DISSERTATION ON

A COMPARATIVE STUDY OF BLOOD GLUCOSE LEVELS IN NEONATES IN SICK NEONATAL CARE UNIT USING GLUCOMETER AND LABORATORY GLUCOSE OXIDASE METHOD IN GOVERNMENT RAJAH MIRASDAR HOSPITAL, THANJAVUR

Dissertation submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfilment of the regulations
for the award of the degree of

DOCTOR OF MEDICINE IN PAEDIATRICS

BRANCH – VII

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THANJAVUR - 613 004

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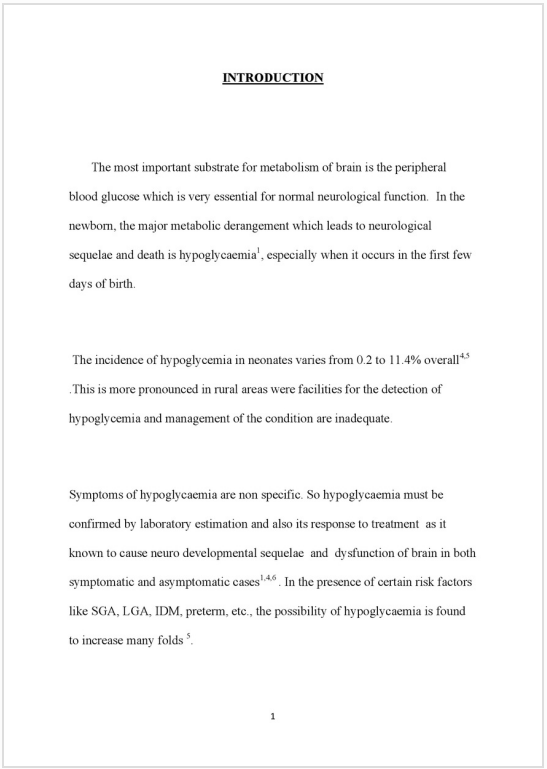

INTRODUCTION

The most important substrate for metabolism of brain is the peripheral blood glucose which is very essential for normal neurological function. In the newborn, the major metabolic derangement which leads to neurological sequelae and death is hypoglycaemia[1], especially when it occurs in the first few days of birth.

The incidence of hypoglycemia in neonates varies from 0.2 to 11.4% overall[4,5] .This is more pronounced in rural areas were facilities for the detection of hypoglycemia and management of the condition are inadequate.

Symptoms of hypoglycaemia are non specific. So hypoglycaemia must be confirmed by laboratory estimation and also its response to treatment as it known to cause neuro developmental sequelae and dysfunction of brain in both symptomatic and asymptomatic cases[1,4,6] . In the presence of certain risk factors like SGA, LGA, IDM, preterm, etc., the possibility of hypoglycaemia is found to increase many folds [5].

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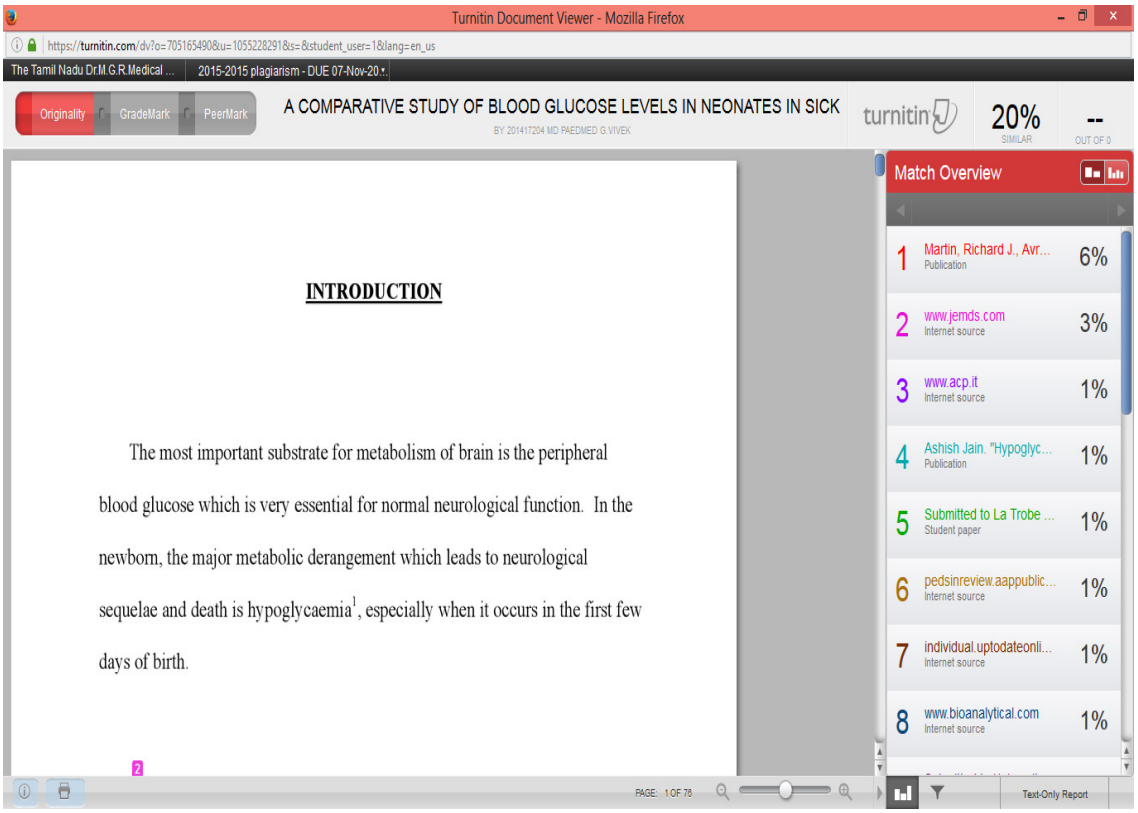
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ABSTRACT

BACKGROUND:

Hypoglycaemia is one of the most common metabolic problems seen in neonatal intensive care unit. The symptoms of hypoglycaemia in neonates are subtle. There is increased risk of neuromotor disability and intellectual disability among the survivors of symptomatic hypoglycaemia[1].

Hence for early detection and treatment of hypoglycemia a reliable device is needed. In the laboratory, the blood glucose estimation is done using glucose oxidase method which is specific and precise for the estimation of glucose but the results are not immediately available. So glucose estimation is done using glucometer in the neonatal intensive care unit for immediate results.

Blood glucose estimated by glucometers correlates well with the laboratory values only in euglycemic and hyperglycemic states but it is less often useful in the hypoglycaemic range according to many studies [2.]

AIMS AND OBJECTIVES

1. To estimate the blood glucose levels in sick newborn infants.
2. To estimate the validate of the glucometer for detection of blood glucose levels in detecting hypoglycaemia.

METHODS:

200 neonates admitted in NICU, GOVERNMENT RAJAH MIRASDAR HOSPITAL,Thanjavur Medical College, Thanjavur during a period of 6 months from January 2016 to July 2016. The glucose oxidase peroxidase method is done in the laboratory using venous sample. The blood glucose estimation was done by capillary and venous method using glucometer. Pearson correlation was used for statistical analysis. Hypoglycaemia is defined as the blood glucose level below 40mg/dl [3]. The glucose oxidase peroxidase method is the gold standard based on which the sensitivity, specificity, and predictive value was calculated.

RESULTS

In our study, of the 200 cases, 31 cases (15.5%) were hypoglycaemic, 164 (82%) cases were euglycemic and 5 cases (2.5%) were hyperglycemic by laboratory glucose oxidase peroxidase method, which is taken as the gold standard.

Capillary Blood glucose estimation using glucometer detected 23 cases (74.2%) of hypoglycaemia, 156 cases (95.1%) of euglycemiaand 5 cases (100%) of hyperglycemia in comparison with laboratory glucose oxidase peroxidase method.

Venous blood glucose estimation using glucometer detected 29 cases (93.5%) of hypoglycaemic, 158 cases (96.3%) of euglycemia and 3 cases

(60%) of hyperglycemia in comparison with laboratory glucose oxidase peroxidase method.

Estimation of capillary blood glucose by glucometer was found to have a sensitivity of 74.19%, specificity of 98.2%, positive predictive value of 88.4% negative predictive value of 95.4% and accuracy of 94.5% with statistically significant P value < 0.05.

Estimation of venous blood glucose using glucometer was found to have a sensitivity of 93.55%, specificity of 98.23 %, positive predictive value of 90.62% negative predictive value of 98.8% and accuracy of 97.5% with statistically significant P value < 0.05.

CONCLUSION:

1. Estimation of blood glucose using capillary and venous blood using glucometer have strong correlation with laboratory oxidase peroxidase method in detecting neonatal hypoglycaemia
2. The sensitivity of detecting neonatal hypoglycemia by glucometer using venous blood is higher than capillary blood.

KEYWORDS

Hypoglycemia, neonates, glucometer, glucose oxidase method.

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ABBREVIATIONS

AGA	-	Appropriate for Gestational Age
IDM	-	Infant of Diabetic Mother
IUGR	-	Intra Uterine Growth Restriction
LBW	-	Low Birth Weight
LGA	-	Large for Gestational Age
NICU	-	Neonatal Intensive Care Unit
NPV	-	Negative Predictive Value
PCV	-	Packed Cell Volume
PIH	-	Pregnancy Induced Hypertension
PPV	-	Positive Predictive Value
SGA	-	Small for Gestational Age
TPN	-	Total Parentral Nutrition

INTRODUCTION

The most important substrate for metabolism of brain is the peripheral blood glucose which is very essential for normal neurological function.In the newborn, the major metabolic derangement which leads to neurological sequelae and death is hypoglycaemia[1], especially when it occurs in the first few days of birth.

The incidence of hypoglycemia in neonates varies from 0.2 to 11.4% overall[4,5].This is more pronounced in rural areas were facilities for the detection of hypoglycemia and management of the condition are inadequate.

Symptoms of hypoglycaemia are non specific. So hypoglycaemia must be confirmed by laboratory estimation and also its response to treatment as it known to cause neuro developmental sequelae and dysfunction of brain in both symptomatic and asymptomatic cases[1,4,6]. In the presence of certain risk factors like SGA, LGA,IDM,preterm, etc., the possibility of hypoglycaemia is found to increase many folds[5].

However, when promptly suspected, diagnosed and treated the complications arising due to this condition can be prevented or minimised.

Hence for the detection of hypoglycaemia many tools were identified and studies were conducted for estimating glucose much sooner to prevent the complications of hypoglycaemia.

OBJECTIVES

1. To estimate the blood glucose levels in sick newborn infants
2. To estimate the validate of the glucometer for detection of blood glucose levels in detecting hypoglycaemia

REVIEW OF LITERATURE

HISTORICAL BACKGROUND

Reduced glucose concentration in the blood is termed as hypoglycemia. It was first described in the children 100 years back and in the newborn baby and older infants it was described 50 years[7] back.

In spite of recent advances and technological development in the care of newborns, the correct definition, significance and treatment of hypoglycaemia in neonates still remains a controversy.

In neonates screening for hypoglycaemia was started only after test for blood glucose using reagent strips was developed in 1970.

GLUCOSE HOMEOSTASIS AT BIRTH

Glucose is the primary fuel for the fetus accounting for approximately 80% of fetal energy consumption. The fetus has a continuous supply of glucose from the mother via the placenta and consequently fetal blood glucose levels are the same as the mothers[8]. The remaining 20% of fetal energy needs is provided by lactate, amino acids, ketones and other means. In uncomplicated pregnancy, the fetus is completely dependent on mother for its supply of glucose, for both energy productions, synthesis of other substrates.

Hepatic glycogen content is low[9] in early gestation; a slow, continuous increase occurs between 15 and 20 weeks; and a rapid accumulation of glycogen in the liver is observed later. Fetal liver contains enzymes which are used in glucose production and glycogen breakdown process. These enzymes are activated by excessive maternal starvation. Fetal liver contains glycogen which is 3 times greater than adult liver. At birth, glycogen stores in the liver accounts for about 1% of the neonate's energy reserve.

Glucose metabolism after birth

Glucose and oxygen are the main metabolic substrate of mature brain. At birth, the infant has to rapidly switch to endogenous gluconeogenesis until feeding is established. At birth the newborns blood glucose falls to approximately 75% of maternal blood glucose level. With the loss of the continuous infusion of glucose through the placenta, plasma glucose concentration in healthy term newborn falls during the first two hours after birth, reaching a nadir no lower than 40mg/dl, and then stabilises by 4-6 hours of age in the range of 45-80 mg/dl[10].

In the neonate the brain can use alternative metabolic fuels such as lactate and ketones. This is why the brain may be able to function normally or near normally, despite very low levels of blood glucose. The plasma glucose is maintained immediately after birth by the breakdown of hepatic glycogen in response to epinephrine and glucagon and it is facilitated by falling insulin levels[11].

TABLE 1: INSULIN EFFECTS IN GLUCOSE METABOLISM

Action	Effects
Glucose Uptake Into the Muscle	Stimulates
Glucose Uptake into the adipose tissue	Stimulates
Adipose tissue {FFA release}	Inhibits
Glucose production	Inhibits
Ketone production	Inhibits

However hepatic glycogen is depleted during 8-12 hours, after which plasma glucose levels are maintained by the synthesis of glucose from lactate, glycerol, amino acids and primarily alanine(gluconeogenesis). As feeds are established and carbohydrate intake is adequate, maintenance of plasma glucose concentrations is no longer solely depend on gluconeogenesis.

