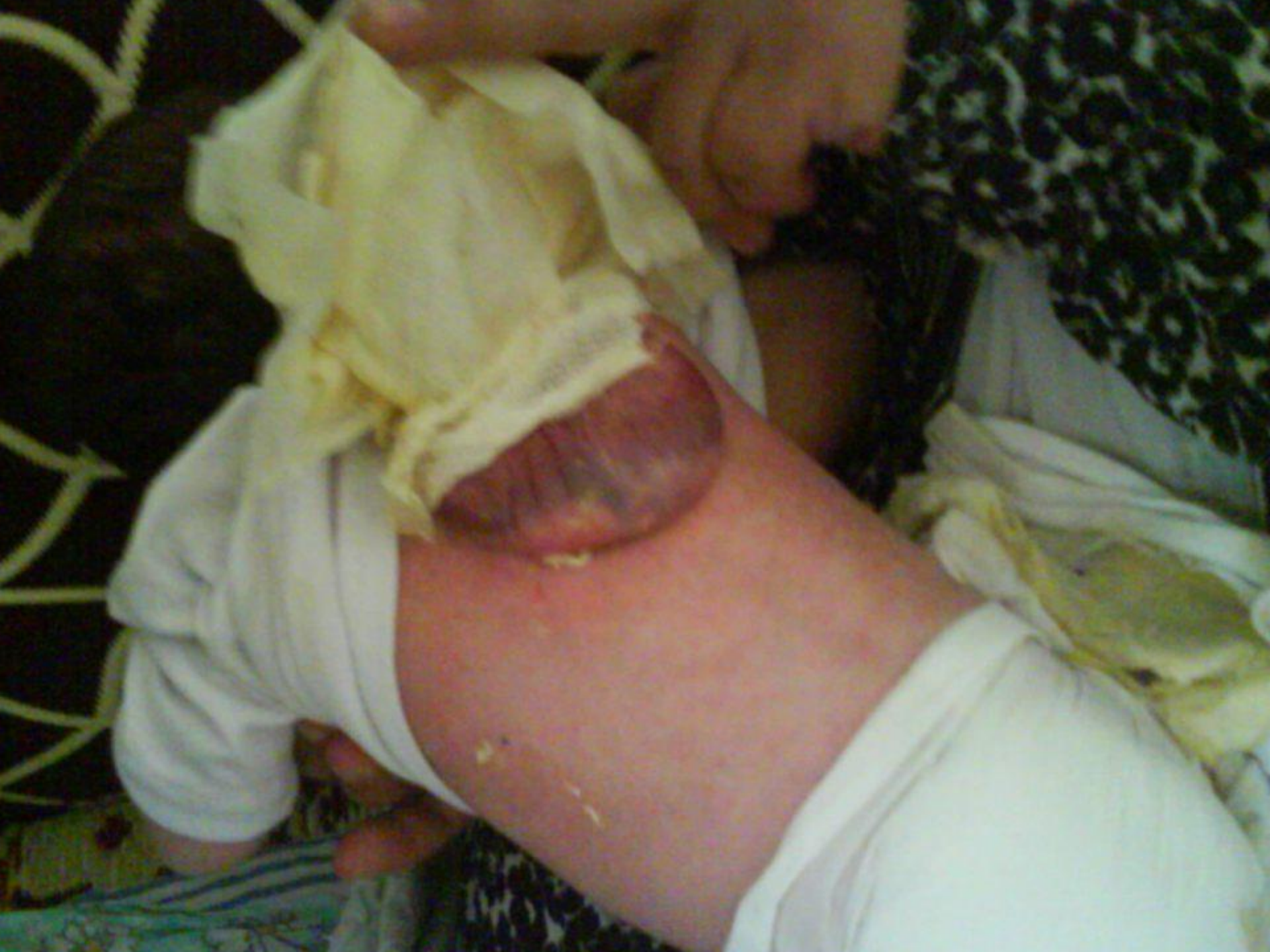
Joint pain

Fitting

Risk factors

- Age
- Low family income
- Attendance at day care
- Head trauma
- Splenectomy
- Chronic disease
- Children with facial cellulitis, periorbital cellulitis, sinusitis, and septic arthritis.
- Maternal infection and pyrexia
- Some malformations.





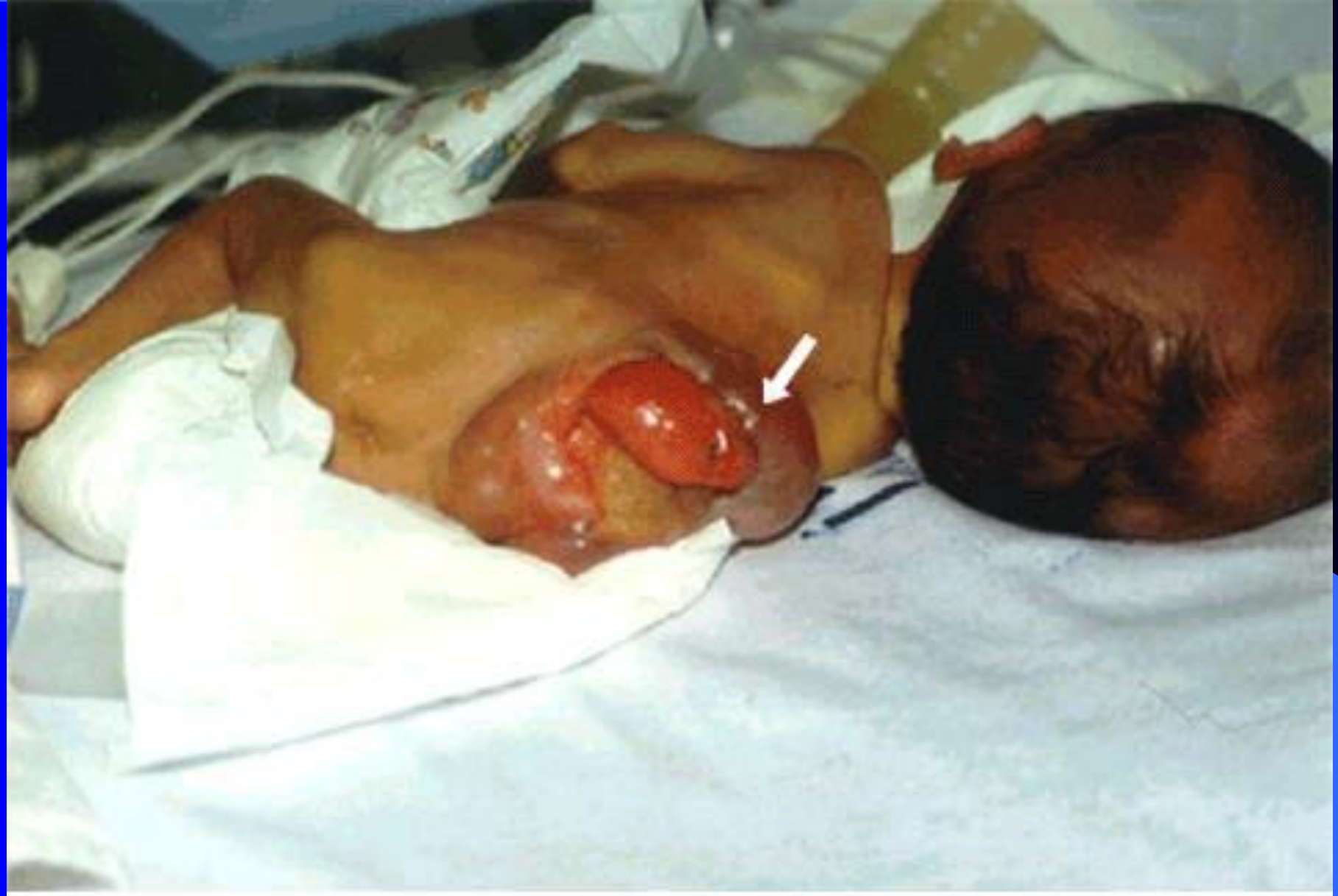
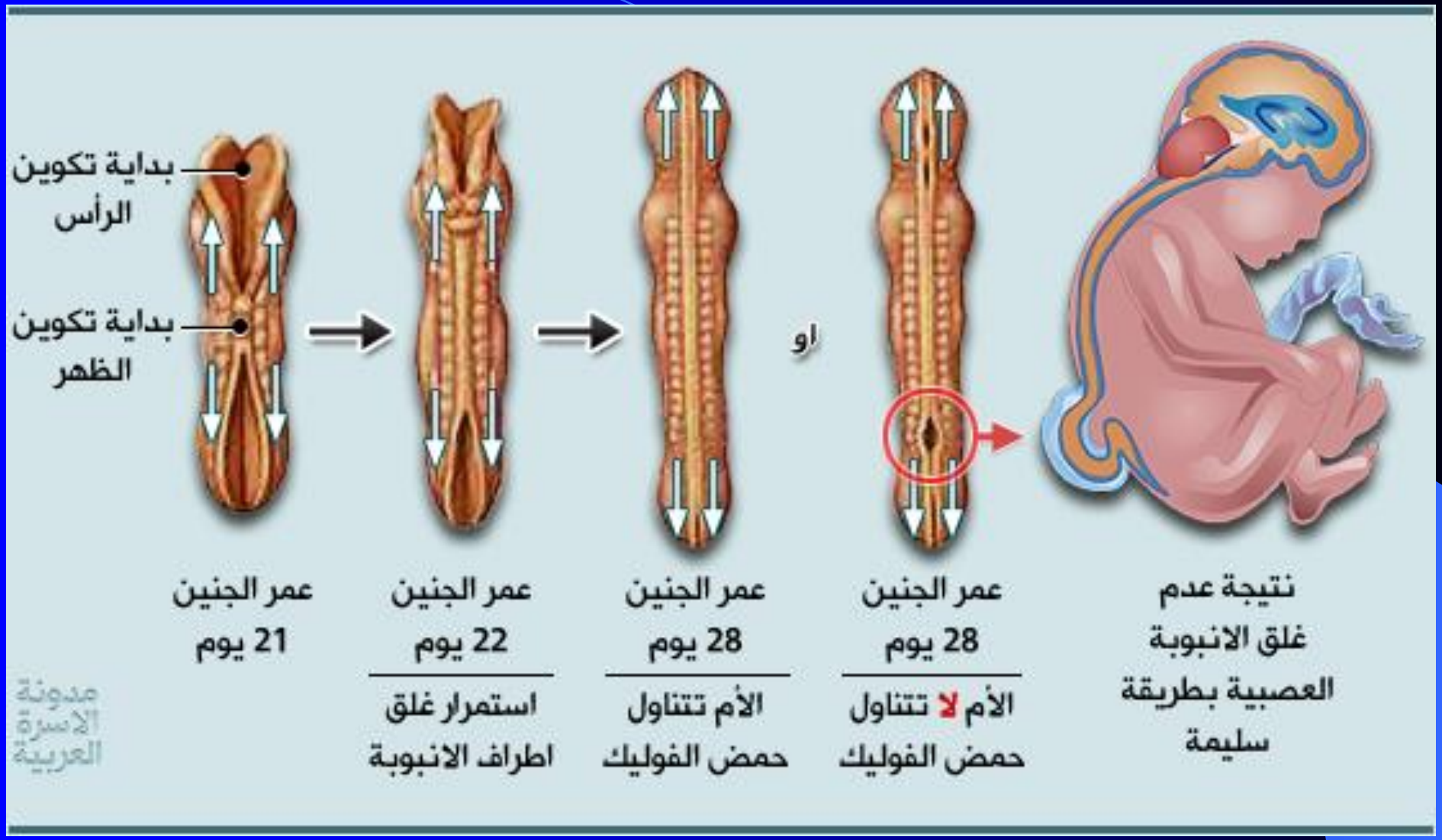
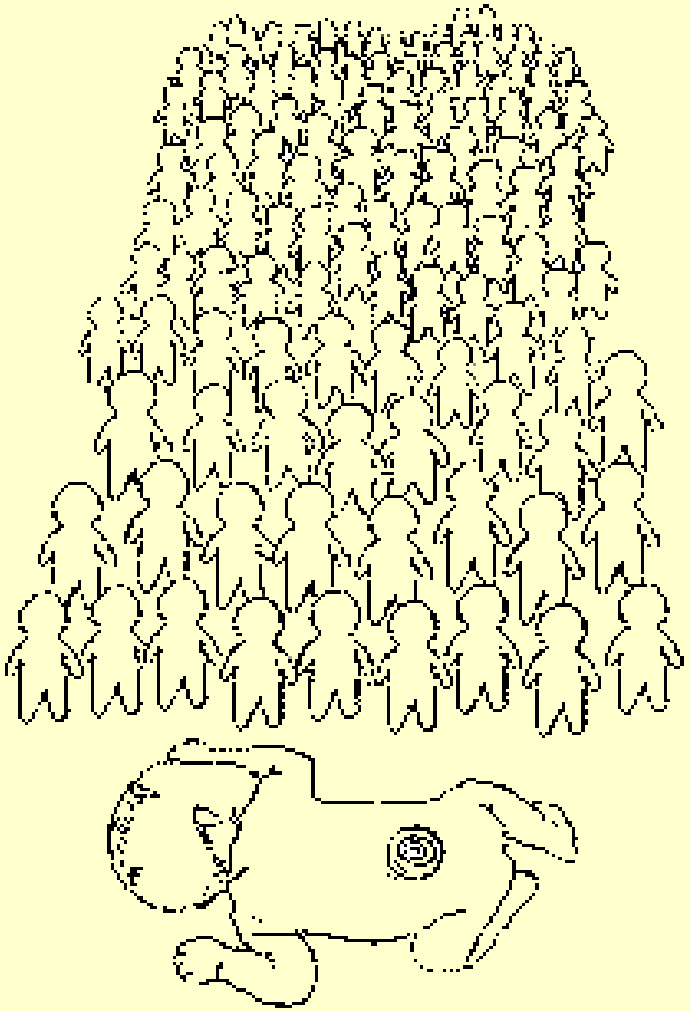


Figure 1 - Pacient's dorsal region. Note the intestinal loops coming through the thoracolumbar deformity, with enteric fistula (arrow) and hydrocephalus.


