Summary

SUMMARY

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, but on ongoing asphyxia causes decrease in cerebral blood flow leading to hypoxic-ischemic-encephalopahty (HIE) resulting in CP *(Avery et al., 1999).*

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

Thyroid hormones play an important part in the 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.

Little is known about the effects of perinatal asphyxia on fetal and neonatal levels of thyroid hormones, despite their importance. This study is focusing on the effect of perinatal asphyxia on TSH and thyroid hormones our Aim is to compare serum concentrations of thyroid hormones –T4, T3 and Free T4 – and thyroid –stimulating hormone (TSH) found in the umbilical cord blood of term newborns

with and without asphyxia and those found in their arterial blood collected between 18 and 24h after birth.

A case –control study was carried out. The case group comprised 20 term newborns (Apgar score ≤ 3 and ≤ 5 at the first and fifth minutes umbilical cord blood pH ≤ 7.15) who required bag and mask ventilation for at least one minute immediately after birth. The control group consisted of 20 normal, term newborns (Apgar score ≥ 8 and ≥ 9 at the first and fifth minutes; umbilical cord blood pH ≥ 7.2). Cord blood and arterial blood samples were collected immediately after birth a1nd 18 to 24h after birth, respectively, and were used in the blood gas analysis and to determine serum concentrations of T4, T3, FT4and TSH by radioimmunoassay.

Gestational age, birth weight, sex, mode of delivery were similar for both groups. No difference were found in mean levels of cord blood TSH, T4 , T3 and FT4 between the groups. In the samples collected 18 to 24h after birth, mean levels of TSH, T4, T3 and FT4 were significantly lower in the asphyxiated group than in the control group. Mean concentrations of arterial TSH, T4 and T3 between 18 and 24h of life were lower than concentration found in the cord blood analysis in asphyxiated newborns, but not in controls.

We concluded that birth asphyxia causes central hypothyroidism decrease in thyroid stimulating hormone resulting in decreased serum levels of thyroid hormones T4, T3 and FT4. This suggests central hypothyroidism secondary to asphyxia.

We recommend improvement of antenatal care, mothers have to attend medical centers for labour having simple facilities for simple resuscitation of asphyxiated babies.

We also recommend assessment of thyroid functions in all asphyxiated neonates.