DISCUSSION

The present study was carried out in the Department of Pediatrics, M.L.B. Medical College, Jhansi, over the period of one year from May 2005 to May 2006, in collaboration with Department of Radiology and Department of Pathology, M.L.B. Medical College, Jhansi.

The present study was undertaken with the following aims:

- To study the ECG changes, Echocardiography, LDH level and CPK-MB level in normal full term and preterm neonates.
- To study the ECG changes, echocardiography, LDH level and CPK-MB level in asphyxiated full term and preterm neonates.

Cardiac manifestations of asphyxia ranged from transient myocardial ischemia to fulminant heart failure which can be diagnosed easily by signs and symptoms of congestive heart failure, shock, supported by abnormal ECG and Echocardiography and elevated CPK-MB and LDH if diagnosed early and treated timely can save many lives.

The present study comprised of total seventy neonates. Out of which fifty were asphyxiated neonates (study group) and twenty were normal neonates (control group). Both study and control group were further subdivided according to gestational age into full
term and preterm cases (table -1). Out of 20 cases in control group 12 (60%) were fullterm and 8 (40%) were preterm. Out of 50 cases in study group 38 (76%) were fullterm and 12 (24%) were preterm. According to table-2, in the control group there were 7 (35%) male and 5 (25%) female full term cases, while in the preterm group there were 5 (25%) male and 3(15%) female cases, however in the study group there were 30(60%) male and 8(16%) female fullterm cases and 11 (22%) male and 1 (2%) female preterm cases. According to table no. –3, out of 20 cases in the control group, 8 (40%) were low birth weight and 12 (60%) were normal birth weight neonates. In the study group, out of 50 cases 21 (42%) were low birth weight and 29(58%) were normal birth weight neonates. According to table no. 4, out of 38 fullterm 12 (34.2%) had mild asphyxia, 15 (39.4%) had moderate asphyxia and 10(26.3%) had severe asphyxia, while out of 12 preterm neonates, 4 (33.3%) had mild asphyxia, 5 (41.6%) had moderate asphyxia and 3 (25%) had severe asphyxia. According to table no. –5, out of 38 full term neonates of study group, 23 (60.5%) were of grade I, 10 (26.3%) were of grade II, and 5(13%) were of grade III of HIE classification. Out of 12 preterm neonates of study group 7 (58.3%) were of grade I, 2 (16.6%) were of grade II and 3 (25%)
were of grade III of HIE classification. According to table no. 6, in the control group there were 8 (40%) vaginal deliveries and 4 (20%) caesarean section done in fullterm cases and 6 (30%) vaginal deliveries and 2 (10%) caesarean sections done in preterm cases, while in the study group there were 20 (40%) vaginal deliveries, 18 (36%) caesarean sections done in fullterm and 10 (20%) vaginal deliveries and 2 (4%) caesarean section done in preterm. According to table no. 7, out of 50 cases of study group 32 (64%) cases had clinical cardiac manifestation, while the remaining 18 (36%) did not manifest any signs of cardiac involvement. According to table no. 8, the most common cardiac manifestation in birth asphyxia is respiratory distress, present in 30 (60%) cases followed by CHF in 22 (44%) cases and then shock which was present in 9 (18%) cases. Murmur was present in 11 (22%) cases.

According to table -9, a significant finding of the present study was that the values of serum CPK-MB an LDH were statistically significant and higher in the study group comprising of asphyxiated babies with the mean value 230±152.7 IU/L; range 62-510 IU/L and 512±159.5 IU/L; range 287-761 IU/L as compared to control group of cases with the mean values of 27.6±13.2 IU/L;
range 6.2-52.6 IU/L and 133±32 IU/L; range 86.5-199.9 IU/L respectively (P<sub>sc</sub> < 0.001 for both CPK-MB and LDH). These observations are in consensus with the hypothesis that elevated serum activity of CPK-MB and LDH in the study group is indicative of release of these enzymes from myocardium as a result of birth asphyxia.

Roberts et al (1975), concluded that the only human tissue containing substantial amount of CPK-MB was myocardium. Further studies by Roberts et al (1978) and Lott et al (1980) showed that in adults, serum CPK-MB activity could be reliably utilized as a marker of myocardial cell injury.

