Al-Rasheed University College / Pharmacy Department

Hospital Training Manual

5th Stage

Pediatric wards

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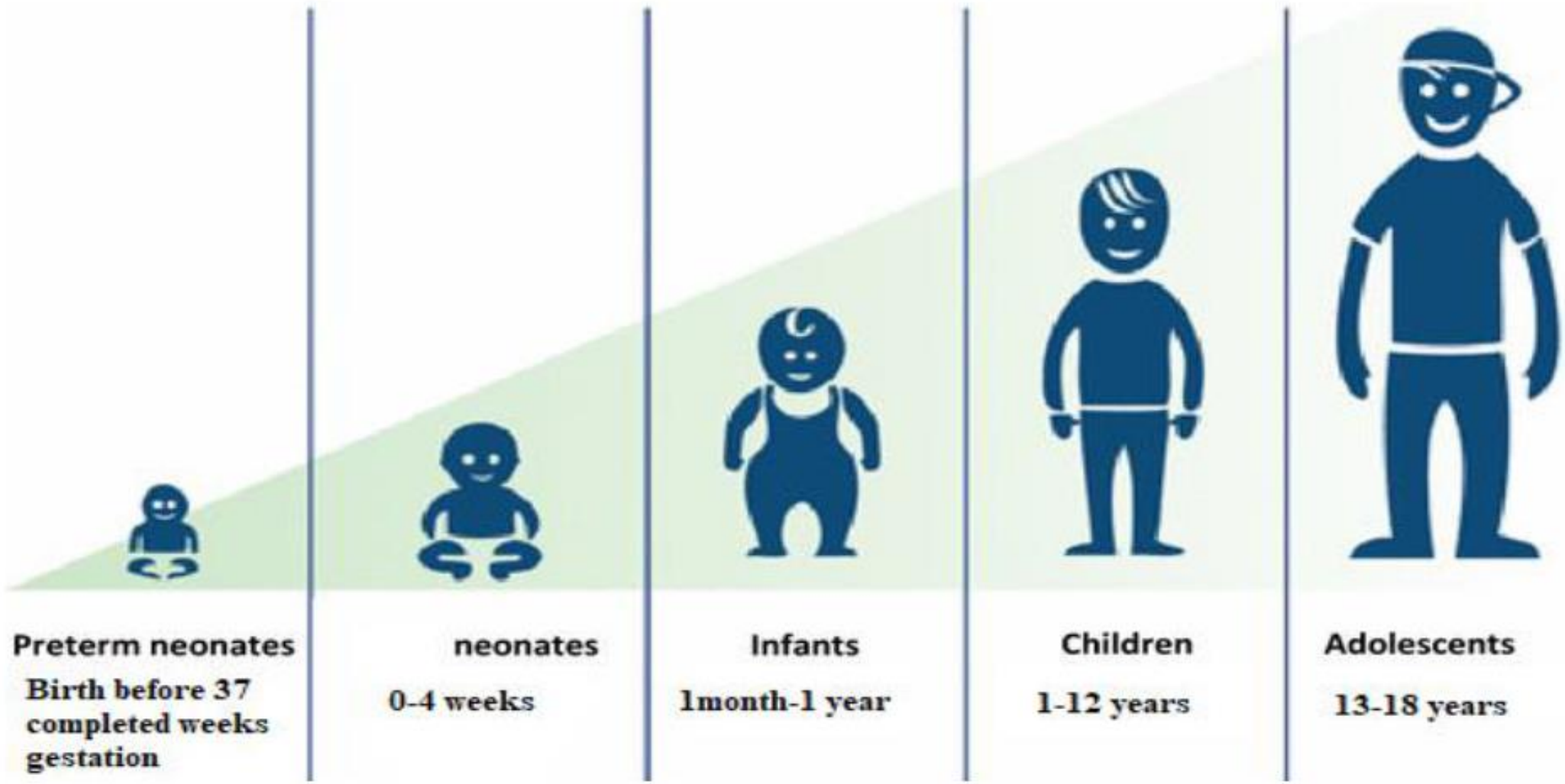
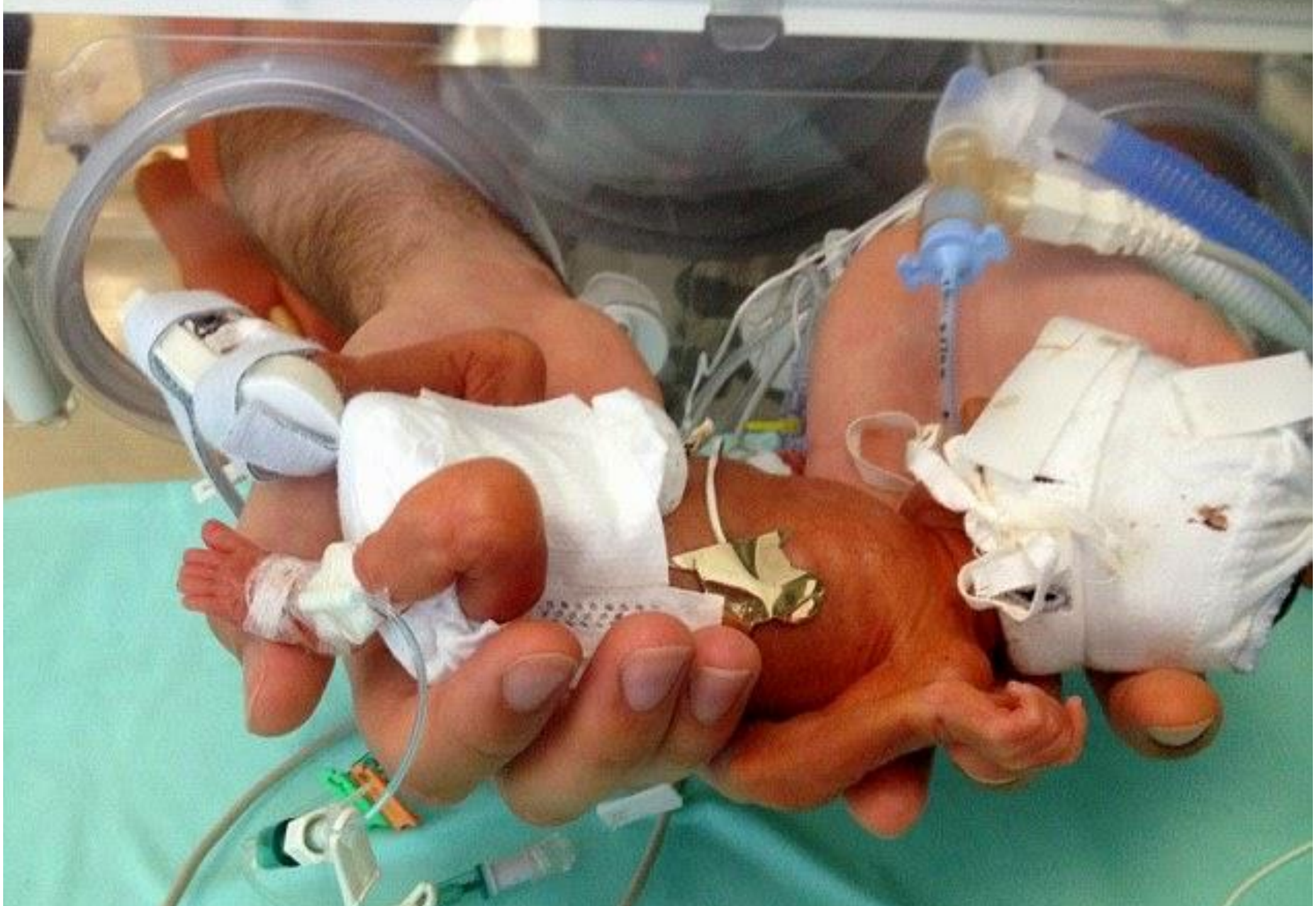
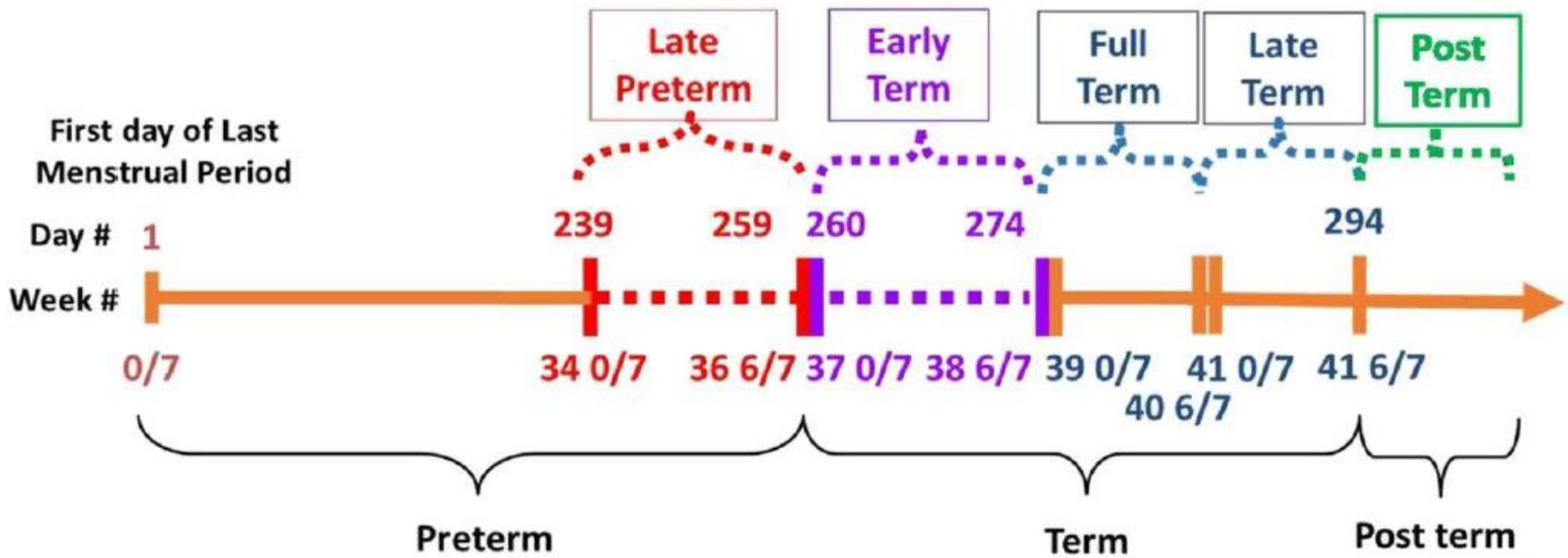
Pediatric age category





