

Introduction

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There are vast rural areas in the tropics where obstetric services are poorly developed or lacking women in these areas tend to be malnourished and during pregnancy continue to undertake hard manual labour (*Saugstad 1998*).

At parturition mothers have to rely on attendants who lack facilities for simple resuscitation of asphyxiated babies, the more this happens the more handicapped children we would have in these tropical areas (*Ringer, 1998*).

Asphyxia at term remains a significant cause of infant death and neurodevelopmental impairment, probably causing 20% of all cases of cerebral palsy (*Volpe, 1995*).

Perinatal asphyxia causes multiple alterations in the fetus and newborn due to failures in the gas exchange system. These include hypoxia, hypercapnia and low blood pH, as well as redistribution of the blood flow with preservation of perfusion in vital organs such as the brain, heart and adrenal glands (*Avery et al., 1999*).

Perinatal asphyxia also triggers rapid alterations in the concentration of several hormones, such as catecholamines and others (*Kaneoka et al., 1979*).

Thyroid hormones play an important role in synthesis of mitochondrial enzymes and structural elements, in addition, they have a role in thermogenesis, water and electrolyte transportation, and in the growth and development of the central nervous system (*Docter et al., 1993*).

Little is known about the effects of perinatal asphyxia on fetal and neonatal levels of thyroid hormones despite their importance.

Laboratory work up of asphyxia, is very important, and confirms the clinical diagnosis of asphyxia and its complications.

The present study is focusing on the evaluation of TSH & Thyroid hormones in asphyxiated newborns.