Nelson et al (1978), compared the serum CPK-MB levels in the normal newborns with the newborns who had asphyxia and found a statistical significant difference between the two. The values of serum CPK-MB in the normal newborns was 0.8 with a range of 0.1-9 IU/L, while in the asphyxiated newborns, the values was 113 IU/L with a range of 29-587 IU/L, these values were more or less analogous to the levels of serum CPK-MB in our study, that is 27.6±13.2 IU/L; range 6.2-52.6 IU/L in control group and 230±152.7 IU/L with a range of 62-510 IU/L in the study group. Reason for the rise of CPK-MB in infants with perinatal asphyxia
may be explained on the basis of transient myocardial ischemia leading to myocardial damage, which is demonstrated in the form of elevated serum CPK-MB levels.

*Sutton et al (1981)*, conducted a study on 26 normal neonates, for CPK-MB activity. They noted that mean value of CPK-MB in the umbilical cord blood was 3.1 IU/L and this value was increased to 19.2 IU/L at 24 hours. *Sutton et al* suggested that this increase in CPK-MB value in normal neonates was due to some myocardial damage associated with birth process or there might be some unidentified source of CPK-MB activity in neonates.

In accordance with table no. –10, serum CPK-MB levels in full term control and study group were 31.4±12.5 IU/L; range 15.2-52.6 IU/L and 225.7±150 IU/L; range 62-510 IU/L respectively. While serum CPK-MB levels in preterm control and study group were 21.8±12.3 IU/L; range 6.2-38.4 IU/L and 243.8±158 IU/L; range 66-501.3 IU/L respectively.

In accordance with table no. –10, no statistical significant difference was observed in serum CPK-MB levels between full term and preterm cases of control or study group (P_{FcPc} = 0.1, P_{FSPS} > 0.1). This is supported by many workers (*Roberts et al, 1978; Lott et al, 1980; Bowek et al, 1981*).
In accordance with table no. –12, serum CPK-MB levels in low birth weight control group and study group were 27.1±14 IU/L; range 6.2-50 IU/L and 241.7±149.6 IU/L; range 62-510 IU/L respectively, while in normal birth weight control and study group were 27.9±12.7 IU/L; range 7.2-52.6 IU/L and 221.6±154.2 IU/L; range 62-501 IU/L respectively. No statistical significant difference was observed in serum CPK-MB levels between low birth weight and normal birth weight cases of control or study group (P_{LcNc} and P_{LsNs} > 0.1).

**Nelson et al (1978),** studied the serum CPK-MB leveling normal term neonates and compared these values with those in asphyxiated term neonates. The serum CPK-MB levels in their study group in the normal term neonate was 0.8 IU/L with a range of 0.1-9 IU/L and in asphyxiated term neonates was 113 IU/L with a range of 29-587 IU/L. These values are lower in contrast to the levels of CPK-MB in our study in full term control newborns that is 31.4±12.5 IU/L; with a range of 15.2-52.6 IU/L and 225.7±150 IU/L; with a range of 62-510 IU/L in asphyxiated babies.

**Britton et al (1980)** reported mean serum CPK-MB levels of 1.7±0.8 IU/L in normal term infants, the level being much higher in our study (31.4±12.5 IU/L).
Sutton et al (1981), reported a mean serum CPK-MB activity of 19.2 IU/L in normal term infants, these values being lower than those found in our study (31.4±12.5 IU/L). Sutton et al suggested that these higher values in normal neonates were associated with some myocardial damage occurring during birth process or some other unidentified source of CPK-MB activity in newborn.

Cuesta et al (1980), reported a mean serum CPK-MB activity of 15.5±13 IU/L in premature healthy neonates. These values were more or less similar to those found in our preterm control group that is 21.8±12.3 IU/L; with a range of 6.2-38.4 IU/L.

Thus our observations revealed that there is no correlation between CPK-MB levels and gestational age, as very well explained by the proven fact detailed by Bowek et al, 1981, and supported by many other workers (Robert et al, 1978, Lott et al, 1980), that the only known human tissue containing substantiated amount of CPK-MB is myocardium and any insult to any organ other than the heart are not associated with increased serum CPK-MB activity, despite marked elevation of total CPK.

As shown in table no. -14, serum CPK-MB levels, bear no correlation with the sex of the newborns ($P_{F_nM_nF_nC_n} > 0.1$, $P_{P_nM_nC_n} > 0.1$, $P_{F_nM_nF_nS_n} > 0.1$). This justifies the hypothesis that it is the presence
and degree of stress that defines the level of serum CPK-MB activity in the newborn. Similarly there is no correlation between serum CPK-MB levels and birth weight of the baby. This has also been substantiated by other workers.

