NEONATAL SEPSIS

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INTRODUCTION

Table 109-8	Definitions of Systemic Inflammatory Respiratory Response Syndrome and Sepsis in Pediatric Patients

SIRS: The systemic inflammatory response to a variety of clinical insults, manifested by 2 or more of the following conditions:
- Temperature instability <35°C (95°F) or >38.5°C (101.3°F)
- Respiratory dysfunction:
  - Tachypnea >2 SD above the mean for age
  - Hypoxemia (PaO_2 <70 mm Hg on room air)
- Cardiac dysfunction:
  - Tachycardia >2 SD above the mean for age
  - Delayed capillary refill >3 sec
  - Hypotension >2 SD below the mean for age
- Perfusion abnormalities:
  - Oliguria (urine output <0.5 mL/kg/hr)
  - Lactic acidosis (elevated plasma lactate and/or arterial pH <7.25)
  - Altered mental status

Sepsis: The systemic inflammatory response to an infectious process

From Adams-Chapman I, Stoll BJ: Systemic inflammatory response syndrome, Semin Pediatr Infect Dis *12:5–16, 2001.*

INTRODUCTION

- **Neonatal sepsis** is **a blood infection that occurs in an infant younger than 90 days old**.

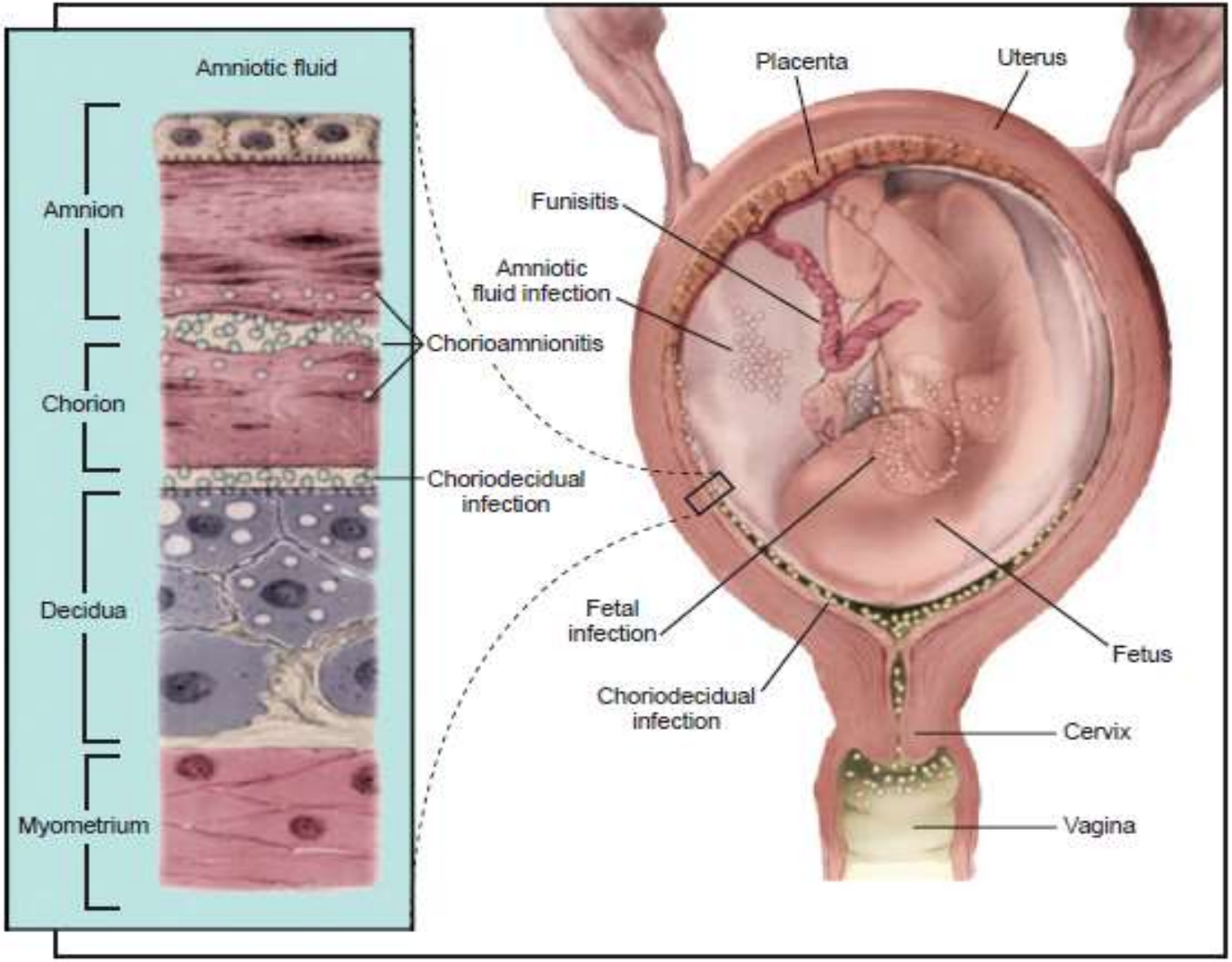
1. **Early-onset sepsis** is onset of sepsis in the 1st week of birth.
2. **Late-onset infections occur in newborns 7 days or older**, with most of these infections appearing in the first 3 months of life

- **Infections early in the first week of life** are typically related to **perinatal risk factors and vertical transmission from the mother**.

