DISCUSSION
Hypoxia, hypercapnia and acidosis represent, at birth, the main symptoms of fetal distress, which manifests clinically in utero as varying fetal heart rate and after delivery as a low Apgar score.

The determination of liver enzymes could offer a simple and rapid laboratory test for establishing the presence of hepatic cellular damage associated with perinatal asphyxia. As a result of change in cellular permeability, hypoxia causes degeneration in tissues, cloudy swelling, vacuolar cytoplasmic degeneration and cellular necrosis (Vogel, 1967) in about 97% of newborn dying from asphyxia.

The effect of asphyxia on the liver and the hepatic functions of the neonate is a relatively unexplored avenue. This study was, therefore, carried out to assess the effect of birth asphyxia on hepatic function of a neonate as also the ultimate outcome of these cases. The study included 30 severely asphyxiated full term neonates. Neonates were considered to have severe birth asphyxia if the Apgar score was $\frac{1}{3}$ and $\frac{1}{5}$ at 1 and 5 minutes respectively.

The infants suffering from significant congenital anomaly, intrauterine infections, heart disease, septicemia, significant hepatosplenomegaly, multiple pregnancies and low birth weight babies were not included in the study.
Preterm infants were excluded from the study as they have more immature liver and are more susceptible to respiratory distress syndrome, infections and other complications which may further complicate the picture of asphyxia.

Asphyxia causes most of system disorders including liver. Damage to liver is reflected by the derangement in its enzymes. In newborns with clinical symptoms of asphyxia, serum transaminase activity increase significantly during first 72 hours of life (Zanardo et al., 1985). Hence in the present study liver function tests (SGOT, SGPT, alkaline phosphatase and serum bilirubin) were measured within first 48-72 hours of life.

The gestational age of neonates was calculated by counting the number of weeks from the first day of last menstrual period till the birth of baby and confirmed by physical and neurological developmental scoring system (modified scoring system for assessment of gestational age of newborn (Meharban Singh et al., 1975).

In the present study the mean gestational age of asphyxiated neonate was 39.50±1.04 weeks and mean birth weight was 2798±206 gms whereas in control group it was 2790±181 gms. Both the groups were well matched as regards to birth weight and gestational age.

In this study the mean level of SGOT in asphyxiated neonates was significantly higher as compared to their controls. The mean value was 112.87±54.70 IU/L in asphyxiated neonates as compared to 48.73±16.08 IU/L
in controls. Out of 30 asphyxiated neonates SGOT was raised in only 73.33% of cases. Similar rise in SGOT level was reported by Zanardo et al (1985) in severely asphyxiated neonates within first 72 hours of birth. They observed a rise to a level of 100±8.9 IU/L in asphyxiated neonates as against 52.0±12.9 IU/L in controls. They also observed that levels of SGOT gradually decreased and become comparable to their controls between 5th and 10th day of life. Between 20th and 30th day of life, the mean values of SGOT in asphyxiated neonates and their controls further decreased and did not exceeded 30 IU/L. Salli et al (1980) also showed similar results with a rise of SGOT levels to 97.8±19.2 IU/L in asphyxiated neonates as compared to 54.8±48.86 IU/L in controls (Mean±2S.D.).

SGPT activity was also raised among asphyxiated neonates in this study as compared to their controls. The mean values of SGPT in asphyxiated neonates and their controls were 51.17±35.03 IU/L and 20.54±6.99 IU/L respectively. Out of 30 asphyxiated neonates the SGPT was raised in 70% cases. Zanardo et al (1985) also reported raised levels of SGPT in severely asphyxiate neonates in first 72 hours of life. The mean levels reported in their study were 54.4±54.4 IU/L in severely asphyxiated neonates and 18.0±6.6 IU/L in their controls. Then after, they obtained a fall in SGPT levels in full term asphyxiated neonates but the value remained significantly raised as compared to controls. According to their study, the initial rise was more marked for SGOT as compared to SGPT but SGPT remained significantly raised even upto 30th day.
of life. Whereas, SGOT levels returned to normal within 5th to 10th day of life in asphyxiated neonates.

Saili et al (1989) reported similar rise in SGPT values during first 72 hours. The level reported by them was 44.09±61.94 IU/L (Mean±2S.D.) in severely asphyxiated newborns and 22.4±32.96 IU/L in their controls.

In this study the levels of alkaline phosphatase were also raised as compared to their controls. The mean value among asphyxiated neonates was 17.64±2.28 KAU/dl and in their control was 14.45±1.53 KAU/dl. Out of 30 asphyxiated neonates, alkaline phosphatase levels were raised in 63.33% of cases. Similar results were observed by Saili et al (1989). The mean value of alkaline phosphatase in their study was 17.64±12.30 KAU/dl in severely asphyxiated neonates and 14.36±9.06 KAU/dl in their controls (mean±2S.D.). The levels of alkaline phosphatase were raised in 58% of cases in their study. But Fitz Simsons (1984) found no significant increase in the values of alkaline phosphatase in asphyxiated neonates in their study.