Adequate glucose output depends on:

- Adequate glycogen stores
- Sufficient gluconeogenetic precursors
- Normally functioning hepatic gluconeogenetic and glycogenolytic system
- Normal endocrine system for modulating these processes

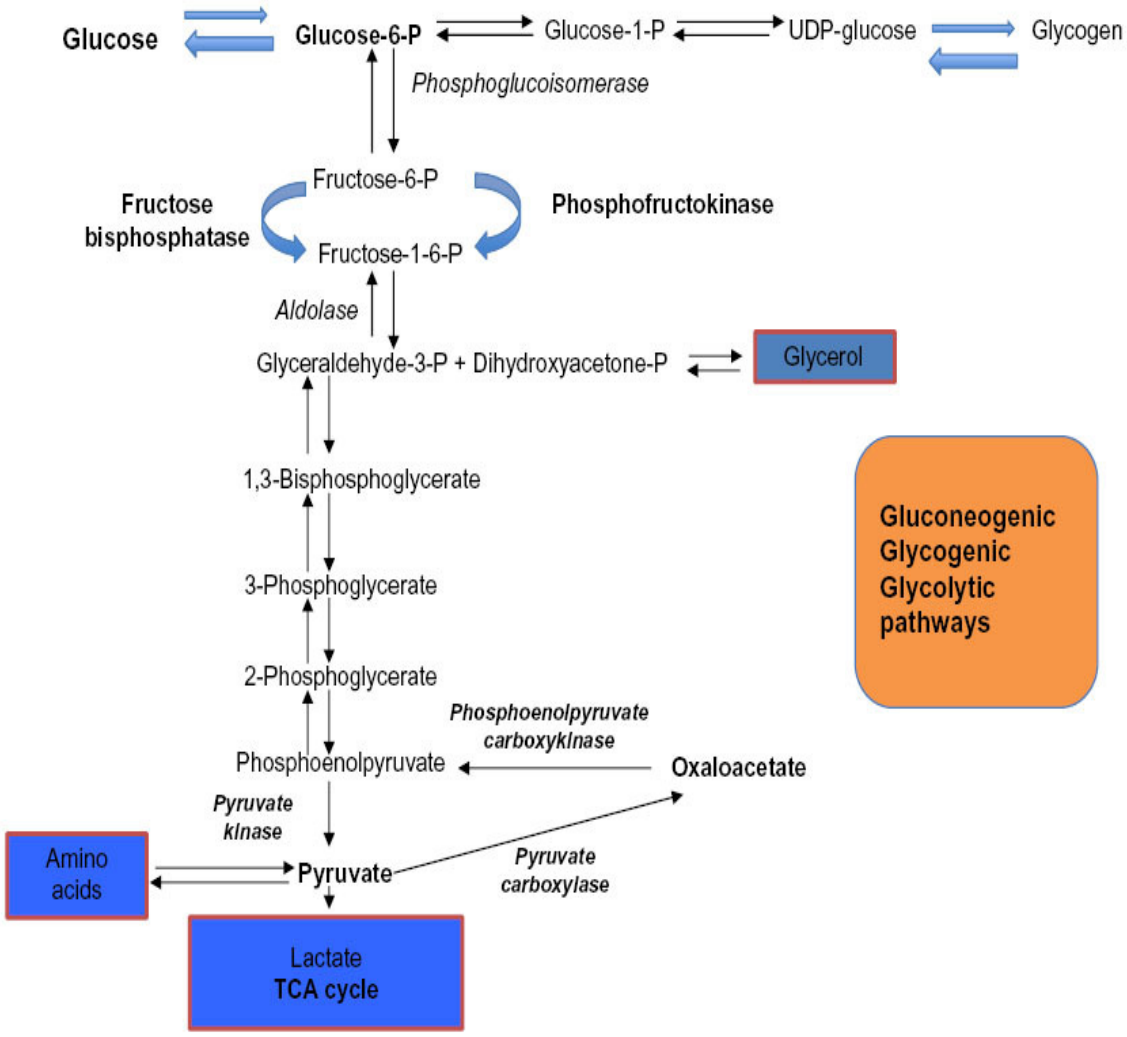
This glucose generation and homeostatic process, partially explains the aetiology of hypoglycaemia in most of the infants.

GLUCONEOGENESIS

The fetus can carry out gluconeogenesis to a limited degree, although it is likely that under normal circumstances it does not need this function. The key gluconeogenetic enzymes namely Pyruvate dehydrogenase, pyruvate carboxylase, phosphoenolpyruvate carboxylase, pyruvate kinase, fructose 1,6 DiPhosphatase which are present in the fetal liver by 2-3 months of gestation[12,13], are the substrate for activation of endocrine system and their inhibition. Activities of these enzymes are believed to increase throughout gestation and the neonatal period. .

The regulatory influences of gluconeogenesis pathway are insulin and glucagon ratio, intracellular accumulation of precursors, concentration of acetyl coA, NAD/NAD+ ratio. Increase in the concentration of intra cellular acetyl co A and NAD/NAD+ ratio results in the conversion of fat and fatty acids into glucose. Gluconeogenesis is also indirectly stimulated by adrenaline.

FIGURE 1 :GLUCONEOGENESIS



Thus all appropriately grown newborns, including the very premature, probably have some degree of gluconeogenic capability. However the growth retarded neonate may have impaired gluconeogenic capability.

Hypoglycemia

Historically, hypogylcemia has been defined as a whole blood glucose concentration of < 1.7 mmol/L in term infants and <1.1mmol/L in preterm term neonates. More recently hypoglycaemia has been defined in term infants as 2.2 – 2.5 mmol/L after the first 24 hours of life.

In a retrospective study Lucas *et al* [14] found that premature infants with birth weight of less than 1.8kg with blood sugar less than 2.6 mmol/L where at increased risk of lower developmental scores, particularly when blood sugar values were below this figure in repeated occasions. Others have shown that a deterioration in neurological function (measured by evoked potential) occurred with blood sugars level < 2.6 mmol/L (Koh*et al* [15]) .According to Heck and Even berg's[16] neonatal hypoglycaemia is defined as

3-24 hours < 2.24mmol/l

>24hrs < 2.5mmol/l[17,18].

The concentration of plasma or whole blood glucose at which clinician should consider intervention is known as operational threshold[19.]

These studies define normoglycemia in new borns as blood sugar levels of 2.6 mmol/L and above, but levels below this does not necessarily

indicate potentially damaging hypoglycaemia. The chemical definition of hypoglycaemia must take into account the methodology of glucose determination. Glucose concentration in whole blood is approximately 10-15% lower than that in plasma. Delay in determination after blood sampling may result in glucose oxidation by erythrocytes causing falsely low values.

Causes of hypoglycaemia

Hypoglycemia is caused either by diminished glucose supply or increased glucose consumption or a combination of both mechanisms. Because normoglycemia initially depends upon glycogenolysis and gluconeogenesis, infants in whom either a substrate is lacking or the metabolic pathway is impaired may develop hypoglycaemia.

CAUSES OF HYPOGLYCAEMIA IN NEONATES:

Diminished production

- ✓ Limited glycogen
- ✓ SGA
- ✓ Prematurity
- ✓ Birth stress
- ✓ Glycogen storage disorders

Limited gluconeogenesis

- SGA
- Inborn errors of metabolism

  Carbohydrate

  - ✓ Galactosemia
  - ✓ Glycogen storage disorders
  - ✓ Fructose intolerance

  Amino acids

  - ✓ Maple syrup urine disease

- ✓ Propionic acidemia
- ✓ Methyl melonicaciduria
- ✓ Hereditary tyrosinemia
- ✓ Glutaricacidemia type II

Fatty acids

- ✓ Defects in carnitine metabolism
- ✓ Acyl co enzyme dehydrogenase defects

Increased glucose utilisation due to hyperinsulinism

- ✓ IDM
- ✓ Beckwith Wiedemann syndrome
- ✓ Nesidioblastosis or pancreatic adenoma
- ✓ Erythroblastosisfetalis
- ✓ Exchange transfusion
- ✓ Drugs: chlorpropamide, benzothiazides, sympathomimetics
- ✓ Malpositioned UA catheter

Increased glucose utilisation without hyperinsulinism

- ✓ Sepsis
- ✓ Hypothermia
- ✓ Polycythemia-Hyperviscosity syndrome
- ✓ Congential cardiac malformations
- ✓ LGA infants who are not IDM
- ✓ Congenital hypopituitarism

Neurohypogylcemia (Hypoglycorrhachia)

Decreased glucose production

Limited glycogen stores

Premature:

The third trimester of pregnancy is an important period for hepatic glycogen deposition. An infant delivered prematurely without the benefit of part of or the entire third trimester will have limited hepatic glycogen stores. The greater the degree of prematurity, the less glycogen will be present.

Small for gestational age(SGA) premature infants are at extremely high risk for development of hypoglycaemia because available nutrients during intrauterine life are used for growth, with little set aside for glycogen storage. For this reason, SGA premature infants have extremely limited glycogen stores.

Perinatal stress:

Hypoxia, acidosis, and alterations in fetal blood pressure and flow can stimulate catecholamine secretion in utero, which in turn will mobilise hepatic glycogen stores. In addition, hypoxia increases the rate of anaerobic glycolysis, thereby accelerating glucose use. These events deplete fetal glycogen stores and place the infant at risk for hypoglycaemia after delivery.

Glycogen storage disorders:

These involve inherited defects in the glycogen metabolic pathway. Some of these like Ia, I, VI may be associated with hypogylcemia[20].

Limited Neoglucogenesis

Small for gestational age infants: In majority of SGA neonates, hypoglycemia is short lived and is caused due to inadequate glycogen stores, high brain: body mass ratio(with corresponding increase in glucose consumption), reduced fat stores. In about 1% infants, hypoglycaemia is prolonged and requires intravenous glucose therapy for several hours to days.

Factorswhich may account for this include a failure of counter regulation (including delayed maturation of gluconeogenesis) and hyperinsulinism[21,22]. These SGA neonates have elevated plasma concentrations of gluconeogenic precursors, suggesting an inability to convert exogenous gluconeogenic precursors, such as alanine, to glucose.

Increased glucose utilisation due to hyperinsulinism

A variety of disorders are associated with fetal and neonatal hyperinsulinism[24]. In some disorders, the mechanisms for heightened beta cell function are well understood, whereas in others, the pathogenesis unclear. In the former category are infants of diabetic mothers (IDM) and

infants with altered pancreatic islets caused by conditions such as nesidioblastosis and pancreatic adenoma.

Those for whom an etiology is not clear include infants with erythroblastosis and Beckwith Wiedemann syndrome. Infants who have erythroblastosisfetalis have increased levels of insulinand an increase in number of pancreatic beta cells. The mechanism for this development is unclear, but one possibility is that glutathione released from hemolysed red cells inactivates insulin in the circulation, which triggers more insulin secretion and upregulates the beta cells. The finding of a hypogylcemic indexwho is macrosomic and requires high rates of glucose infusion(10 to 20 mg/kg body weight per minute) suggests a hyperinsulinemic state.

Infant of diabetic mother(IDM):

IDM are at greater risk for development of hypoglycaemia as a result of the carryover of the fetal hyperinsulinemic state into neonatal life. They have elevated plasma insulin concentrations and release insulin briskly in response to glucose challenge.[24]

Malposition of umbilical artery catheter:

The tip of an umbilical artery catheter located at the level between the tenth thoracic and second lumbar vertebrae may result in glucose stimulated hyperinsulinism. Several infants have been reported in hypogylcemia was relived only the tip of the umbilical artery catheter was repositioned. It has been proposed that glucose from the malpositioned catheter flows into the celiac axis, thereby stimulating insulin secrtion.

Increased glucose utilisation without hyperinsulinism

Sepsis:

Sepsis in neonate is often heralded by hypoglycaemia or hyperglycemia. The mechanisms for this are not understood. Several studies have indicated rapid glucose disposal rates after intravenous challenge in septic term neonates. Although this suggests a hyperinsulinemic state, insulin secretion in these neonates was normal. These hyperglycemia and hypoglycemia that often precede the other signs of sepsis in premature infants may be catecholamine mediated. Hyperglycemia is a presentation of neonatal sepsis in some cases[25]; hypoglycemia with sepsis should be considered an indicator of fulminating infection. Transient hypoglycaemia which occurs in sepsis usually resolves in few days.

Hypothermia:

Hypothermia induced hypoglycaemia is due to rapid depletion of energy stores secondary to rapid heat production mediated by increased availability of catecholamines. Tissue use of glucose might also be increased under these conditions.

Polycythemia:

A negative correlation between plasma hematocrit and glucose concentration has been repeatedly reported. Though the exact pathogenesis is not known, it has been suggested that hyperviscosity possibly acts as an independent variable to depress plasma glucose concentration[26].

Congenital cardiac malformations:

Neonates with congenital cyanotic heart disease or congestive cardiac failure are noticed to have lower mean blood glucose concentration than that in healthy controls. Decreased glycogen stores secondary to chronic hypoxia may be a contributing factor, though the exact mechanism is unknown.

Neurohypoglycemia (Hypoglycorrhachia):

Glucose is transported across blood brain barrier via a carrier mediated diffusion process. At least five such membrane carrier proteins known as glucose transporters have been identified in various tissues. The transport protein that facilitates glucose transport across brain microvessels is termed as GLUT1 and has the same properties as the one transports glucose into red blood cells. Mutations of the genes coding for GLUT1 can lead to seizures due to low brain and cerebrospinal fluid glucose levels inspite of normal blood glucose levels.

PERSISTENT HYPOGLYCEMIA:

Most hypoglycaemia will resolve in 2-3 days. In cases of intractable hypoglycaemia requiring glucose infusions rates higher than 12mg/kg/min or requiring intravenous glucose for more than seven days, rare causes should be considered and investigated

This involves sending simulataneousSamples of blood for insulin and glucose estimation. An insulin : glucose ratio of >0.4 is suggestive of hyperinsulinemia. If insulin level is normal for blood glucose level then other causes of persistent hypoglycaemia should be considered and appropriate samples sent.