- **Nosocomial infections** are more often related to **patient colonization and/or environmental risk factors**.

INTRODUCTION

- There are several mechanisms by which bacteria can reach the fetus or newborn and initiate infection.
  1. **Maternal bloodstream infections**, can reach the fetus and cause infection.
  2. **Acquired from the vagina, cervix, or fecal contamination of the birth canal**.
  3. **Infection can occur via aspiration of birth canal contents or colonization of mucosal surfaces during passage through the birth canal, leading to pneumonia, followed by bacteremia and sepsis on day 1 or later**.
  4. Acquired after birth from human contact or from contaminated equipment mainly for late onset sepsis.



Potential sites of bacterial infection within the uterus

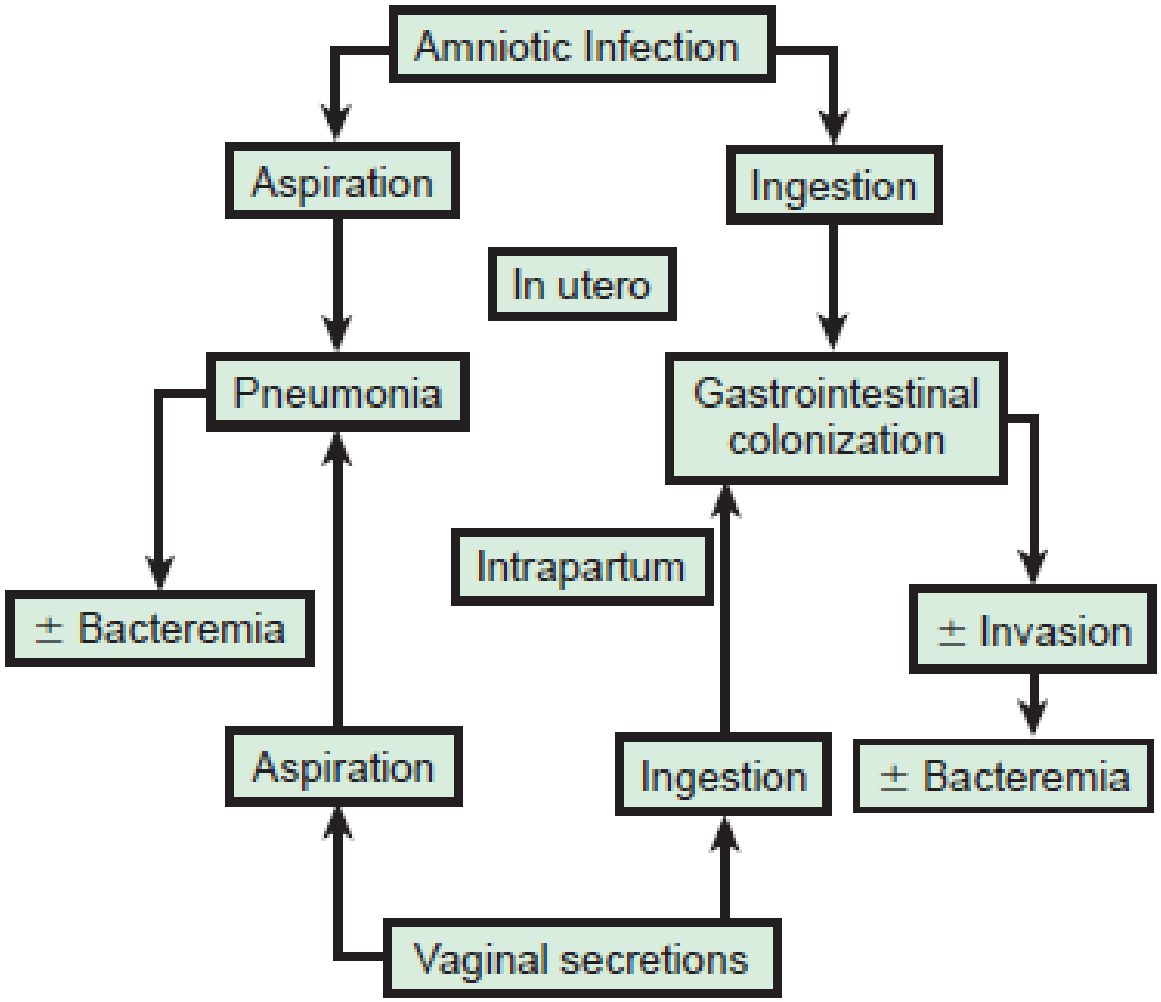


Figure 109-2 Pathways of ascending or intrapartum infection.

Risk factors for early onset sepsis

1. **Young maternal age,**
2. **Maternal fever**
3. **History of bacterial vaginosis**.
4. **Colonization with well-known pathogens (e.g., GBS)**
5. **The presence of maternal urinary tract infections,**
6. **Chorioamnionitis**.
7. **Prolonged labor and low APGAR score**
8. **Prolonged rupture of membranes (≥18 hours),**
9. **Internal scalp fetal monitoring,**
10. **Meconium aspiration**
11. **Prematurity** is considered the single greatest risk factor for early-onset bacterial infections.