According to table no. –29, serum CPK-MB level in echopositive group was 379.5±148 IU/L; range 130-510 IU/L and in echonegative group was 187.8±124 IU/L; range 62-490 IU/L. It was found that there was a statistical significant difference evident between two groups, with the values being higher in those with positive echofindings ($P_{\text{EPEn}} < 0.001$).

_Nelson et al (1978)_ reported that the mean value of serum CPK-MB in asphyxiated newborns without tricuspid insufficiency was 13 IU/L and in asphyxiated newborns suffering from myocardial dysfunction with tricuspid insufficiency, the mean value of CPK-MB was 113 IU/L. These values were lower than the values in our study that is 379.5±148 IU/L; range 130-510 IU/L in echopositive asphyxiated newborns and 187.8±124 IU/L range 62-490 IU/L in echonegative asphyxiated newborn. The most probable reason of lower range of CPK-MB in Nelson et al study was small sample size.
Herdy et al (1988), documented that from a total of 90 cases serum CPK-MB levels were significantly elevated in 26 (28%) patients having clinical evidence of heart hypertrophy and failure.

In accord to table no. –16, in full term neonates, the levels of serum CPK-MB and LDH in mild asphyxia were 78.5±13.5 IU/L; range 62-95.3 IU/L and 337.2±47.8 IU/L; range 287-411.2 IU/L, in moderate asphyxia were 206.4±54 IU/L; range 130-311 IU/L and 513±40.7 IU/L; range 453-571 IU/L and in severe asphyxia were 445.8±67 IU/L; range 310-510 IU/L and 735.4±18.9 IU/L; range 703.2±760 IU/L (P_{ab}<0.001, P_{bc}<0.001, P_{ac}<0.001 for both CPK-MB and LDH). In accord to table no. –17, in preterm neonates, the levels of serum CPK-MB and LDH in mild asphyxia were 79.1±11.6 IU/L; range 66-90.3 IU/L and 334.7±44.9 IU/L; range 289-382.4 IU/L, in moderate asphyxia were 240.3±81.6 IU/L; range 135-365 IU/L and 517.8±59.3 IU/L; range 451.3-600 IU/L, and in severe asphyxia were 469.2±42.6 IU/L; range 410-501.3 IU/L and 746.7±20.6 IU/L; range 721.3-761 IU/L (P_{ab}<0.01, for both CPK-MB and LDH, P_{bc}<0.01 for CPK-MB, P_{bc}<0.001 for LDH, P_{ac}=0.001 for CPK-MB, P_{ac}<0.001 for LDH).

According to table no. –18, in full term neonates the levels of serum CPK-MB and LDH, in grade I of HIE classification were
151.6±85.9 IU/L; range 62-350 IU/L and 438±125.8 IU/L; range 287—709.3 IU/L; in grade II were 264±130 IU/L; range 94-450.1 IU/L and 568.8±120.9 IU/L; range 401.3-746 IU/L and in grade III were 500±11.8 IU/L; range 490-510 IU/L and 748±30.8 IU/L; range 740-760 IU/L. (P<sub>ab</sub> < 0.01 for both CPK-MB and LDH, P<sub>bc</sub> < 0.001 for CPK-MB, P<sub>bc</sub> < 0.01 for LDH, P<sub>ac</sub> < 0.001 for both CPK-MB and LDH).

According to table no. –19, in preterm neonates, the levels of serum CPK-MB and LDH in grade I of HIE classification were 121.8±54 IU/L; range 66-203.5 IU/L and 395±82 IU/L; range 289-525 IU/L, in grade II were 355±55 IU/L; range 300-410 IU/L and 641.1±80.2 IU/L; range 560.9-721.3 IU/L and in grade III were 454.2±63.6 IU/L; range 365-501.3 IU/L and 706±78.2 IU/L; range 600-761 IU/L. (P<sub>ab</sub> < 0.01 for both CPK-MB and LDH, P<sub>bc</sub> > 0.1 for both CPK-MB and LDH, P<sub>ac</sub> < 0.001 for both CPK-MB and LDH).

These observations correlate well with the hypothesis that greater was the degree of asphyxia, higher were serum LDH and serum CPK-MB values. Our findings are similar to the observations of various other workers in the field who have ascribed that highest values in severe asphyxia is due to increased cellular damage and thus increased efflux of CPK-MB and LDH.
from their respective tissues resulting in increased levels of CPK-MB and LDH activity in the blood.