CAUSES OF PERSISTENT HYPOGLYCEMIA AND THEIR RELEVANT INVESTIGATIONS:

CAUSES	INVESTIGATIONS
Congenital hypopituitarism	serum growth hormone, cortisol
Adrenal insufficiency	serum cortisol
Hyperinsulinemic states	Insulin : glucagon ratio >0.4 Insulin level >0.6 C peptide level >0.2mmol/l Pro insulin level >5pmol/l
Galactosemia	presence of non glucose reducing substances in urine Measurement of galactose 1 phosphate uridyltransferase
Glycogen storage disorders	lactic acidosis Hyperuricemia

Maple syrup urine disease	Normal serum ammonia Positive urine for ketones Quantitative analysis of plasma amino acids (increasedleucine, isoleucine, valine)
Mitochondrial disorders	lactic acidosis

Symptoms of hypoglycemia

The clinical features of neonatal hypoglycaemia as in most neonatal disorders are non specific, so it is not possible to confidentially diagnose neonatal hypoglycaemia clinically. They are caused due to neuroglycopenia and may range from seizures to no symptom at all.

In addition careful attention should be given to underlying cause of hypoglycaemia, which may be suggested by characteristic physical features or other suggestive symptoms[27].

- Jitteriness
- Tremors
- Apnea
- Cyanosis
- Limpness/lethargy
- Seizures
- Abnormal crying
- Irritability
- Feeding difficulty

- Grunting/ tachypnea
- Hypothermia
- Hypotonia
- Tachycardia

It is unusual for a newborn with hypoglycaemia to have a classical autonomic nervous system response, sweating, pallor, tachycardia as occurs in adults. A low blood sugar detected by stick test should be checked by a laboratory blood assay for glucose.

Blood glucose screening

The high risk neonates who warrant screening of blood glucose have been highlighted in the following table

TABLE 2: HIGH RISK SITUATIONS REQUIRING BLOOD GLUCOSE ESTIMATION

- Preterm infants
- Small for gestational age infants (Birth weight <10^{th} percentile)
- Large for gestational age infants (Birth weight >90^{th} percentile)
- IDM
- Infants with Rh-hemolytic disease
- Infants born to mothers receiving terbutaline, propanolol, oral hypoglycaemic agents
- Infants with growth retardation (i.e three or more loose skin folds in gluteal region, decreased subcutaneous fat, and head to chest circumference difference >3cm)
- Sick neonate(Perinatal asphyxia, polycythemia, sepsis,shock)
- Infant receiving total parenteral nutrition

TABLE 3 : SCHEDULE OF BLOOD GLUCOSE MONITORING

Risk factor/Etiology	**Frequency**
Infant of diabetic mother	Cord blood, 1h,2h,3h,6h then 6 hourly for 48 hours
Preterms / Small for gestational age	1h,2h,3h,6h then 6 hourly for 72 hours
Sick babies/ babies on Intravenous fluids/ TPN	6 hourly individualised

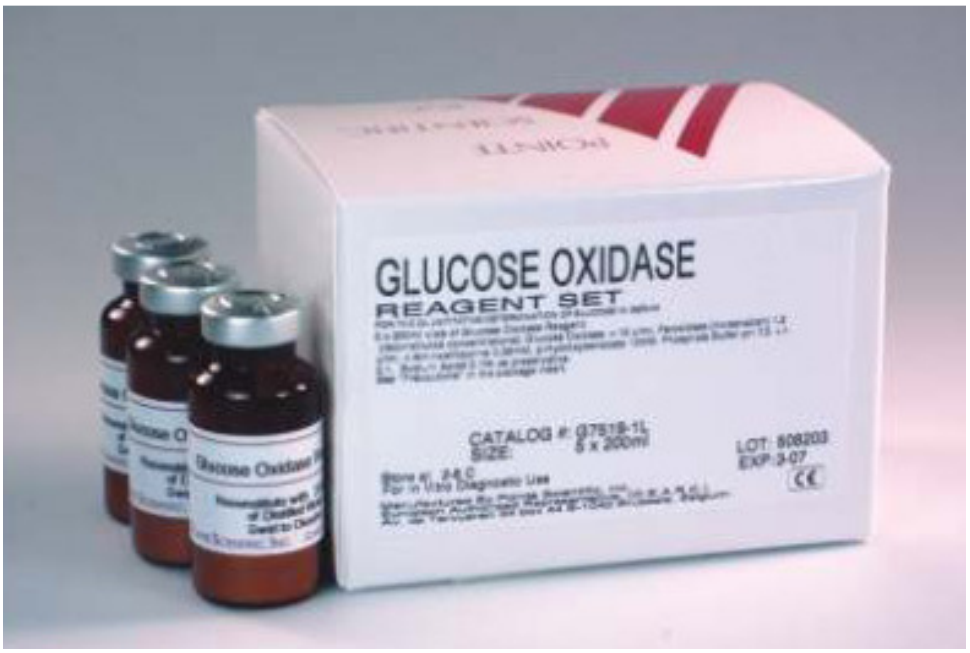
EVALUATION

Estimation of blood glucose

Laboratory diagnosis:

This is the most accurate method of estimation of glucose. The most frequently used method for glucose determination in the laboratory is an automated analysis technique with glucose oxidase or commercial glucose oxidase immobilised electrode[28]. In this method Glucose is oxidised to glucuronic acid & H_2O_2 which is catalysed by glucose oxidase. The resulting concentration of H_2O_2 is determined by Peroxidase or a coloured O2 acceptor [29].

FIGURE 2 :GLUCOSE OXIDASE PEROXIDASE METHOD



$$\text{Glucose} + 2H_2O + O_2 \xrightarrow{\text{Glucose Oxidase}} \text{Gluconic acid} + 2H_2O_2$$

Addition of the enzyme peroxidase and a chromogenic oxygen acceptor, such as *o*-dianisidine, results in the formation of a colored compound that can be measured:

$$\underset{\text{(Colorless)}}{\text{o-Dianisidine}} + H_2O_2 \xrightarrow{\text{Peroxidase}} \underset{\text{(Colored)}}{\text{Oxidized } o\text{-Dianisidine}} + H_2O_2$$

Plasma or serum glucose concentrations are determined, and the results are very accurate. It is also important to consider the differences in glucose content of plasma and blood.

Plasma glucose values are higher than those of whole blood by about 14%; the difference may be greater at very low glucose values (less than 30mg/dl). Whole blood glucose content also varies in accordance with the hematocrit. Neonatal red blood cells contain high concentrations of glycolytic intermediates such as reduced glutathione therefore whole blood must be deproteinized with zinc hydroxide before analysis.

Capillary blood samples should be collected from a warm heel. Also, the samples should be immediately tested or kept on ice because the rate of in vitro glycolysis is increased in red blood cells at room temperature, and the whole blood glucose values may drop 15 to 20mg/dl per hour if the sample is allowed to stand at room temperature.

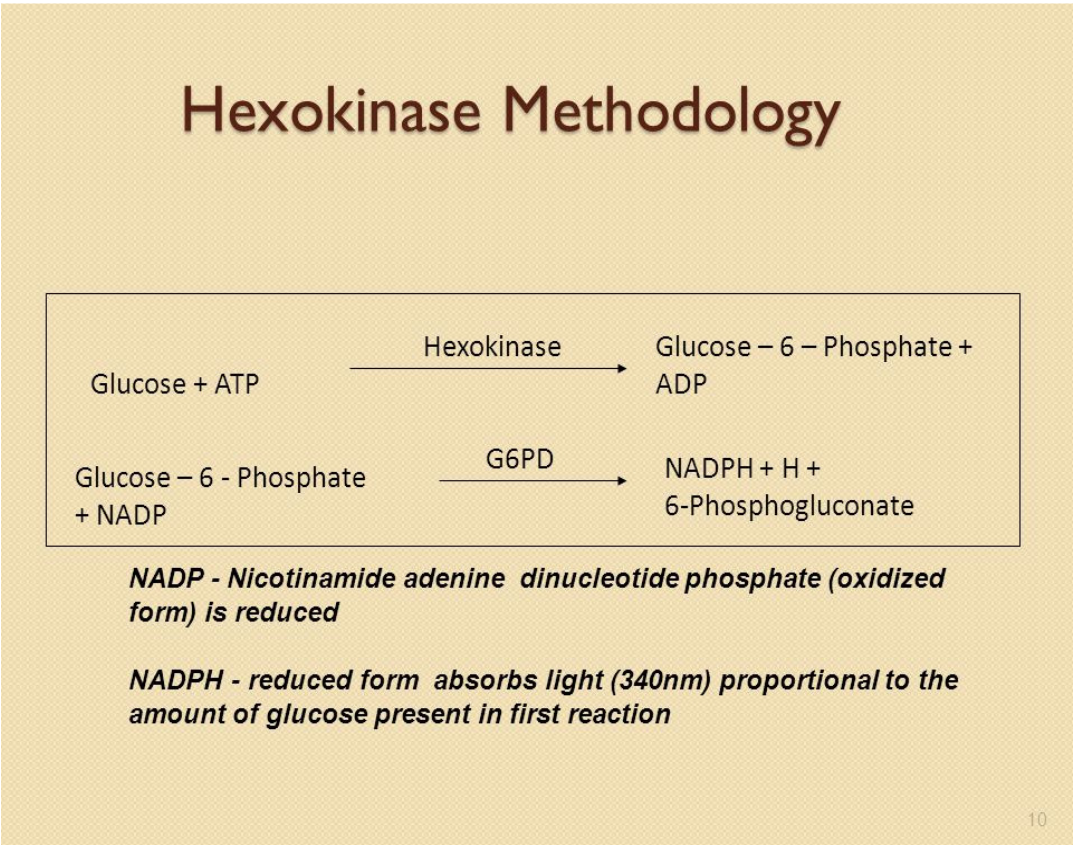
Reductiometric methods:

The principle of this traditional method depends on the glucose reducing property. eg: ferricyanide method. By this method, total reducing sugar concentration can be determined[5]. Reductiometric methods when compared with enzymatic methods, the detection of blood glucose by the latter is found to be accurate[2].

Hexokinase method

This method is more accurate and highly specific for blood glucose estimation

FIGURE 3 : HEXOKINASE METHOD



In this method, the NADPH/H+ yielded by the phosphorylation of glusose, which is catalysed by hexokinase is determined using suitable spectrophotometric analysis[30].

Reagent strips:

This involves whole blood glucose concentration estimation using glucose oxidase and peroxidase chromogen test strip either alone or with a reflectance colorimeter. Though widely use , they are unreliable especially at blood glucose values < 40 – 50 mg/dl[31-35].

They are useful for screening purposes and a low value obtained on them should always be confirmed by laboratory estimation. However a concurrent treatment should be instituted while awaiting a laboratory confirmation[36].

If the sample viscosity is higher it may interfere with the diffusion of the plasma into the strip, so in order to overcome this, heparinised micro hematocrit tube is used[37]. Some of the commercially available paper strips are dextrostix, Chemstrip, BM test Glycemia[38, 39, 40.]

Glucose electrode system:

This system uses electrode based analyser for measuring the level of glucose in the plasma using 25µL sample of uncentrifuged blood. This method is mostly used by nurses in clinical settings[41.]

The linear correlation between this method and laboratory glucose oxidase peroxidase methodis good between the concentration0 to 100 mg/dL. The main advantage of this method is that the assay is not affected by the sample hematocrit and bilirubin[42]. This method is costly compared with other reflectance meters.

Other bedside systems

Hemocue glucose photometer:

When compared to reagent strip or electrode methods, the cost is high in HemoCue. Room temperature variation and cuvette storage temperature will produce many errors, even though they are more useful when glucose concentration is higher.

A recent study in Nepal[43] showed that this method is not suitable for the detection of hypoglycaemia(<2mmol/L) since it over estimated the neonatal blood glucose concentrations.

FIGURE 4: HEMOCUE PHOTOMETER FOR GLUCOSE ESTIMATION

MANAGEMENT OF HYPOGLYCEMIA:

The occurrence of hypoglycaemia should prompt consideration of the cause. It is particularly important to note that term breastfed babies do not develop symptomatic hypoglycaemia as a result of simple underfeeding.

Presence of hypoglycaemia in this group is likely to be a manifestation of underlying illness, for example sepsis. Detection and treatment of the cause is as important as correction of the blood glucose concentration.

Healthy Asymptomatic Infants

It has been suggested that as many as 10 % of the healthy infants who are the appropriate size for gestational age develop transient asymptomatic hypoglycaemia, which in most cases is managed by the initiation of early feeding.

This type of hypoglycaemia is often transient and recovers spontaneously. However, in clinical practice it is often treated with early feedings. Controlled studies examining the benefits or impact of this early feeding on recovery from hypoglycaemia have not been performed.

Direct breast feeding is the best option among early feed initiation. If the baby is unable to suck, expressed breast milk may be used. If breast milk is not available, then formula milk may be given. Such a supplementation may be tried in infants for whom blood glucose is 20 – 40 mg/dl and who are otherwise asymptomatic.

SYMPTOMATIC INFANTS / VERY LOW GLUCOSE CONCNETRATION IN ASYMPTOMATIC INFANTS

Symptomatic babies should be treated with parenteral glucose infusion. Also, the infants who have blood glucose concentration <20mg/dl should be managed with intravenous glucose. These infants should be administered a mini bolus of 200mg/kg glucose as 2ml/kg of 10% dextrose over 1 minute. This should be immediately followed by an infusion of 10% dextrose at a rate of 6mg/kg/min.

Subsequently, blood glucose should be checked after 30 minutes, and if it has normalised (>50mg%), then infusion should be continued at the same rate and blood glucose monitored 6^{th} hourly. If the blood glucose continues to be low, increase infusion rate by 2mg/kg/min every 30 minutes to a maximum of 12mg/kg/min.

If two or more blood glucose values are >50mg/dl after 24 hours of parenteral glucose and the primary underlying condition has been taken care of, and there is no contraindication to enteral feeds, the baby should be initiated on oral feeds. Simultaneously, the glucose infusion can be tapered by 2mg/kg/min every 6 hours. Once the infusion rate is 4mg/kg/min, and oral intake is adequate, the parenteral glucose infusion can be stopped.

The intravenous infusion of the glucose should never be stopped abruptly; it leads to severe rebound hypoglycaemia. Also, infusion of glucose concentration >12.5% in a peripheral vein may lead to thrombophlebitis and hence a central venous catheter should be inserted if glucose concentration >12.5% is required. In addition, while increasing the glucose infusion rate one should also ensure that the baby does not go into fluid overload.

