Pelvic infection following cesarean section is the most common hospital-acquired infection facing the Obstetrician-Gynecologist today. The most important contributing factor is the dramatic rise in the cesarean section rate. Cesarean births in the United States have increased almost threefold, from 5.5% of all deliveries (195,000 operations) in 1970 to 15.2% (510,000) in 1978 [1]. Although the prevalence of infection following cesarean section is not uniform throughout the United States, maternal morbidity rates are generally five to ten times higher after cesarean birth than after vaginal delivery, occurring in 10% to 65% of cases [1-4]. (see Fig. 1).

The operation itself is always a contaminated procedure which results in conditions that favor the proliferation of bacteria and suppression of host defense mechanisms. These conditions include: 1) impairment of local vas-

![Fig. 1. Incidence of post-cesarean endomyometritis in various university hospitals (1971-1978) (Adapted from the Consensus Development Conference on Cesarean Childbirth NICHD-NIH, 1980).](image)
cular supply resulting from vascular trauma; 2) presence of foreign bodies (i.e., sutures, talc, lint, etc.); 3) destruction of tissue by surgical manipulation (necrosis of tissue at suture line); and 4) hematoma formation [5]. The two most common causes of infectious morbidity following both cesarean and vaginal delivery are endometritis and urinary tract infection, with both complications occurring more commonly after cesarean birth [1]. Since urinary tract infections are discussed elsewhere in this volume, the scope of this chapter will focus upon post-cesarean endomyometritis.

The fact that rates of infection following cesarean section vary widely suggests that a woman's risk of developing infection postoperatively is dependent upon a variety of predisposing factors. Among these are internal fetal heart rate monitoring, prolonged rupture of the amniotic membranes, and lower socio-economic status. Although a wide variety of other clinical considerations, including frequency of pelvic exams, low hematocrit, obesity, general anesthesia, experience of the surgeon, and parity have been considered as predisposing factors to post-cesarean section febrile morbidity [6–22], insufficient data is available to permit critical evaluation. In addition, since many of these 'factors' occur concurrently, their individual assessment, in most cases, has been inconclusive.

**INTERNAL FETAL HEART RATE MONITORING**

The impact of internal fetal heart rate monitoring upon infectious morbidity in patients undergoing cesarean section remains one of the most important questions in operative obstetrics. When controls were used to reduce population heterogeneity, Wiechetek found no difference between the febrile morbidity in patients who were monitored during labor versus those who were not monitored [12]. Similarly, Hagen found no difference in the role of febrile morbidity in monitored and unmonitored lower socio-economic class patients who underwent cesarean section [23]. However, he observed that the infection rate among monitored private patients after cesarean section was higher than that among nonmonitored private patients. Consequently, he raised the possibility that the infection rate among indigent patients may be sufficiently high so as to obfuscate comparative studies of gross morbidity when comparing private and nonprivate patient studies. In concurrence, Gibbs et al. found no enhancement of morbidity when internal fetal heart rate monitoring was employed in a population of military dependents [10].

However, contrasting information was reported from studies utilizing a large population of indigent urban patients. Gassner and Ledger noted an increase from 20% to 40% in the incidence of endometritis when internal fetal heart rate monitoring preceded cesarean section [7] but found no statistical