Risk Factors for Development of Nosocommual Infection (Late onset sepsis)

1. **Lower gestational age and birthweight.**
   - Overall, VLBW infants are more vulnerable to infection, because of both the **immaturity of their immune response and their greater need for invasive devices**.
2. **Intravascular catheters,**
3. **Endotrachial intubation and Mechanical ventilators,**
4. **Ventriculoperitoneal shunts,**
5. **Urinary catheters.**
6. **Lack of enteric feeding and use of formula feedings.**
7. **Inborn error of metabolism**
8. **Exposure to broad spectrum antibiotics.**
9. **Underlying GI condition** such as necrotizing enterocolitis, gastroschisis, or omphalocele may be predisposed to mucosal barrier injury.
10. **Use of Histamine-blocking agents, proton pump inhibitors**, and **postnatally corticosteroids**.
11. **Prolonged parenteral nutrition.**

Epidemiology of **Early-Onset**

- Even in an era of intrapartum antibiotic prophylaxis of GBS-colonized mothers, **GBS remains the most common bacterial pathogen in neonatal centers followed by *E. coli*.**

- Regional differences exist.

- It is clear that the **incidence has declined as a result of intrapartum antibiotic therapy.**

Nosocommual Infection Epidemiology in the NICU

- **Rates of late-onset sepsis declined** among VLBW infants of all gestational ages.

- **CLABSIs (central line associated bacterial systemic infections) constitute most of the infections in the NICU**.

- The remaining cases involve the respiratory tract, eye, ear, nose, throat, GI tract, bone or joint infections or urinary tract or may be Skin and Soft Tissue Infections.

Bacterial Pathogens in Early-Onset Infections

Group B Streptococcal Sepsis in Neonates

- There are nine antigenically distinct GBS serotypes, based on their capsular polysaccharide analysis.

- Most infections in newborns occur **within the first week** of life and are designated as *early-onset disease*.

- **Treatment of GBS-colonized women during pregnancy only temporarily eradicates the organism**, and most women are recolonized within several weeks so the antibiotics protects from early-onset sepsis but not late-onset.

Bacterial Pathogens in Early-Onset Infections

Group B Streptococcal Infections

- Approximately **20%–35% of pregnant women are asymptomatic carriers of GBS** in the **genital tract and gastrointestinal tract** during pregnancy and at the time of delivery.

- A small number of GBS-infected infants acquired their bacteremia because of **hematogenous** transmission through the placenta causing **signs and symptoms of chorioamnionitis**.

- As recommended in the CDC, the optimal time for **performing prenatal cultures is between 35 and 37 weeks' gestation (36 through 37 weeks; CDC)**, and culture yield is obtained from **the lower vaginal area and anal or rectal sites.**

TABLE 3. Indications and nonindications for intrapartum antibiotic prophylaxis to prevent early-onset group B streptococcal (GBS) disease

Intrapartum GBS prophylaxis indicated	Intrapartum GBS prophylaxis not indicated
• Previous infant with invasive GBS disease	• Colonization with GBS during a previous pregnancy (unless an indication for GBS prophylaxis is present for current pregnancy)
• GBS bacteriuria during any trimester of the current pregnancy*	• GBS bacteriuria during previous pregnancy (unless an indication for GBS prophylaxis is present for current pregnancy)
• Positive GBS vaginal-rectal screening culture in late gestation† during current pregnancy*	• Negative vaginal and rectal GBS screening culture in late gestation† during the current pregnancy, regardless of intrapartum risk factors
• Unknown GBS status at the onset of labor (culture not done, incomplete, or results unknown) and any of the following: -- Delivery at <37 weeks' gestation§ -- Amniotic membrane rupture ≥18 hours -- Intrapartum temperature ≥100.4°F (≥38.0°C)¶ -- Intrapartum NAAT** positive for GBS	• Cesarean delivery performed before onset of labor on a woman with intact amniotic membranes, regardless of GBS colonization status or gestational age