The present study also registered increased serum bilirubin in all neonates of both study and control groups. The mean value of serum bilirubin in asphyxiated neonates was 6.47±2.61 mg/dl, while mean value in their controls was 5.32±2.49 mg/dl. Similar results were reported by Saili et al (1989). In their study the mean level of serum bilirubin among severely asphyxiated neonates was 4.78±
6.62 mg/dl and in their controls was 4.50±6.12 mg/dl (Mean±2S.D.).

The mean levels of transaminases are significantly higher among normal healthy full term neonates as compared to older children and adults. The adult levels of transaminases is achieved by the end of neonatal period. The higher transaminase activity in neonatal blood has been attributed to seepage from the hepatocytes of neonates, which, being immature, have more permeable membranes. Increased transaminases in newborns may also be the result of increased biosynthesis, physiologic hemolysis of erythrocytes and skeletal muscle trauma during birth (Wolf, 1981).

Due to asphyxia the liver may be so damaged ("Shock Liver") that it may not provide its basic functions (Cloherty et al, 1985). In the event of hepatic cellular injury due to perinatal hypoxia, transaminases activity in the blood increases due to changes in cell membrane permeability or cellular necrosis of hepatocytes (King et al, 1959). In the presence of liver cell necrosis, both the cytoplasmic and mitochondrial enzymes are increased. Since SGOT is also present in myocardium, kidneys and RBCs while SGPT is primarily released from the liver, therefore, this enzyme is more specific for liver damage or injury. Alkaline phosphatase also rises in the liver damage but it is less specific and sensitive.

The serum bilirubin values are not taken into account as there is physiological rise even in healthy
neonates due to multifactorial etiology during first few
days of life and the difference in study and control
group was found to be insignificant.

Asphyxiated neonates were divided into the
following two groups:

Group A: Asphyxiated neonates who expired in the hospital.
Group B: Asphyxiated neonates who improved and survived
and were discharged from the hospital.

Out of 30 asphyxiated neonates in our study,
14 neonates expired (46.67%) and 16 survived (53.33%).
The mean values of liver enzymes of those two groups
were calculated and compared (Table V).

Similar results were observed by Saini et al
(1989). Levels reported in their study for SGOT, SGPT
and alkaline phosphatase among neonates who expired, were
146.2±102.4 IU/L, 61.1±51.52 IU/L and 20.3±12.2 KAU/dl
respectively. Whereas levels of these enzymes among
survivors of asphyxiated neonates in their study were
62.9±73.6, 31.7±58.0 IU/L and 15.7±11.18 KAU/dl for
SGOT, SGPT and Alkaline phosphatase respectively.

In this study, SGOT levels were raised in 100% of neonates, SGPT levels were raised in 92.86% of
neonates and alkaline phosphatase levels were raised
in 70.57% of non survivors from the study group. Whereas
serum bilirubin was raised in 100% of cases. Out of 14,
neonates, who expired, only 78.57% of neonates had raised
levels of all the three enzymes viz. SGOT, SGPT and
alkaline phosphatase.

Among 16 asphyxiated neonates, who survived, SGOT, SGPT and alkaline phosphatase were deranged in only 50% of cases. Only 37.5% of these cases had all three enzymes viz. SGOT, SGPT and alkaline phosphatase deranged.

No significant difference was observed in the mean values of SGOT, alkaline phosphatase and serum bilirubin levels between controls and asphyxiated neonates who survived. However, SGPT values were significantly raised in asphyxiated neonates who survived as compared to their controls (Table VI).

This study thus conclude that liver damage due to asphyxia is minimal in neonates who survived as compared to neonates who expired in the hospital.

It was interesting to note that asphyxiated neonates having deranged liver enzymes had poor prognosis as compared to neonates having normal levels of liver enzymes (Table IV). Out of 23 neonates who showed deranged liver enzymes, 60.87% expired in contrast to neonates having normal liver enzymes, all of whom survived. Saiili et al (1989) recorded deranged liver enzymes in 60% of cases, who expired due to asphyxia.

On further analysis, it was found that enzyme derangement was present in 23 (76.67%) neonates, Out of 30 asphyxiated neonates. All the 14 non-survivors felt in this group. Rest of neonates (23.33%) had normal liver function tests, none of them expired.
It could be concluded from our observations that when all the enzymes are deranged in an asphyxiated neonate a guarded prognosis have to be explained to the relatives.