Newborn age classification



1st: Neonatal Sepsis and Meningitis

- Neonatal sepsis is a clinical syndrome in an infant 28 days of life or younger, manifested by systemic signs of infection and isolation of a bacterial pathogen from the bloodstream
- Bacterial meningitis is more common in the first month than at any other time of life, The clinical presentation of neonatal meningitis is indistinguishable from that of neonatal sepsis without meningitis

A. Signs and symptoms

i. General: Temperature instability, feeding intolerance, lethargy, grunting, nasal flaring, apnea

ii. More likely to be associated with meningitis: Bulging fontanelle and seizures

B. Early versus late onset neonatal sepsis

a) Onset

i. Early: Within 72 hours of birth

ii. Late: After the first 72 hours of life

b) Risk factors

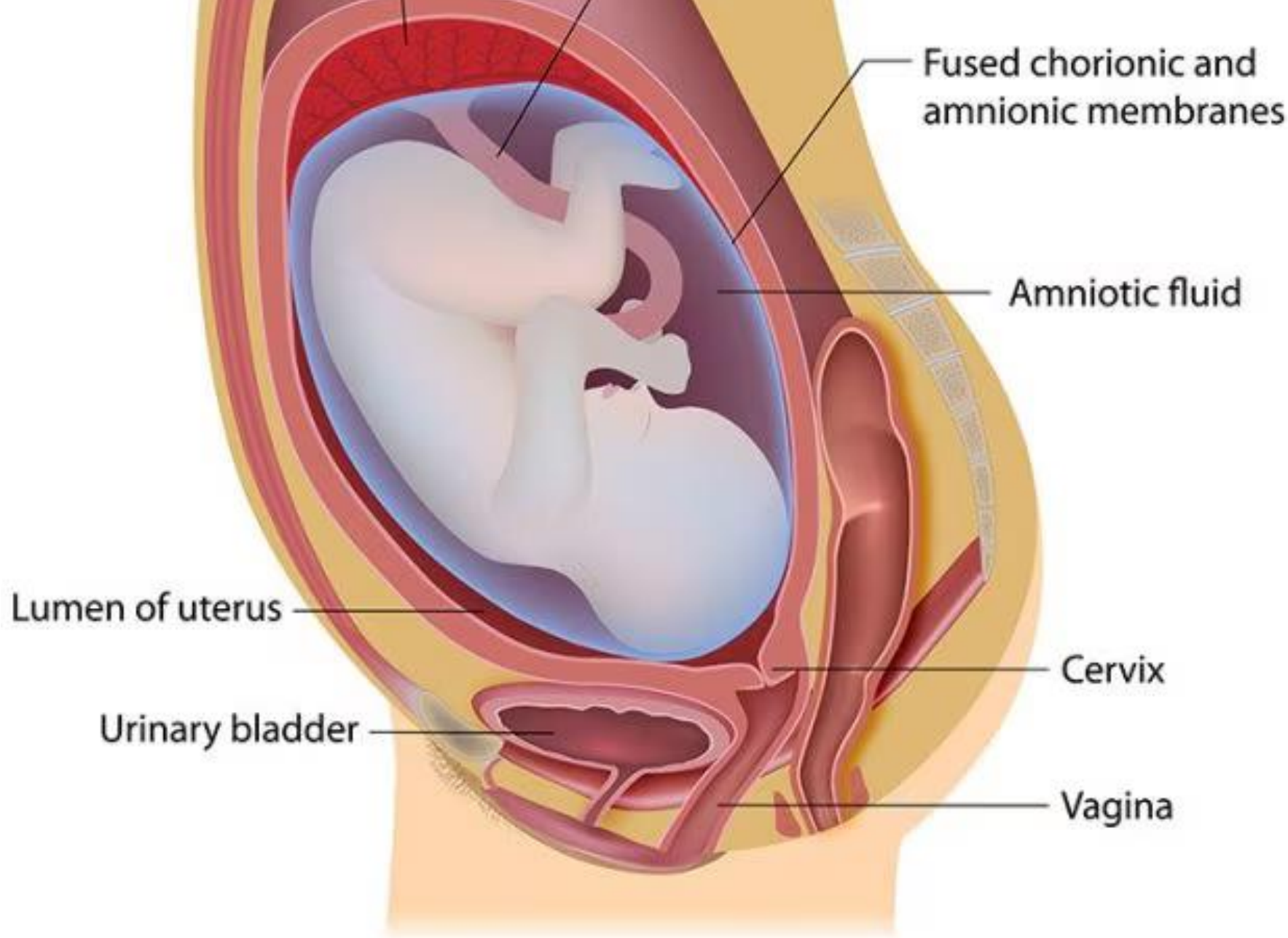
i. Early

Usually related to maternal or obstetric risk factors: very low birth weight (less than 1500 g), prolonged rupture of amniotic membranes (greater than 18 hours), maternal fever, prolonged labor, maternal genitourinary tract colonization with group B *Streptococcus*, maternal endometritis, or chorioamnionitis

ii. Late

a. Unrelated to obstetric risk factors

b. Usually related to iatrogenic/nosocomial factors (e.g., endotracheal tubes, central venous catheters)


Fused chorionic and amnionic membranes
Amniotic fluid
Lumen of uterus
Cervix
Urinary bladder
Vagina