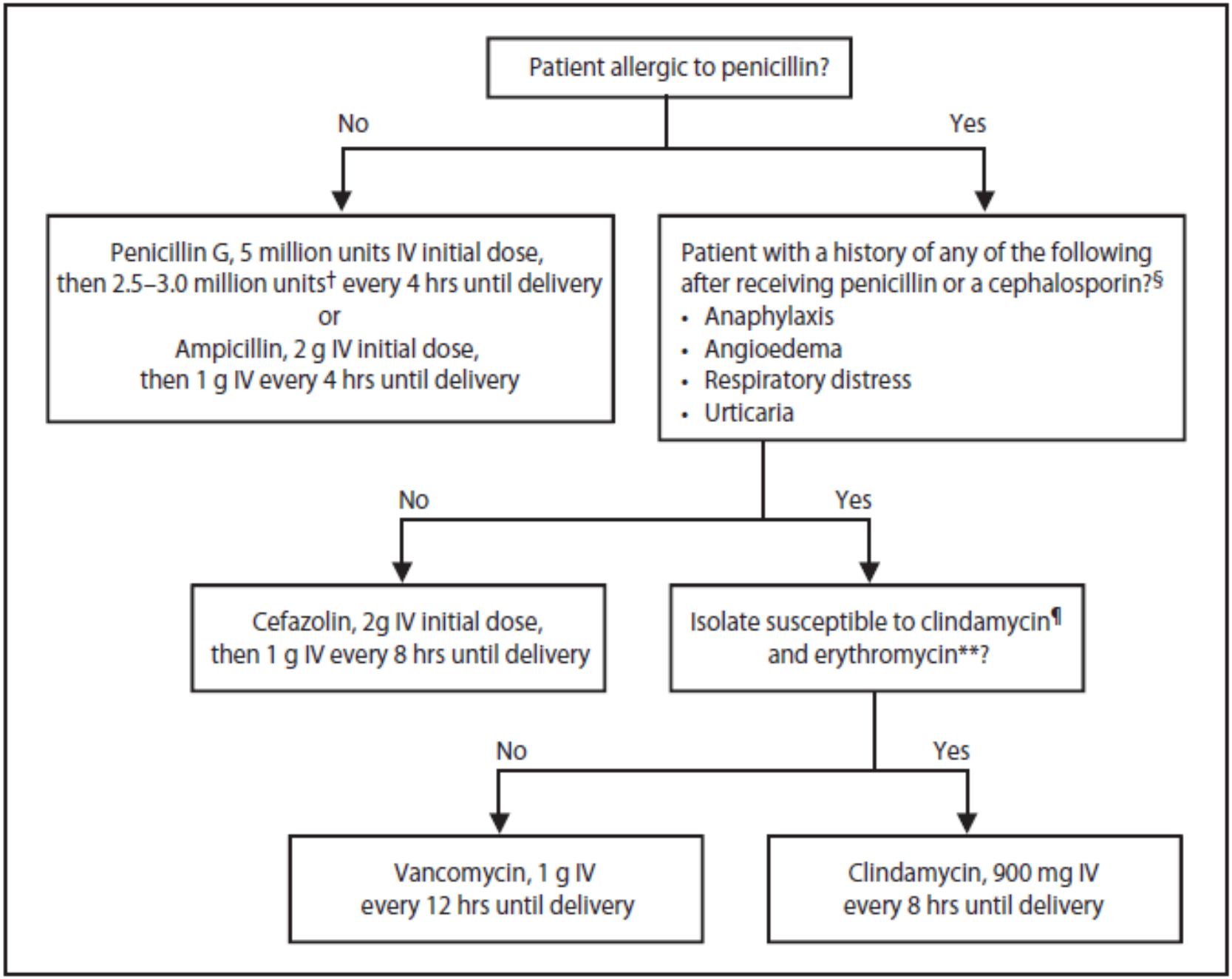
Abbreviation: NAAT = Nucleic acid amplification tests

* Intrapartum antibiotic prophylaxis is not indicated in this circumstance if a cesarean delivery is performed before onset of labor on a woman with intact amniotic membranes.

† Optimal timing for prenatal GBS screening is at 35--37 weeks' gestation.

§ Recommendations for the use of intrapartum antibiotics for prevention of early-onset GBS disease in the setting of threatened preterm delivery are presented in Figures 5 and 6.

¶ If amnionitis is suspected, broad-spectrum antibiotic therapy that includes an agent known to be active against GBS should replace GBS prophylaxis.


Patient allergic to penicillin?
No
Yes
Penicillin G, 5 million units IV initial dose, then 2.5–3.0 million units† every 4 hrs until delivery
or
Ampicillin, 2 g IV initial dose, then 1 g IV every 4 hrs until delivery
Patient with a history of any of the following after receiving penicillin or a cephalosporin?§
• Anaphylaxis
• Angioedema
• Respiratory distress
• Urticaria
No
Yes
Cefazolin, 2g IV initial dose, then 1 g IV every 8 hrs until delivery
Isolate susceptible to clindamycin¶ and erythromycin**?
No
Yes
Vancomycin, 1 g IV every 12 hrs until delivery
Clindamycin, 900 mg IV every 8 hrs until delivery

Bacterial Pathogens in Early-Onset Infections

Escherichia Coli Infections

- *E. coli* has been **the second most common** pathogen causing sepsis, bacteremia and meningitis in newborns.

- *Acquired through the genital tract.*

- Among preterm infants, however, the incidence of *E. coli* infections increased significantly.

Bacterial Pathogens in Early-Onset Infections

Listeria Monocytogenes Infections

- *L. monocytogenes* is a small, facultative anaerobic, gram-positive motile bacillus can be confused with GBS unless a careful Gram stain, a catalase reaction, and other tests are performed.

- Transmission to the fetus occurs through either a **hematogenous (transplacental)** route or via an **ascending infection through the birth canal**.

- Frequently, infections with *Listeria* spp. **early in gestation result in abortion**; **later in pregnancy**, infection with *Listeria* spp. can result in **premature delivery of a stillborn or infected newborn**.

- *Listeria* spp. infection may have either an **early-onset or a late-onset** presentation.

