INTRODUCTION
Foetus is totally dependent for its oxygen supply on the blood supplied through placenta. In any case if blood supply through placenta is hampered it leads to asphyxial injury. The incidence of perinatal asphyxia is about 1.0 to 1.5 percent in most centres and is usually related to gestational age and birth weight. It occurs in 9.0 percent of infants less than 36 weeks of gestation and 0.5 percent of infants of more than 36 weeks gestation, accounting for 20 percent of perinatal deaths or as high as 50 percent of deaths if still borns are included. The incidence is higher in term infants of diabetic or toxemic mothers. These factors correlate less well in preterm infants.

Ninety percent of asphyxial insult occurs in antepartum/intrapartum period as a result of placental insufficiency, resulting in an inability to provide $O_2$ and to remove $CO_2$ and $H^+$ from foetus.

During normal labour there is reduced blood flow to placenta, hence decreased $O_2$ delivery to the foetus. Because there is concomitant increase in $O_2$ consumption by both mother and foetus, foetal $O_2$ saturation falls. Maternal dehydration and maternal alkalosis from hyper-ventilation may further decrease maternal and foetal $O_2$ saturation. Some degree of cord compression also occurs
in many deliveries. Uterine contractions decrease
placental blood flow. These events cause most babies
to be born with little \( O_2 \) reserve.

In addition to the normal factors mentioned
above any process which (i) impairs maternal oxygenation.

(2) Decreased blood flow from mother to placenta or from
placenta to foetus. (3) Impairs gas exchange across the
placenta or at the foetal tissue. (4) Increases foetal
\( O_2 \) requirement, will exacerbate perinatal asphyxia.

In the presence of hypoxic ischemic challenge
to the foetus, reflexes are initiated causing shunting
of blood to the brain, heart, and adrenals and away from
lungs, gut, liver, kidneys, spleen, bone, skeletal muscle
and skin (diving reflex).

In mild hypoxia there is decreased heart rate,
slight increase in blood pressure to maintain cerebral
perfusion, increased central venous pressure and little
change in cardiac output. As asphyxia progresses, with
severe hypoxia and acidosis, there is decreased heart rate,
decreased cardiac output and initially increased then fall
in blood pressure as oxidative phosphorylation falls and
energy reserves become depleted. During asphyxia
anaerobic metabolism produces lactic acid which because of
poor perfusion remains in local tissues systemic acidosis
may actually be mild until perfusion is restored and these
local acid stores are mobilized.
Apgar score was developed to identify quickly the new born in need of resuscitation. It should be noted that Apgar score is partially dependent on the age maturity of the newborn. Immature infants are more likely to be hypotonic to have cyanotic extremities and to have decreased responsiveness. Therefore, a score of 7 may be "Maximum" for a normal premature infant. Hypoxia in utero due to hypoperfusion, a fibrotic placenta, premature placental separation, or problems with the umbilical cord, may be responsible for low Apgar score. But it must be remembered that factors other than hypoxia in utero may affect the Apgar score as well as the infant's future prognosis. Such factors include prematurity, central nervous system (CNS) abnormalities cardiac and respiratory problems and maternal medications. Prior infection, abnormalities of development of the fetal CNS or insult to it, also may be a cause of perinatal asphyxia, perinatal problems and subsequent deficit.

In the past, the clinical course of the full term infant who experienced intrapartum asphyxia was thought primarily to reflect altered brain function, it is now known, however, that the infants can have a different clinical course due to variable involvement of various organ systems. The variation in clinical signs is due in part to the ability of the foetus to redistribute blood flow to protect vital organs. It has been documented that the infants with low Apgar score, have had problems with their
pulmonary, cardiovascular, central nervous, gastrointestinal and renal systems. The effects of asphyxia on the liver and the hepatic functions of the neonate is a relatively unexplored avenue. This study is being carried out to assess the effect of birth anoxia on hepatic functions of a neonate as also the ultimate outcome of these cases.

The liver plays a central role in the synthesis, degeneration and regulatory metabolism. Due to asphyxia the liver may be so damaged ("shock Liver") that it may not provide its basic functions. Among parameters of liver cell dysfunction the serum activity of glutamic pyruvic transaminase (SGPT) and glutamic oxaloacetic transaminase (SGOT) is most specific in adults and infants (Kove et al, 1957). The release of these enzymes from damaged tissue into the blood stream is the principal factor responsible for increased serum transaminase activity in the presence of hepatic cellular injury, due to both cellular necrosis and reversible injuries of permeability of cellular or intracellular membranes (King et al, 1959).

Therefore, during perinatal hypoxia which causes a reversible increase in cellular membrane permeability, there is a release into the blood stream of cytoplasmic enzymes (serum glutamic oxaloacetic transaminase, serum glutamic pyruvic transaminase, lactic dehydrogenase). In the presence of liver cell necrosis both the cytoplasmic and mitochondrial enzymes are increased. Since SGOT is
also present in myocardium, kidney and RBC while SGPT is
grimly released from the liver, therefore, this
enzyme is more specific for liver damage or injury.
Alkaline phosphatase also rises in liver damage but it
is less specific and less sensitive.

AIMS OF STUDY

1. To assess the liver functions in healthy newborns.

2. To assess the liver function in severe birth
asphyxiated babies and compare with the controls.

3. To collect the information about severity of
liver damage and their prognostic value.

4. The drugs that are detoxified by the liver must
have their levels monitored closely because in
a damaged liver, the drug metabolism may be altered.