_Cuesta et al (1980)_ showed in their data, large increases of serum CPK-MB, CPK-BB and CPK-MM in asphyxiated infants. A rise in CPK-MB was attributed to the transient myocardial dysfunction and ischemia occurring in stressed or asphyxiated newborns. They opined that the correlation between the magnitude of significant elevation of serum CPK-BB following severe neonatal asphyxia and neonatal mortality suggests a quantitative relationship between the extent of CPK-BB elevation and the severity of cerebral damage in a manner analogous to the relation between serum CPK-MB activity and myocardial infarction size.

_Thangavel et al (1982)_ documented elevation of all the isoenzymes of CPK in two newborns with perinatal asphyxia and they suggested that MB fraction was increased on the second day. The more likely mechanism for this was hypoxic or ischemic cardiac injury. The serum CPK level in first newborn with no reliable score was 796 IU/L, and isoenzyme fractions were: MM 87.8%, MB 2.7% & BB 9%, while the other newborn with Apgar score one at one minute and five minute, had serum CPK levels of 460 IU/L with fractions as MM 94.7%, MB 4.7% & BB 2.5%.
Stevenson in 1943 and Jones and McCance in 1949 observed that the serum LDH level in the newborns were different from that in the adults. Zimmer-man (1958) noted a higher value of serum LDH in the cord blood samples. Stewart and Birkbeck (1962) suggested that LDH was higher in premature newborns and it could from the basis of biochemical definition of maturity.

Sinha et al 1978 reported that LDH activity was found to be higher in the samples collected 24 hr after delivery compared to the cord blood, then a gradual fall was observed on subsequent estimation.

Barberi et al (1999), studied three groups of neonates – group I- 22 healthy newborns with 5 min Apgar scores > 9 and pH > 7.3; group II- 15 neonates with moderate respiratory distress with 5 min Apgar scores 7-9 and pH 7.2-7.3; and group III- 13 neonates with severe asphyxia with 5 min Apgar scores < 7 and pH < 7.2. The ECGs were evaluated according to the 4 grade classification proposed by Jedeikin et al. On the echocardiograms fractional shortening and aortic flow curve parameters were taken in to account. Serum creatinine kinase, creatinine kinase-MB isoenzyme and lactate dehydrogenase (LDH) were determined. All neonates of group I and II survived, but 5 out of 13 babies in group
Ill died within the first week. Grade 3 or 4 ECG changes were observed only in group III patients, while all babies in group II and 3 patients of group I showed grade 2 ECG changes. Fractional shortening, peak aortic velocity and mean acceleration were significantly reduced in group III, whereas the only abnormality found in group II was a reduced fractional shortening. CPK, CPK-MB, CPK-MB/CPK ratio and LDH were all increased in group III, while in group II only CPK-MB and the CPK-MB/CPK ratio were abnormal. Severely asphyxiated newborn infants reflect relevant ischemic ECG changes, depressed left ventricular function and marked cardiac enzyme increase. These alterations are far less pronounced in neonates with mild respiratory distress.

According to table no. –11, serum LDH levels in full term control group were 135.4±34.8 IU/L; range 96.5-199.9 IU/L and in preterm control group were 129.3±27.4 IU/L; range 86.5±179.8 IU/L. Sinha et al (1978), reported that serum LDH activity in normal full term newborn was 300.3±29.6 IU/L; range 198-372 IU/L and in preterm normal newborn 305.1±18.9 IU/L; range 274-346 IU/L. The values of LDH reported by Sinha et al were higher compared to our study. Stewart & Birkbeck (1962) suggested that LDH activity was higher in premature newborns. But in our study in
control group LDH activity was slightly higher in full term newborns.

According to Sinha et al (1978), the values of serum LDH in newborns with respiratory distress were high compared to those in control group i.e. 397±26.3 IU/L; range 364-460 IU/L. Nelson, Avery and Naeye (1973) had similar observations to make. They had suggested that LDH level approaching 700 IU/L had an ominous prognostic value. The explanation given by them with regard to such a high LDH activity were two folds. Nelson (1973) was of the view that there is excessive breakdown and necrosis of the alveolar cells per se whereas Avery (1973) suggested that lack of surfactant exposed the cells to traumatic injury even by movement of air leading to release of LDH from cells. Another view is that acute respiratory distress puts the cardiac muscles in excessive strain triggering the release of extra LDH in circulation (Naeye, 1973).