Bacterial Pathogens in Early-Onset Infections

Other Bacterial Pathogens

- *Enterococcus* spp.,
- viridans group *Streptococcus* spp.,
- *Klebsiella* spp.,
- *Enterobacter* spp.,
- *Haemophilus influenzae* (typeable and nontypeable),
- *S. aureus,*
- *Streptococcus pneumoniae*,
- group A streptococcus and other beta-hemolytic streptococci,
- coagulase-negative staphylococci.

Bacterial pathogens in Late-onset sepsis

TABLE 40.3 Pathogens Associated With Late-Onset Sepsis (2002–2008)

Organism	Singletons	Multiples
Gram-positive bacteria[a]	2916 (76.8%)	905 (75.7%)
• *Staphylococcus*, coagulase negative[a]	2020 (53.2%)	588 (49.2%)
• *Staphylococcus aureus*	408 (10.7%)	137 (11.5%)
• Group B streptococcus	69 (1.8%)	25 (2.1%)
• Other streptococci[a]	138 (3.6%)	68 (5.7%)
• Other gram-positive bacteria	281 (7.4%)	87 (7.3%)
Gram-negative bacteria[a]	597 (15.7%)	222 (18.6%)
• *Escherichia coli*	171 (4.5%)	61 (5.1%)
• *Klebsiella* species	151 (4.0%)	63 (5.3%)
• *Enterobacter* species	102 (2.7%)	42 (3.5%)
• *Pseudomonas* species	85 (2.2%)	27 (2.3%)
• *Serratia* species	44 (1.2%)	13 (1.1%)
• Other gram-negative bacteria	44 (1.2%)	16 (1.3%)
Fungi[a]	284 (7.5%)	69 (5.8%)
• *Candida albicans*[a]	172 (4.5%)	35 (2.9%)
• *Candida parapsilosis*	73 (1.9%)	23 (1.9%)
• Other fungi	39 (1.0%)	11 (0.9%)

Bacterial pathogens in Late-onset sepsis

Coagulase-Negative Staphylococci

- **CoNS** (such as *S. epidermidis, S. capitis, S. hominis, S. warneri*, and *S. haemolyticus*), while commonly thought of as skin commensals, are **the most common endemic nosocomial pathogen**.

- Most CoNS infections are **bloodstream infections**.

- CoNS are **lower-virulence pathogens**, with low mortality rates noted.

Bacterial pathogens in Late-onset sepsis

❖*Staphylococcus aureus*

- *S. aureus* has caused **epidemics of Skin Soft Tissue Infections in NICUs** and causes 10%.
- The skin, nares, and umbilicus are the most common sites of colonization.
- **MSSA vs MRSA**

❖*Enterococcus*

- Enterococci (*Enterococcus faecalis, Enterococcus faecium*) are responsible for both **endemic and epidemic late-onset sepsis in the NICU**.
- The **GI tract** is often the primary source of infection; also spread via **the hands of healthcare workers** or through **environmental contamination**.
- The widespread use of antibiotics has led to the emergence of **VRE (vancomycin Resistant Enterococcus)**.

❖Group B Streptococcus

- Group B streptococcus remains an important cause of early-onset and late-onset infection in neonates.

Bacterial pathogens in Late-onset sepsis

Gram-Negative Bacteria

- Gram-negative organisms are a particularly important cause of nosocomial bloodstream infections, pneumonia, and meningitis because they **generally cause severe disease**.
- ***Escherichia coli*** **is the most common gram-negative pathogen**.
- Other gram-negative organisms responsible for HAI include *Klebsiella, Pseudomonas, Enterobacter, Acinetobacter, Serratia, Haemophilus, Citrobacter*, and *Salmonella* spp.
- The **GI tract** is thought to serve as the reservoir for these bacteria, and **prolonged antibiotic therapy** may promote selection of these bacteria.
- The attributable **mortality is much higher for gram-negative infections than for gram-positive infections**.

Bacterial pathogens in Late-onset sepsis

Fungi

Risk factors for fungal infections:

1. The smallest and most premature infants appear to be at the highest risk, particularly when they are exposed to broad-spectrum antibiotics and long courses of antibiotics.
2. prolonged mechanical ventilation,
3. prolonged use of CVCs,
4. use of lipid emulsions,
5. antenatal antibiotics,
6. the use of histamine H2-receptor antagonists.

Bacterial pathogens in Late-onset sepsis

Chlamydia

- Associated with *preterm deliveries*
- Neonatal *chlamydial conjunctivitis*
  - 1st few days to several weeks after birth
  - Not prevented by routine eye prophylaxis
- Pneumonitis occurs between 2-19 weeks after birth
- Infants born to untreated mothers should be treated with oral erythromycin for 14 days
- Neonatal chlamydial conjunctivitis: Oral erythromycin no Topical therapy.

Bacterial pathogens in Late-onset sepsis

Gonorrhea (GC)

- Associated with Preterm deliveries
- All pregnant women should have routine cervical cultures for GC
- ALL infants should receive routine eye prophylaxis regardless of maternal history: 1% tetracycline, 0.5% erythromycin.
- Infants born to mothers with gonorrhea: Routine eye prophylaxis + Single dose of ceftriaxone or cefotaxime.
- Ophthalmia Neonatorum: leading cause of *acquired blindness treated with* Crystalline penicillin G for 7-10 days.
- Arthritis or septicemia: Ceftriaxone or Cefotaxime for 7 days.
- Meningitis: Ceftriaxone or cefotaxime for 10-14 days.