Mortality: Increases with decreasing gestational age

- 1.6% in term neonates

- 30% in preterm neonates 25–28 weeks

- 50% in preterm neonates 22–24 weeks

C. Cerebrospinal fluid (CSF) findings: see (table 1)

Table 1. Cerebrospinal Fluid Findings

Laboratory Value	Normal Child	Normal Newborn	Bacterial Meningitis	Viral Meningitis
WBC (cells/mL)	0–6	0–30	>1000	100–500
Neutrophils (%)	0	2–3	>50	<40
Glucose (mg/dL)	40–80	32–121	<30	>30
Protein (mg/dL)	20–30	19–149	>100	50–100
RBC (cells/mL)	0–2	0–2	0–10	0–2

للحفظ

D. Common Pathogens for Sepsis and Meningitis: see (table 2)

Table 2. Common Pathogens

Age	Organism
0–1 mo	Early onset: Group B *Streptococcus* (*Streptococcus agalactiae*) – 40%–45% of cases in term and late-preterm neonates (20% of cases in preterm neonates < 34 weeks' gestation) *Escherichia coli* – 10%–15% of cases in term and late-preterm neonates (50% of cases in preterm neonates < 34 weeks' gestation) *Listeria monocytogenes* – < 2% of cases in term and late-preterm neonates Late onset: Viral (e.g., herpes simplex virus) Coagulase-negative *Staphylococcus* (nosocomial) Gram-negative bacteria (e.g., *Klebsiella* spp., *Pseudomonas* spp., *Enterobacter* spp.; nosocomial)

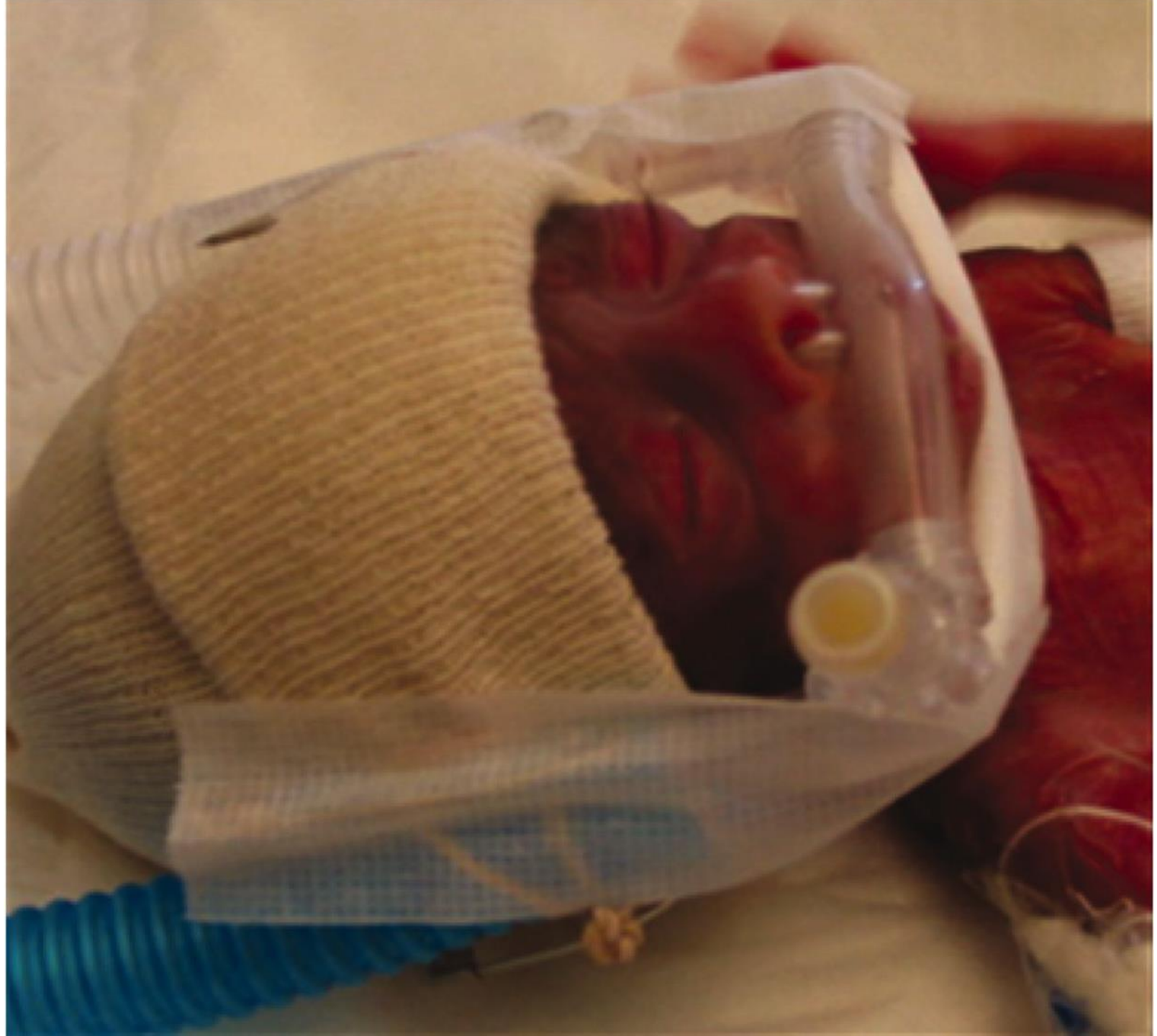
E. Sequelae of Meningitis

Hearing loss	Visual impairment	Hydrocephalus
Mental retardation and learning deficits	Seizures	Mortality 10%–20% in neonates

F. Treatment of sepsis and meningitis: it's important to know the **right duration**, preferred **empiric therapy**, and **drug of choice** for specific pathogens. The optimum dose per age and kg was illustrated in (Table 3).