بداية تكوين
الرأس
بداية تكوين
الظهر
او
عمر الجنين
21 يوم
عمر الجنين
22 يوم
استمرار غلق
اطراف الانبوبة
عمر الجنين
28 يوم
الأم تتناول
حمض الفوليك
عمر الجنين
28 يوم
الأم لا تتناول
حمض الفوليك
نتيجة عدم
غلق الانبوبة
العصبية بطريقة
سليمة
مدونة
الاسرة
العربية

عدد الأطفال المصابون بالظهر المشقوق في السنة



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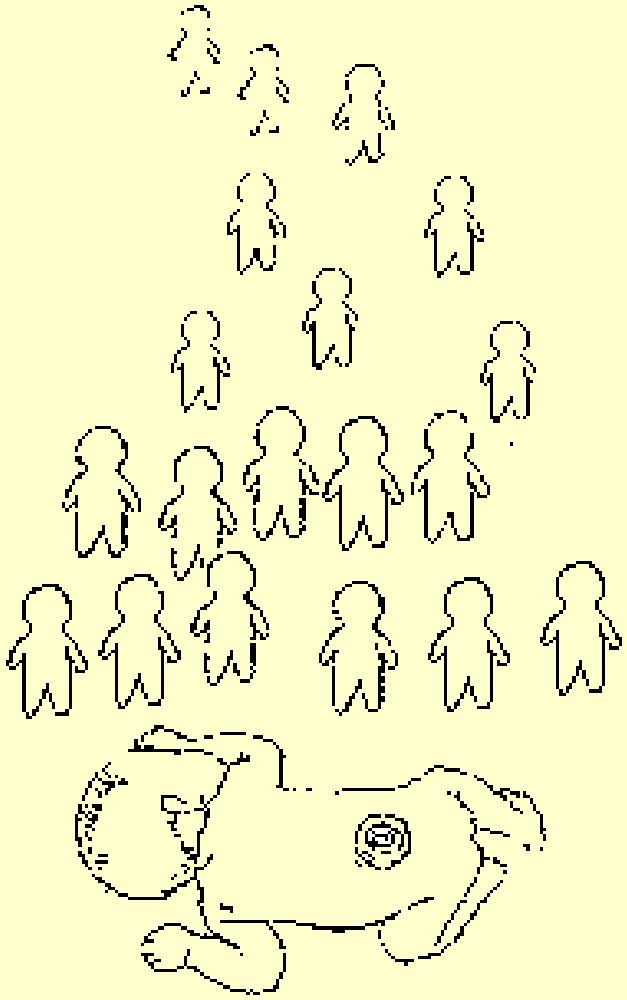

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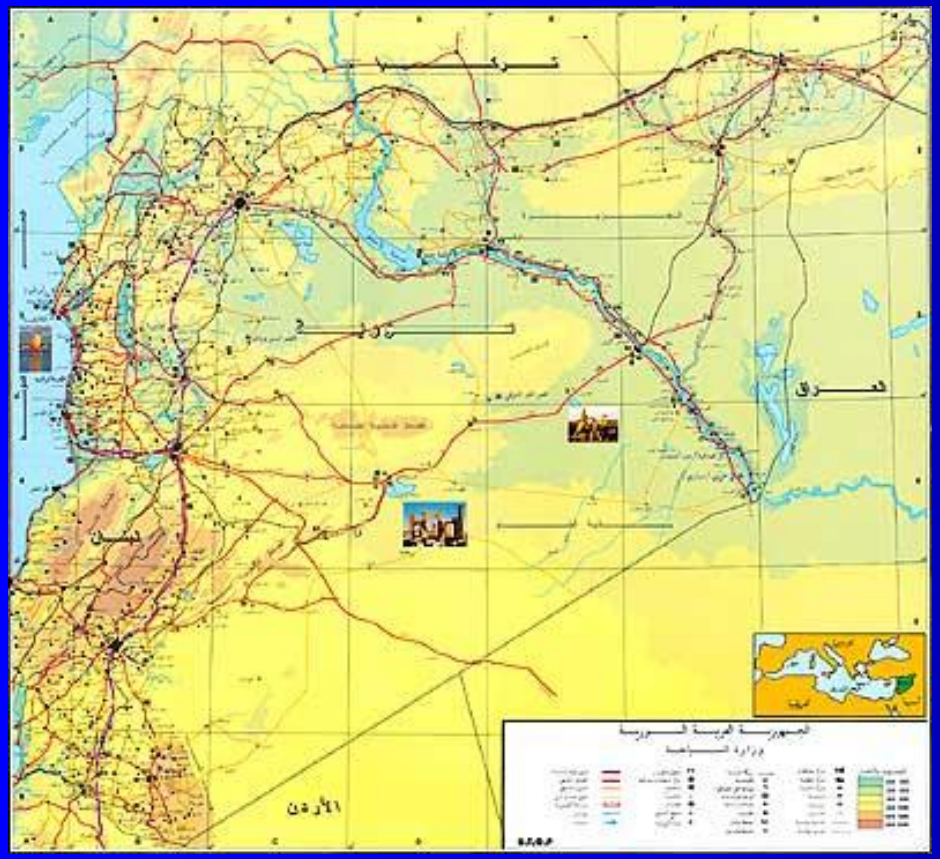
Frequency



Syria

- **United States**
- Approximately 30% of newborns with clinical sepsis have associated bacterial meningitis.
- Since the initiation of intrapartum antibiotics in 1996, a decrease has occurred in the national incidence of early-onset GBS infection from approximately 1.8 cases per 1000 live births in 1990 to 0.32 case per 1000 live births in 2003.


NICU

Cinical: Physical

- Generalized or focal seizures are observed in as many as 33% of patients. Seizures that occur during the first 3 days of illness usually have little prognostic significance. However, prolonged or difficult-to-control seizures, especially when observed beyond the fourth hospital day, are predictors of a complicated hospital course with serious sequelae.

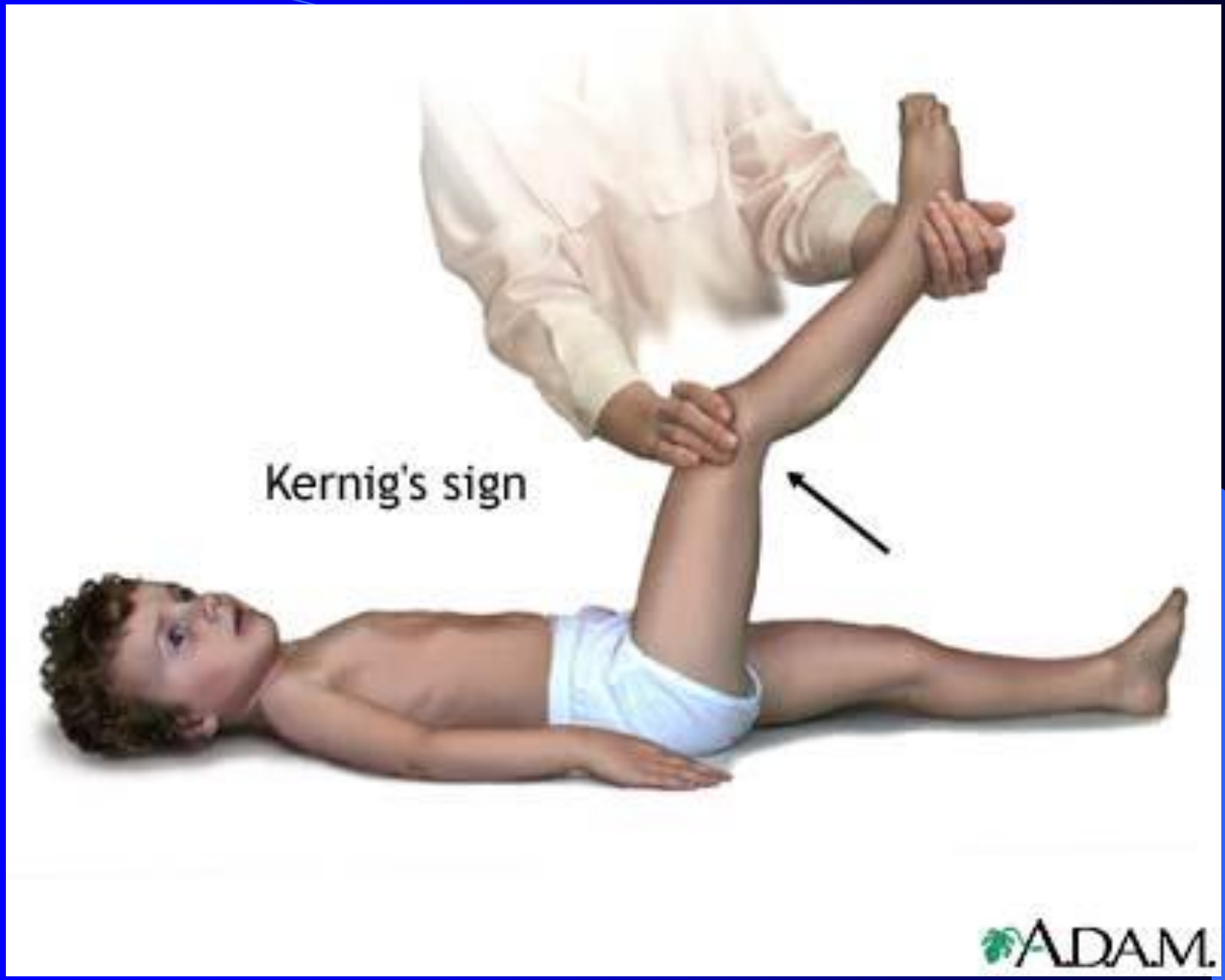
Cinical: Physical

- Approximately **6%** of affected infants and children show signs of **disseminated intravascular coagulopathy and endotoxic shock.** These signs are indicative of a poor prognosis.

Signs associated with neck stiffness

Brudzinski sign – flexion of the neck with the child supine causes flexion of the knees and hips

Kernig sign – with the child lying supine and with the hips and knees flexed, there is back pain on extension of the knee


Kernig's sign
A.D.A.M.


Brudzinski's neck sign
A.D.A.M.

العلامة السورية

الطفل بوضعية الاستلقاء

العلامة السورية

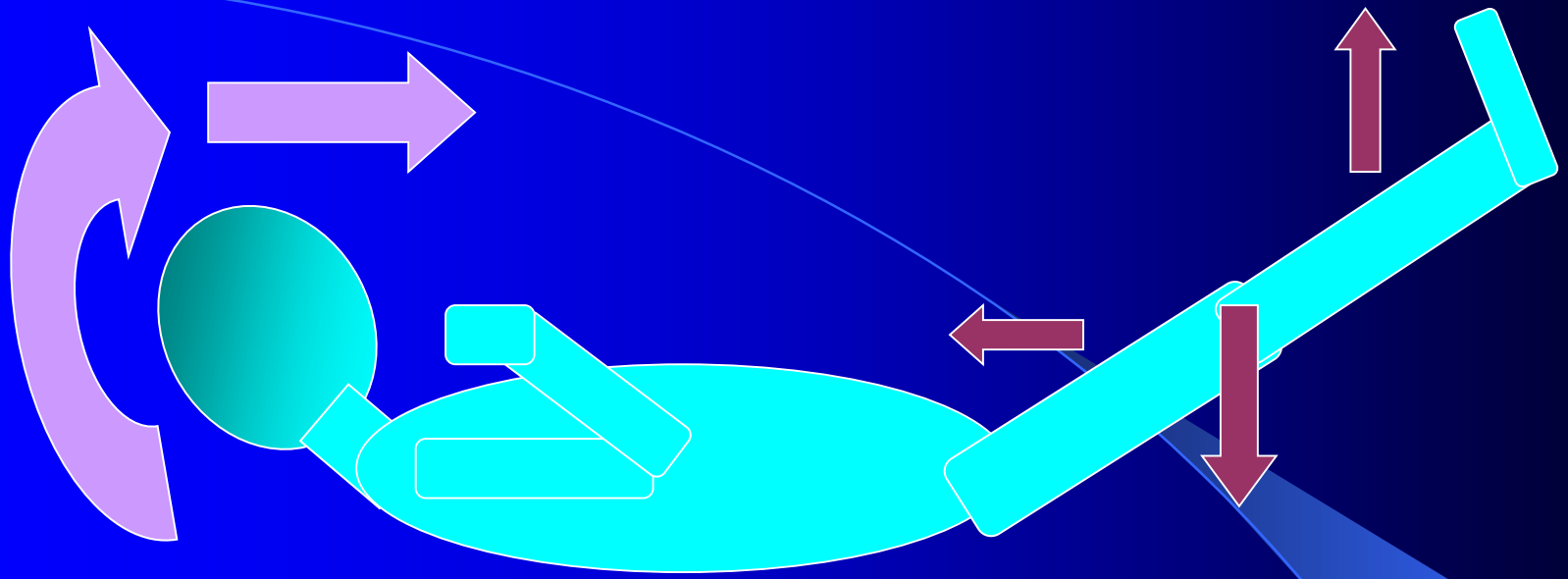
علامة برودزينسكي

THE SYRIAN SIGN

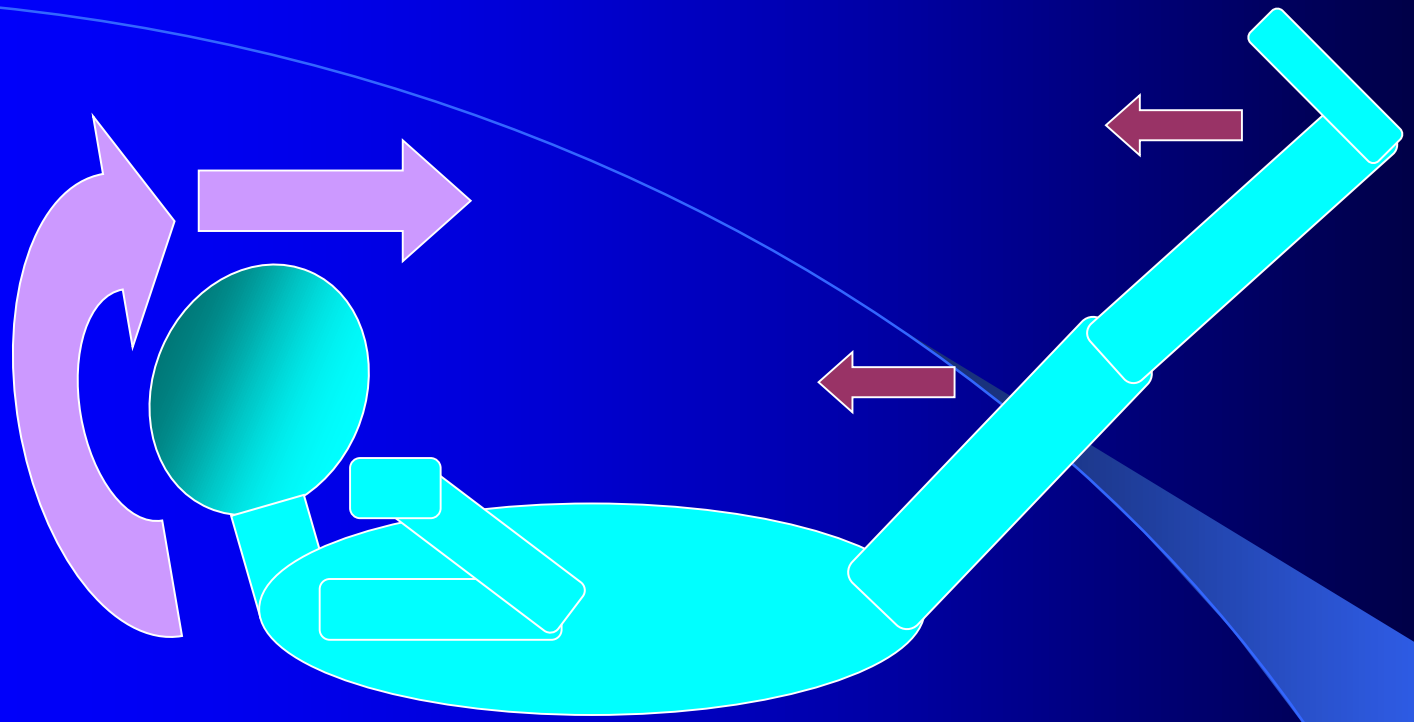
Child in Supine Position

Brudzinski's Sign

The Syrian Sign



*تحري علامة برودزنسكي والاستجابة المعاكسة لها بالبسط بدل العطف عند مريض مما ينفي التهاب السحايا لديه.



*استمرار عملية التحري واشتداد وضوح الإيجابيات.

الشروط

- شروط تطبيق العلامة:
- ١- الخبرة بتحري العلامة.
- ٢- أن يكون المريض غير معالج سابقاً من أجل التهاب السحايا سواء بشكل كامل أو جزئي أو متعرض لمعالجة ناقصة.
- ٣-أن لايكون هناك أي إيجابية لبرودزنسكي.
- ٤- أن يحصل بسط الساقين على مستوى الركبة سواء كانت اللحظة البدئية للتحري عطف بسيط أو حالة استواء.