MANAGEMENT OF PERSISTENT HYPOGLYCEMIA

In cases of intractable hypoglycaemia, after sending the requisite investigations, central venous line maybe inserted to enable infusion of higher concentration and higher rates of glucose. Besides increase in glucose infusion, following drugs may also be tried in refractory cases.

- **HYDROCORTISONE:**

5 – 15 mg/kg/day intravenously in two divided doses. It is most useful in hypoglycaemia due to adrenal insufficiency and rarely used nowadays for other indications.

- **DIAZOXIDE**

10 – 25mg/kg/day per orally in three divided doses for treatment of hypoglycaemia due to hyperinsulinism. It acts by reducing insulin secretion and increasing catecholamine release. It can be used for prolonged periods without any significant side effects.

- **GLUCAGON**

30 -300 mcg/kg/dose intravenous, intramuscular or subcutaneously. It can be used to increase blood glucose levels in acute situations before intravenous glucose infusion can be started. It acts by increasing both glycogenolysis and gluconeogenesis. However the effect is short lasting and glucose infusion is required to maintain blood glucose levels for a longer period. It should not be used in SGA infants who are normally glycogen depleted.

- **OCTREOTIDE**

5-10mcg/kg subcutaneously, Every 6 to 8 hourly. It suppresses the secretion of various hormones including insulin.

MATERIALS AND METHODS

STUDY DESIGN

This is an analytical cross sectional study .

STUDY SETTING

Neonatal intensive care unit, Government Rajah Mirasdar Hospital, Thanjavur Medical College,Thanjavur.

STUDY POPULATION

200 Newborns admitted in NICU,Government Rajah Mirasdar Hospital, Thanjavur Medical College during the period of 6 months from January 2016 to July 2016.

INCLUSION CRITERIA :

- Neonates admitted in Neonatal Intensive Care Unit Level 3, Department of Paediatrics, Thanjavur Medical College Thanjavur.

EXCLUSION CRITERIA :

- Infants >28 days.
- Neonates with PCV less than 40% and more than 65%

SAMPLING PROCEDURE

Consecutive sampling

SAMPLE SIZE

200 neonates admitted in Neonatal Intensive Care unit, Government Rajah Mirasdar Hospital during the period of 6 months from January 2016 to July 2016

TOOLS

1. Glucose oxidase peroxidase method

Principle:

The β D glucose present in the plasma is acted upon by the glucose oxidase enzyme to produce hydrogen peroxide. This in turn is acted upon by peroxidase enzyme to produce water and oxygen. This oxygen reacts with oxygen acceptor like ortho toluidine to produce a coloured product that is estimated by calorimeters.

2. Glucometer(ACCU CHECK ACTIVE/Sensor glucometer)

Meter type - ACCU-CHEK Active Meter Measuring range- 10-600 mg/dL Sample size - 1µL Measuring time - Approximately 5 seconds

Samples: venous, arterial, finger stick capillary.

Principle:

The test strip containing glucose dehydrogenase catalyzes a selective electron-transfer reaction between glucose in the sample and potassium ferricyanide in the reagent layer. Each molecule of glucose reduces two molecules of ferricyanide, creating two molecules of ferrocyanide.The final ferrocyanide concentration is directly correlated to the sample glucose concentration.

Procedure:

Hold the test strip so the arrows printed on it and the orange square faces upwards and push it into the machine. After heel prick with lancet, apply a drop of blood to the centre of the orange field. An hourglass indicates the test is in progress. The test is complete after approximately 5 seconds and results appears on the display.

DATA COLLECTION PROCEDURE

Antenatal History:

History regarding regular antenatal checkups was enquired. History of medical illness like diabetes, fever during first trimester or third trimester was asked. History of obstetric complications like PIH, eclampsia, antepartum hemorrhage, oligo or polyhydramnios was noted.

Perinatal History:

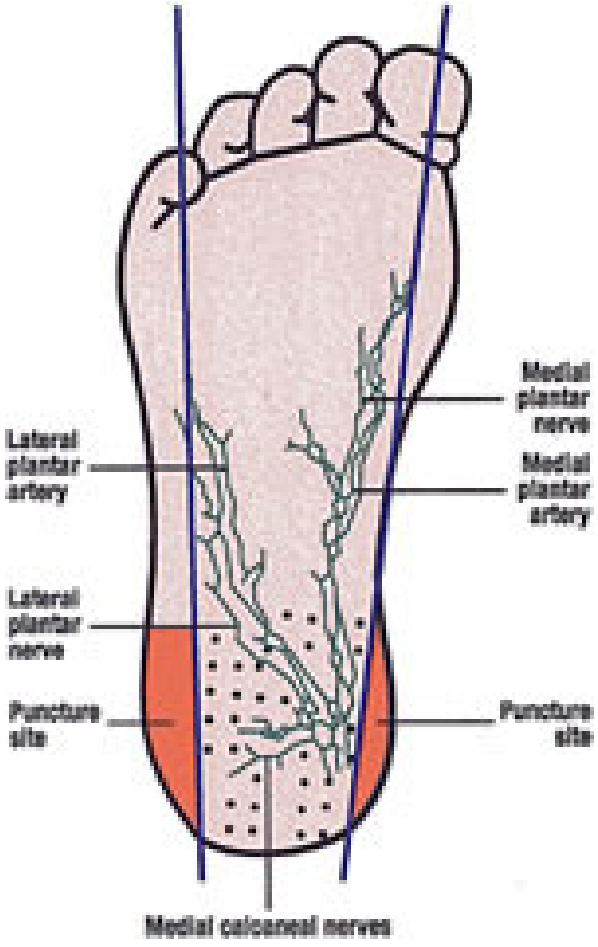
History of Premature rupture of membranes, prolonged second stage of labour, Meconium staining of liquor, place of delivery, type of delivery and indication for forceps and caesarean section was noted. After delivery whether baby cried immediately or not, was it meconium stained and any resuscitation done was noted. Apgar score at 1minute and 5minutes of birth was documented in inborns and if mentioned in the referral was noted in the outborns.

Sample collection

After washing hands,the infant's heel is held with a moderately firm grip. The forefinger is placed at the arch of the foot and the thumb below the puncture site at the ankle.After selection of the puncture site, (the ideal site being lateral/medial edges of the plantar surface-to avoid damage to calcaneus),the area is cleaned with warm water & gauze.

The sites punctured using a lancet with a quick controlled stroke.The first drop of blood is wiped off with a cotton ball or gauze and discarded as it may be contaminated with skin cells, alcohol or excess tissue fluid, which may distort test results. The heel is held in a dependent position and gentle pressure applied to facilitate blood flow. Excessive squeezing of the heel may cause haemolysis or contamination of the specimen from interstitial

fluid leakage & bruising.Gentle pressure with a cotton ball or gauze is applied,once adequate blood volume has been collected,until bleeding stops.



Then 2 ml of blood drawn from the peripheral vein was used for estimating the glucose levelin the laboratory by glucose oxidase method.Blood from the sample was used to estimate the venous blood glucose level by glucometer.Complete blood count was also done for PCV estimation with the sample collected from peripheral vein.

Examination

The venous blood glucose was estimated by the glucometer and in the laboratory by the glucose oxidase peroxidase method. Estimation of the capillary blood glucose level by glucometer was also done. Values obtained by laboratory glucose oxidase method were taken as the gold standard. The samples were collected in the newborn babies who were admitted in NICU level 3.Pearson correlation was used for the statistical analysis.

INVESTIGATIONS

1. Blood glucose level using glucometer Accu-check (venous sample)
2. Blood glucose level using glucose oxidase peroxidase method in the laboratory (venous sample)
3. Blood glucose level using glucometer (capillary sample by heel prick method)
4. Complete blood count.(For PCV estimation)

DATA ANALYSIS

Analysis of Cases and Results:

200 neonates who were admitted to NICU level 3, RAJAH MIRASDAR HOSPITAL, THANJAVUR MEDICAL COLLEGE, THANJAVUR, during the period of 6 months from January 2016 to July 2016.

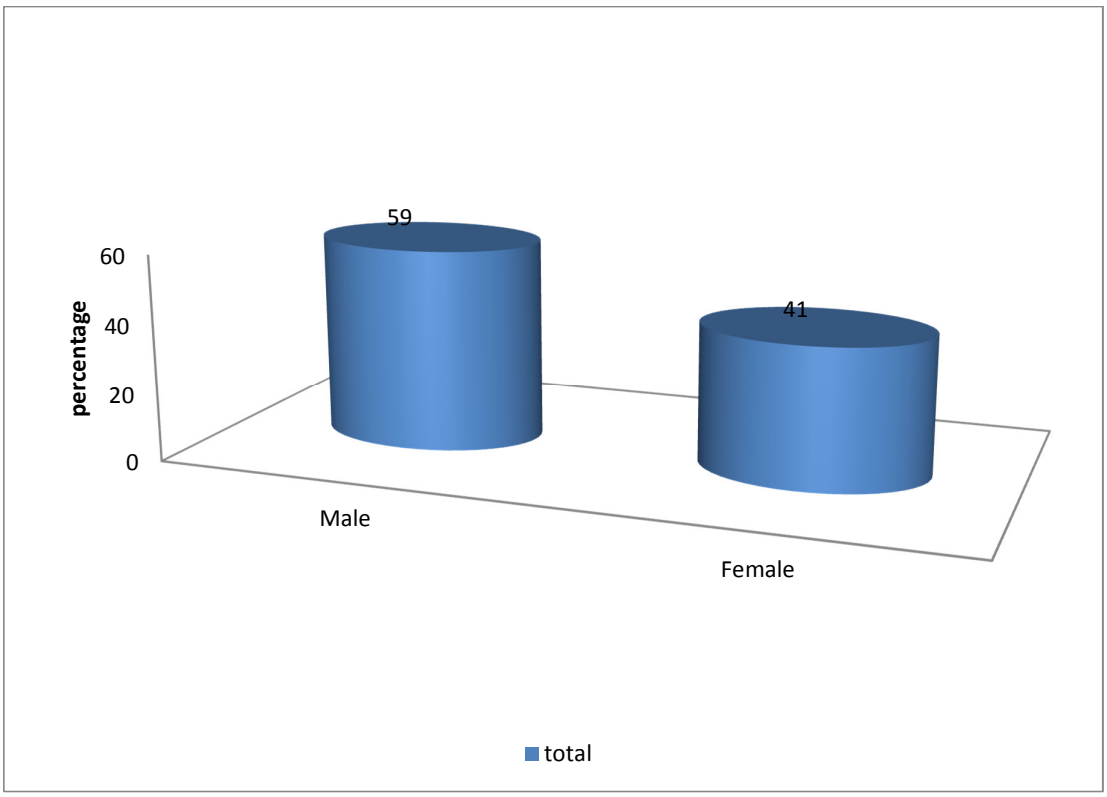
Sex Distribution:

TABLE 4: DISTRIBUTION OF CASES ACCORDING TO SEX

Sex	No.of cases (n=200)	Percentage (100%)
Male	118	59.0
Female	82	41.0

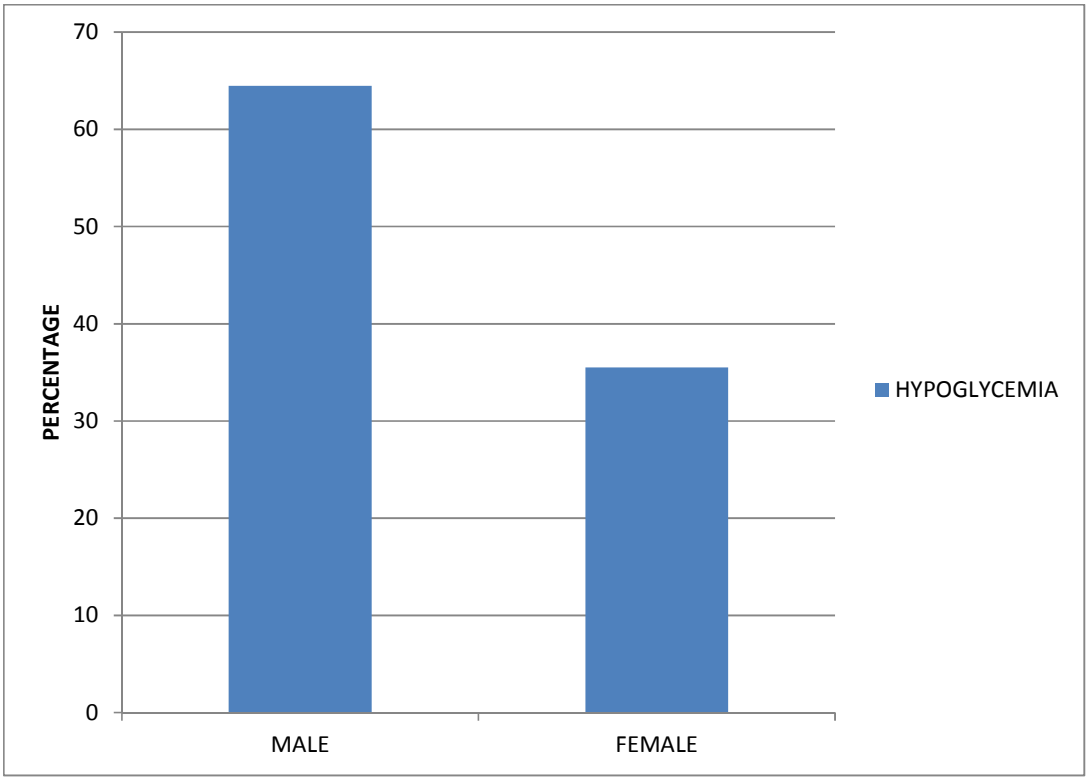
In our study, 118 (59%)cases were male babies and 82(41%) cases were female babies with the male to female ratio of 1.4:1.

