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Perinatal asphyxia is an insult to the fetus or newborn due to lack of oxygen (hypoxia) and or lack of perfusion (ischaemia) to various organs of sufficient magnitude and duration to produce functional and or biochemical change. The effects of hypoxia and ischaemia may not be identical but they are difficult to separate clinically. Both factors probably contribute to asphyxial injury. It is one of the commonest medical emergencies of newborns and is one of the most important causes of neonatal morbidity and mortality.

Apgar scoring system devised by Dr. Virginia Apgar in 1953 and modified later on, has been widely used to identify birth asphyxia and to grade it into mild, moderate or severe.

The incidence of perinatal asphyxia is about 1-1.5% in most centres and usually related to gestational age and birth weight. It occurs in 9% of infants less than 36 weeks of gestation and in 0.5% of infants more than 36 weeks of gestation; accounting for 20% of perinatal deaths. (Ohlsson et al, 1987).

The incidence is higher in term infants of diabetic or toxaemic mothers (Snyder et al, 1992). In both term infants and preterm infants, intrauterine growth retardation and breech
presentation are associated with an increased incidence of asphyxia.

It is a known fact that seeds of neonatal morbidity are sown in the labour room, therefore effective management of newborn after birth, with early establishment of respiration is associated with improved survival and outcome.

In term neonates, 90% of asphyxial insults occur in the antepartum or intrapartum periods as a result of placental insufficiency. The remainders are postpartum. The proportion of postpartum events is higher in premature neonates, especially the E.L.B.W. babies.

During normal labour, uterine contractions and some degree of cord compression result in reduced blood flow to the placenta and hence decreased O₂ delivery to the fetus. Because there is a concomitant increase in O₂ consumption by both mother and fetus, fetal O₂ saturation falls. Maternal dehydration and maternal alkalosis from hyperventilation may further reduce placental blood flow, maternal hypoventilation may also contribute to decreased maternal and fetal O₂ saturation. These normal events cause most babies to be born with little O₂ reserve.
At birth as a consequence of normal events, most of the babies are born with little oxygen reserve so any factor impairing oxygenation or increasing its demand exacerbate perinatal asphyxia. In the presence of hypoxic- ischaemic challenge to the fetus, reflexes are initiated, causing shunting of blood to the brain, heart and adrenals and away from the lungs, gut, liver kidneys, spleen, bone, skeletal muscle and skin (Diving reflex).

Target organs of perinatal asphyxia are the brain, heart lungs, kidneys, liver, bowel and bone marrow.

Perlman et al (1989) conducted a study on asphyxiated newborns and reported that 34% had no evidence of organ injury, 23% had an abnormality confined to one organ, 34%, involved two organs and 9% had three affected organs.

The most frequent abnormalities involved the kidney (50%), central nervous system (28%), cardiovascular system (25%) and pulmonary system (23%). The degree of asphyxia required to cause permanent neurological impairment is close to that which cause death from multisystem failure (Freeman et al 1988).

Grossly, the following cerebral lesions may be seen after moderate or severe asphyxia (i) focal or multifocal cortical necrosis
(ii) watershed infarcts (iii) selective neuronal necrosis (iv) necrosis of thalamic nuclei and basal ganglia (status marmoratus).

In the past the clinical course of full term infant who experienced intrapartum asphyxia was thought primarily to reflect altered brain function, it is now known that the infant can have a different clinical course due to variable involvement of various organ system.

Adamsons 1963, Mueller Heubach 1968 and Myers 1971 have opined that severe partial asphyxia leads to diminished ventricular contractility and diminished cardiac output. Thus arterial hypotension which develops, contributes to development of brain damage, because of diminished cerebral perfusion. At the same time, impaired circulation to the heart increases the probability of myocardial ischaemia, which further impairs circulation. A vicious cycle is thus set up leading to fetal death, either within a relatively short time or sometimes delayed for some hours after birth. Thus one factor contributing to both brain injury and fetal death is asphyxiated myocardial decompression.

Cardiac manifestations of birth asphyxia ranges from transient myocardial ischaemia, to congestive cardiac failure. In mild asphyxia there is increased heart rate, slight increase in blood
pressure to maintain cerebral perfusion, increased CVP 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 a fall in blood pressure. The decreased cardiac output and hypotension so caused may lead to features of myocardial infarction due to inadequate coronary perfusion, but the ability of newborn to survive longer episodes of hypoxia than older children or adults, combined to produce a myocardial dysfunction syndrome limited to newborns. Stressed newborns having myocardial dysfunction may present with signs of congestive heart failure such as tachycardia, tachypnea, enlarged liver and gallop rhythm. Many infants have a systolic murmur at the lower left sternal border and radiating toward the right sternal border (Tricuspid insufficiency) and some have a murmur at the apex (Mitral insufficiency). Perlman J.M. et al (1989) conducted a study on moderately to severely asphyxiated newborns and reported that left ventricular dysfunction occurred in <10% of infants and right ventricular dysfunction was found in 30% of infants.

This cardiac dysfunction syndrome can thus be accurately diagnosed with signs and symptoms mentioned above, supported
by abnormal ECG, Echo study and elevated levels of LDH and CPK-MB. It is known that at present three different CPK isoenzymes were detected by means of electrophoresis or chromatography (Burger et al 1964, Rosalki et al 1965). The first being brain type (CPK-BB), second muscular type (CPK-MM), and third one cardiac type (CPK-MB). The only human tissue containing substantial amount of CPK-MB is myocardium (Roberts et al 1975), so the increased activity of serum CPK – MB is a useful indicators of myocardial injury. The sites of storage of LDH are the brain, kidney, heart, skeletal muscle and erythrocyte so in myocardial injury, serum LDH level elevates.

Rudolph et al (1966), the first worker, studied the relationship of birth asphyxia to the elevated levels of serum CPK and its isoenzyme fraction MM. Further studies by Nelson et al 1978 and Thangavel et al 1982 reported elevated levels of CPKMB in newborn who had severe birth asphyxia.

Present study was under taken to study myocardial dysfunction in birth asphyxia by ECG changes, echocardiography, LDH estimation and CPK-MB estimation.