الشروط

- ٥- إن حصول العطف بعد بسط بدئي يعني إيجابية برودزنسكي وليس علامتنا.
- ٦- العلامة وكأي علامة طبية أخرى لاتحمي غير الخبير ولا الخبيرولاتعفيه من المسؤولية التشخيصية و العلاجية والاستشارية والقانونية والأخلاقية اتجاه مريضه، ولذلك للطبيب الحرية في اعتمادها بشكل أساسي أو بشكل مساعد للوصول للتشخيص وله القرار في اتخاذ مايلزم من اختبارات واجراءات أخرى.
- ٧- ألا يكون المريض مصاباً بشلل سفلي.
- ٨- العلامة مساعدة جداً في السنوات الثلاث الأولى من العمر بدءاً من عمر شهر ونصف ولكن إيجابيتها في أعمار أكبر لها نفس الأهمية التشخيصية.

Investigations

The essential investigations are listed in Fig. 15.4. A lumbar puncture is performed to obtain CSF to confirm the diagnosis, identify the organism responsible, and its antibiotic sensitivities. Characteristic findings are shown in Fig. 15.4. However, exceptions can occur, e.g. lymphocytes can predominate in bacterial meningitis, e.g. in Lyme disease, and glucose levels can be low in viral meningitis, e.g. enterovirus meningitis. If any of the contraindications for performing a lumbar puncture are present, as listed in Fig. 15.4, it should not be performed, as under these circumstances, the procedure carries a risk of coning of the cerebellum through the foramen magnum. In these circumstances, a lumbar puncture can be postponed until the child's condition has stabilized. Even without a lumbar puncture, bacteriological diagnosis can be achieved in about half of the cases from the blood by culture or polymerase chain reaction (PCR), and rapid antigen screens can be performed on blood and urine samples. Throat swabs should also be obtained for bacterial culture and viral PCRs. A serological diagnosis can be made on convalescent serum 4 weeks to 6 weeks after the presenting illness if necessary.

Investigations

Full blood count and differential count
Blood glucose and blood gas (for acidosis)
Coagulation screen, C-reactive protein
Urea and electrolytes, liver function tests
Culture of blood, throat swab, urine, stool for bacteria
Rapid antigen test for meningitis organisms (can be done on blood, CSF, or urine)
Samples for viral PCRs (e.g. throat swab, nasopharyngeal aspirate, conjunctival swab, stool sample)
Lumbar puncture for CSF unless contraindicated (see below for tests on CSF)
Serum for comparison of convalescent titres
PCR of blood and CSF for possible organisms
If TB suspected: chest X-ray, Mantoux test

Culture of blood, throat swab, urine, stool for bacteria

Rapid antigen test for meningitis organisms (can be done on blood, CSF, or urine)

Samples for viral PCRs (e.g. throat swab, nasopharyngeal aspirate, conjunctival swab, stool sample)

Lumbar puncture for CSF unless contraindicated (see below for tests on CSF)

Serum for comparison of convalescent titres

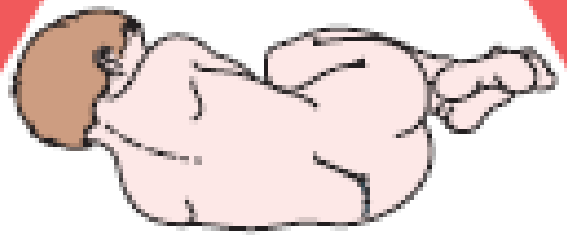
PCR of blood and CSF for possible organisms

If TB suspected: chest X-ray, Mantoux test and/or Interferon-gamma release assay, gastric aspirates or sputum for microscopy and culture (and PCR if available)

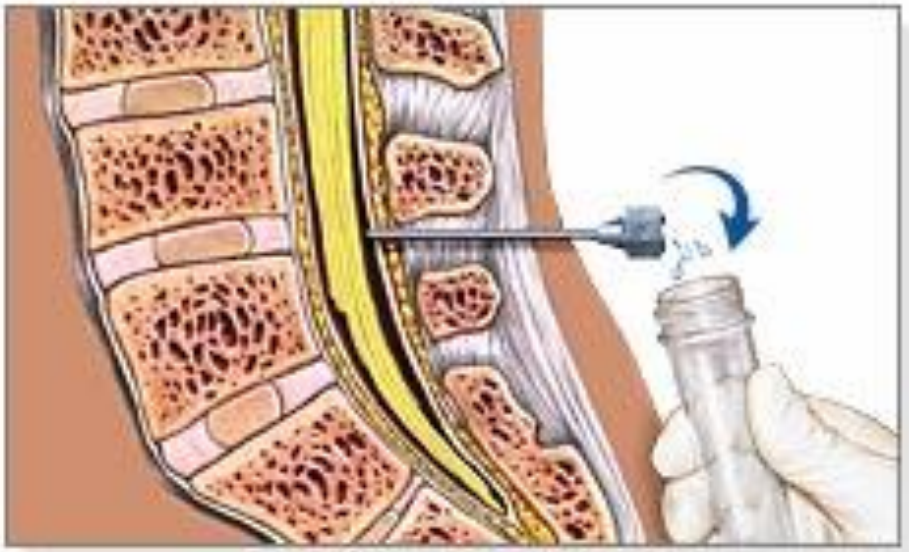
Consider CT/MRI brain scan and EEG

Contraindications to lumbar puncture:

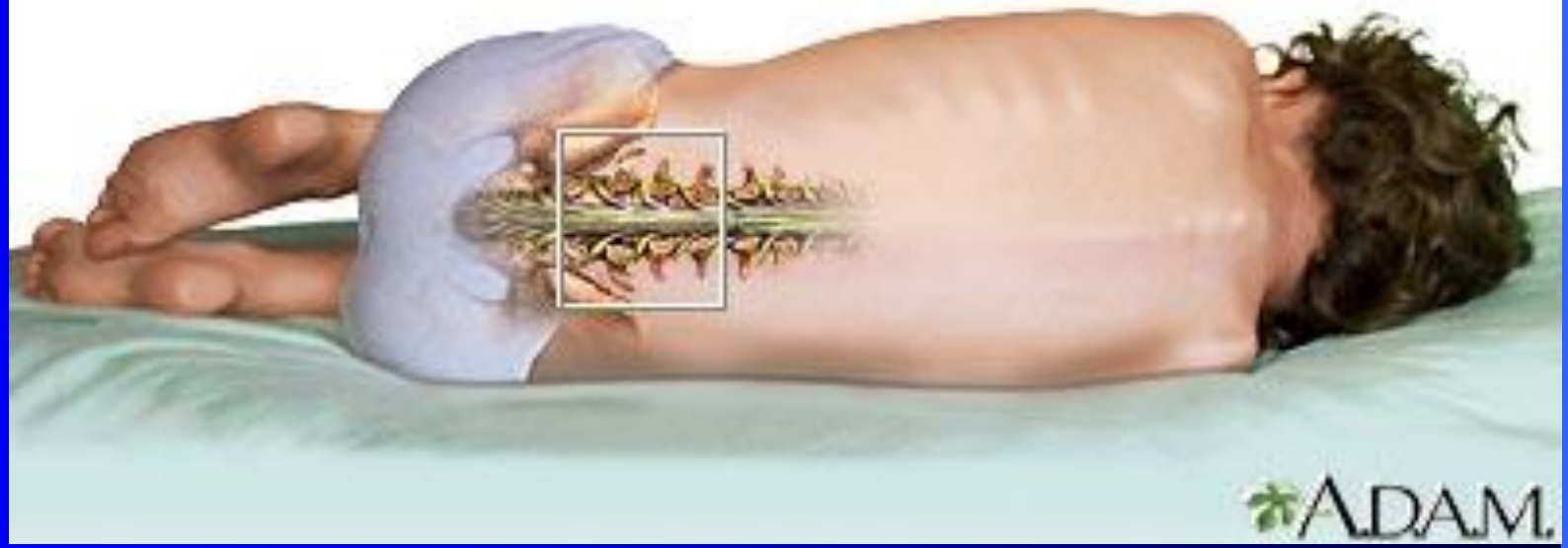
- Cardiorespiratory instability
- Focal neurological signs
- Signs of raised intracranial pressure, e.g. coma, high BP, low heart rate or papilloedema
- Coagulopathy
- Thrombocytopenia
- Local infection at the site of LP
- If it causes undue delay in starting antibiotics

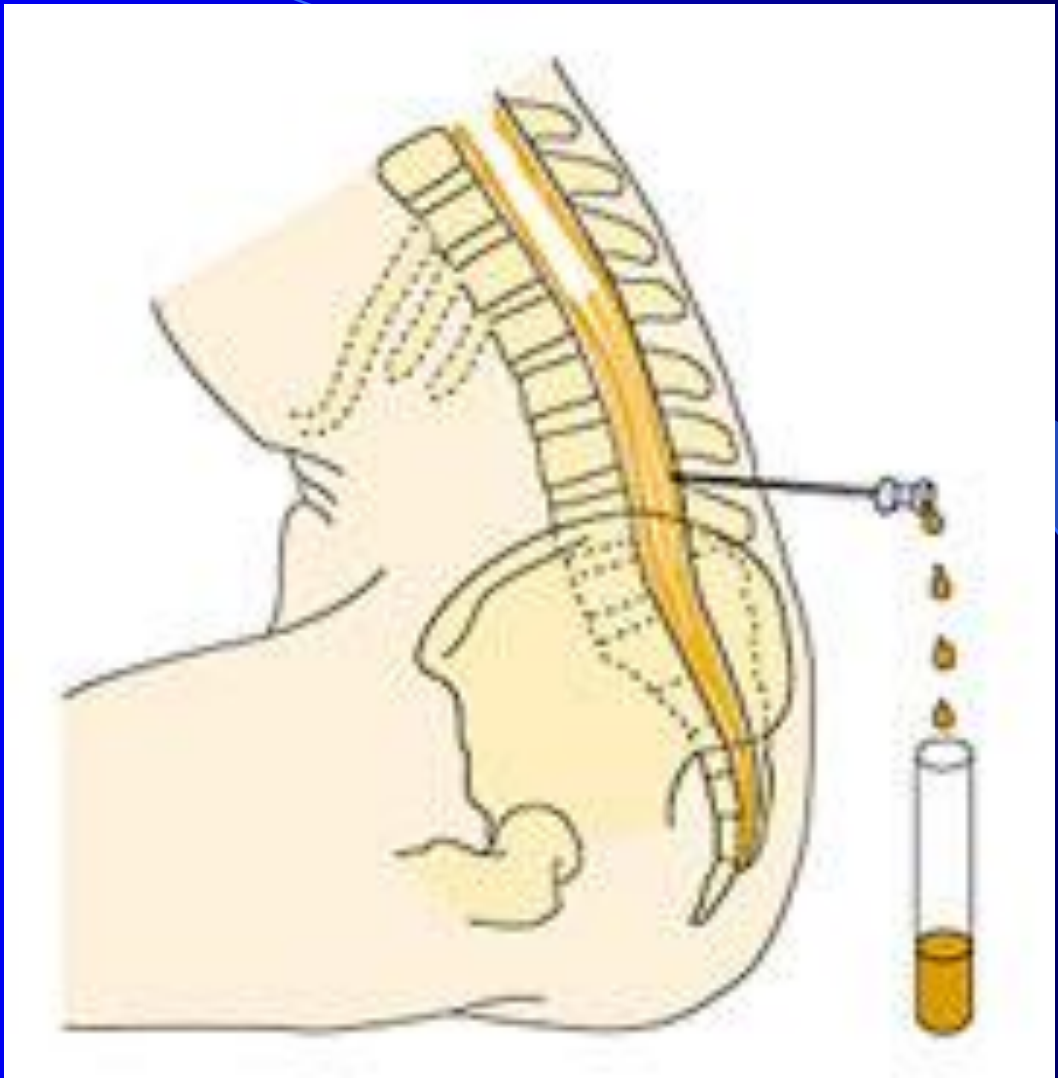


Best time for LP?
Diagnostically useful
but potentially dangerous

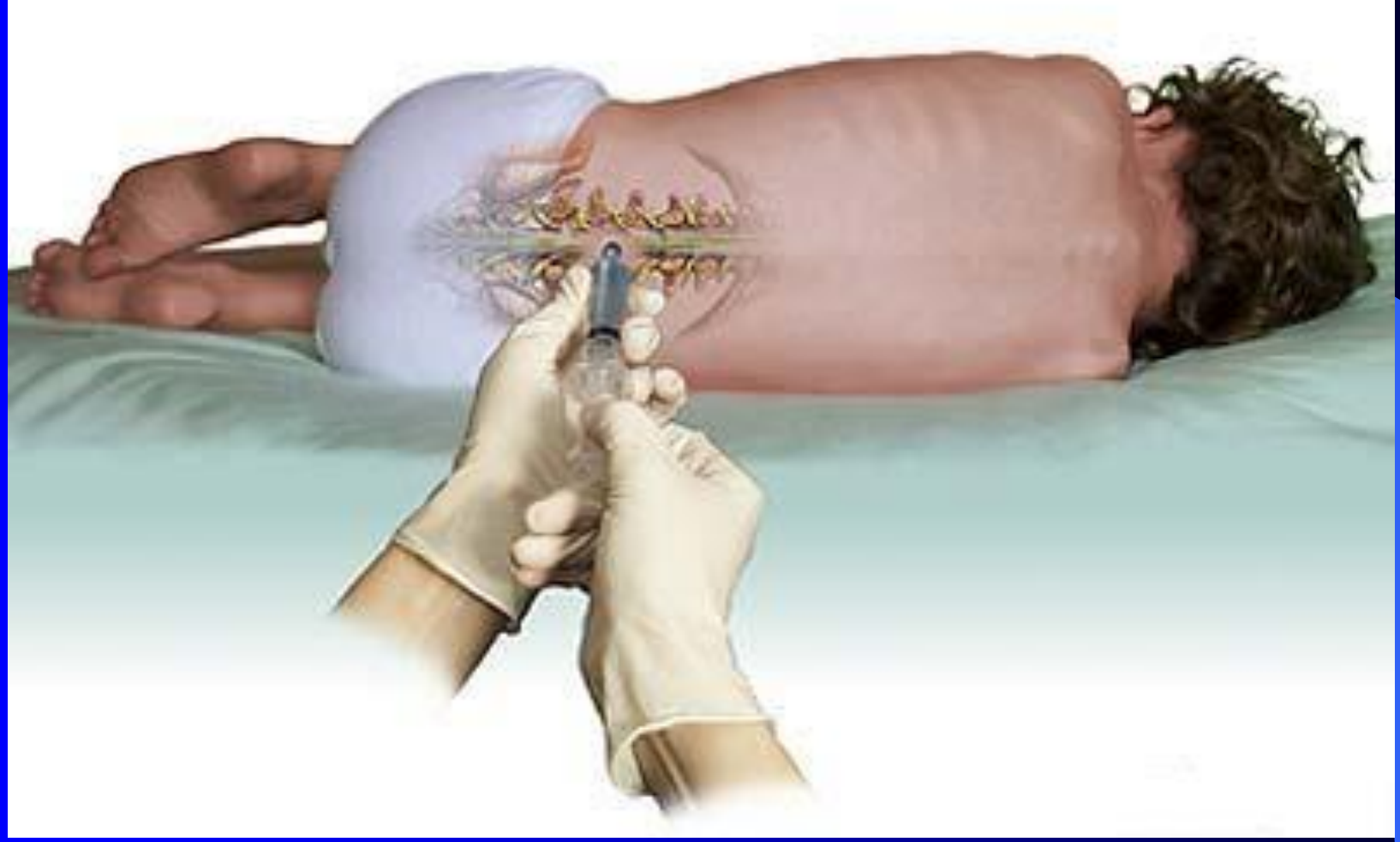


Spinal fluid is collected for testing





سحب السائل الشوكي من بين فقرتين



Diagnostic Considerations

- The presence or absence of classic meningeal signs and symptoms should not be used as the sole criteria for referring patients for further diagnostic testing

Blood and other Studies

- Some data suggest that procalcitonin may be a useful biomarker for distinguishing bacterial meningitis from aseptic meningitis. a level of 0.5 ng/mL to have a sensitivity of 99% and a specificity of 83% for differentiating bacterial from aseptic meningitis

CSF studies

- In about 2-3% of bacterial meningitis cases, bacterial cultures may be positive even when the Gram stain is negative and the cell counts and glucose and protein levels are normal.
- White blood cell (WBC) counts higher than 1000/µL are usually caused by bacterial infections. Counts of 500-1000/µL may be bacterial or viral and call for further evaluation. Lower counts are usually associated with viral infections.