In accordance with table no. -11, serum LDH levels in full term control and study group were 135.4±34.8 IU/L; range 96.5-199.9 IU/L and 511.4±158.4 IU/L; range 287-760 IU/L respectively. While serum LDH levels in preterm control and study group were 129.3±27.4 IU/L; range 86.5-179.8 IU/L and 514±162.7 IU/L; range
289-761 IU/L respectively. In accordance with table no. –11, no statistically significant difference was observed in serum LDH levels between fulterm and preterm cases of control or study group (P_FcPc > 0.1, P_FsPs > 0.1).

In accordance with table no. –13 serum LDH levels in low birth weight control and study group were 136.2±30.3 IU/L; range 86.5-179.8 IU/L and 528.5±156.9 IU/L; range 287-761 IU/L respectively. While in normal birth weight control and study group were 130.8±33.2 IU/L; range 96.5-199.9 IU/L and 500±160.5 IU/L; range 289-758 IU/L respectively. No statistically significant difference was found in serum LDH levels between low birth weight and normal birth weight cases of control or study group (P_LcNc > 0.1, P_LsNs > 0.1).

According to table no. –15, serum LDH levels bear no correlation with the sex of newborns (P_FcmFcf > 0.1, P_PcmPcf > 0.1, P_FsmFsf > 0.1). This justifies the hypothesis that it is the presence and degree of stress that defines the level of serum LDH activity in the newborn.

Karunatilaka (2000) documented that an upward trend in serum LDH values was seen with the increasing severity of HIE which however was not statistically significant. But in our study a
statistical significant difference was observed between mild and moderate asphyxia, moderate and severe asphyxia and mild and severe asphyxia. In our study in full term serum LDH levels in mild, moderate and severe asphyxia were 337.2±47.8 IU/L; range 287-411.2 IU/L, 513±40.7 IU/L; range 453-571 IU/L and 735.4±18.9 IU/L; range 703.2-760 IU/L respectively (according to table no. 16).

The median values of serum LDH in their study were 3876 IU/L in mild, 5505 IU/L in moderate and 7653 IU/L in severe asphyxia. These values are quite higher in contrast to the values in our study viz 337.2 IU/L in mild, 513 IU/L in moderate and 735.4 IU/L in severe asphyxia. The difference of serum LDH levels in our study and in above study is probably due to the less number of cases included in the above study.

Our observations regarding the correlation of CPK-MB and LDH levels to the grade of birth asphyxia according to Apgar score (table 16 & 17) and HIE classification (table 18 & 19) in full term and preterm babies, reveals, that severe asphyxia manifest with higher values of both CPK-MB and LDH levels than the other groups.

ECG findings of myocardial ischemia suggested by Myung K Park in neonates were ST depression, Q waves > 2mm and T
wave inversion or flattening in the leads facing the ischemic area. Since ST segment changes were often accompanied by 'T' wave changes, a combination of ST depression and T wave flattening or inversion, if supported by elevated CPK-MB levels were sufficient enough to diagnose myocardial ischaemia. He further stated that Q wave >2mm is observed if ischaemia has progressed to infarction and necrosis.

In our present study commonest ECG abnormalities were flat or inverted T wave in mild asphyxia; Q wave >2mm with T wave inversion or flattening (with or without ST depression) in limb leads and right precordial leads in moderate asphyxia and ST depression and T wave inversion or flattening in severe asphyxia (table no -22). CPK-MB and LDH levels showed gradually increasing levels from mild to severe asphyxia. Similar ECG findings were observed by other workers also.

Rowe and Hoffman (1972) published a case report of 3 neonates who were admitted within 24 hr of birth with clinical features of respiratory distress, cyanosis, CHF and striking impairment of left ventricular output. Scalar electrocardiogram were recorded, chest radiography was performed. Blood gases were analysed. Cardiac catheterization and angiocardioigraphy
were performed to rule out congenital heart disease and coronary artery obstruction. In first neonate ECG at 24 hrs demonstrated abnormal T waves in I, aV\textsubscript{R}, aV\textsubscript{L}, V3-V6 as well as Q waves in V3R and V1. It was interpreted as showing right atrial and right ventricular overload, and nonspecific T wave changes. In second neonate, ECG at 48 hours showed flat T waves in leads I, II aV\textsubscript{R} and aV\textsubscript{L} and in third neonate, ECG showed abnormal T waves in V6. To explain this they advanced the concept of transient myocardial ischemia. In this supposition it can be argued that the primary effect could be an exaggerated response to hypoxia by a particularly sensitive pulmonary vascular bed. This in turn would create excessive demands on the right coronary arterial supply through increasing right ventricular work. Perfusion to those areas of distribution of this artery at most risk i.e. subendocardial zones of the right ventricle and the posterior portion of the left ventricle, could thus be impaired.