Clinical Signs of Bacterial Sepsis

- Most neonates exhibit respiratory distress in the **first 12 hours** of life, frequently immediately after birth. In these neonates the **progression may be rapid, with cardiovascular instability, shock, and death**.

- **Early Presentation** suggests that the infection with pneumonia and bacteremia occurred **at or near the time of birth or during the immediate postnatal period**.

- Because the signs of sepsis can be relatively nonspecific, such as **poor feeding and increased sleepiness**, they can be overlooked.

Clinical Signs of Bacterial Sepsis

Table 109-5	Initial Signs and Symptoms of Infection in Newborn Infants

GENERAL
Fever, temperature instability
"Not doing well"
Poor feeding
Edema

GASTROINTESTINAL SYSTEM
Abdominal distention
Vomiting
Diarrhea
Hepatomegaly

RESPIRATORY SYSTEM
Apnea, dyspnea
Tachypnea, retractions
Flaring, grunting
Cyanosis

RENAL SYSTEM
Oliguria

CARDIOVASCULAR SYSTEM
Pallor; mottling; cold, clammy skin
Tachycardia
Hypotension
Bradycardia

CENTRAL NERVOUS SYSTEM
Irritability, lethargy
Tremors, seizures
Hyporeflexia, hypotonia
Abnormal Moro reflex
Irregular respirations
Full fontanel
High-pitched cry

HEMATOLOGIC SYSTEM
Jaundice
Splenomegaly
Pallor
Petechiae, purpura
Bleeding

Clinical Signs of Bacterial Sepsis

- Presenting sites of Early-onset sepsis:
  - Occult bacteraemia
  - **pneumonia**
  - and *occasionally meningitis or UTI*

- Presenting sites of Late-onset sepsis:
  - Occult bacteraemia
  - **Meningitis**
  - **UTI**
  - Cellulitis
  - osteomyelitis
  - septic arthritis

Diagnostic testing

1. Blood culture
2. Lumber pumcture
3. Urine analysis and culture
4. CBC and differential
5. I:T ratio, absolute neutrophil count
6. Platelet count
7. CRP and procalcitonine (PCT); Cytokines IL-1ß, IL-6, IL-8,IL-10 and TNF
8. CXR
9. Swab culture of skin, eye, secretions or sputum cultures.
10. bilirubin, glucose, electrolytes and KFT provide supportive evidence for sepsis.
11. Placental cultures for evidence of chorioamnionitis.
12. Coagulation profile for septic shock and DIC.

Laboratory Testing

Blood Cultures

- The **gold standard for detection of bacteremia in newborns** with suspected sepsis is a positive blood culture result.
- Most blood culture results are **detected within 24–48 hours** with use of the new technology.
- The use of **intrapartum antibiotic prophylaxis in mothers can reduce the ability to detect bacteremia** in newborns.
- The decision to discontinue treatment with antibiotics should include **the assessment of the infant's clinical condition and should not rely solely on a negative blood culture result**.

Laboratory Testing

Urine Cultures

- Infants with **late-onset sepsis tend to have a higher rate of positive urine culture results**.

- **In the first 72 hours of life**, because the yield from urine cultures is low, it is **not generally recommended to obtain urine specimens**.

- In the newborn **older than 72 hours**, a **urine sample collected by an aseptic technique** (urinary catheter or suprapubic bladder aspiration) is an essential part of the sepsis work-up.

Laboratory Testing

Cerebrospinal Fluid

- The gold standard for diagnosis of meningitis is the analysis of the **CSF, including the WBC count, glucose and protein levels, Gram stain, and culture and latex test.**

- Although an **increase is expected in the number of neutrophils** with bacterial meningitis, one **may see a predominance of lymphocytes within a conversion to PMNs**.

- It is especially important to **repeat the CSF examination before antibiotic therapy is stopped in:**

1. **patients with complicated courses,**
2. **patient has not responded clinically,**
3. **is experiencing seizures or continued fever**
4. **for enteric gram negative bacterial meningitis**.

- **Lumbar punctures are deferred in infants with any instability or uncorrected bleeding disorders.**

Normal CSF analysis

	WBCs (/microL)	Protein (mg/dL)	Glucose (mg/dL)	RBCs
Normal in Children	0 - 5 (Lymphocytes)	20 – 40	50 – 100 (1/2 - 2/3) of serum glucose	0
Normal in Neonates	0-28 (60% Lymphocytes)	15-135	50 – 100 (1/2 - 2/3) of serum glucose	0

Laboratory Testing

White Blood Cell Count and Neutrophil Indices

- Normal white blood cell (**WBC**) counts range from **9000–32,000 cells** per microliter at the time of birth.
- The **absolute neutrophil count (ANC)**, the **absolute band count of immature neutrophils**, and **the ratio of immature neutrophils to total neutrophils (I/T)** are **more useful than total leukocyte counts in the diagnosis of neonatal sepsis**.
- **The optimal time to obtain WBC counts is after 4 hours of age**, and most recommendations are to obtain the first counts at 6–12 hours of age.
- **Neutropenia is the best predictor of sepsis**, whereas neutrophilia does not correlate well.
- The **I/T ratio is considered to have the best sensitivity of all of the neutrophil indices (normal value < 20%).**

Laboratory Testing

Platelet Counts

- Approximately 25%–30% of infants exhibit **thrombocytopenia** at the time of diagnosis of sepsis and usually it is a **late sign of sepsis**.