CSF studies

- The use of a corrected ratio of WBCs to red blood cells (RBCs)—that is, 1:500—or the percentage of neutrophils to “normalize” the cell count was shown to have limited utility in predicting which patients would have meningitis. The “corrected CSF” was shown to underestimate the true WBC count, causing clinicians to underdiagnose borderline meningitis cases.

Typical changes in the CSF in meningitis or encephalitis, beyond the neonatal period

	Aetiology	Appearance	White blood cells	Protein	Glucose
Normal	—	Clear	0–5/mm^3	0.15–0.4 g/L	≥50% of blood
Meningitis	Bacterial	Turbid	Polymorphs:↑↑	↑↑	↓↓
	Viral	Clear	Lymphocytes:↑ (initially may be polymorphs)	Normal/↑	Normal/↓
	Tuberculosis	Turbid/clear/ viscous	Lymphocytes:↑	↑↑↑	↓↓↓
Encephalitis	Viral/unknown	Clear	Normal/↑ lymphocytes	Normal/↑	Normal/↓

gure 15.4 Assessment and investigation of meningitis and encephalitis.

Management

It is imperative that there is no delay in the administration of antibiotics and supportive therapy in a child with meningitis. The choice of antibiotics will depend on the likely pathogen. A third-generation cephalosporin, e.g. ceftriaxone, is the preferred choice to cover the most common bacterial causes. Although still relatively rare in the UK, pneumococcal resistance to penicillin and cephalosporins is increasing rapidly in certain parts of the world. The length of the course of antibiotics given depends on the causative organism and clinical response. Beyond the neonatal period, there is some evidence suggesting that dexamethasone administered with the antibiotics reduces the risk of long-term complications such as deafness.

Medical Care

- **Infants and children: Management of acute bacterial meningitis involves both appropriate antimicrobial therapy and supportive measures. All patients should have an audiologic evaluation upon completion of therapy .**

Medical Care

- **Fluid and electrolyte management**
  - **Closely monitor patients by checking vital signs and neurologic status and by ensuring an accurate record of intake and output .**

– Fluid and electrolyte management

- By prescribing the correct type and volume of fluid the risk of development of brain edema can be minimized. The child should receive fluids sufficient to maintain systolic blood pressure at around ٨٠ mm Hg, urinary output of ٥٠٠ mL/m hguohtlA .noisufrep eussit etauqeda dna ‹d/٢ gnitardyhrednu ‹tnatropmi si HDAIS diova ot erac noisufrep larberec desaerced fo ksir dna tneitap eht .llew sa gninrecnoc yllauqe era
- Dopamine and other inotropic agents may be necessary to maintain blood pressure and adequate circulation.

Antimicrobial therapy for infants and children

- vancomycin and either ceftriaxone or cefotaxime
- Of note, ceftazidime has poor activity against pneumococci and should not be substituted for cefotaxime or ceftriaxone .

Antimicrobial therapy for infants and children

- Because vancomycin poorly penetrates the CNS, a higher dose of ٦٠ mg/kg/d is recommended when vancomycin is used to treat CNS infections. Cefotaxime or ceftriaxone is adequate if pneumococci are susceptible to cefotaxime. However, if *S pneumoniae* isolates have a higher MIC for cefotaxime and fall in the intermediate resistance group, there have been concerns regarding prompt sterilization of the CSF ،
- and a high dose of cefotaxime (٣٠٠ mg/kg/d) with vancomycin (٦٠ mg/kg/d) may be preferred .

Duration of antimicrobial therapy

- *N meningitidis*٧ - days
- *H influenzae*٧ - days
- *S pneumoniae* ١٤-١٠ - days
- *S agalactiae* ٢١-١٤ - days
- Aerobic gram-negative bacilli - ٢١ days or ٢ weeks beyond first sterile culture (whichever is longer)
- *L monocytogenes*٢١ - days or longer

Dexamethasone administration

– Follow-up examination demonstrated a significant decrease in the incidence of neurologic and audiologic sequelae, with evidence of clinical benefit being greatest for overall hearing impairment. As a result, the IDSA guidelines recommend the use of adjunctive dexamethasone in cases of *H influenzae* type b meningitis to be initiated ١٠-٢٠ minutes prior to or at least concomitant with the first antimicrobial dose at ٠,١٥ mg/kg q٦h for ٢-٤ days .

Dexamethasone administration

- Likewise, data are insufficient to recommend adjunctive steroids in neonates with bacterial meningitis .

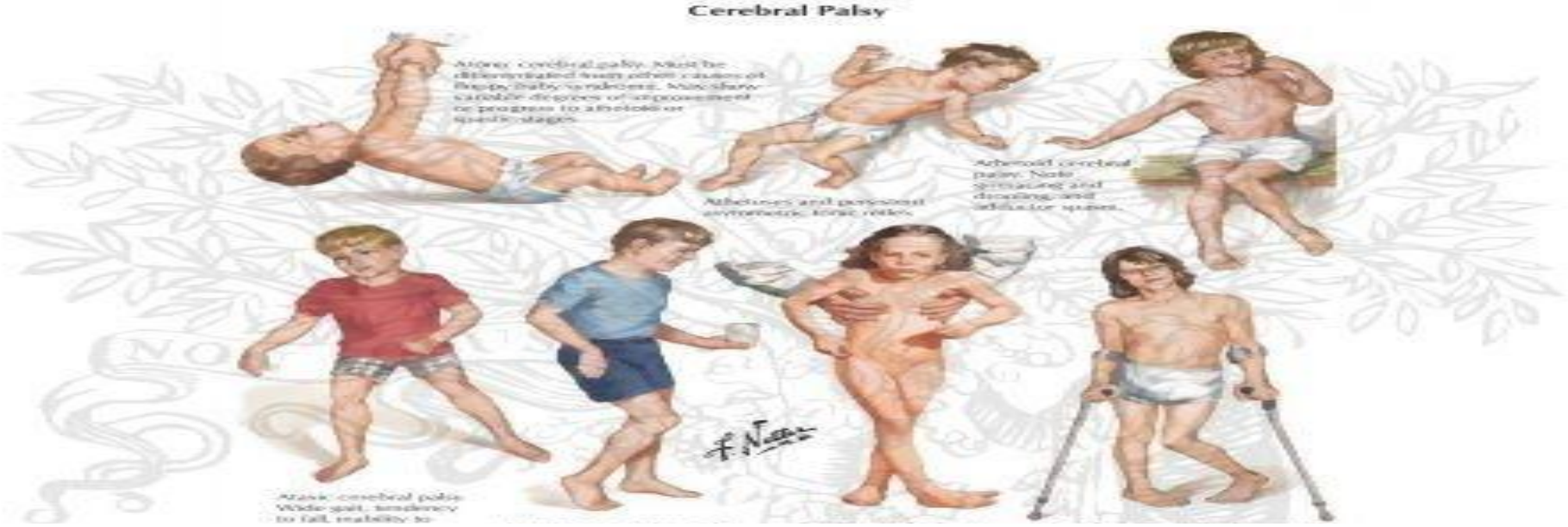
Cerebral complications

These include:

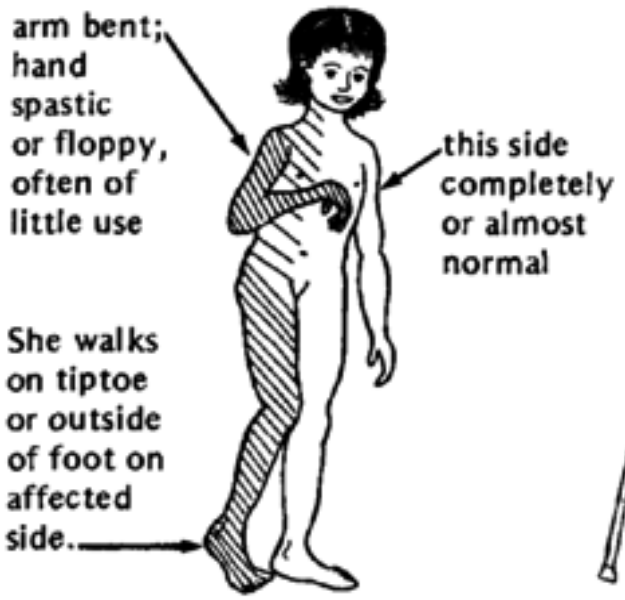
- *hearing impairment* – inflammatory damage to the cochlear hair cells may lead to deafness. All children who have had meningitis should have an audiological assessment done promptly, as children with hearing impairment may benefit from hearing amplification or a cochlear implant
- *local vasculitis* – this may lead to cranial nerve palsies or other focal neurological lesions
- *local cerebral infarction* – this may result in focal or multifocal seizures, which may subsequently result in epilepsy
- *subdural effusion* – particularly associated with *H. influenzae* and pneumococcal meningitis. This is confirmed by cranial CT or MRI scan. Most resolve spontaneously, but some require neurosurgical intervention
- *hydrocephalus* – may result from impaired resorption of CSF (communicating hydrocephalus) or blockage of the cerebral aqueduct or ventricular

outlets by fibrin (noncommunicating hydrocephalus). A ventricular shunt may be required

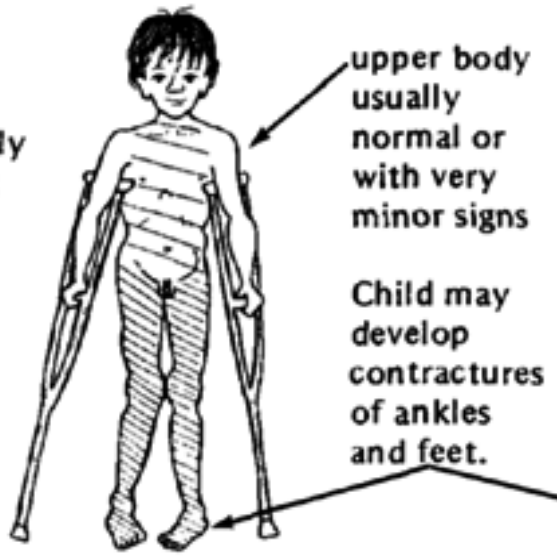
- *cerebral abscess* – the child's clinical condition deteriorates with or without the emergence of signs of a space-occupying lesion. The temperature will continue to fluctuate. It is confirmed on cranial CT or MRI scan. Drainage of the abscess is required.



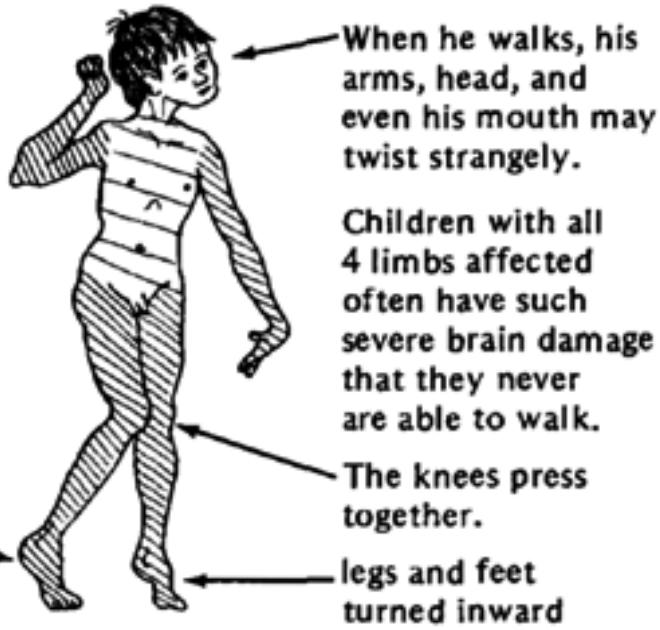
ARM AND LEG ON ONE SIDE (HEMIPLEGIC)