Table 3: Antimicrobial dosage for neonates للاطلاع والفهم

	Weight <1,200 g	Weight 1,200–2,000 g		Weight >2,000 g	
Drug	0–4 weeks PNA (mg/kg)[a]	0–7 days PNA (mg/kg)[a]	8–28 days PNA (mg/kg)[a]	0–7 days PNA (mg/kg)[a]	8–28 days PNA (mg/kg)[a]
Amphotericin B					
Deoxycholate	1 every 24 hours	1 every 24 hours	1 every 24 hours	1 every 24 hours	1 every 24 hours
Lipid complex/Liposomal	5 every 24 hours	5 every 24 hours	5 every 24 hours	5 every 24 hours	5 every 24 hours
Ampicillin					
Meningitis	100 every 12 hours	100 every 8 hours	75 every 6 hours	100 every 8 hours	75 every 6 hours
Other diseases	50 every 12 hours	50 every 12 hours	50 every 8 hours	50 every 8 hours	50 every 6 hours
Cefotaxime[b]	50 every 12 hours	50 every 12 hours	50 every 8 hours	50 every 12 hours	50 every 8 hours
Fluconazole	6 every 72 hours	12 every 24 hours	12 every 24 hours	12 every 24 hours	12 every 24 hours
Metronidazole	7.5 every 48 hours	7.5 every 24 hours	15 every 24 hours	15 every 24 hours	15 every 12 hours
Oxacillin/Nafcillin[b]	25 every 12 hours	25 every 12 hours	25 every 8 hours	25 every 8 hours	25 every 6 hours
Penicillin G crystalline					
Meningitis[c]	50,000 units every 12 hours	100,000 units every 12 hours	100,000 units every 8 hours	100,000 units every 8 hours	100,000 units every 6 hours
Other diseases	25,000–50,000 units every 12 hours	25,000–50,000 units every 12 hours	25,000–50,000 units every 8 hours	25,000–50,000 units every 12 hours	25,000–50,000 units every 8 hours
Vancomycin	15 every 24 hours	15 every 12–18 hours	15 every 8–12 hours	15 every 8–12 hours	15 every 6–8 hours



2nd: Apnea of prematurity

Types:

1. Central: Reduced sensitivity to hypercarbia of the central respiratory center results in a lack of respiratory effort.

2. Obstructive: the inability to maintain upper airway (i.e., larynx and pharynx) patency results in obstruction of airflow
3. Mixed – The most common type of apnea of prematurity, a combination of central and obstructive apnea

Clinical Presentation: Cessation of breathing for at least 20 seconds or pauses in breathing of less than 20 seconds if associated with bradycardia (heart rate less than 100 beats/minute), cyanosis, or pallor

Risk factors: a. Prematurity **&** b. Extremely low birth weight (less than 1000 g)

Treatment

1. Methylxanthines (i.e., theophylline/aminophylline and caffeine)

✓ **Mechanism of action**:

- CNS stimulant
- Increase respiratory drive
- Increase sensitivity to hypercapnia

✓ Treatment duration

Most neonates will “outgrow” apnea of prematurity **by 34–36 weeks’** postmenstrual age because of maturation of the central respiratory center, so methylxanthine therapy can be discontinued at that time.

3rd: TORCH infections

Infections acquired in utero or during the birth process are a significant cause of fetal and neonatal mortality and an important contributor to early and later childhood morbidity

T - TOXOPLASMA

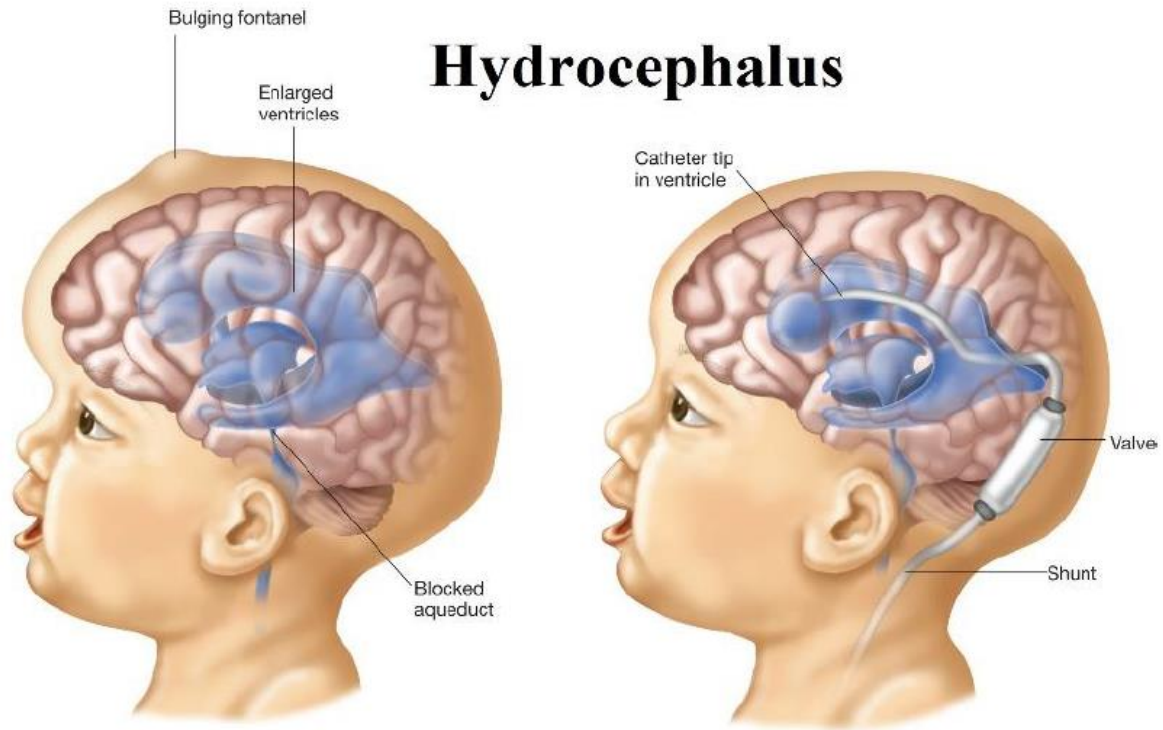
O - OTHER PATHOGENS (SYPHILIS)

