



INTRODUCTION AND AIM OF THE WORK

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Neonatal sepsis is a clinical syndrome characterized by systemic signs and symptoms and bacteremia during the first month of life (*Odio, 1995*). In spite of the use of the potent antibiotics and intensive supportive care, sepsis is still a major cause of morbidity and mortality during the neonatal period (*Adriaanse, 1996*).

Bacterial sepsis leads to many metabolic disturbances including disturbances in glucose which is the main source of energy for brain growth and metabolism, insulin hormone which plays a central role in glucose homeostasis and lactate which is an intermediary in carbohydrate metabolism (*Bailey et al., 1990*).

Yeung, (1970) first demonstrated hypoglycemia in term neonates with gram-negative infection. Again, *Yeung et al., (1973)* reported that in 8-day old term infected infants, a more rapid glucose disappearance rate has been reported, hyperinsulinemia was not present to explain the increased glucose disappearance. Almost a decade later, increased glucose disappearance rate again without hyperinsulinism was reported in infants with sepsis at a mean age of 21 days by *Leake and Fiser, (1981)*. The proposed mechanisms of sepsis associated hypoglycemia include increased metabolic rate, altered glucose production and increased insulin sensitivity (*Pildes, 1986*).

Lactic acidosis is frequently seen in critically ill - patients but has not been emphasised in neonatal sepsis. The causes of elevated lactic acid concentration include tissue hypoperfusion (i.e. hypoxic - anaerobic metabolism), liver dysfunction including impaired gluconeogenesis and sepsis (*Mizock and Falk, 1992*).

The aim of this study is to clarify the effects of sepsis on blood glucose, lactate and insulin in newborn infants with bacterial sepsis.