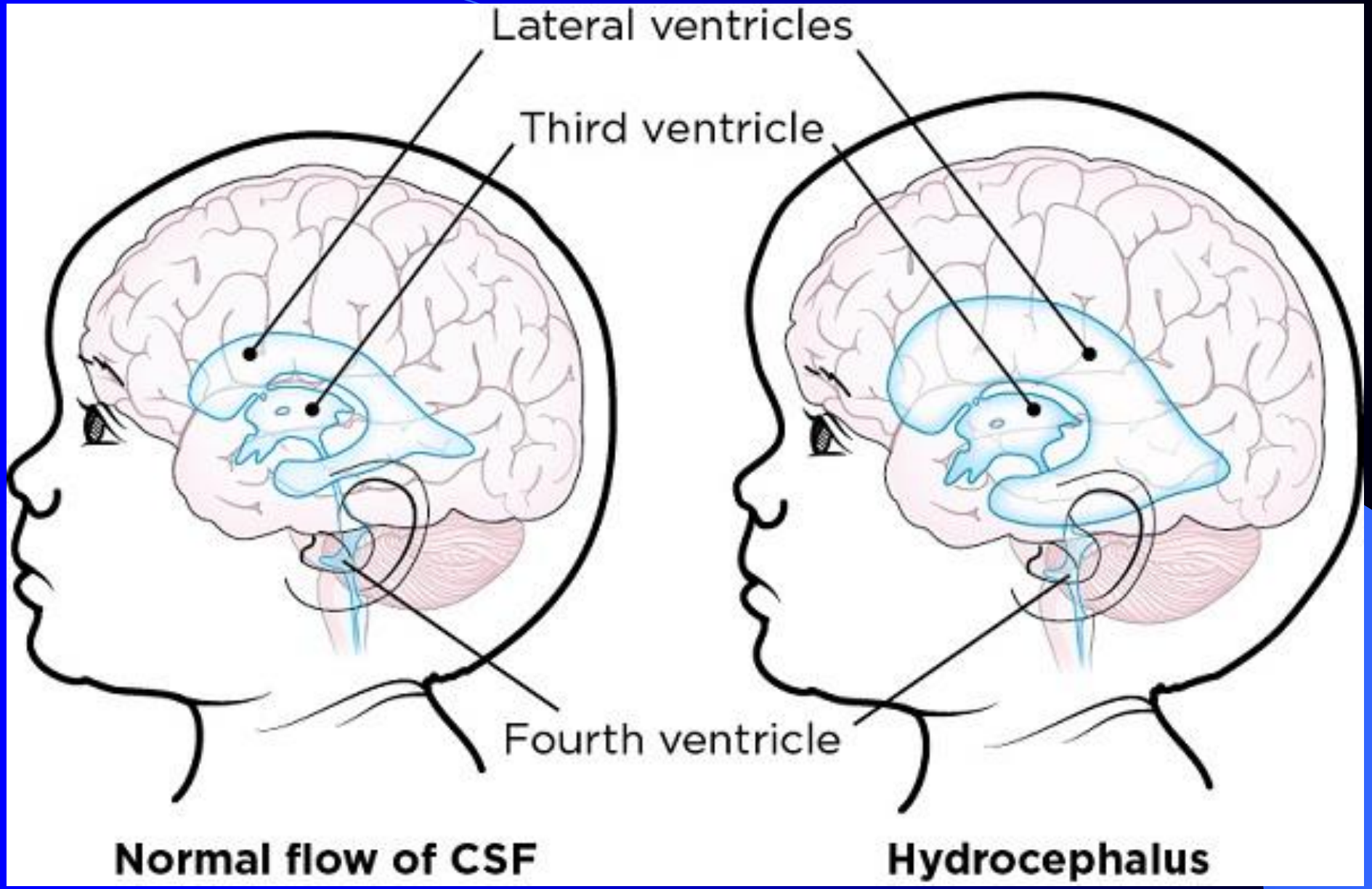


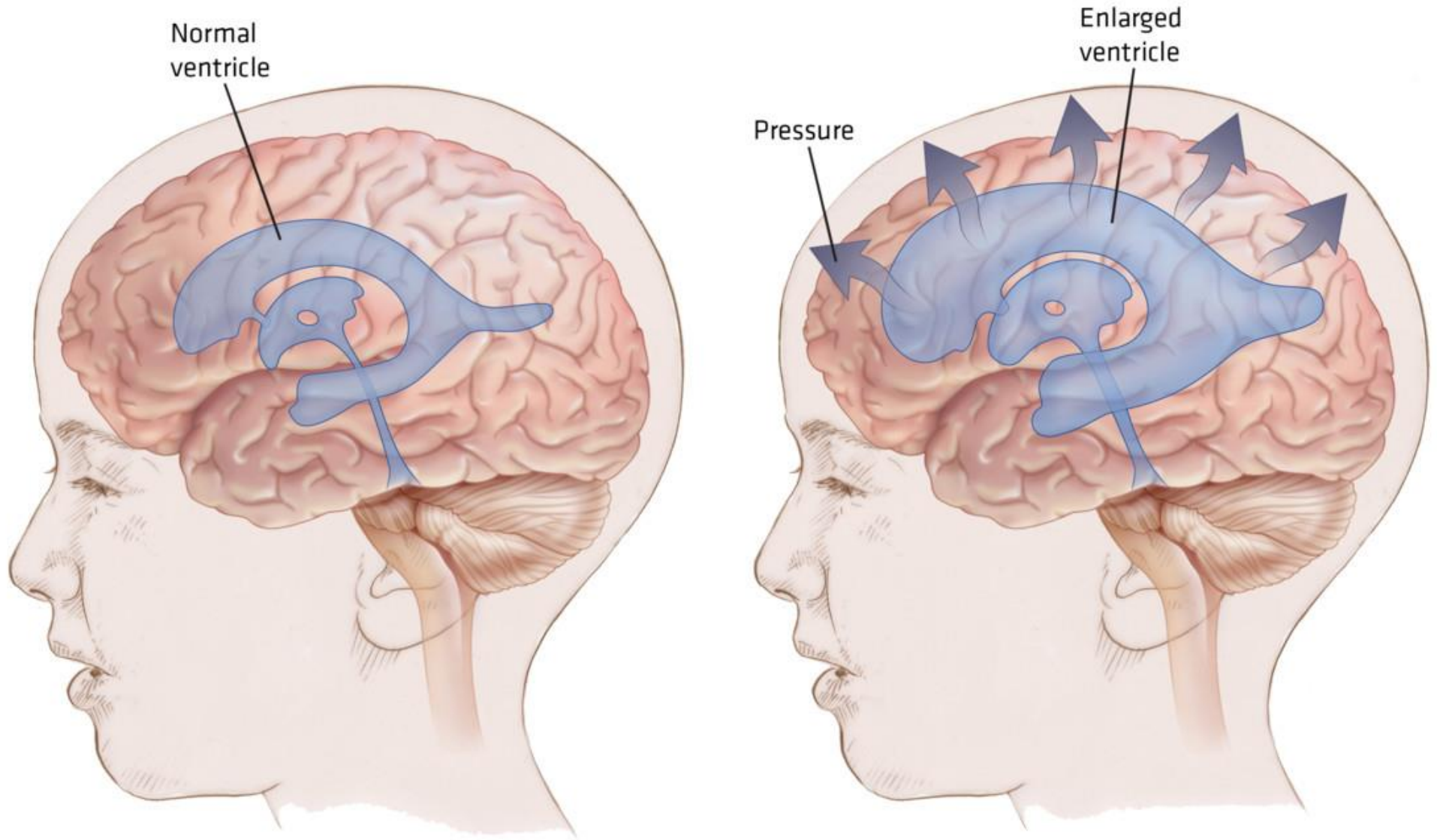
BOTH LEGS ONLY (PARAPLEGIC) or with slight involvement elsewhere (DIPLEGIC)

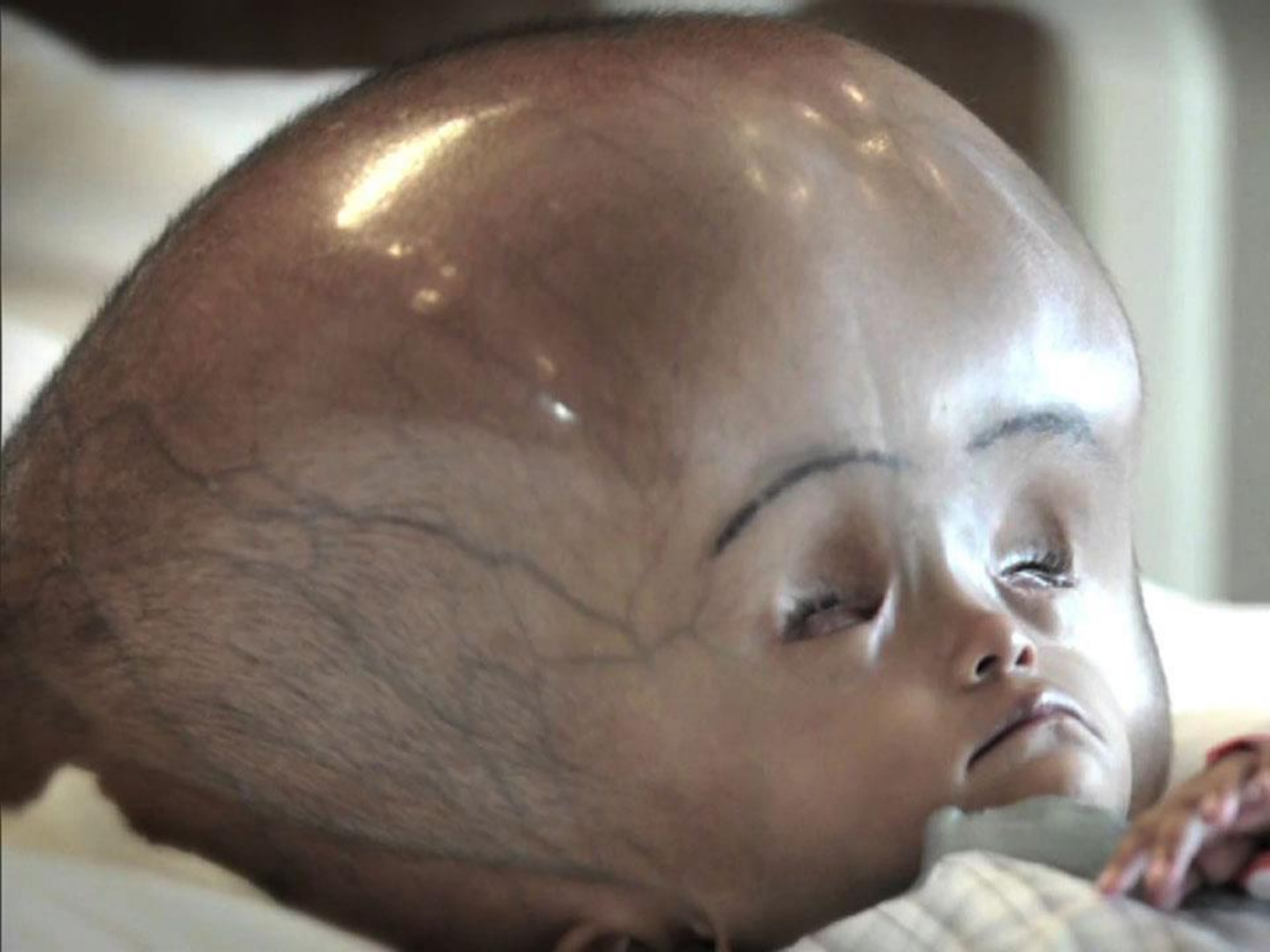


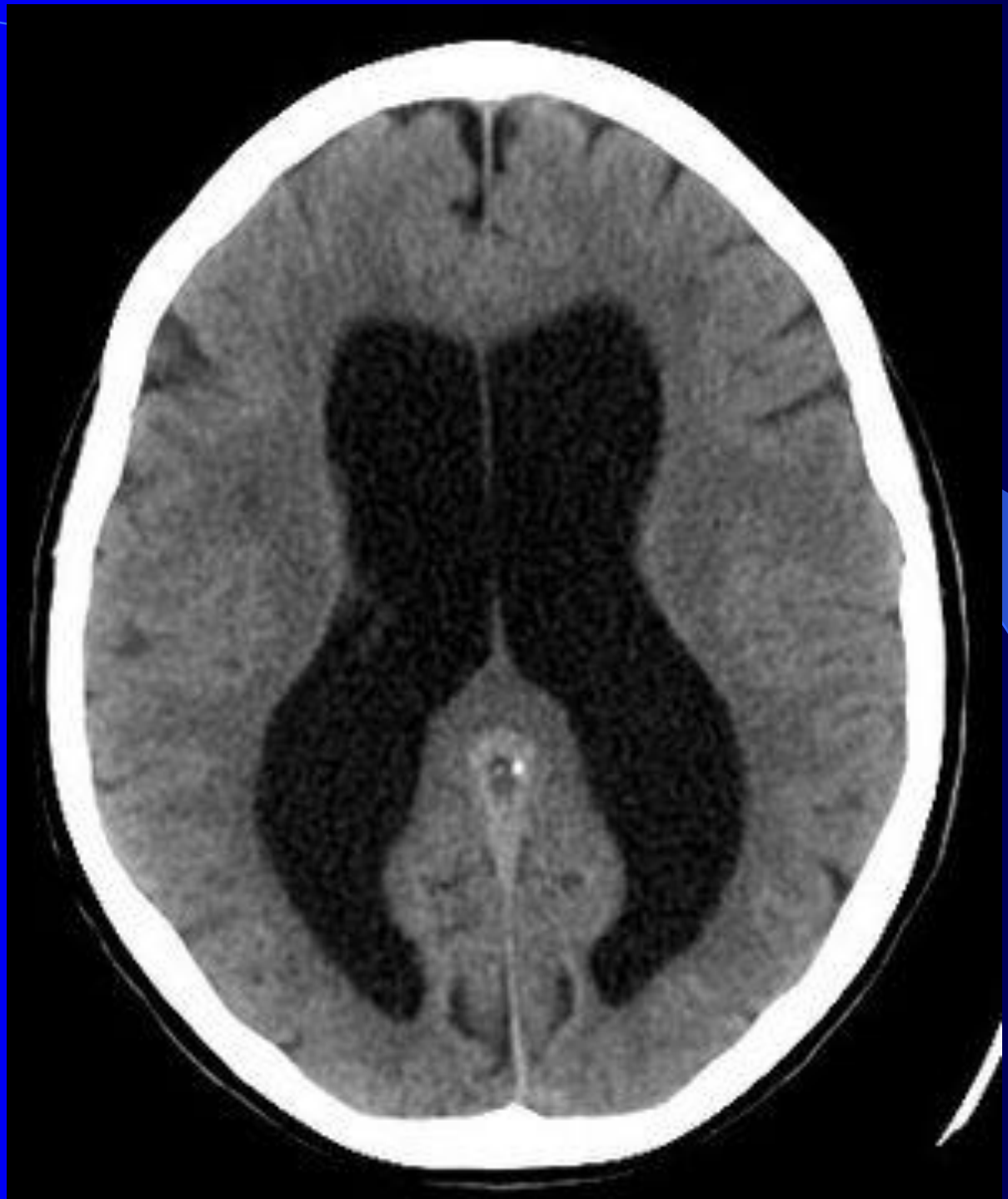
BOTH ARMS AND BOTH LEGS (QUADRIPLEGIC)

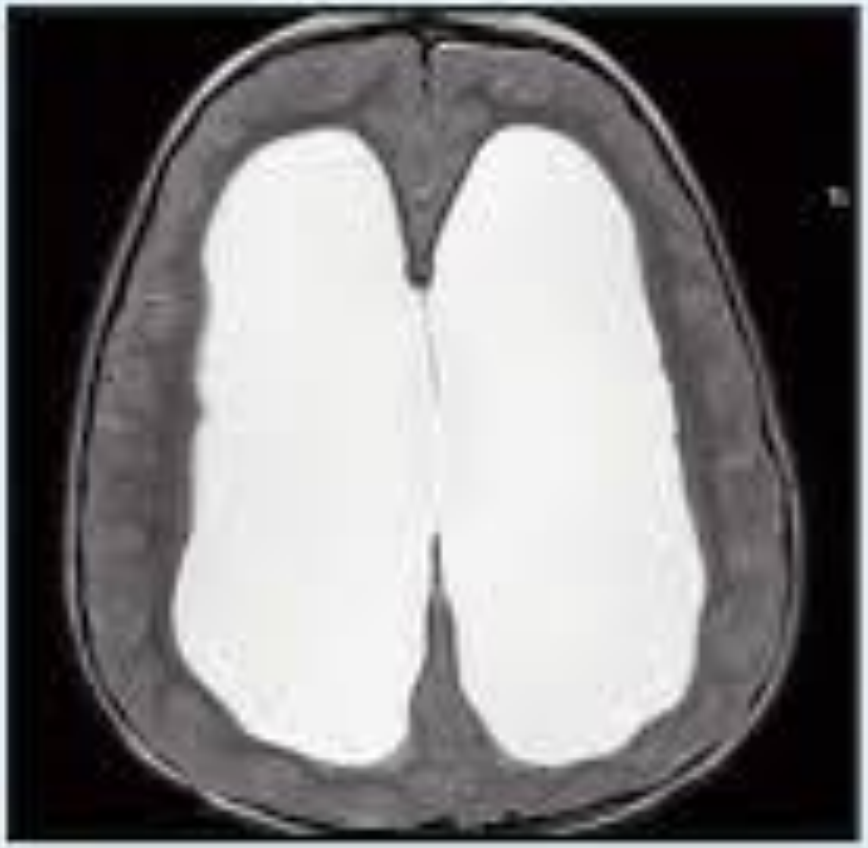


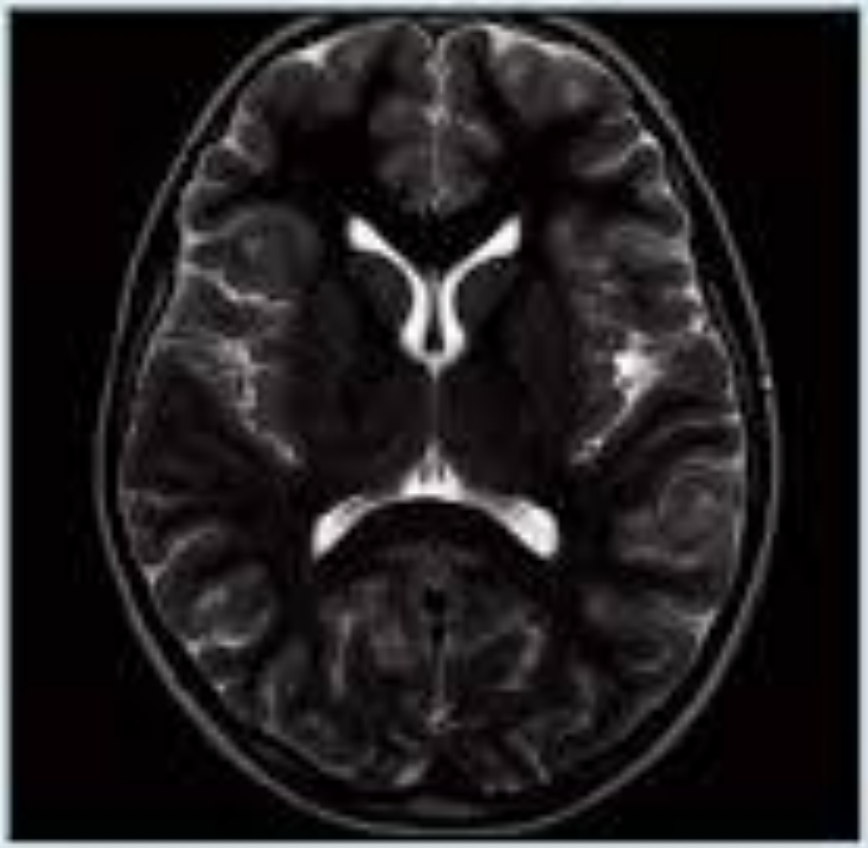

Lateral ventricles
Third ventricle
Fourth ventricle
Normal flow of CSF
Hydrocephalus