R - RUBELLA

C - CYTOMEGALOVIRUS

H - HERPES SIMPLEX VIRUS

Congenital toxoplasmosis: Toxoplasmosis is caused by the protozoan parasite Toxoplasma gondii. Primary infection during pregnancy can result in congenital disease. Presentation: Chorioretinitis, Intracranial calcifications, Hydrocephalus, Abnormal cerebrospinal fluid, Jaundice, Thrombocytopenia, Hepatosplenomegaly, Lymphadenopathy, Pneumonitis, Rash, Seizures, Microphthalmia, Microcephaly. **The preferred antiparasitic regimen consists of pyrimethamine plus sulfadiazine (or sulfamerazine or sulfamethazine) and folinic acid (leucovorin)**



Congenital syphilis: Congenital syphilis occurs when the spirochete Treponema pallidum is transmitted from a pregnant woman to her fetus. Infection can result in stillbirth, hydrops fetalis, or prematurity and associated long-term morbidity. **Parenteral penicillin is the drug of choice for the treatment of congenital syphilis The single-dose regimen for treatment of congenital syphilis is as follows: Penicillin G benzathine (50,000 units/kg, intramuscularly [IM] as a single dose), Ten-day regimens — There are two alternative 10-day penicillin regimens for the treatment of congenital syphilis :Aqueous penicillin G 50,000 units/kg intravenously (IV) every 12 hours (for infants ≤7 days of age) and every 8 hours (for infants >7 days of age) for a total of 10 days.**

Hydrops fetalis

Congenital cytomegalovirus: Congenital cytomegalovirus (CMV) infection is the leading cause of nonhereditary sensorineural hearing loss and can cause other long-term

neurodevelopmental disabilities, including cerebral palsy, intellectual disability, vision impairment, and seizures. **Intravenous (IV) ganciclovir and its orally available prodrug, valganciclovir, are the first-line antiviral agents of choice for treatment of congenital CMV disease.**

Congenital Herpes simplex virus: Intrauterine HSV infection is rare and usually results from maternal viremia associated with primary HSV infection during pregnancy. Live-born infants with congenital HSV infection may exhibit a characteristic triad of skin vesicles, ulcerations, or scarring; eye damage; and severe CNS manifestations, including microcephaly or hydranencephaly. **Acyclovir is the antiviral agent of choice for the treatment of all categories of neonatal herpes simplex virus (HSV) infections, including skin, eye, and mouth (SEM), central nervous system (CNS), and disseminated disease**


NORMAL HEAD
MICROCEPHALY
wikiHow

4th: Necrotizing enterocolitis (NEC)

the most common gastrointestinal emergency in the newborn, is a disorder manifested by ischemic necrosis of the intestinal mucosa.