FIGURE 5: DISTRIBUTION OF CASES ACCORDING TO SEX



Out of 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 20(64.5%) were males and 11(35.5%) were female neonates.

FIGURE 6 :DISTRIBUTION OF HYPOGLYCEMIA CASES ACCORDING TO SEX



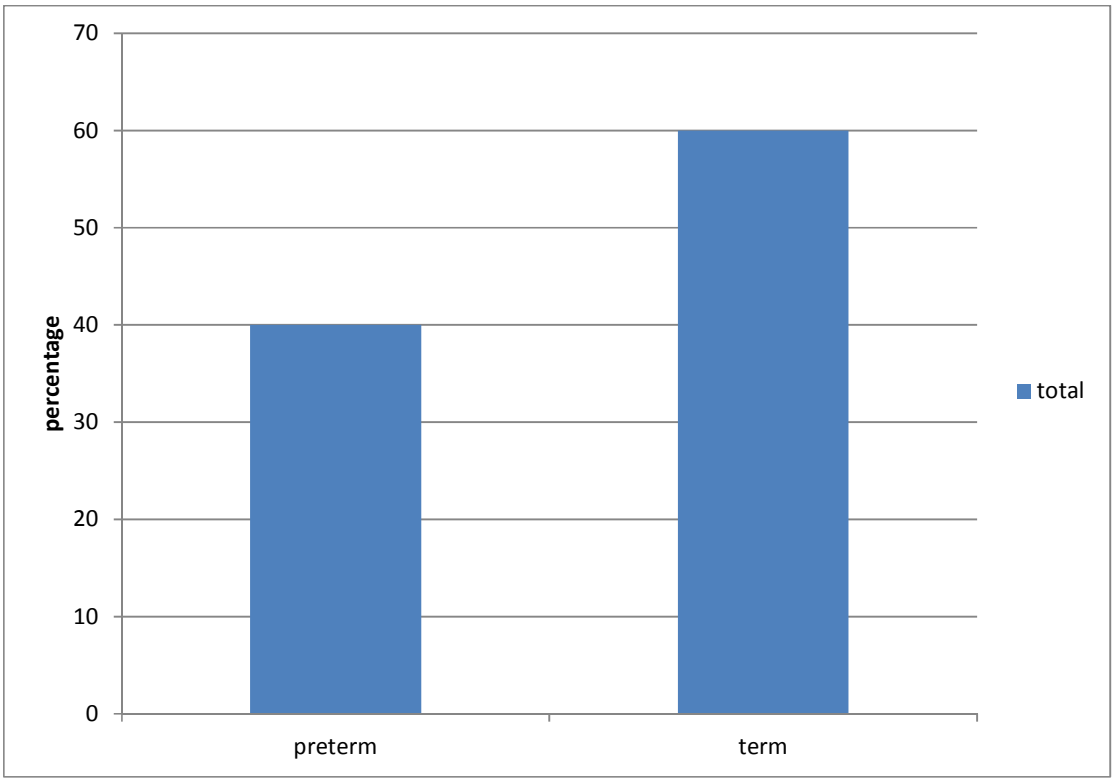
GESTATIONAL AGE:

TABLE 5:DISTRIBUTION OF CASES IN RELATION TO GESTATIONAL AGE

Gesttional age	**No.of cases (n=200)**	**Percentage (100%)**
Preterm	80	40.0
Term	120	60.0

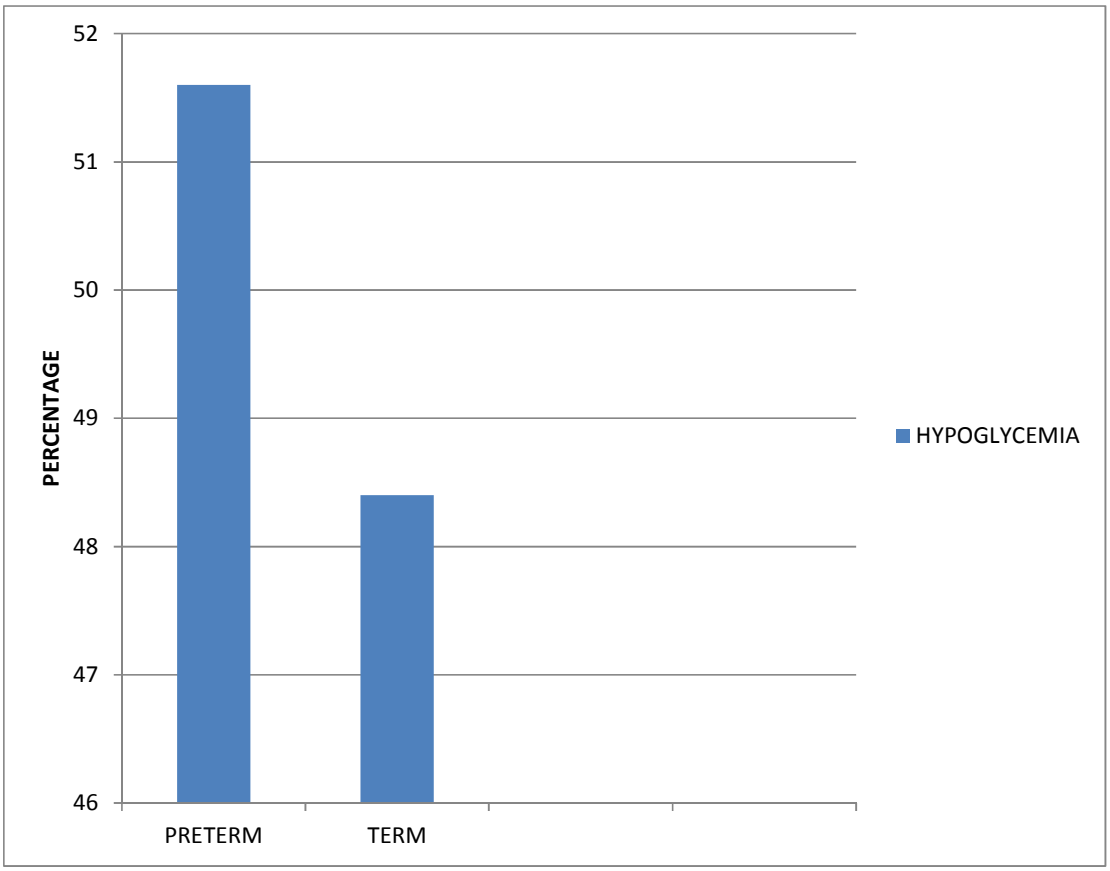
In the present study of the 200 babies, 120 were full term (60%) and 80((40%) were preterm neonates.

FIGURE 7: DISTRIBUTION OF CASES IN RELATION TO GESTATIONAL AGE



Of the 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 16 (51.6%) were preterm and 15(48.4%) were term neonates.

FIGURE 8: DISTRIBUTION OF HYPOGLYCEMIA CASES IN RELATION TO GESTATIONAL AGE



PLACE OF DELIVERY:

TABLE 6: DISTRIBUTION OF CASES IN RELATION TO PLACE OF DELIVERY

Place of delivery	**No.of cases (n=200)**	**Percentage (100%)**
Outborn	61	30.5
inborn	139	69.5

Of the 200 cases studied, 61(30.5%) were outborn and 139 (69.5%) were inborn.

FIGURE 9: DISTRIBUTION OF CASES IN RELATION TO PLACE OF DELIVERY

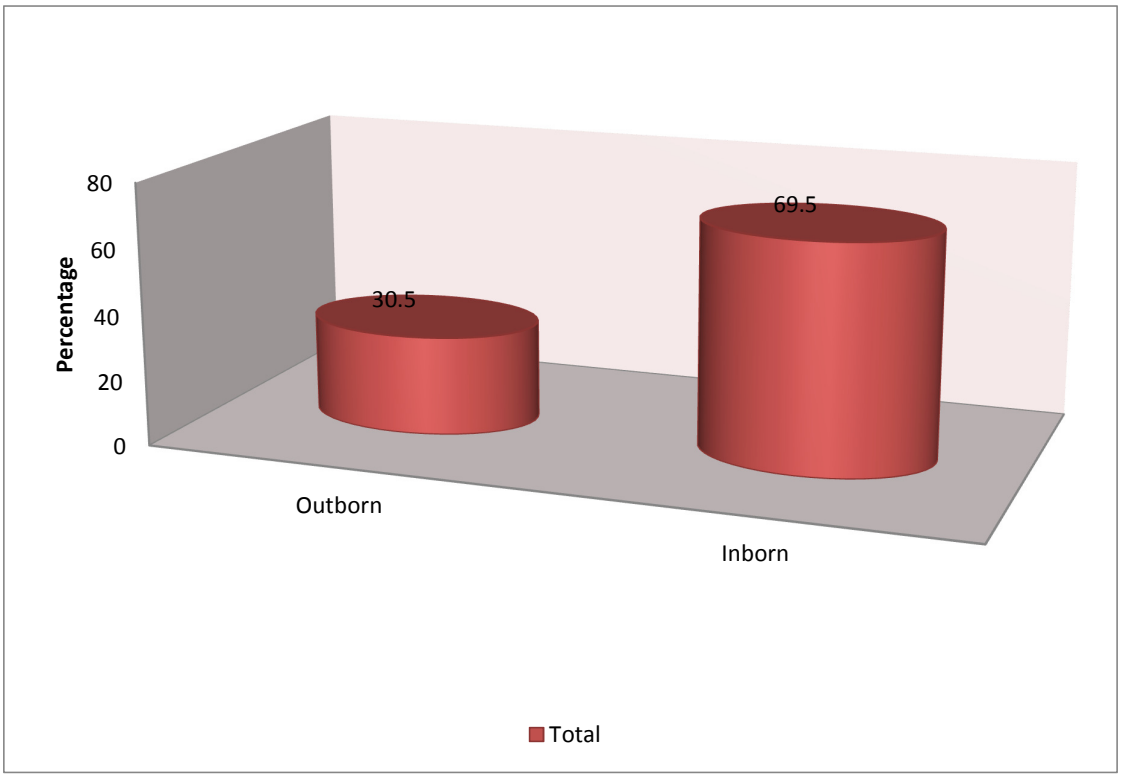
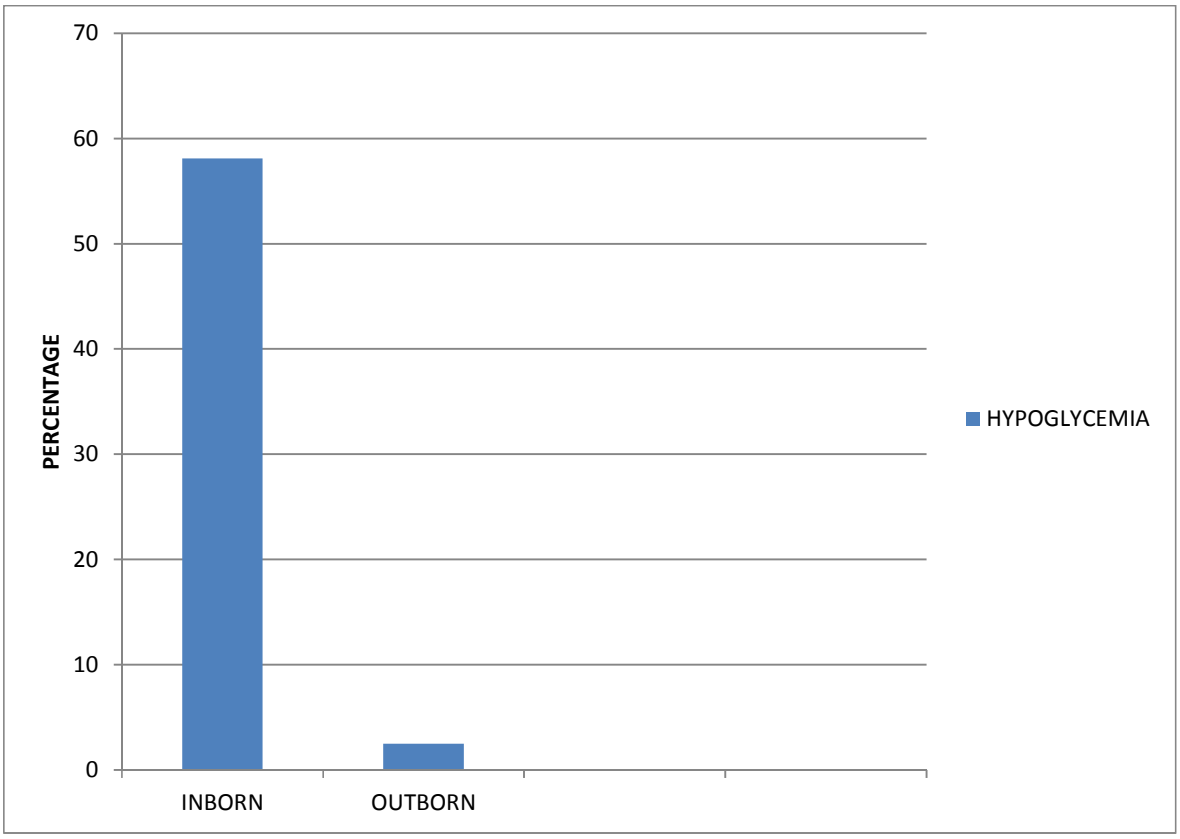


FIGURE 10: DISTRIBUTION OF HYPOGLYCEMIA CASES IN RELATION TO PLACE OF DELIVERY



Of the 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 13(41.9%) were outborn, and 18(58.1%) were inborn neonates.