Normal
ventricle
Enlarged
ventricle
Pressure





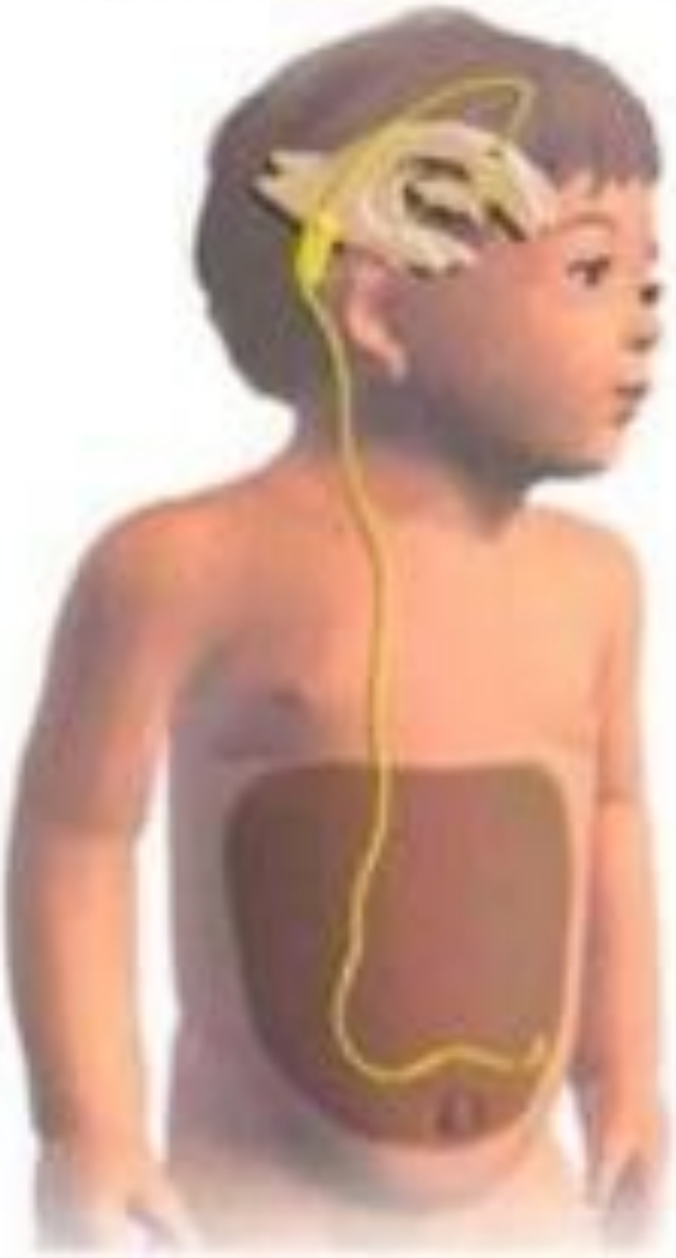




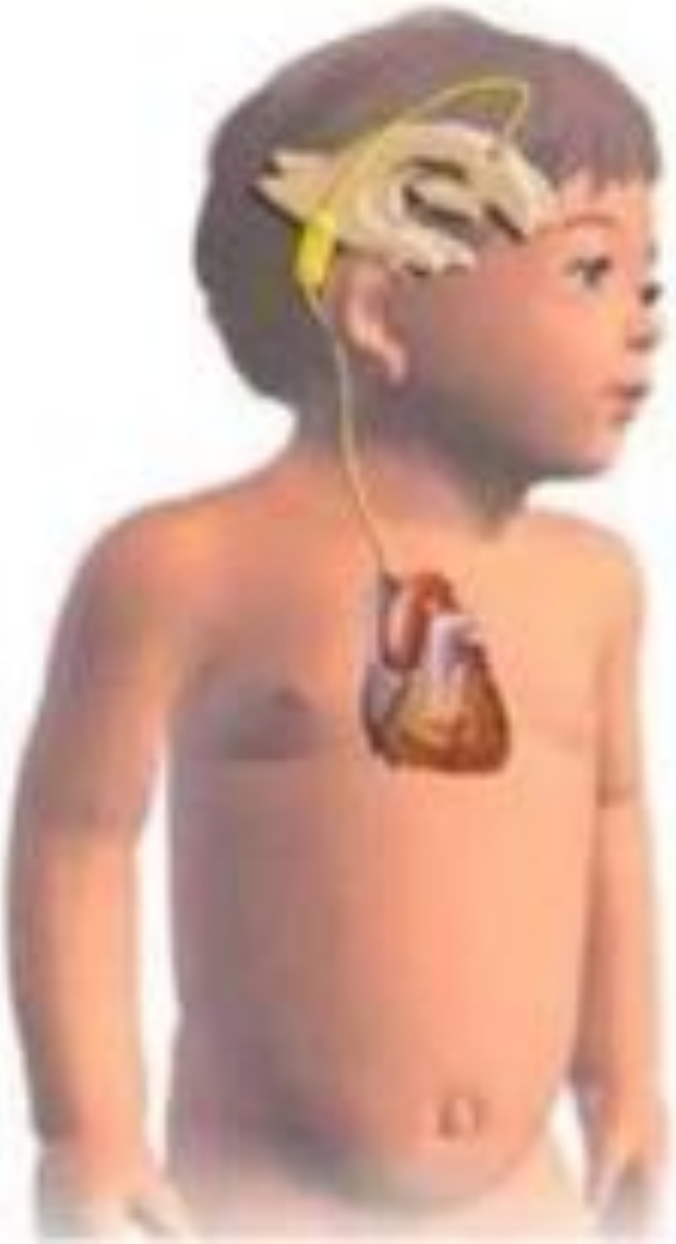
Enlarged ventricles **Normal ventricles**

MRI scans showing the ventricles from the top view.

VP SHUNT



VA SHUNT




32 33 34 35 36 37 38 39 40 41 42 43 44

Prophylaxis

Prophylactic treatment with rifampicin or ciprofloxacin to eradicate nasopharyngeal carriage is given to all household contacts for meningococcal meningitis and Hib infection. It is not required for the patient if given a third-generation cephalosporin, as this will eradicate nasopharyngeal carriage. Household contacts of patients who have had group C meningococcal meningitis should be vaccinated with the meningococcal group C vaccine.

Partially treated bacterial meningitis

Children are frequently given oral antibiotics for a nonspecific febrile illness. If they have early meningitis, this partial treatment with antibiotics may cause diagnostic problems. CSF examination shows a markedly raised number of white cells, but cultures are usually negative. Rapid antigen screens and PCR are helpful in these circumstances. Where the diagnosis is suspected clinically, a full course of antibiotics should be given.

Viral meningitis

More than two-thirds of central nervous system (CNS) infections are viral. Causes include enteroviruses, Epstein–Barr virus (EBV), adenoviruses, and mumps. Mumps meningitis is now rare in the UK due to the measles, mumps, and rubella (MMR) vaccine. Viral meningitis is usually much less severe than bacterial meningitis and most cases make a full recovery. Diagnosis of viral meningitis can be confirmed by culture or PCR of CSF, stool, urine, nasopharyngeal aspirate, and throat swabs, as well as serology.

Uncommon pathogens and other causes

Where the clinical course is atypical or there is failure to respond to antibiotic and supportive therapy, unusual organisms, e.g. *Mycoplasma* species or *Borrelia burgdorferi* (Lyme disease), TB, or fungal infections need to be considered. Uncommon pathogens are particularly likely in children who are immunocompromised (i.e. with immunodeficiency or receiving chemotherapy or immunosuppressive medication). Recurrent bacterial meningitis may occur in the immunodeficient children or in those with structural abnormalities of the skull or meninges that facilitate bacterial access. Aseptic meningitis may be seen in malignancy or autoimmune disorders.

Summary

Meningitis

- Predominantly a disease of infants and children.
- Incidence has been reduced by immunization.
- Clinical features: nonspecific in children under 12 months – fever, poor feeding, vomiting, irritability, lethargy, drowsiness, seizures, or reduced consciousness; late signs – bulging fontanelle, neck stiffness, and arched back (opisthotonos).
- Septicaemia can kill in hours; good outcome requires prompt resuscitation and antibiotics.
- Any febrile child with a purpuric rash should be given intramuscular benzylpenicillin immediately and transferred urgently to hospital.
- The meningococcal vaccines now included in the infant and adolescent immunization schedule in the UK should further reduce the incidence of meningococcal sepsis and meningitis.

Neonatal meningitis

See Chapter 11.

Encephalitis/encephalopathy

In meningitis there is inflammation of the meninges, whereas in encephalitis there is inflammation of the brain substance, although the meninges are often also affected. Encephalitis may be caused by:

- direct invasion of the brain by a neurotoxic virus (such as HSV)
- delayed brain swelling following a dysregulated neuroimmunological response to an antigen, usually a virus (postinfectious encephalopathy), e.g. following chickenpox
- a slow virus infection, such as HIV infection or subacute sclerosing panencephalitis (SSPE) following measles.

In encephalopathy from a noninfectious cause, such as a metabolic abnormality, the clinical features may be similar to infectious encephalitis.

The clinical features and investigation of encephalitis are described in Fig. 15.4. Most children present with fever, altered consciousness, and often seizures. Initially, it may not be possible to clinically differentiate encephalitis from meningitis, and treatment for both should be started. The underlying causative organism is only detected in fewer than half of the cases. In the UK, the most common causes of encephalitis are enteroviruses, respiratory viruses (influenza viruses), and herpesviruses [e.g. HSV, varicella zoster virus (VZV), and human herpesvirus 6 (HHV-6)]. Worldwide, microorganisms causing encephalitis include *Mycoplasma*, *B. burgdorferi* (Lyme disease), *Bartonella henselae* (cat scratch disease), rickettsial infections (e.g. Rocky Mountain spotted fever), and arboviruses.

HSV is a rare cause of childhood encephalitis but it can have devastating long-term consequences. All children with encephalitis should therefore be treated initially with high-dose intravenous aciclovir (acyclovir) until this diagnosis has been ruled out, because this is a very safe treatment. Most affected children do not have outward signs of herpes infection, such as cold sores, gingivostomatitis, or skin lesions. PCR is used in the majority of laboratories to detect HSV in CSF. As HSV encephalitis is a destructive infection, the electroencephalogram and CT/MRI scan may show focal changes, particularly within the temporal lobes either unilaterally or bilaterally (Fig. 15.5). These tests may be normal initially and need to be repeated after a few days if the child is not improving. Later confirmation of the diagnosis may be made from HSV antibody production in the CSF. Proven cases of HSV encephalitis or cases where there is a high index of suspicion should be treated with intravenous aciclovir for 3 weeks, as relapses may occur after shorter courses. Untreated, the mortality rate from HSV encephalitis is over 70% and survivors usually have severe neurological sequelae.

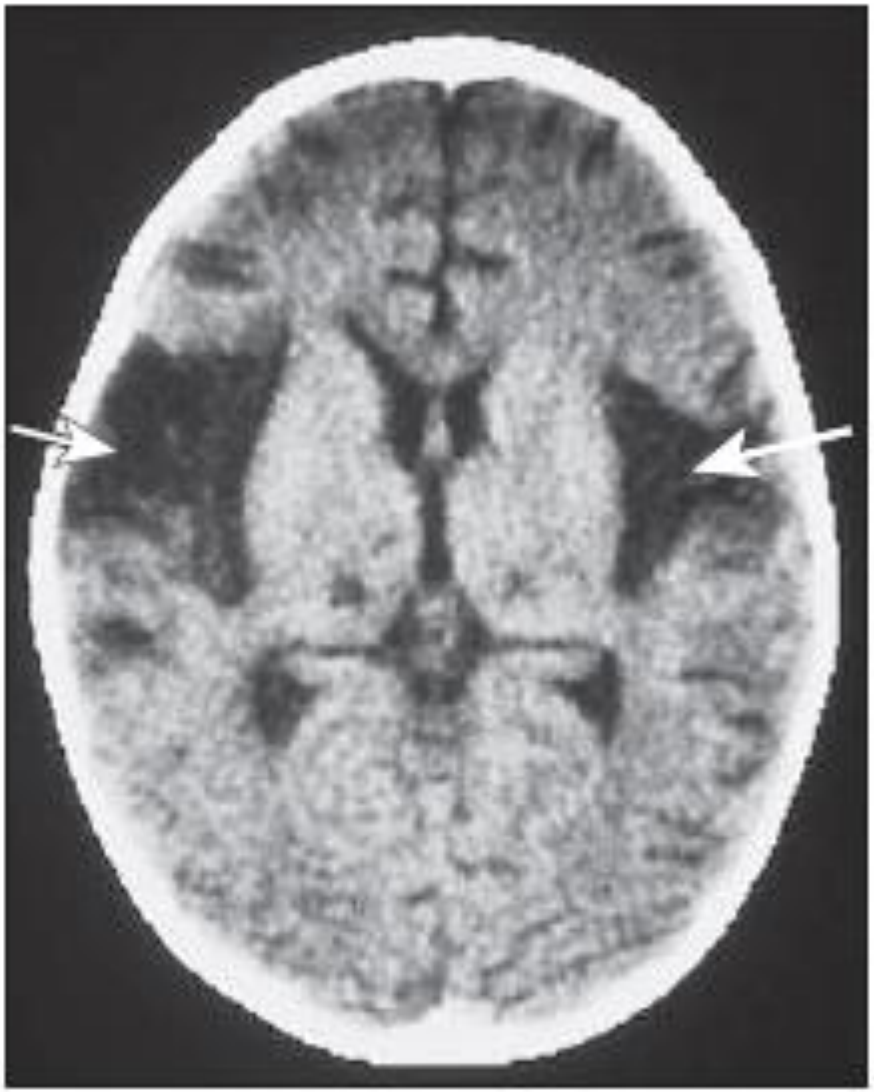


Figure 15.5 Herpes simplex encephalitis. The computed tomography scan shows gross atrophy from loss of neural tissue in the temporoparietal regions (arrows).

Summary

Encephalitis

- Onset can be insidious and includes behavioural change.
- Consider if HSV could be the cause.
- Treat potential HSV with parenteral high-dose aciclovir until this diagnosis is excluded.