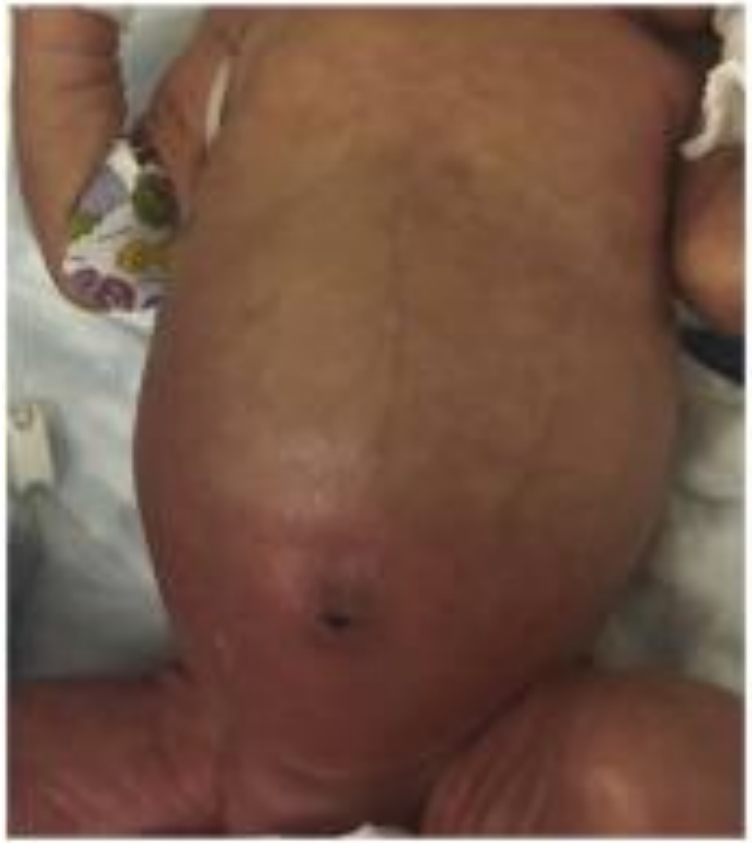
1. Risk factors

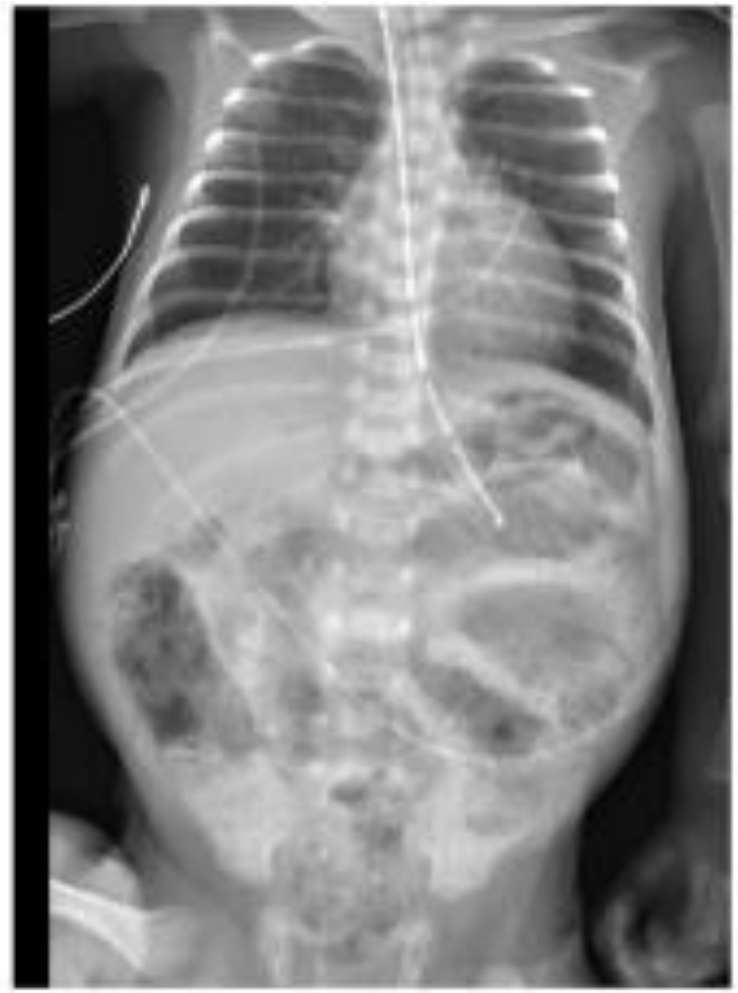
a. **Immaturity of the GI tract:** results in intestinal **hyperpermeability**, **decreased peristalsis** increases the time intestinal bacteria are in contact with carbohydrate substrates, and **decreased immunoglobulin A (IgA) production** by the immature intestine

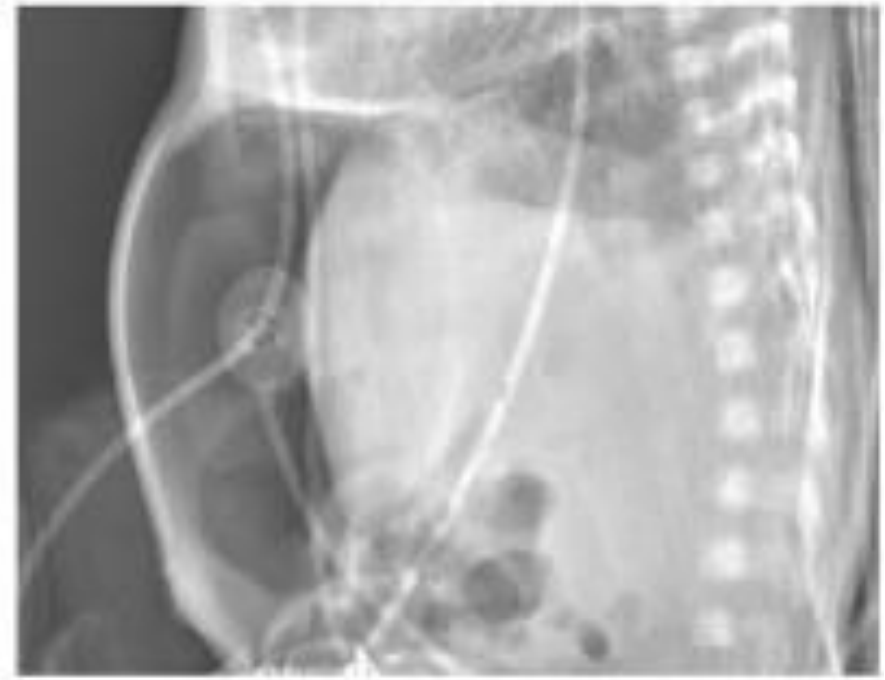
b. **Very low (less than 1500 g) or extremely low (less than 1000 g) birth weight**

