In the 1970s, Group B Streptococcus (GBS) replaced *Escherichia coli* as the leading infectious cause of early neonatal morbidity and mortality in the United States (1–4). Prior to preventive screening and treatment, an estimated 7,500 cases of neonatal GBS disease occurred annually in the United States (5). A great reduction in disease occurrence resulted from an increase in preventive measures such as recommendations for intrapartum prophylaxis in order to prevent perinatal GBS disease issued in 1996 by the American College of Obstetricians and Gynecologists (ACOG), Centers for Disease Control and Prevention (CDC), and in 1997 by the American Academy of Pediatrics (AAP) (6-9). Disease occurrence declined even further after the publication of CDC guidelines for prevention in 2002 which recommended a universal culture-based screening for all pregnant women at 35–37 weeks’ gestation to optimize the identification of women who should receive intrapartum antibiotic prophylaxis (10, 11). Despite recommendations for prevention, GBS remains the leading infectious cause of morbidity and mortality among newborns in the United States (12, 13). GBS has been recognized as one of the most important pathogens in obstetric patients and can cause: urinary tract infections, amnionitis, post-partum endometritis, wound infection, as well as intrapartum and postpartum bacteremia (14, 15). GBS infection may also lead to premature rupture of membranes (PROM) and preterm delivery (16-18).

**Epidemiology**

- Group B Streptococcus, or *Streptococcus agalactiae*, is a Gram-positive bacterium that causes invasive disease primarily in infants, pregnant or postpartum women and older adults, with the highest incidence among young infants (12, 19–25).

- Asymptomatic vaginal colonization with GBS occurs in approximately 20% (range 4.6% to 40.6%) of pregnant women (26, 27-30).

- The CDC estimates that in recent years, GBS has caused approximately 1,200 cases of early-onset invasive disease per year (31); approximately 70% of cases are among babies born at term (≥37 weeks’ gestation) (12).

**Pathogenesis**

- Early-onset infections are acquired vertically through exposure to GBS from the vagina of a colonized woman. Neonatal infection occurs primarily when GBS ascends from the vagina to the amniotic fluid after onset of labor or rupture of membranes, although GBS also can invade through intact membranes (32, 33).

- Pregnant women with GBS colonization were >25 times more likely than pregnant women with negative prenatal cultures to deliver infants with early-onset GBS disease (34).

- Approximately 10% to 30% of pregnant women are colonized with GBS in the vagina or rectum (35–37). GBS colonization during pregnancy can be transient, intermittent, or persistent (38–40).

- Although some women with GBS colonization during a pregnancy will be colonized during subsequent pregnancies, a substantial proportion will not (41, 42).

- In the absence of any intervention, an estimated 1% to 2% of infants born to colonized mothers develop early-onset GBS infections (7, 34).

- In addition to maternal colonization with GBS, other factors that increase the risk for early-onset disease include: gestational age <37 completed weeks, longer duration of membrane rupture, intra-amniotic infection, young maternal age, black race, and low maternal levels of GBS-specific antcapsular antibody (43–50).

**Clinical Significance**

- Two distinct clinical syndromes related to age were described as acute and delayed or early and late-onset (51,52).

- Infants with early-onset GBS disease generally present with respiratory distress, apnea, or other signs of sepsis within the first 24–48 hours of life (3,53,54).
Women who do not need screening at 35–37 weeks’ gestation:

- GBS isolated from the urine at any time during the current pregnancy
- Previous infant with invasive GBS disease
- Women with symptomatic or asymptomatic GBS urinary tract infection detected during pregnancy

Women who do need to be screened at 35–37 weeks’ gestation:

- All other pregnant women should be screened at 35–37 weeks’ gestation for vaginal and rectal GBS colonization

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Table 1. Summary of Group B Streptococcus (GBS) Screening Guidelines (64)

<table>
<thead>
<tr>
<th>Women who do not need screening at 35–37 weeks’ gestation:</th>
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Table 2. Indications and nonindications for intrapartum antibiotic prophylaxis to prevent early-onset Group B Streptococcal (GBS) disease (64).

### Intrapartum GBS prophylaxis indicated:

- Previous infant with invasive GBS disease
- GBS isolated from the urine at any time during the current pregnancy
- Positive GBS vaginal-rectal screening culture in late gestation during current pregnancy
- 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

### Intrapartum GBS prophylaxis not indicated:

- Colonization with GBS during a previous pregnancy (unless an indication for GBS prophylaxis is present for current pregnancy)
- GBS bacteriuria during previous pregnancy (unless an indication for GBS prophylaxis is present for current pregnancy)
- Negative vaginal and rectal GBS screening culture in late gestation during the current pregnancy, regardless of intrapartum risk factors

* 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.
** NAAT testing for GBS is optional and might not be available in all settings. If intrapartum NAAT is negative for GBS but any other intrapartum risk factor (delivery at <37 weeks’ gestation, amniotic membrane rupture at ≥18 hours, or temperature ≥100.4°F [≥38.0°C]) is present, then intrapartum antibiotic prophylaxis is indicated.
Abbreviation: IV = intravenously.

- Broader spectrum agents, including an agent active against GBS, might be necessary for treatment of chorioamnionitis.
- Doses ranging from 2.5 to 3.0 million units are acceptable for the doses administered every 4 hours following the initial dose. The choice of dose within that range should be guided by which formulations of penicillin G are readily available to reduce the need for pharmacies to specially prepare doses.
- Penicillin-allergic patients with a history of anaphylaxis, angioedema, respiratory distress, or urticaria following administration of penicillin or a cephalosporin are considered to be at high risk for anaphylaxis and should not receive penicillin, ampicillin, or cefazolin for GBS intrapartum prophylaxis. For penicillin-allergic patients who do not have a history of those reactions, cefazolin is the preferred agent because pharmacologic data suggest it achieves effective intraamniotic concentrations.
- Vancomycin and clindamycin should be reserved for penicillin-allergic women at high risk for anaphylaxis.
- If laboratory facilities are adequate, clindamycin and erythromycin susceptibility testing (Box 3) should be performed on prenatal GBS isolates from penicillin-allergic women at high risk for anaphylaxis. If no susceptibility testing is performed, or the results are not available at the time of labor, vancomycin is the preferred agent for GBS intrapartum prophylaxis for penicillin-allergic women at high risk for anaphylaxis. Resistance to erythromycin is often but not always associated with clindamycin resistance. If an isolate is resistant to erythromycin, it might have inducible resistance to clindamycin, even if it appears susceptible to clindamycin. If a GBS isolate is susceptible to clindamycin, resistant to erythromycin, and testing for inducible clindamycin resistance has been performed and is negative (no inducible resistance), then clindamycin can be used for GBS intrapartum prophylaxis instead of vancomycin.

### Figure 1. Recommended regimens for intrapartum antibiotic prophylaxis for prevention of early-onset Group B Streptococcus (GBS) disease* (64).

<table>
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<th>Patient allergic to penicillin?</th>
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<tr>
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<td>- Urticaria</td>
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<tr>
<td>No</td>
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<tr>
<td>Cefazolin, 2 g IV initial dose, then 1 g IV every 8 hours until delivery</td>
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<tr>
<td>Yes</td>
</tr>
<tr>
<td>Isolate susceptible to clindamycin and erythromycin?</td>
</tr>
<tr>
<td>No</td>
</tr>
<tr>
<td>Vancomycin, 1 g IV every 12 hours until delivery</td>
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<tr>
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References:

7. CDC. 1996. MMWR 45(No. RR-7).
64. CDC. 2010. MMWR Recomm Rep 59(RR-10):1-36.