- It occurs as Accelerated platelet destruction and possibly depressed production caused by bacterial products on the bone marrow.

- **Disseminated intravascular coagulation** may be seen in some infants with severe sepsis.

Laboratory Testing

Acute-Phase Reactants

- C-reactive protein (**CRP**) is **produced by the liver and** induced by proinflammatory cytokines (**cytokine IL-6)**, and **its level rises to a maximum at 12–24 hours**.

- **Monitoring CRP levels** has been widely used **to diagnose neonatal infection and to adjust the duration of antibiotic therapy** in infants with suspected versus proven sepsis.

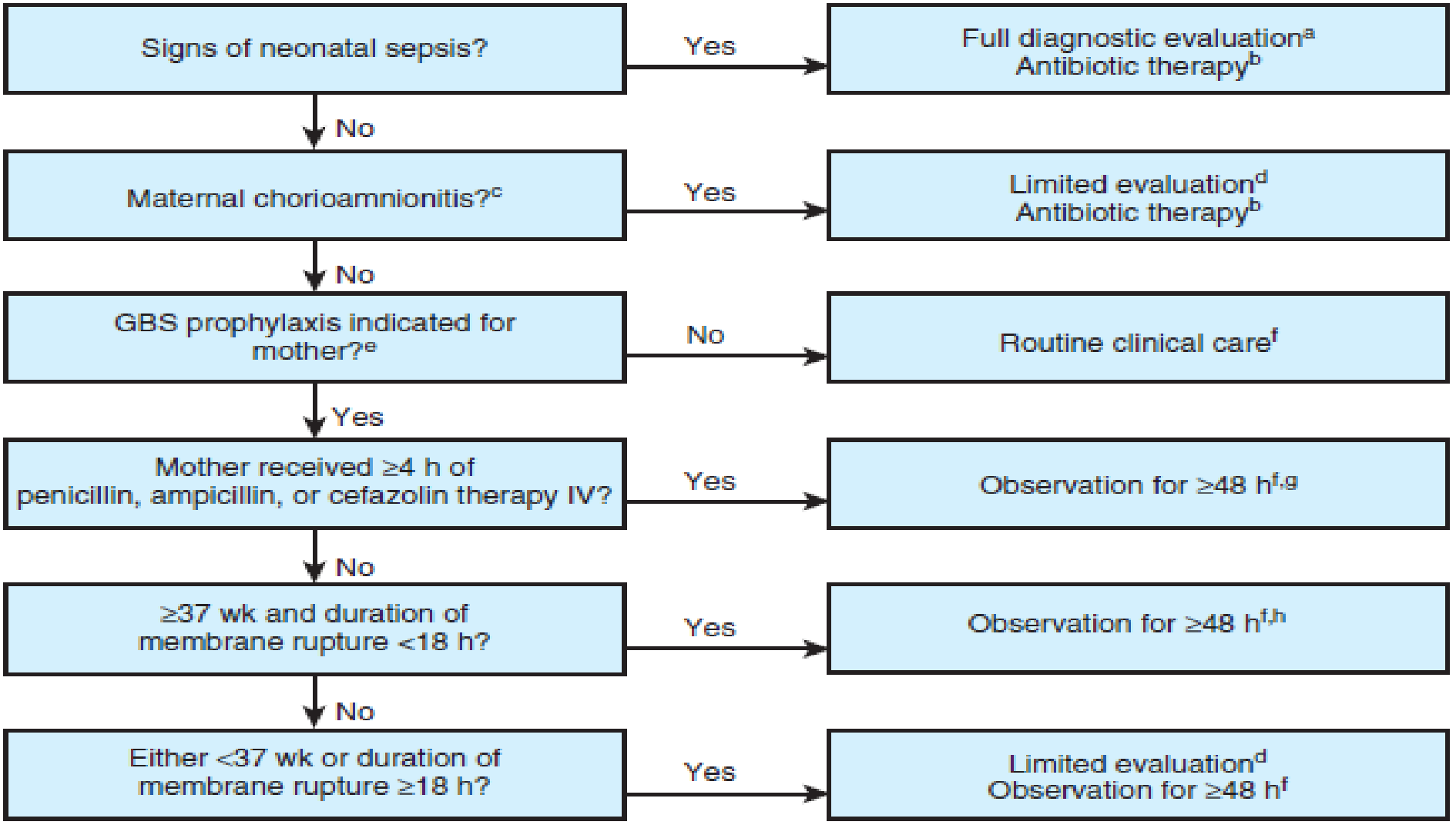
- **Depending on the laboratory**, **a CRP value of 5 mg/dL is considered the upper limit of normal.**

Laboratory Testing

Acute-Phase Reactants

- PCT is produced by cells such as **hepatocytes, nephrons, and monocytes**.
- **Procalcitonin (PCT**) concentration **rises at 4 hours**, peaks at 6 hours, and plateaus 8–24 hours after a stimulus.
- Most srudies have concluded that **PCT levels are superior to CRP levels in the early diagnosis of neonatal sepsis.**
- **PCT concentrations rise much faster than CRP concentrations**.
- **In healthy newborns, plasma PCT concentrations increase gradually after birth**, decrease to normal values by 48–72 hours of age.