Meningococcal infection

Meningococcal infection is a disease that strikes fear into both parents and doctors, as it can kill previously healthy children within hours (Case History 15.1). However, of the three main causes of bacterial meningitis, meningococcal infection has the lowest risk of long-term neurological sequelae, with most survivors recovering fully. The septicaemia is usually accompanied by a purpuric rash, which may start anywhere on the body and then spreads. The rash may or may not be present in cases with meningococcal meningitis. Characteristic lesions are nonblanching on palpation, irregular in size and outline, and may have a necrotic centre (Fig. 15.8a,b). Any febrile child who develops a purpuric rash should be treated immediately, at home or in the general practitioner's surgery, with intramuscular penicillin or intravenous third-generation cephalosporin before urgent admission to hospital. Following the inclusion of the meningococcus C vaccine into the routine immunization schedule in the UK,

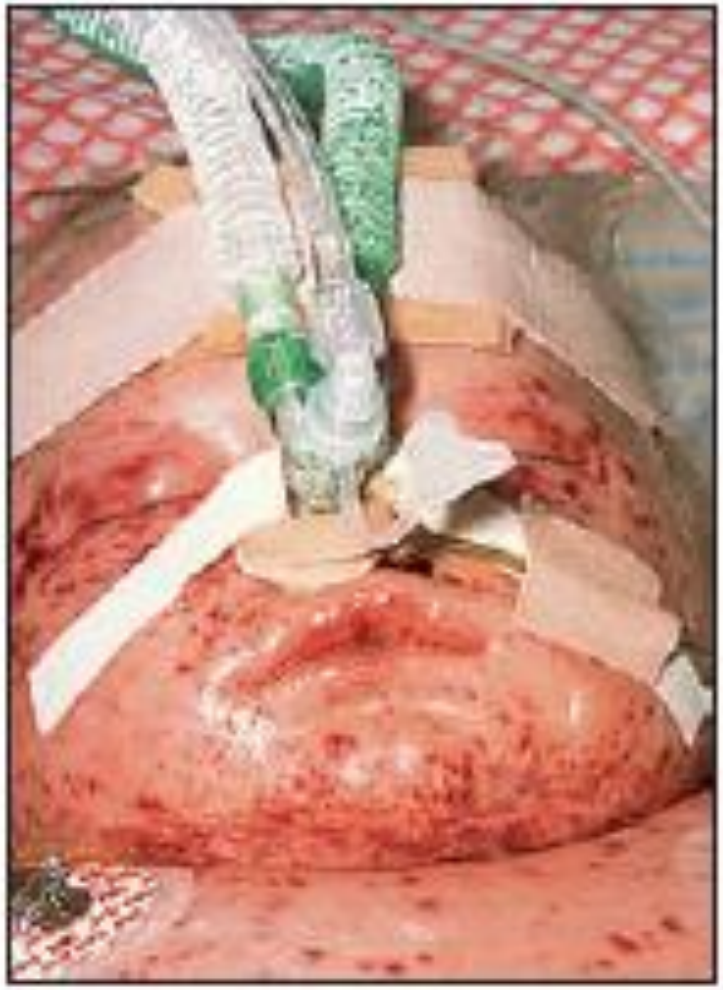
group B meningococci causes the majority of disease. As polysaccharide conjugate vaccines against group A, B, and C meningococci have now been developed and incorporated into the immunization schedule, the incidence of meningococcal disease should decline further.

Case history 15.1

Meningococcal septicaemia

This 7-month-old boy presented with a 12-hour history of lethargy and a spreading purpuric rash. In hospital, he required immediate resuscitation and transfer to a paediatric intensive care unit for multiorgan failure (Fig. 15.7a). The gross oedema is from leak of capillary fluid into the tissues. He required colloid and inotropic support and peritoneal dialysis for renal failure. He made a full recovery (Fig. 15.7b).

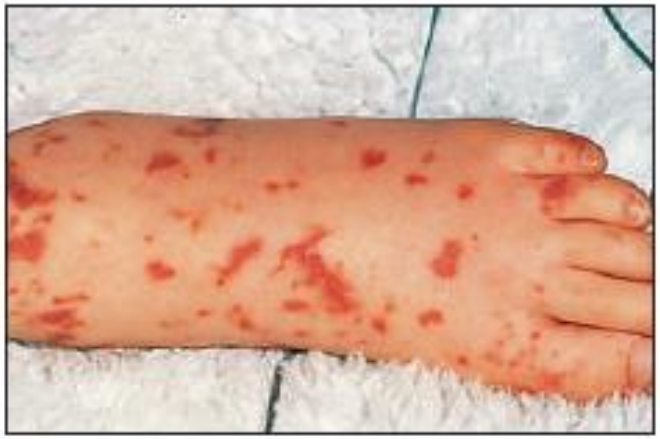
Meningococcal septicaemia can kill children in hours. Optimal outcome requires immediate recognition, prompt resuscitation and antibiotics



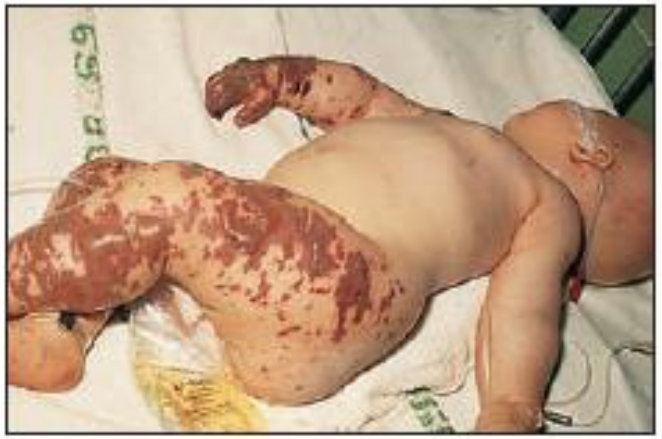
(a)

(b)

Figure 15.7 (a) A boy with meningococcal septicaemia receiving intensive care. (b) After full recovery. (Courtesy of Dr Parviz Habibi.)



(a)



(b)

Figure 15.8 Rash of meningococcal infection. (**a**) Characteristic purpuric skin lesions, irregular in size and outline and with a necrotic centre; and (**b**) the lesions may be extensive, when it is called *purpura fulminans*.

Any febrile child with a purpuric rash should be given intramuscular benzylpenicillin immediately and transferred urgently to hospital

Chronic meningitis

- slow-growing organisms
- develops over two weeks or more.

Fungal meningitis

- uncommon and causes chronic meningitis.
- isn't contagious from person to person.
- Cryptococcal meningitis is a common
- immune deficiencies, such as AIDS.

Fungal meningitis

- In 2012, fungal meningitis made the news because contaminated corticosteroid injections caused a multistate outbreak.

Other meningitis causes

- Tuberculosis.

Other meningitis causes

- chemical reactions, drug allergies, some types of cancer and inflammatory diseases such as sarcoidosis.

Other meningitis causes

- Central nervous system (CNS) leukemia
- Lead encephalopathy
- Hypersensitivity to drugs (eg, trimethoprim-sulfamethoxazole, intravenous immune globulin, and antithymocyte globulin)
- Disorders associated with vasculitis (eg, Kawasaki disease and collagen vascular disease)

Other problems to be considered include the following:

- Brain abscess
- Subdural/epidural abscess
- Brain tumors

Causes

- Viruses associated with aseptic meningitis
  - EV 71, EV 70, EV 7518,12
  - Polioviruses types 1, 2, and 3
  - Coxsackievirus type A (23 serotypes), coxsackievirus type B (6 serotypes)
  - Echoviruses (31 serotypes; see image below)

Causes

- Human parechoviruses (HPeV) (6 serotypes; HPeV types 1 and 2, previously classified as echovirus types 22 and 23 within the genus Enterovirus
- Arbovirus (eastern, western, and Venezuelan equine encephalitis viruses; Powassan virus; California group viruses [primarily LaCrosse virus]; St. Louis encephalitis virus; West Nile virus; Colorado tick fever)

Causes

- Mumps
- HSV types 1 and 2
- Cytomegalovirus (CMV)
- Epstein-Barr virus (EBV)
- Human herpesvirus type 6 (HHV6), human herpesvirus type 7 (HHV7)
- Varicella zoster virus (VZV)
- Adenoviruses (types 3 and 7)

Causes

- Human immunodeficiency virus (HIV)
- Lymphocytic choriomeningitis (associated with contact with guinea pigs, hamsters, and pet mice)
- Rhinovirus
- Measles
- Rubella
- Influenza A and B
- Parainfluenza
- Parvovirus B19
- Rotavirus
- Coronavirus
- Variola virus

Causes

***Vaccines related to aseptic meningitis**

- **Mumps19**
- **Measles, mumps, rubella (MMR)**
- **Polio**
- **Rabies**
- **Yellow fever**

***Bacterial infection associated with aseptic meningitis (partially treated bacterial meningitis or brain abscess)**

Causes

***Nonpyogenic bacteria associated with aseptic meningitis**

- **Mycobacterium tuberculosis**
- **Leptospira**
- **Treponema pallidum**
- **Borrelia (relapsing fever, Lyme disease)**
- **Nocardia**
- **Bartonella**
- **Atypical mycobacteria**
- **Brucella**

Causes

* **Parasites associated with aseptic meningitis**
  - **Roundworms**
  - **Tapeworms**
  - **Flukes**
  - **Amoebae**
  - **Toxoplasma**

Causes

* **Atypical organisms associated with aseptic meningitis**
  - **Chlamydia**
  - **Rickettsia**
  - **Mycoplasma**

Causes

- **Other organisms associated with aseptic meningitis**
  - ***Blastomyces dermatitidis***
  - ***Coccidioides immitis***
  - ***Alternaria* species**
  - ***Aspergillus* species**
  - ***Cephalosporium* species**
  - ***Cladosporium trichoides***
  - ***Drechslera hawaiiensis***
  - ***Paracoccidioides brasiliensis***
  - ***Petriellidium boydii***
  - ***Sporotrichum schenckii***
  - ***Ustilago* species**
  - ***Zygomycetes* species**

Causes

* **Diseases associated with aseptic meningitis**
  - **Leukemia**
  - **Behçet disease**
  - **Systemic lupus erythematosus (SLE)**
  - **Sarcoidosis**
  - **CNS tumor**
  - **Kawasaki disease**
  - **Recurrent benign endothelioleukocytic aseptic meningitis (Mollaret Meningitis)**
  - **Neonatal-onset multisystem inflammatory disorder (one of the cryopyrin-associated periodic syndromes [CAPS])**

Causes

- **Other associations with aseptic meningitis**
  - **Immunoglobulin replacement therapy**
  - **Heavy metal poisoning**
  - **Intrathecal agents**
  - **Foreign bodies (eg, shunt or reservoir)**
  - **Drugs**

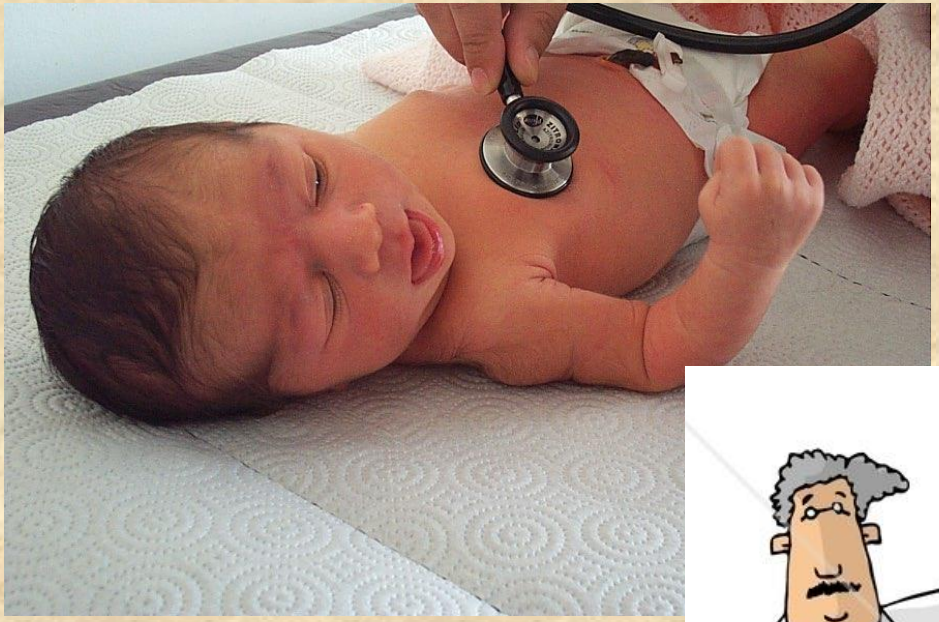

مجمع الأسد الطبي

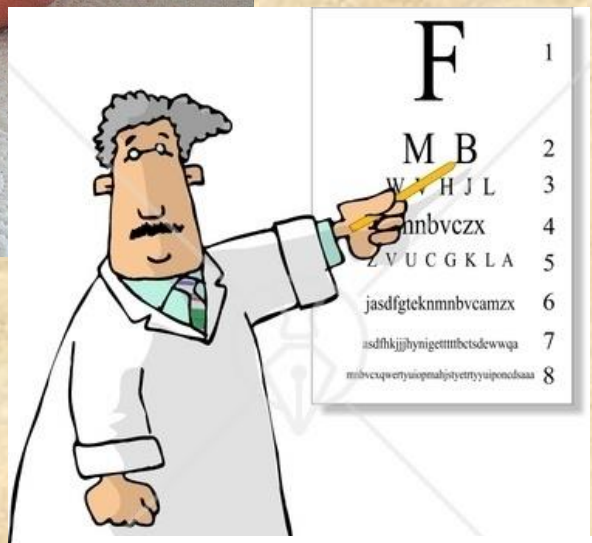
Consultations

- **Referral to a specialized pediatric ICU is appropriate if the level of consciousness is reduced and the airway cannot be maintained.**

Prognosis

- **Full recovery** is usual after uncomplicated viral aseptic meningitis. Most cases resolve within **7-10 days**.
- **Psychological sequelae** may be more common than in bacterial meningitis.
- **Recurrence** is possible (known as **Mollaret, or benign recurrent meningitis**). Associated viruses include Epstein-Barr virus (EBV), coxsackieviruses B5 and B2, echoviruses 9 and 7, herpes simplex virus (HSV)-1 and HSV-2, and human immunodeficiency virus (HIV).

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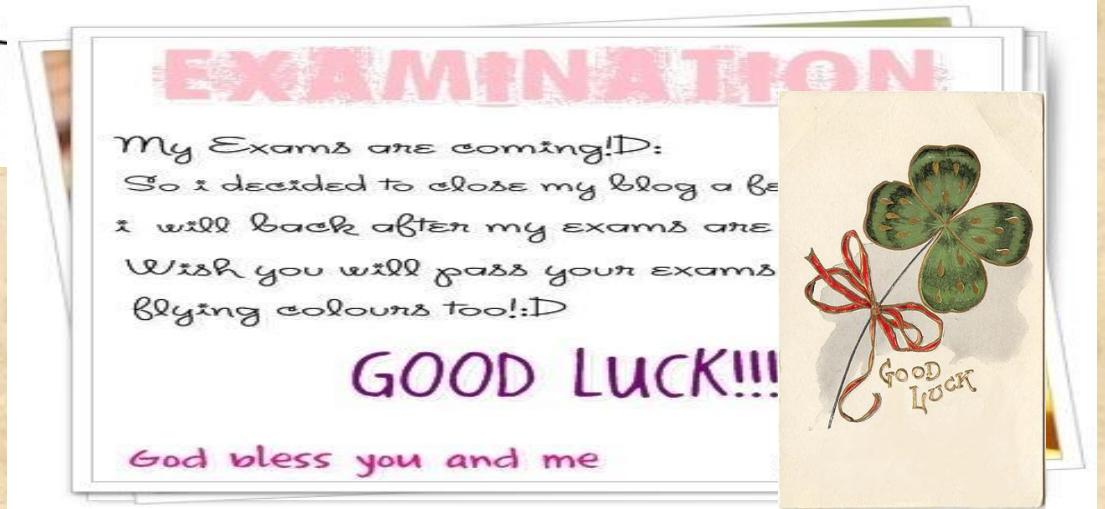
GOOD LUCK!


