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Prevention of Neonatal Sepsis

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Traditionally, neonatal sepsis has been a focus of concern for pediatric caregivers; however, obstetric practitioners can have a tremendous impact on reducing the burden of this serious disease through interventions that are applied during pregnancy or childbirth. In the last decade, the use of intrapartum antimicrobial prophylaxis has increased in the United States, primarily for group B streptococcal (GBS) disease prevention, and to a lesser extent for preterm premature rupture of membranes (pPROM). During the 1990s, the incidence of laboratory-confirmed GBS infections in newborns declined dramatically. Although neonatal sepsis is a leading cause of neonatal mortality around the world, and the neonatal period contributes disproportionately to child mortality in resource-poor countries [1], this article focuses on prevention of neonatal sepsis in industrialized countries. The scope of the article also is restricted to infections that develop in the first days of life, because perinatal interventions may have the most impact on these infections.

Burden of neonatal sepsis

Severe bacterial infections (ie, sepsis or meningitis) during the neonatal period typically are divided into early- and late-onset syndromes. Early-onset sepsis is acquired through vertical transmission by ascending spread from the lower genital tract, through transplacental transmission after maternal bacteremia (eg, *Listeria monocytogenes*), or through neonatal acquisition during passage through the birth canal. Early-onset sepsis becomes clinically evident within the first few

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days of life, and has been defined in most reports as occurring within the first week or the first 72 hours of life. Late-onset sepsis presents thereafter, with an outer limit of 28, 30, or 90 days in various reports. Late-onset infections may be acquired intrapartum during passage through the birth canal, through horizontal spread within hospital settings, or from maternal or other sources in the home or community. Prevention strategies have not been identified to reduce late-onset sepsis.

The pathogens that are identified most frequently from early-onset sepsis have varied over the past 60 years and may differ from hospital to hospital or country to country [2,3]. The relative contribution of the leading etiologies of neonatal sepsis from four multicenter U.S. reports from the past decade is shown in Fig. 1 [4–7]. Since its emergence in the 1970s, GBS has ranked consistently as the leading cause of early-onset sepsis in U.S. surveillance populations. Before prevention efforts were adopted in the United States, rates of laboratory-confirmed early-onset GBS disease ranged from 1.4 [4] to 1.7 [8] per 1000 live births in the general population. The case fatality ratio was influenced strongly by gestational age. Rates in the very low birth weight (<1500 g) population in the early 1990s were 5.9 per 1000 births [2,7]. *Escherichia coli* and other gram negatives accounted for a large proportion of non-GBS cases. One multicenter study that was performed in the mid-1990s reported a case fatality ratio of 6.7% for cases of early-onset GBS cases compared with 22.3% for non-GBS cases [4]. Prematurity was linked closely to the higher case fatality ratio for non-GBS cases in that report.

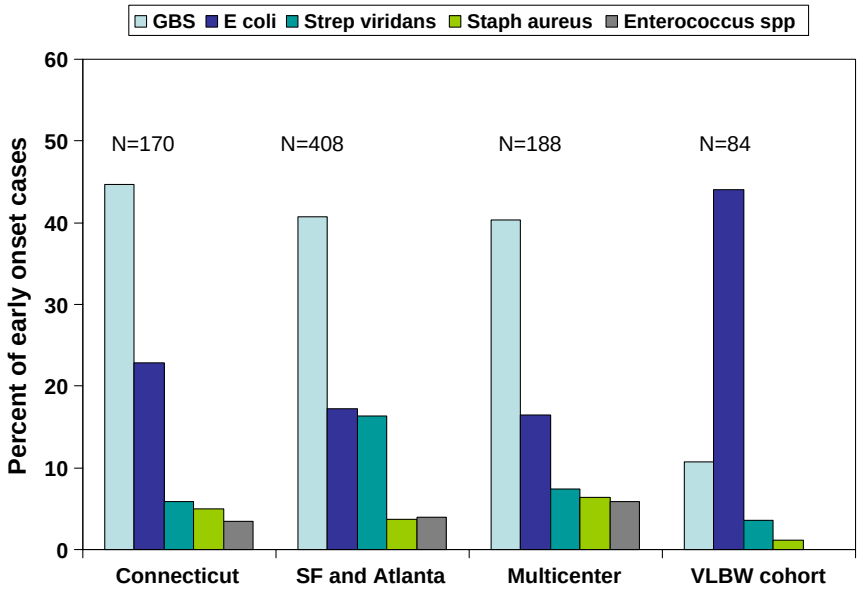

Fig. 1. Leading etiologies of neonatal sepsis from four U.S. reports. SF, San Francisco; Staph, *Staphylococcus*; Strep, *Streptococcus*; VLBW, very low birth weight. (*Data from* Refs. [1–4].)