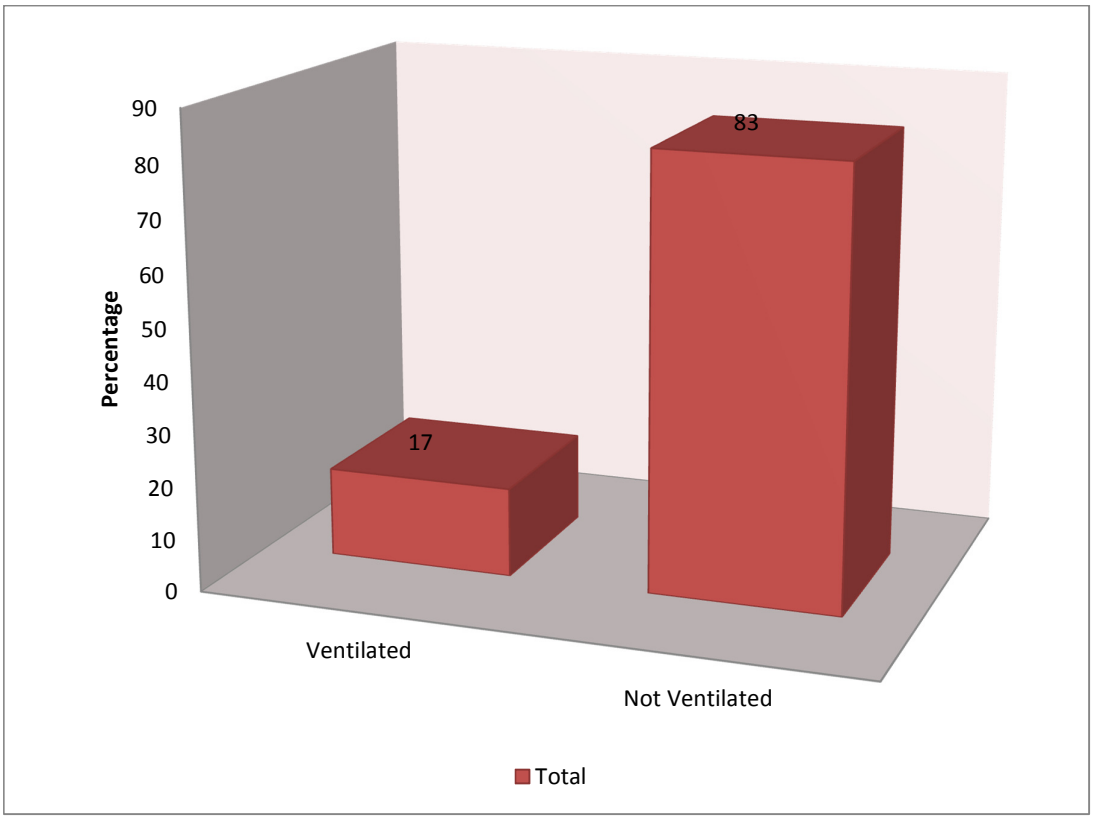
MECHANICAL VENTILATION:

TABLE 7: DISTRIBUTION OF CASES IN RELATION TO MECHANICAL VENTILATION

Mechanical ventilation	**No.of Cases (n=200)**	**Percentage (100%)**
Yes	34	17.0
No	166	83.0

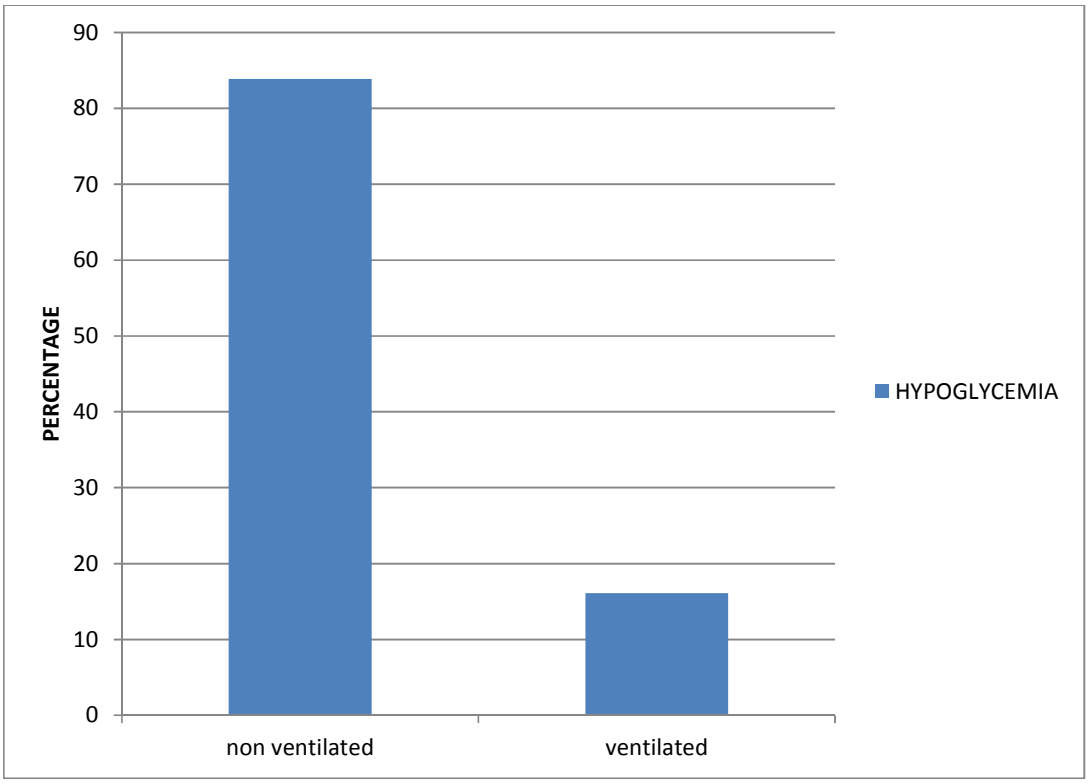
In our study of the 200 cases,34 cases(17%) were mechanically ventilated and 166 cases(83%) did not require mechanical ventilation.

FIGURE 11: DISTRIBUTION OF CASES IN RELATION TO MECHANICAL VENTILATION



Of the 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 26(83.9%) cases were not ventilated and 5(16.1%) were ventilated.

FIGURE 12: DISTRIBUTION OF HYPOGLYCEMIA IN VENTILATED AND NON VENTILATED CASES



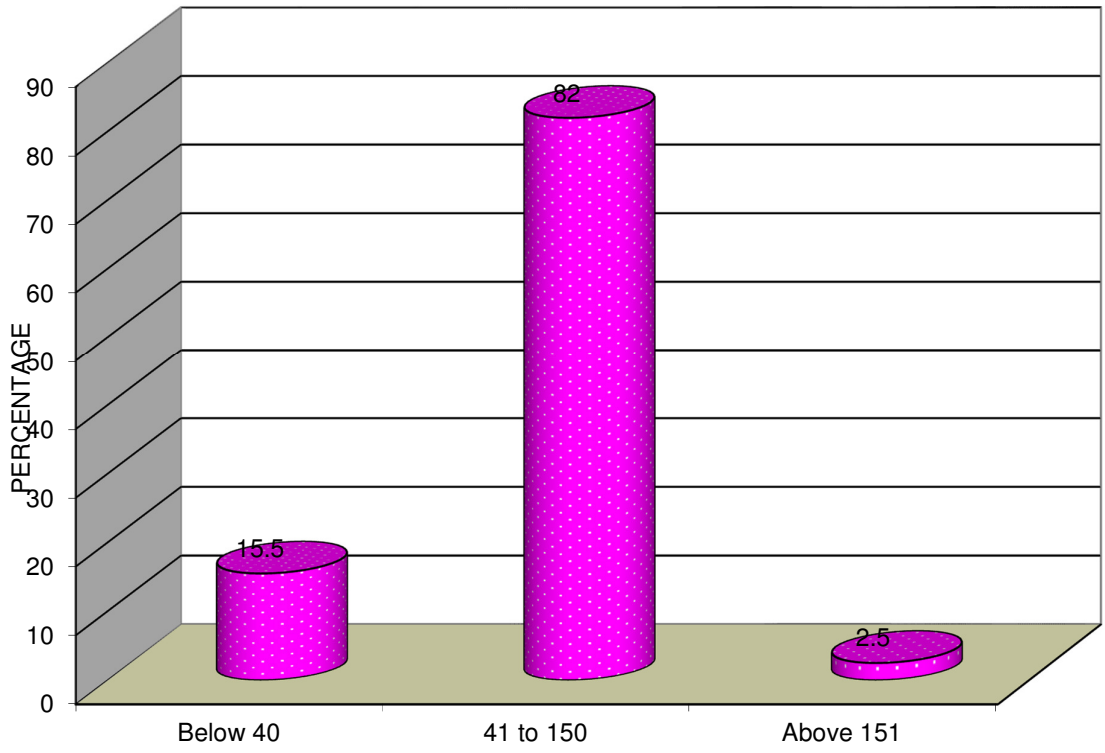
GLUCOSE OXIDASE METHOD

TABLE 8: DISTRIBUTION OF CASES ACCORDING TOLAB VALUES BY GLUCOSE OXIDASE PEROXIDASE METHOD

Lab values (mg/dl)	**No. of cases (n=200)**	**Percentage (100%)**
Below 40	31	15.5
41 to 150	164	82.0
Above 151	5	2.5

In our study,of the 200 cases,blood glucose values were less than or equal to 40mg/dl in 31 cases (15.5%), between 41-150mg/dl in 164 (82%) cases and above 151mg/dl in 5 cases(2.5%) by glucose oxidase peroxidase method.

FIGURE 13: DISTRIBUTION OF CASES ACCORDING TOLAB VALUES BY GLUCOSE OXIDASE PEROXIDASE METHOD



Glucose oxidase peroxidase method was taken as gold standard in this study.

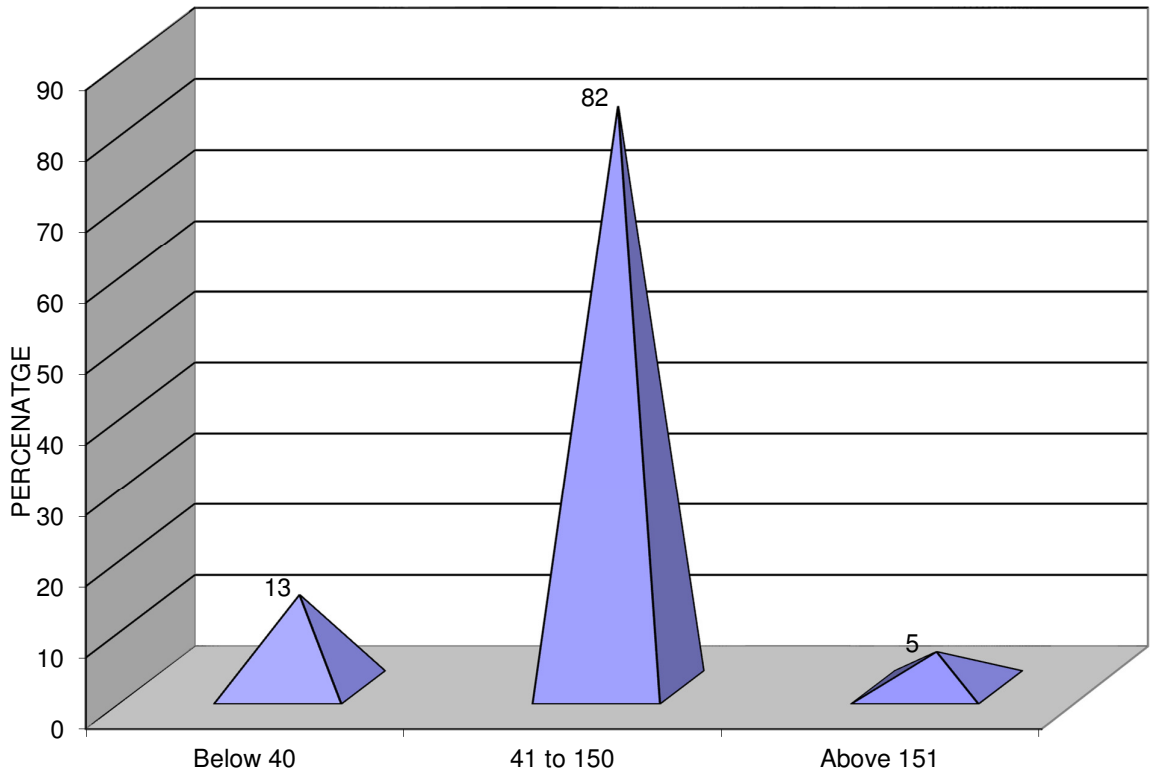
CAPILLARY ESTIMATION OF BLOOD GLUCOSE BYGLUCOMETER

In our study, of the 200 cases,26 cases (13%) had blood glucose values less than or equal to 40mg/dl, 164 (82%) had blood glucose values of 41-150mg/dl.10 cases (5%) had blood glucose values above 151mg/dl, when capillary blood glucose was estimated using glucometer. Of the 31 cases of hypoglycaemia detected by laboratory method, 23 cases of hypoglycaemia (less than or equal to 40mg/dl) were detected by capillary blood glucose method.

TABLE 9: DISTRIBUTION OF CASES ACCORDING TO CAPILLARY BLOOD GLUCOSE BY GLUCOMETER

Values (mg/dl)	No.ofcases (n=200)	Percentage (100%)
Below 40	26	13.0
41 to 150	164	82.0
Above 151	10	5.0

FIGURE 14: DISTRIBUTION OF CASES ACCORDING TO CAPILLARY BLOOD GLUCOSE BY GLUCOMETER



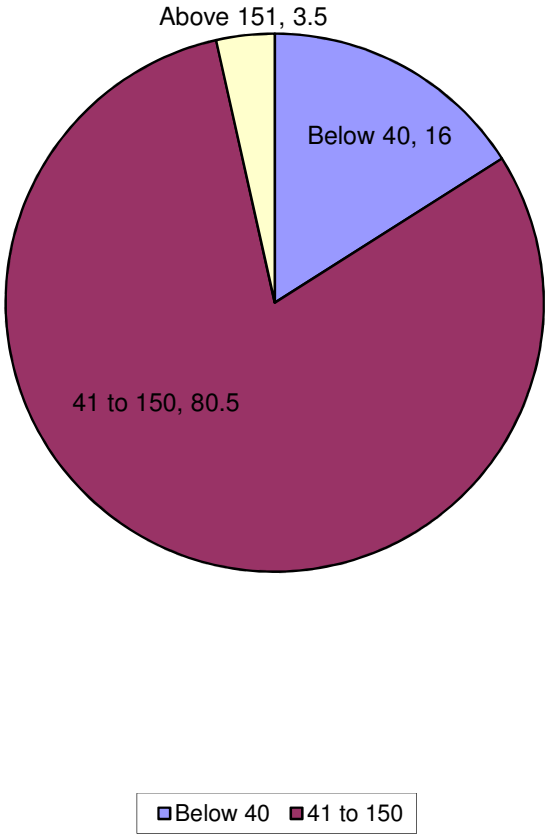
VENOUS BLOOD GLUCOSE ESTIMATION BY GLUCOMETER

In our study, of the 200 cases, 32 cases (16%) had blood glucose values less than or equal to 40mg/dl ,161 (80.5%) had blood glucose values of 41-150mg/dl. 7 cases(3.5%) had blood glucose values above 151mg/dl, when venous blood glucose was estimated using glucometer.

