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This Week's Citation Classic

Franciosi R A, Knostman J D & Zimmerman R A. Group B streptococcal neonatal and infant infections. *J. Pediatrics* 82:707-18, 1973.
[Streptococcal Dis. Sect., Ecol. Invest. Program, Ctr. for Dis. Control, Hlth. Serv. and Mental Hlth. Admin., Public Hlth. Serv., US Dept. HEW, Fort Collins, CO and Dept. Pathol., Children's Hosp., Minneapolis, MN]

Epidemiologic studies of group B streptococcal (GBS) infection revealed an incidence of three per 1,000 live births and a mortality of one per 1,000 live births. Vaginal cultures for GBS were positive in 4.6 percent of women at delivery, and 1.2 percent of their infants. Two distinct types of sepsis occurred; one type presented within hours of birth with respiratory distress and the second presented as meningitis in the later neonatal. [The *SCI*® indicates that this paper has been cited in over 305 publications since 1973.]

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"My experience with group B streptococcal (GBS) infection began in July 1969 when I joined the pathology staff at the Children's Hospital in Denver, Colorado. My curiosity was aroused quickly by observing in the first eight months of work five newborns who died of GBS infection. This experience caused me to review the problem of neonatal sepsis at Children's Hospital.[1] These five cases accounted for 45 percent of the neonatal septic mortality that year.

"Robert A. Zimmerman, chief of the Ecological Investigation Program of the Streptococcal Disease Section of the Center for Disease Control in Fort Collins, Colorado, directed the reference laboratory for streptococcal identification. He confirmed the isolates from our cases as group B streptococcus. When asked if his laboratory would support a study of this potential problem, he readily agreed. Zimmerman's work in the area of streptococcal research was well known and he was able to enlist the aid of Rebecca Lancefield. James D. Knostman was an internist with a special interest in renal disease assigned as a public health officer to Zimmerman. He was very interested in helping with the epidemiologic aspects of this study.

"Our study of GBS was designed to address these questions: What is the prevalence of GBS in maternal vagina at delivery and the incidence of neonatal infection? What is the prevalence of GBS in newborns at delivery and discharge from the hospital? What is the prevalence of GBS vaginally positive nonpregnant women? What is the prevalence of GBS in the male urethra? Is antepartum prevention possible? What are the clinical, laboratory, and pathologic findings in GBS infection?

"Three months were needed to recruit the necessary cooperation, i.e., four obstetrical units, one private obstetric and gynecology clinic, three neighborhood health centers, and one state penal institution. During the two and a half years of study, cultures were collected from over 1,200 deliveries, 350 nonpregnant women, 100 nursery staff, and 130 men; 43 clinical and autopsy records of GBS cases were abstracted; 500 placentas were examined; and 800 paired maternal and infant serums were collected.

"We were able to conclude that GBS sepsis was a significantly underestimated problem. Clinically there were two distinct presentations of GBS sepsis, one defined as acute onset occurring within 48 hours of birth and the other delayed onset usually occurring a week or more after birth. The pathogenesis of acute onset GBS infection was colonization of the newborn *in utero* or in transit through the birth canal of a vaginally colonized mother.

"In 1979, I reviewed the published cases of acute onset GBS infection.[2] It was clear that the morbidity and mortality could be reduced by earlier diagnosis and treatment. In 1980, I proposed a hypothesis to explain the rising incidence of acute onset GBS infection.[3]

"A recent review on GBS infection indicates that this is a worldwide problem.[4] With so many talented researchers working on this problem, the next decade should see a marked reduction in the 12,000-15,000 GBS cases per year in the US. I feel that our original paper illustrated the epidemiology and pathogenesis of GBS infection in newborns. For this reason, it is often quoted as a reference. In addition, the paper is referred to as a model for approaching the problem of neonatal bacterial infection."

1. **Franciosi R A, Zimmerman R A, Favara B E & Butterfield J.** Neonatal infection due to group B streptococcus. *Rocky Mt. Med. J.* 68:48-52, 1971.
2. **Franciosi R A.** Infant at risk for early onset group B streptococcal infection. *Minn. Med.* 62:801-4, 1979.
3. ----------. Hypothesis to explain the emergence of early onset group B streptococcal infection in newborns. *Minn. Med.* 63:267-9, 1980.
4. **Christensen K K, Christensen P, Hägerstrand I, Lindén V, Nordbring F & Svenningsen N.** The clinical significance of group B streptococci. *J. Perinatal Med.* 10:133-46, 1982.