INTRODUCTION

Hypoxic- ischaemic encephalopathy **(HIE)** is one of the main preventable causes of morbidity and mortality in the full term newborn ***(Figueras, J. et al 1992).*** In the last twenty years, the incidence of perinatal asphyxia has decreased; however, it is responsible for 10-20% of all cases of cerebral palsy **(Patel & Edward, 1997.** ***Breat & Rumeau, 1996 and perlaman, 1997).*** Traditionally, assessment of perinatal asphyxia had relied on a combination of clinical observation, such as Apgar score, and measurement of systemic indices of tissue ischaemia, such as serum creatinine. There are weakness in such methods.

Clinical scoring systems generally reflect neurological state (encephalopathy) which may influenced by factors other than perinatal asphyxia, such as metabolic and chromosomal disorders. Objective measurement, such as serum creatinine are insufficiently sensitive to be generally useful. The first form of assessment lacks specificity in the context of perinatal asphyxia; the latter lacks sensitivity. ***(Mehta, 1991).***Perinatal asphyxia causing organ damage is an important neonatal problem, its study is challenging because it is difficult to measure.

Perinatal asphyxia occur when fetal and newborn gas exchange fails (Ploom,R.S.1992).blood and tissue pH and partial oxygen pressure (Po_2)fall, but the tissue oxygen consumption persists leading to a further decrease in Po_2 .anaerobic metabolism lead to the