TABLE 10: DISTRIBUTION OF CASES ACCORDING TO VENOUS BLOOD GLUCOSE BY GLUCOMETER

Values(mg/dl)	**No. ofcases (n=200)**	**Percentage (100%)**
Below 40	32	16.0
41 to 150	161	80.5
Above 151	7	3.5

FIGURE 15:DISTRIBUTION OF CASES ACCORDING TO VENOUS BLOOD GLUCOSE BY GLUCOMETER



Of the 31 cases of hypoglycaemia detected by lab method, 29 cases of hypoglycaemia (less than or equal to 40mg/dl) were detected by venous blood glucose method.

TABLE 11: DIAGNOSTIC VALUE OF CAPILLARY BLOOD GLUCOSE ESTIMATION BY GLUCOMETER IN COMPARISION TO LAB VALUE IN DETECTING THE HYPOGLYCEMIA

VALUE	PERCENTAGE
True Positive (a)	**23**
True Negative (d)	**166**
False Positive (b)	**3**
False Negative (c)	**8**
Sensitivity	**74.19**
Specificity	**98.2**
PPV	**88.4**
NPV	**95.4**
ACCURACY	**94.5**

Sensitivity = a/a+c = 74.19%

Specificity = d / b+d = 98.2%

PPV = a / a+b = 88.46%

NPV = d / c+d = 95.4%

Accuracy = a+d /a+b+c+d = 94.5

Capillary blood glucose monitored by glucometer has sensitivity of 74.19% and specificity of 98.2% , positive predictive value of 88.4% , negative predictive value of 95.4% and accuracy of 94.5% with statistically a significant P value< 0.05

TABLE 12: DIAGNOSTIC VALUE OF VENOUS BLOOD GLUCOSE ESTIMATION BY GLUCOMETER IN COMPARISION TO LAB VALUE IN DETECTING THE HYPOGLYCEMIA

VALUE	PERCENTAGE
True Positive (a)	29
True Negative(d)	166
False Positive(b)	3
False Negative(c)	2
Sensitivity	93.55
Specificity	98.23
PPV	90.62
NPV	98.8
ACCURACY	97.5%

Sensitivity = a/a+c = 93.55%

Specificity = d / b+d = 98.23%

PPV = a / a+b = 90.62%

NPV = d / c+d = 98.8%

Accuracy = a+d / a+b+c+d = 97.5%

Venous blood glucose monitored by glucometer has sensitivity of 93.55% and specificity of 98.23% , positive predictive value of 90.62% , negative predictive value of 98.8% and accuracy of 97.5% with statistically a significant P value < 0.05.

EXPECTED OUTCOMES

DISCUSSION

In the present study, 200 neonates admitted in level 3 NICU were studied for a period of 6 months. Both intramurally and extramurally delivered babies were included in this study.

In our study, we evaluated the efficacy of glucometer as a good screening tool in estimating the blood glucose level in newborn (both capillary and venous) in comparison with laboratory glucose oxidase peroxidase method. The study was also used to determine the sensitivity, specificity, predictive value and accuracy of glucometer compared to glucose oxidase peroxidase method.

Laboratory glucose oxidase peroxidase method of blood glucose estimation was taken as gold standard.

In this prospective study, 200 neonates were studied with varied symptomatology. Of these, 59% of the cases were male babies, 41% of the cases were female babies.Of the 200 babies, 40% babies were born preterm and 60% babies were term.

Hypoglycaemia was defined as blood glucose level less than 40 mg/dl[3].

In this study, out of 200 babies, 31 babies (15.5%) were found to be hypoglycaemic by laboratory glucose oxidase peroxidase method which is comparable with previous studies by PK SINGHAL et al[44]& PK MISHRA & BINA SHARMA [45]where the incidencewas 4.8% & 9.7% in their studies respectively.

This basal variation in the incidence of hypoglycaemia could be attributed to the lack of uniform definition of hypoglycaemia, variable sample size and multiple risk factors.

In the PK SINGHAL et al[44] study hypoglycaemia was defined as blood glucose level less than 30mg% while in the PK MISHRA AND BINA SHARMA[45] study it was taken as 20mg%

COMPARISON OF OVERALL INCIDENCE OF HYPOGLYCEMIA WITH OTHER STUDIES

STUDIES	INCIDENCE
PK SINGHAL et al[44]	4.8%
PK MISHRA & BINA SHARMA[45]	9.7%
OUR STUDY	15.5%

Hypoglycaemia detected by capillary blood glucose and venous blood glucose using glucometer was 23 cases (11.5%) and 29 cases (14.5%) respectively which was in concordance with laboratory oxidase peroxidase method which detected 31 cases(15.5%) of hypoglycaemia in our study population. This is comparable with previous study HAMID mhet Al[46] which detected hypoglycaemia by laboratory method in 11% and by capillary blood glucose method in 11% cases.

In our study, euglycaemia detection by glucose oxidase peroxidase method were 164 cases (82%) while glucometer detected euglycaemia by Capillary blood glucose method in 156 cases(78%) and by venous blood glucose method in 158 cases(79%) which is in concordance with glucose oxidase method.

Capillary blood glucose estimated by glucometer has sensitivity of 74.19% and specificity of 98.2% , positive predictive value of 88.4% , negative predictive value of 95.4% and accuracy of 94.5% and a statistically significantP value < 0.05.

COMPARISON OF SENSITIVITY AND SPECIFICITY OF THE ESTIMATION OF CAPILLARY BLOOD GLUCOSE BY GLUCOMETER WITH OTHER STUDIES

STUDIES	SENSITIVITY	SPECIFICITY
Dahlberg et al [47]	100%	84%
Mehta et al[48]	86%	89%
H O Ht et al [49]	92.3%	
M Ellis et al [50]	83%	62%
Hamid MH et al[46]	98%	63%
Our study	74.19%	98.2%

Venous blood glucose monitored by glucometer has sensitivity of 93.55% and specificity of 98.23%, positive predictive value of 90.62% , negative predictive value of 98.8% and accuracy of 97.5% with a statistically significant P value < 0.05.

These figures indicate that measuring Capillary blood glucose and venous blood glucose using glucometer is a reliable tool to diagnose neonatal hypoglycaemia. Further blood glucose estimated by glucometer using venous bloodwas more sensitive than capillary blood.

Moreover, capillary blood glucose monitoring by glucometer has the added advantages as follows:

Quick performance within few minutes

Least required preparation

No risk for the patient

Cheaper and cost effective.

Less blood to be drawn

Less trauma to baby

SUMMARY

- In our study, of the 200 neonates118 (59%) cases were male and 82(41%) cases were female with the male to female ratio of 1.4:1. Out of 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 20(64.5%) were males and 11(35.5%) were female neonates.

- In the present study of the 200 babies, 120were term (60%) and 80(40%) were preterm.Of the 31 cases of hypoglycaemiadetected by glucose oxidase peroxidase method in the laboratory using venous blood 16 (51.6%) were preterm and 15(48.4%) were term neonates.

- Of the 200 cases studied, 61(30.5%) were outborn and 139 (69.5%) were inborn.Of the 31 cases of hypoglycaemia detected by glucose oxidase peroxidase method in the laboratory using venous blood, 13(41.9%) were outborn, and 18(58.1%) were inborn neonates.

- In our study, of the 200 cases, 34 cases (17%) were mechanically ventilated and 166 cases (83%) did not require mechanical ventilation.Of the 31 cases of hypoglycaemia detected by glucose

oxidase peroxidase method in the laboratory using venous blood, 26(83.9%) cases were not ventilated and 5(16.1%) were ventilated**.**

- In our study, of the 200 cases ,31 cases (15.5%) were hypoglycaemic, 164(82%)cases were euglycemic and 5 cases (2.5%) were hyperglycemic by laboratory glucose oxidase peroxidase method, which is taken as the gold standard.

- Capillary Blood glucose estimation using glucometer detected 23 cases (74.2%) of hypoglycaemia, 156 cases (95.1%) of euglycemiaand 5 cases (100%) of hyperglycemia in comparison with laboratory glucose oxidase peroxidase method.

- Venous blood glucose estimation using glucometer detected 29 cases (93.5%) of hypoglycaemic, 158 cases (96.3%) of euglycemia and 3 cases (60%) of hyperglycemia in comparison with laboratory glucose oxidase peroxidase method.

- Estimation of capillary blood glucose by glucometer was found to have a sensitivity of 74.19% , specificity of 98.2 % , positive predictive value of 88.4% , negative predictive value of 95.4% and accuracy of 94.5% with statistically significant P value < 0.05

- Estimation of venous blood glucose using glucometer was found to have a sensitivity of 93.55% , specificity of 98.23 % , positive predictive value of 90.62% negative predictive value of 98.8% and accuracy of 97.5% with statistically significant P value < 0.05.

CONCLUSION

1. Estimation of blood glucose using capillary and venous blood using glucometer have strong correlation with laboratory oxidase peroxidase method in detecting neonatal hypoglycaemia.

2. The sensitivity of detecting neonatal hypoglycemia by glucometer using venous blood is higher than capillary blood.

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PROFORMA

BABY OF :

DOA :

IP NO. :

BIRTH WEIGHT :

GESTATIONAL AGE :

DAY OF LIFE :

SEX :

MODE OF DELIVERY :

DATE AND TIME OF BIRTH :

PLACE OF BIRTH :

MATERNAL COMPLICATIONS :

APGAR SCORE AT 1ST MINUTE :

5TH MINUTE :

INTRAMURAL/EXTRAMURAL :

CRIED SOON AFTER BIRTH/NOT :

INDICATION FOR NICU ADMISSION :

ON MECHANICAL VENTILATION/NOT:

INVESTIGATIONS:

BLOOD GLUCOSE LEVEL BY LABORATORY GLUCOSE OXIDASE METHOD	**GLUCOMETER (ACCU-CHECK ACTIVE) VENOUS SAMPLING**	**GLUCOMETER (ACCU-CHECK ACTIVE) CAPILLARY SAMPLING**

S.NO	IP NO	B WT (kg)	G.A	SEX	D.O.L	M.O.D	P.O.B	D.O.B	MAT COMP	APGAR	IM/EM	CR AFT BIRTH	IND NICU ADM	M.V	CBG (mg/dl)	VBG(mg/dl)	LAB (mg/dl)
1	427653	2.5	38	f	1	lscs	mannarkudi gh	15,6	cpd	na,na	EM	yes	resp distress of nb	no	92	84	78
2	427382	2.75	37	f	1	forceps	rmh	14.6.	cpd	7,8	im	yes	resp dist/erbs	no	86	92	108
3	424968	3	39	m	3	lscs	puthukottai gh	10.6.	failed induction	na,na	EM	yes	resp distress of nb	no	106	98	84
4	427831	3	39	m	1	lscs	rmh	17.6	gdm	7,8	im	yes	idm	no	74	68	54
5	407907	1.9	32	m	1	lscs	rmh	17.6.	oligohydraminos	4,8	im	yes	rds/pt/lbw	no	114	106	118
6	427617	2.5	30	m	1	lscs	rmh	15.6.	prom	2,5	im	yes	birth asphyxia	no	114	92	68
7	424679	2.7	38	m	3	lscs	rmh	09.6.	pih	na,na	im	no	sepsis	no	112	90	76
8	427968	3	38	m	1	lscs	jeyamkon gh	16.6.	prev lscs	7,8	EM	yes	resp distress of nb	no	86	70	58
9	427761	3	39	m	1	nvd	puthukottai gh	18.6.	nil	7,8	EM	yes	resp distress of nb	no	140	132	112
10	427888	3	39	m	1	lscs	rmh	18.6	nil	5,8	im	yes	hydrocephalus	no	136	120	98
11	427761	2.8	38	f	1	nvd	rmh	16.6	nil	3,7	im	yes	sepsis	no	118	124	104
12	428823	2.6	41	m	1	nvd	rmh	23.6	nil	na,na	im	no	birth asphyxia	no	76	82	94
13	427497	1	28	f	8	nvd	rmh	15,6	nil	4,7	im	yes	rds/pt/lbw	no	172	160	132
14	427490	2.8	38	m	1	lscs	rmh	23,4	pih	4,8	im	yes	sepsis	yes	72	68	54
15	427242	1.3	41	m	1	lscs	puthukottai gh	14.6	pih	4,7	EM	yes	iugr/lbw/resp dis	no	34	32	32
16	418653	1.4	30	f	1	nvd	rmh	24.4	idm	na,na	im	yes	rds/pt/lbw	no	86	72	50
17	417539	2.7	38	f	1	nvd	rmh	25,4	anemia	na,na	im	no	birth asphyxia	no	92	88	76
18	423924	2.8	40	m	1	nvd	rmh	31,5	nil	7,8	im	yes	resp distress of nb	no	88	60	75
19	416927	2.8	40	m	3	nvd	rmh	22.6	nil	8,8	im	yes	sepsis	no	43	42	44
20	423397	2.96	40	m	1	lscs	pvt hos	31,5	prom	4,6	EM	no	birth asphyxia	no	102	90	75