\textit{Bucciarelli et al (1977)} studied 14 full term asphyxiated newborns. They observed ECG findings in the form of ST depression in mid precordium and T wave inversion in left precordium (suggestive of myocardial ischemia) in 9 out of 14 asphyxiated neonates. In our study ST depression and T wave
inversion both were found in 23 newborns out of 50 asphyxiated newborns.

*John P Finley et al (1979)* observed ECG changes in the form of Q wave or ST - T wave abnormalities suggestive of myocardial ischaemia in stressed newborns.

*Daga et al (1983)* stated that myocardial dysfunction should be anticipated following birth anoxia. The presentation may be in the form of respiratory distress, shock, CHF, presence of murmur. ECG is an useful aid in the diagnosis. Eight asphyxiated neonates were included in their study. All but one babies survived. Clinical recovery preceded the reversion to normal of the ECG changes. Commonest ECG findings in this study were T wave flattening and ST depression which are in the agreement with our findings but they could not grade the ECG findings with the degree of asphyxia (table no.-21).

*Gidwani et al (1990)* studied 25 asphyxiated neonates and found ECG changes consistent with the degree of asphyxia. In their study group 25 neonates were included. Out of 25 asphyxiated neonates, 7 had mild, 10 had moderate and 8 had severe degree of asphyxia. Criteria for the diagnosis of asphyxia were same as in our study. They reported wide spread T wave
flattering in mild asphyxia. Significant Q waves in lower limb leads and ST-T wave changes in chest leads were observed in moderate asphyxia. Qs pattern and ST elevation or depression were observed in severe asphyxia.

*Herdy et al (1998)* conducted a study to evaluate the severity of cardiac complications of neonatal asphyxia. They included 90 babies in their study. The main cardiological findings were systolic murmur in 46(50%) signs of hypertrophy in 18 (20%) and heart failure in 8(9%). But in our study main cardiological findings were respiratory distress in 30(60%), CHF in 22 (44%), murmur in 11(22%) and shock in 9(18%) (table no.- 8). In their study main ECG findings were ST and T wave abnormalities, as same with our study. In their study Echocardiographic findings were PDA in 20(22%), Tricuspid regurgitation in 6(7%), pulmonary hypertension in 6(7%), dyskinesia and ventricular dilatation in 4(5%). In our study echocardiographic findings were PDA in 7(14%), Tricuspid regurgitation in 4(8%) and pulmonary hypertension in 4(8%) (table no. – 26).

*Ranjit (2000)* studied cardiac abnormalities in birth asphyxia. These abnormalities were : (1) Transient tricuspid regurgitation (2) Transient mitral regurgitation  (3) Transient myocardial ischemia.
This would be suspected in any baby with apshyxia, respiratory distress and poor pulses especially if a murmur was audible ECG was very important for diagnosis of transient myocardial ischemia and showed changes ranging from T wave inversion in one lead to a classical segmental infarction pattern with abnormal Q waves. CPK-MB was raised and echocardiography showed impaired left ventricular function, mitral and or tricuspid regurgitation and at times, wall motion abnormalities of left ventricle (4) Persistent pulmonary hypertension of the newborn. Cardiac abnormalities in asphyxiated newborns were often under diagnosed and high index of suspicion was required. ECG and ECHO helped in early recognition and hence better management of these cases.

It is evident from our observations on serum CPK-MB and LDH and ECG and Echo findings and from the observations of other workers, regarding serum CPK-MB and LDH, and ECG and Echo findings in these group of neonates that there was some variations of the results in different studies which may be possibly because of different populations studied and individual analytical laboratory differences.

Thus we also shared the view that myocardial dysfunction in asphyxiated newborns is not uncommon and should be suspected
in all stressed neonates presenting with respiratory distress, CHF, shock, murmur of tricuspid insufficiency in varying combination. If the clinical features and ECG and Echo findings are supported by serum CPK-MB and LDH estimation, can diagnose myocardial ischemia in asphyxiated neonates fairly accurately. Since the myocardium of newborn has a greater ability to recover, early diagnosis and supportive therapy for shock, hypoxemia and acidemia can save many more asphyxiated neonates.