Diagnostic Approach to Neonates With Suspected Sepsis



Diagnostic Approach to Neonates With Suspected Sepsis

- Obviously, **all symptomatic newborns must be carefully evaluated** for the possibility of bacterial sepsis **and treated with antibiotics.**

- Although the **presence of various risk factors should increase the suspicion of sepsis**, **the absence of risk factors in the symptomatic infant does not indicate that sepsis risk can be dismissed**.

Diagnostic Approach to Neonates With Suspected Sepsis

- In any newborn who is **symptomatic other than early tachypnea or is still symptomatic 6 hours after birth**, a **diagnostic evaluation with a complete blood cell count and differential, a blood culture, and, as appropriate, a lumbar puncture and a chest radiograph** should be strongly considered.

- **Antibiotic therapy can be stopped** when

1. the **physical findings are normal**,
2. the **clinical suspicion of sepsis is low**,
3. the **screening results for sepsis**, including the blood culture results, remain negative.

Diagnostic Approach to Neonates With Suspected Sepsis

- Recommendations replacing the diagnosis of **chorioamnionitis** with a classification of "intrauterine infection or inflammation or both," which it calls "**Triple I**." dividing maternal fever into three categories:

1. Isolated fever.

2. **Suspected Triple I** is defined as **fever without a source** combined with
    1. baseline fetal tachycardia,
    2. maternal WBC count greater than 15,000,
    3. and/or purulent fluid from the cervical os.

3. **Confirmed Triple I** requires symptoms compatible with **suspected Triple I plus biologic or microbiologic amniotic fluid results consistent with microbial invasion of the amniotic cavity**.

Treatment of early-onset sepsis

Antimicrobial Therapy

- The **choice of antibiotic** for an infant with suspected early-onset sepsis depends on the predominant **bacterial pathogens and the antibiotic susceptibility** profiles.

- If sepsis is highly suspected in an infant, antibiotics should be considered for a full course even if the culture results are negative.

- **Empiric therapy for early-onset sepsis** generally consists of **combinations of antibiotics** effective against gram-positive pathogens (e.g., GBS, *L. monocytogenes*) and gram-negative pathogens (e.g., *E. coli*).

- The two most commonly used combinations are
  1. **ampicillin with an aminoglycoside**, usually gentamicin,
  2. **ampicillin with a third-generation cephalosporin**, usually cefotaxime.

Treatment of early-onset sepsis

Antimicrobial Therapy

- ***Ceftriaxone is contraindicated in neonatal period.***

- In infants with **GBS sepsis**, **gentamicin is frequently combined with ampicillin** or penicillin as synergetic effect during the first few days of therapy and then to continue the full course of therapy with ampicillin or penicillin alone.

- In most hospitals, **48 hours is sufficient to determine whether a blood culture result** is negative, assuming that no antibiotics were being given when the culture was obtained.

- Infants with **proven bacteremia, but without meningitis**, are commonly treated for **7–10 days**.

- The use of **antibiotics with nephrotoxicity** (i.e., aminoglycosides) should be monitored with the use of appropriate **drug levels**.

Treatment of Late-onset sepsis

- So we give emperically for **nosocomial late onset infections** **Vancomycine** (to cover the resistant strains of the gram positive bacteria) **+ Aminoglycoside** (to cover the gram negative bacteria) **or Carbapenem** (for resistant gram negative bacteria).

- For **outpatient babies with late onset sepsis** we give **Ampicillin and Aminoglycoside or cefotaxim** because they don't have high risk of resistant bacteria.

- Antibiotic use should be discontinued if infection is not proved and is not likely after 48 hrs.

Complications

1. Respiratory: need for oxygen support.
2. Cardiovascular: need for fluids and inotrops.
3. Hematological: DIC.
4. CNS: seizures and SIADH.
5. Metabolic: hypo and hyperglycemia, hyponatremia and metabolic acidosis.

Prevention

- Orally administered **lactoferrin and Probiotic** **for prevention of sepsis and necrotizing enterocolitis** suggests a decrease in the development of late-onset sepsis.

- **Intravenous Immune Globulin and** G-CSF **for Prevention of Early-Onset Sepsis: not suggested**

Prevention of Health Care–Associated Infection

• BOX 40.2 Principles for the Prevention of Health Care–Acquired Infection in the Neonatal Intensive Care Unit

Observe recommendations for standard precautions with all patient contact.
Observe recommendations for transmission-based precautions (gowns, gloves, masks, isolation, as indicated).
Use good nursery design and engineering.
Appropriate nurse-to-patient ratio
Avoidance of overcrowding and excessive workload
Improve hand hygiene compliance (Box 40.3).
Minimize risk of contamination of central lines—adopt care bundles.
Provide meticulous skin care.
Encourage early and appropriate advancement of enteral feedings.
Perform continuous monitoring and surveillance of health care–acquired infection rates in the neonatal intensive care unit.
Provide education and feedback to nursery personnel.