c. **Formula feeding (as opposed to breast milk)**

d. **Abnormal bacterial colonization: because of prolonged antibiotic exposure**

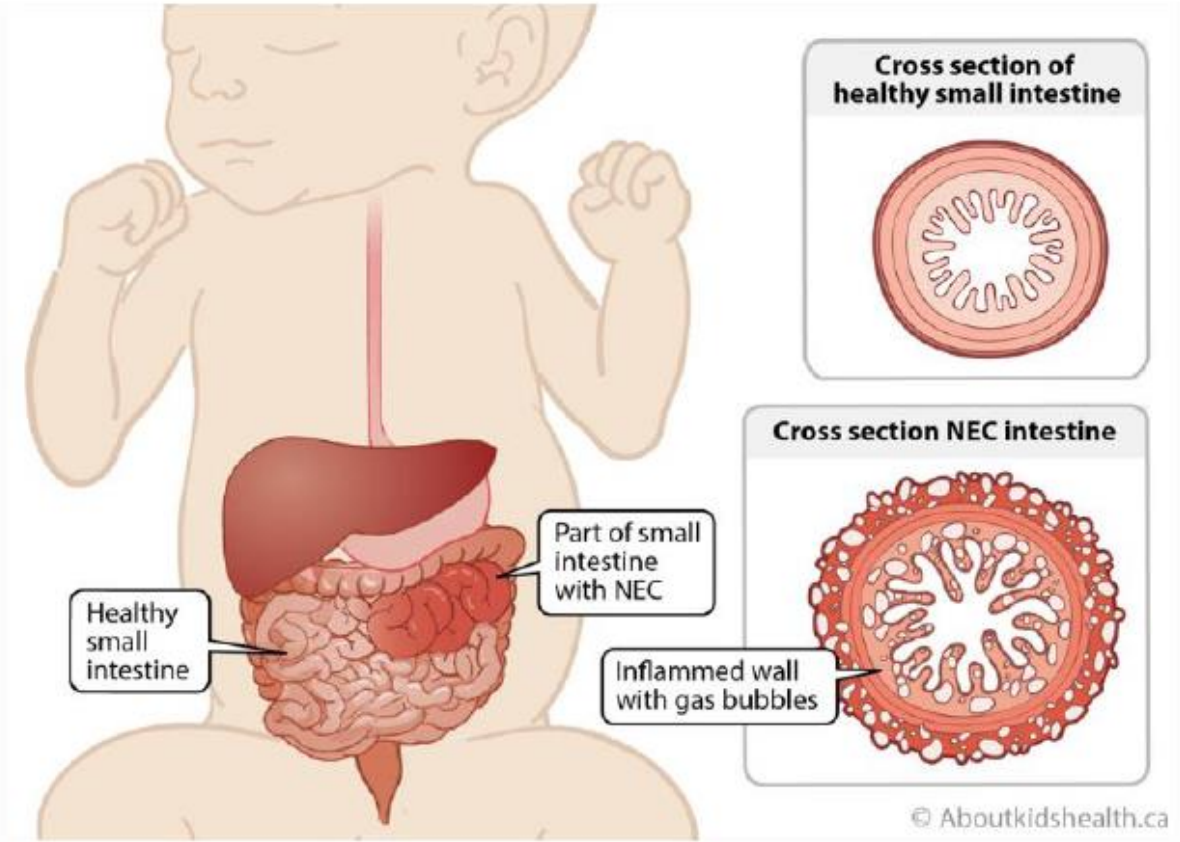






Clinical Presentation:

a. GI signs include feeding intolerance (e.g., gastric residuals or emesis), abdominal distension, bloody stool, discoloration of abdominal wall, apnea, bradycardia, lethargy, temperature instability, Radiologic findings include dilated loops of bowel, pneumatosis intestinalis (hallmark sign of NEC).



b. Mortality is high (up to 30% overall), especially with fulminant NEC (more than 90%).

3. Treatment

1- Nonpharmacologic: Bowel rest and abdominal decompression with gastric tube **&** Peritoneal drains and/or surgery

2- Pharmacologic

A- Supportive care: Intravenous fluids/parenteral nutrition

B- Antibiotics: The goals are to prevent ongoing GI mucosal injury, prevent translocation of enteric bacteria into the bloodstream, and reduce mortality. **Gram-positive** coverage can be provided by ampicillin or vancomycin. **Gram-negative** coverage can be provided by an aminoglycoside or a third-, or fourth- generation cephalosporin. **Anaerobic** coverage (e.g., clindamycin, metronidazole)

Prevention

a. Antenatal corticosteroids: Shown to augment intestinal maturation
b. Feeding protocols that emphasize the use of early trophic feedings (to promote peristalsis) and breast milk (contains maternal IgA) may help decrease NEC.
c. Probiotics – Augment intestinal maturation, reduce growth and colonization by potentially pathogenic organisms, increase amounts of anti-inflammatory cytokines. Consist mainly of strains of Lactobacillus, Bifidobacterium, and Saccharomyces

5th: Neonatal jaundice

- ✓ Neonatal jaundice is yellowish discoloration of the **skin, conjunctiva, and sclera** due to elevated serum or plasma **bilirubin** in the newborn period. Neonatal jaundice is typically a mild and transient event. However, it is imperative to identify newborns with jaundice that present with more **severe jaundice** or whose jaundice does not resolve in a typical manner.
- ✓ Unconjugated hyperbilirubinemia in neonates is due to **either physiologic or pathologic** causes. Over 75% of neonatal unconjugated hyperbilirubinemia is due to physiologic causes. Physiologic jaundice is also referred to as non-pathologic jaundice, and it is mild and transient