EXAMINATION
My Exams are coming!D:
So i decided to close my blog a
i will back after my exams are
Wish you will pass your exams
flying colours too!:D
GOOD LUCK!!!
God bless you and me
GOOD LUCK

Patient Education

- For excellent patient education resources, visit eMedicine's Brain and Nervous System Center and Children's Health Center. Also, see eMedicine's patient education articles Meningitis in Adults and Meningitis in Children.
- For more information, visit the Meningitis Foundation of America Web site.
- The Meningitis Research Foundation has excellent sections for nonexperts, parents, and health care professionals.

Mother

Mother

أ- قلق.

ب- أرق.

ج- اكتئاب.

د- تعب.

هـ- عدم رضا.

و- حزن.

Discharge

أ- البداية وليس نهاية المطاف.

ب- بالإنتظار صديقات و مجتمع و إعلام و ...

Family

أـ الكل معني بالأمر.

بـ البيت مدرسة.

جـ آباء و أمهات المستقبل.

Society

أ- الفضائيات.

ب- الإعلام.

د- الإنترنت.

School

المدرسة.

Doctor

المتخرج حديثاً.

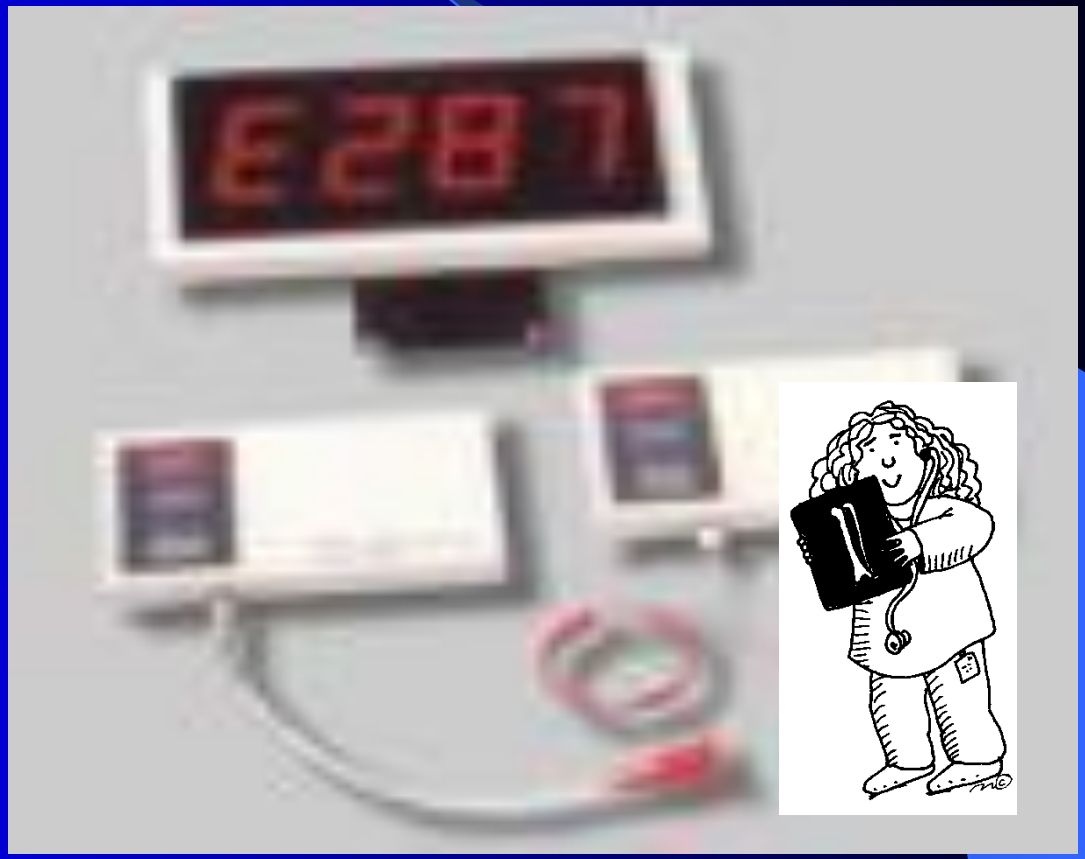
الخوف من هذه الحالات!

ضرورة عدم الخجل من تحويلها.

Doctor

ثقافة الإبرة.

Nurse



Time

الوقت.

Take it E&S

مليء بالورد و ليس الأشواك.

ESE

الأخلاق والعلم والثقافة.

الصحة العالمية للطفل

Abdul Muttaleb Alsah (Ph.D, MBBCH, MA, AMRCPCH)

First Consultant Paediatrician/ Genitest

Member of The Genetics Society of America

Member of The European Society for Human Genetics

Member of The International Society for Low Vision Research and Rehabilitation

Global child health

Features of global child health are:

- the number of deaths of children <5 years of age per year has halved in the last 25 years, but there is huge intercountry and intracountry variation in mortality rates
- poverty; poor maternal education; conflict; and inadequate access to safe water, sanitation and healthcare are the main determinants of child mortality
- the number of children dying from infectious diseases has declined markedly
- neonatal mortality accounts for an increasing proportion of child deaths, but could be significantly reduced by implementing basic neonatal care.

- 5.9 million children <5 years of age die each year
- 45% of all deaths in children <5 years of age occur in the neonatal period (first 4

After the neonatal period, about half of all child deaths are due to just five preventable and treatable conditions: pneumonia, diarrhoea, malaria, measles and HIV (human immunodeficiency virus)

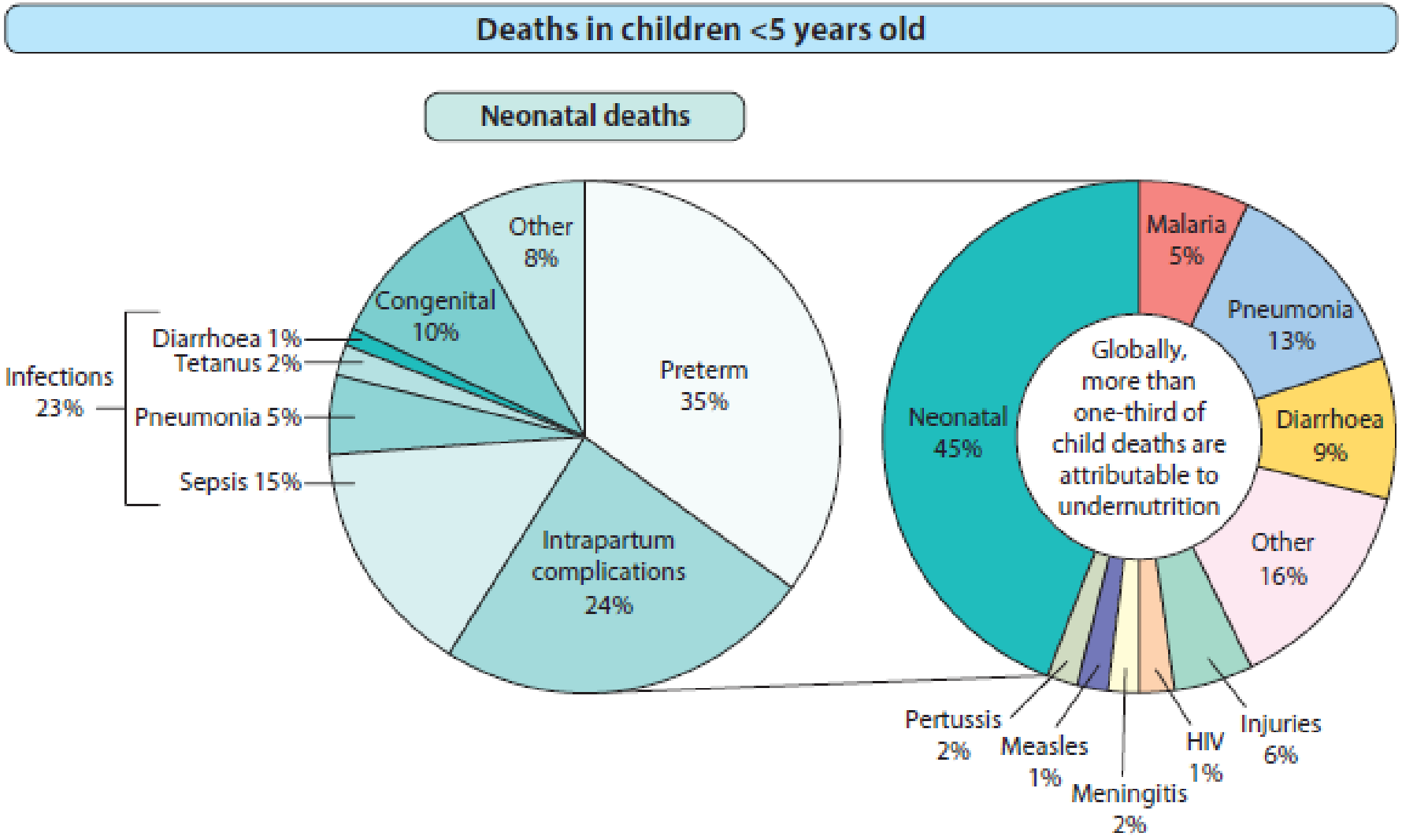


Figure 31.1 Causes of the 5.9 million deaths among children under 5 years of age in 2015. (Data from WHO fact sheet: Available at: www.who.int/mediacentre/factsheets/fs178/en/ and Liu L, Oza S, Hogan D, Perin J, Rudan I, et al: Global, regional, and national causes of child mortality in 2000–13, with projections to inform post-2015 priorities: an updated systematic analysis. *Lancet* 385:430–440, 2015 – Erratum in: *Lancet* 385:420, 2015.)

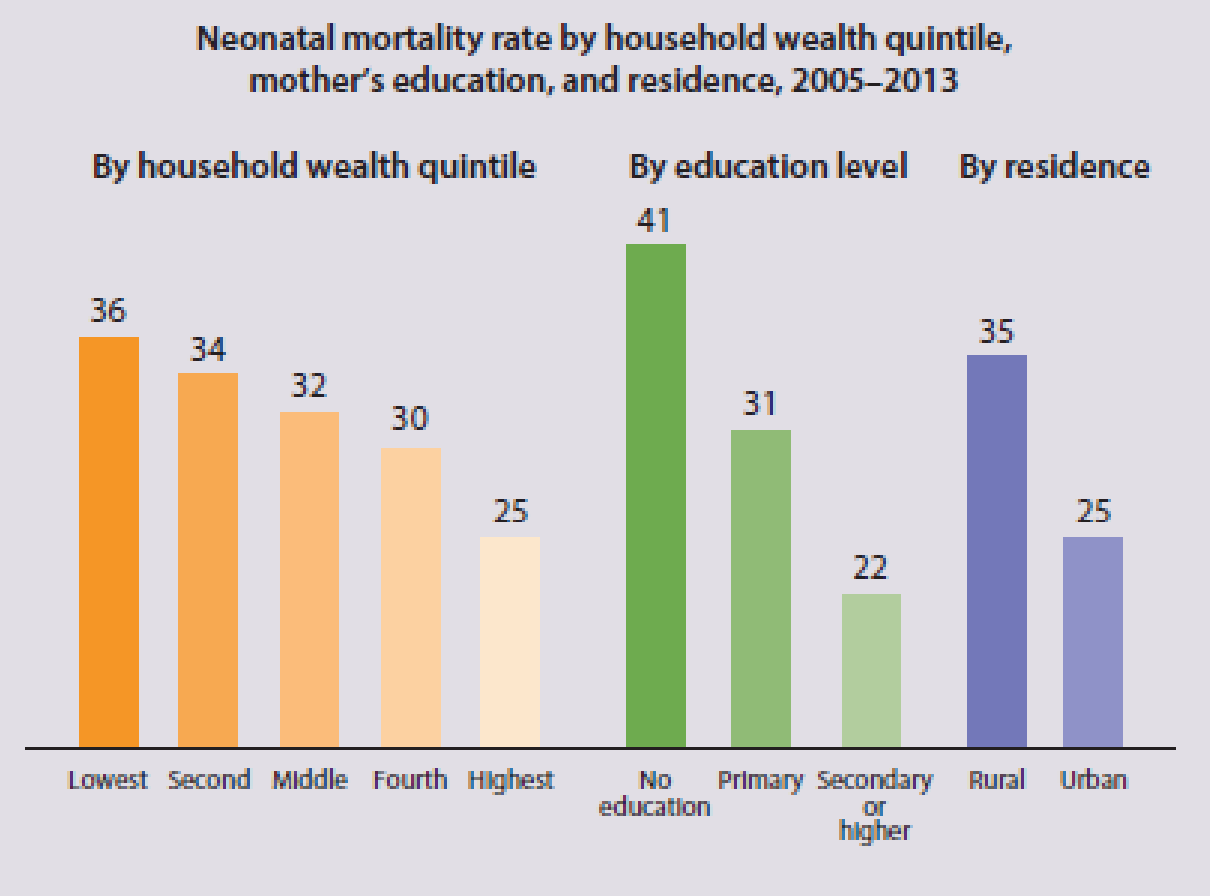


Figure 31.3 Children born to poorer households, to mothers with no formal education, and living in rural areas face a higher risk of dying in the first 28 days of life. (Data from Committing to Child Survival: a promise renewed. Progress report 2014, UNICEF.)

Why is child mortality so high?

Children depend on their environment as well as the provision of healthcare for their survival, as described in Chapter 1 (The child in society). The environment can have major detrimental effects on their chances of survival:

- poverty and social determinants – pervade almost all aspects of health and healthcare. In almost all low-resource countries, there is a marked difference in health outcomes between high-income and low-income groups (Fig. 31.3)
- maternal education – the higher the level of maternal education, the higher the chance of her child's survival (Fig. 31.3)

- poor sanitation and unclean water – increase in diarrhoea and infection
- poor food security – malnutrition increases vulnerability and severity of illness, especially infection [see Ch. 13 (Nutrition)]. Chronic malnutrition is reflected in stunting of children
- inadequate housing, air pollution, and overcrowding – promotes the spread of respiratory pathogens (Fig. 31.3)
- conflict – see the 'Children affected by conflict' section
- geographical variation in diseases – large intercountry and inter-regional differences, e.g. malaria is responsible for 15% of deaths in children under-5-years-old in Sub-Saharan Africa compared with 1% in Southeast Asia.

The provision of healthcare in many low and middle-income countries is poor and adversely affects survival:

- limited finance available for healthcare – whereas Sweden spends US$4244/person per year on health (10% of gross domestic product), in 34 low-resource countries it is less than US$50/person per year (WHO 2013)
- disease prevention, e.g. vaccination, insecticide-treated net distribution for protection from malaria – lower coverage in poor areas
- access – difficult to get to health facilities; cost and affordability may be major limiting factors
- community care is limited by lack of trained community health workers
- health centres and hospitals – care compromised by lack of trained staff, equipment, drugs, suitable buildings, etc.
- medical care – lack of trained doctors is a serious problem in almost all these countries. The contrast between the proportion of doctors compared with deaths in children is shown in Figs 31.4 and 31.5.

Reducing child mortality

There has been a major effort by many low and middle-income countries to improve child health. This has been done in conjunction with the international community,

Summary

Global child health

- The number of child deaths globally has decreased to 5.9 million under-5-year-old deaths per year, but the proportion of neonatal deaths has risen to 44%.
- Over half of all child deaths occur in just five countries: India, Nigeria, Democratic Republic of Congo, Pakistan, and China.
- The main causes of neonatal mortality are preterm birth, infection, and intrapartum-related conditions.
- The main causes of postneonatal child mortality are pneumonia, diarrhoea, malaria, and injuries.
- Malnutrition contributes to one-third of child deaths.
- Trauma, conflict, being refugees, mental health, and the coexistence of multiple pathologies pose significant and ongoing threats to the wellbeing of children in low and middle-income countries.
- The Sustainable Development Goals, approved by the United Nations in 2015, are setting the agenda for improving global child health until 2030.