21	423956	2.5	38	m	2	nvd	sirkali gh	4,6	chd	7,8	EM	yes	meningomyeocee	no	86	72	60
22	423751	2.9	41	f	1	lscs	rmh	3,6	cord prolapse	3,5	im	no	birth asphyxia	no	34	44	53
23	418145	3.5	39	m	2	lscs	puthukottai gh	24,5	gdm	6,7	EM	yes	a.v malform/sepsis	no	80	72	65
24	418362	1	30	m	1	nvd	rmh	25,5	prom	8,9	im	yes	rds/pt/lbw	yes	96	82	60
25	418962	2.5	37	m	4	lscs	rmh	25,4	nil	6,	im	yes	sepsis	no	47	38	24
26	423519	2.9	40	m	18	lscs	rmh	16,5	cpd	7,8	im	yes	sepsis	yes	98	90	70
27	423529	1.75	32	m	1	nvd	rmh	2,6	nil	8,9	im	yes	rds/pt/lbw	no	134	110	120
28	423680	2.5	37	m	1	nvd	rmh	4,6	pih	3,6	im	no	birth asphyxia	no	94	78	65
29	423245	1.4	34	m	5	lscs	rmh	17,5	pih	7,8	im	yes	rds/pt/lbw	yes	94	82	68
30	421624	2.1	37	m	1	lscs	rmh	21,5	idm	8,8	im	yes	sepsis	no	88	64	72
31	418760	4.6	38	m	1	lscs	rmh	29,4	idm	8,8	im	yes	idm	no	60	82	67
32	418458	1.1	30	m	1	nvd	rmh	30,4	nil	6,8	im	yes	rds/pt/lbw	yes	90	72	66
33	421029	1.41	31	m	1	nvd	rmh	17,5	nil	6,7	im	yes	rds/pt/lbw	no	362	388	342
34	428849	1.5	32	f	1	lscs	rmh	23,6	pih	6,8	im	yes	lbw	no	84	102	90
35	485699	2.7	39	m	4	nvd	rmh	2,6	nil	5,8	im	yes	sepsis	no	94	88	71
36	422845	2.3	40	f	2	nvd	patukot gh	26,5	anemia	5,7	EM	yes	sepsis	no	36	40	36
37	424125	1.25	32	m	28	nvd	manarkudi gh	8,5	anemia	7,8	EM	yes	rds/pt/lbw	no	104	92	71
38	423258	2.1	36	f	2	nvd	rmh	31,5	pih	7,9	im	yes	iugr/lbw/resp dis	no	40	32	21
39	421567	1.7	32	m	3	nvd	rmh	17.6.16	nil	8,9	im	yes	sepsis	no	84	78	56
40	423883	1.65	32	f	8	lscs	rmh	28,5	pih	8,9	im	yes	rds/pt/lbw	no	94	80	72
41	428902	3.6	40	f	4	nvd	outborn	20.6.16	nil	6,9	EM	no	sepsis	no	32	66	48
42	421901	1.75	34	f	1	lscs	rmh	24,6	twin	6,9	im	yes	rds/pt/lbw	no	100	94	72

43	421901	3	38	f	1	nvd	rmh	23,5	prom	4,6	im	no	birth asphyxia	yes	94	88	76
44	428870	3.1	39	m	1	lscs	rmh	23,6	gdm	7,8	im	yes	idm	no	28	35	24
45	428486	2.6	37	m	1	lscs	rmh	20,6	nil	7,8	im	yes	msaf/resp dist	no	168	162	146
46	428824	2.1	35	f	1	nvd	rmh	23,6	hypothyroid	6,8	im	yes	resp distress of nb	yes	140	134	122
47	428160	2.4	38	m	4	nvd	rmh	19,6	nil	6,8	im	yes	sepsis		104	96	84
48	427953	3	40	m	1	lscs	rmh	18,6	gdm	8,9	im	yes	idm	no	82	70	58
49	427862	3.7	39	f	1	nvd	rmh	17,6	nil	5,7	im	yes	chd	yes	124	110	92
50	427930	3	39	m	7	lscs	rmh	16,6	prev lscs	4,8	im	yes	sepsis	no	124	110	94
51	428904	2.6	38	f	1	lscs	rmh	23,6	nil	6,9	im	yes	resp distress of nb	no	152	146	136
52	427168	2.8	37	m	6	nvd	rmh	13,6	nil	7,8	im	yes	sepsis	no	92	84	72
53	428634	2.75	38	f	2	lscs	rmh	21,6	oligohydramino s	8,8	im	yes	sepsis	no	142	134	126
54	427467	2.1	36	m	3	lscs	rmh	14,6	oligohydramino s	8,9	im	yes	seizures	no	170	146	174
55	424593	2.4	37	f	2	nvd	thiruvarur gh	8,6	nil	4,6	EM	no	birth asphyxia	no	144	132	120
56	429003	2.6	38	m	1	nvd	rmh	24,6	nil	8,9	im	yes	resp distress of nb	yes	122	102	94
57	428630	3	38	m	1	nvd	rmh	24,6	hypothyroid	7,8	im	yes	sepsis		110	92	106
58	429020	1.75	34	m	1	nvd	rmh	24,6	nil	7,8	im	yes	lbw/resp distress		106	134	112
59	412679	1.8	39	m	2	nvd	rmh	17.3	nil	6,7	im	yes	anomalous baby	yes	86	92	46
60	413438	3.15	39	m	1	nvd	pvt hos	20.3	nil	8,8	EM	yes	resp distress of nb	no	127	94	93
61	412627	1,2	30	m	2	nvd	pvt hos	15.3	hypothyroid	7,8	EM	yes	rds/pt/lbw	yes	93	78	62
62	412619	3	38	f	6	nvd	pvt hos	10.3	nil	8,8	EM	yes	sepsis	no	89	84	64
63	412682	2.5	40	f	1	nvd	rmh	15,3	nil	8,8	EM	yes	resp distress of nb	no	144	132	88
64	412761	1.2	28	m	1	nvd	rmh	16.3	anemia	6,7	im	yes	rds/pt/lbw	no	322	286	289

65	412655	1.2	32	m	1	nvd	home	15,3	nil	8,8	EM	yes	rds/pt/lbw	no	84	72	70
66	412475	3	38	m	6	lscs	rmh	14.3	short primi	8,8	im	yes	nnh	no	70	71	58
67	412043	3	38	f	1	outlet forceps	rmh	11.3	nil	8,8	im	yes	msaf/resp dist	no	130	122	86
68	412471	2.7	38	m	18	nvd	vellankudi phc	26,2	nil	8,8	EM	yes	sepsis	no	48	32	85
69	411605	3.5	38	m	1	lscs	rmh	8.3	nil	6,8	im	yes	resp distress of nb	no	58	70	116
70	411305	2.5	39	m	1	nvd	ammapettai phc	5.3	nil	5,6	EM	no	birth asphyxia	no	98	178	75
71	411682	2.6	38	f	2	outlet forceps	rmh	8.3	nil	8,8	im	yes	resp distress of nb	no	151	115	90
72	411681	4.1	39	m	2	lscs	rmh	8.3	nil	8,8	im	yes	resp distress of nb	no	98	81	90
73	416264	1.6	30	m	1	lscs	rmh	21.5	abrubtion	7,8	im	yes	resp distress of nb	yes	128	116	92
74	419288	3.1	40	m	4	lscs	rmh	29.4	nil	8,8	im	yes	sepsis	no	36	34	68
75	419274	2.3	40	m	1	lscs	rmh	2.5	nil	7,7	im	yes	resp distress of nb	no	136	108	92
76	417832	3	40	m	7	nvd	rmh	25.4	nil	8,8	im	yes	resp distress of nb	no	92	74	84
77	419262	1.8	32	f	5	lscs	pvt hos	27.4	pih	7,8	EM	yes	hypoglycemia	no	92	54	64
78	412343	1.7	34	f	1	lscs	rmh	3,5	anemia	8,8	im	yes	rds/pt/lbw	no	108	94	78
79	419245	2.3	37	m	1	lscs	rmh	3.5	prom	8,8	im	yes	resp distress of nb	no	108	146	116
80	412422	3	38	m	2	lscs	rmh	19.5	nil	8,8	im	yes	resp distress of nb	yes	60	62	54
81	1187	2.2	34	m	8	lscs	rmh	13.5	nil	6,7	im	no	birth asphyxia	no	86	72	54
82	1191	2.5	37	f	8	lscs	rmh	11.5	nil	5,7	im	yes	sepsis	no	52	78	64
83	1145	2.9	40	m	3	nvd	rmh	18.5	nil	5,8	im	yes	resp distress of nb	no	86	72	64
84	421316	3	40	m	3	nvd	rmh	18.5	nil	8,8	im	yes	sepsis	no	346	389	380
85	1213	2.9	40	m	5	lscs	rmh	16.5	nil	3,6	im	no	birth asphyxia	no	92	104	68
86	418983	2.7	39	m	3	lscs	rmh	1.5	nil	8,8	im	yes	sepsis	no	94	138	101

87	1123	1.95	37	m	14	lscs	rmh	8.5	pih	1,3	im	no	birth asphyxia	yes	96	108	60
88	420576	1.3	30	f	8	lscs	rmh	13.5	opacenta previa	6,8	im	yes	resp distress of nb	yes	60	60	58
89	421007	1.5	32	m	1	lscs	rmh	17.5	pih	6,7	im	yes	nnh	rd	49	72	60
90	420140	2.3	38	f	10	lscs	pvt hos	9.5	nil	6,8	EM	no	birth asphyxia	yes	88	90	62
91	411959	3	38	f	4	nvd	rmh	10.3	nil	6,7	im	no	birth asphyxia	no	60	51	72
92	412681	2.75	38	f	1	nvd	rmh	17,3	prom	8,8	im	yes	meningomyeocee	no	113	115	108
93	412796	2.5	38	m	4	lscs	pvt hos	15.3	oligohydraminos	8,8	EM	yes	anomalous baby	no	43	27	40
94	424692	2.5	38	f	1	nvd	rmh	9.6	pih	na,na	im	yes	resp distress of nb	no	72	50	59
95	427077	1.2	32	f	1	nvd	kumbakonam gh	4.6	nil	na,na	EM	yes	rds/pt/lbw	yes	33	32	36
96	424717	1.5	34	m	23	nvd	mannarkudi gh	17.5	nil	na,na	EM	yes	rds/pt/lbw	yes	23	23	18
97	423751	2.9	38	f	1	lscs	rmh	3.6	prev lscs	4,6	im	no	birth asphyxia	yes	72	64	53
98	424480	2	38	f	4	nvd	ariyalur gh	4.6	nil	na,na	EM	yes	thrombocytopenia	no	90	86	72
99	412627	1,2	30	m	2	nvd	pvt hos	15.3	hypothyroid	7,8	EM	yes	rds/pt/lbw	yes	98	76	67
100	428849	1.5	32	f	1	lscs	rmh	23,6	pih	6,8	im	yes	lbw	no	76	100	86
101	434026	1.5	30	f	9	lscs	rmh	29.6	nil	8,8	im	yes	rds/pt/lbw	no	26	62	37
102	424662	2.7	40	f	1	nvd	jayamkondam gh	9.6	nil	5,6	EM	no	birth asphyxia	no	62	54	42
103	427066	2.4	36	m	14	nvd	jayamkondam gh	23.5	nil	7,8	EM	yes	sepsis	no	84	64	70
104	424813	1.8	36	m	4	nvd	pvt hos	6.6	pih	8,8	EM	yes	hypoglycemia	no	45	40	28
105	423519	2.9	40	m	16	lscs	pvt hos	16.5	cpd	8,8	EM	yes	sepsis	no	72	56	70
106	427455	2.5	38	m	1	nvd	rmh	14.6	prom	na,na	im	yes	sepsis	no	68	74	68
107	525053	1.8	34	m	1	nvd	pvt hos	13-Jun	nil	5,7	EM	yes	rds/pt/lbw	no	112	112	148
108	425062	2.5	35	m	1	nvd	rmh	14.6	nil	na,na	im	yes	rds/pt/lbw	no	38	22	40

109	433384	1.5	33	f	1	nvd	tiruvarur gh	28.6	nil	na,na	EM	yes	rds/pt/lbw	no	78	66	49
110	434726	1	38	f	1	nvd	rmh	5.6	nil	2,4	im	no	birth asphyxia	no	66	78	58
111	434026	1.5	32	f	8	lscs	rmh	29.6	pih	4,6	im	yes	rds/pt/lbw	no	90	98	72
112	435026	1.8	30	f	1	nvd	pvt hos	7.6	abrubtion	3,8	EM	yes	resp distress of nb	yes	98	88	78
113	417539	2.3	38	f	1	nvd	rmh	22.4	nil	na,na	im	yes	resp distress of nb	no	82	74	68
114	421567	1.8	35	m	5hrs	lscs	rmh	20.5	preeclampsia, hypothyroid		im	yes	rds/pt/lbw	no	96	80	62
115	433290	2.3	36	f	1	nvd	rmh	24.5	gdm	3,6	im	yes	rds/pt/lbw	no	110	94	90
116	435303	2.72	38	f	4	nvd	ariyalur gh	5.6	nil	5,6	EM	yes	sepsis	yes	36	68	34
117	435383	2.5	38	f	3	nvd	pvt hos	6.6	nil	5,7	EM	yes	sepsis	no	98	76	50
118	435253	1.2	30	m	1	nvd	pvt hos	9.6	abrubtion	5,6	EM	yes	rds/pt/lbw	yes	72	56	54
119	435254	1.4	28	m	1	nvd	pvt hos	9.6	abrubtion	5,8	EM	yes	rds/pt/lbw	no	42	24	31
120	435348	2.8	38	m	1	nvd	rmh	8,8	abrubtion	7,8	im	yes	resp distress of nb	no	96	90	92
121	435379	1.5	32	m	1	lscs	pvt hos	9.6	nil	5,7	EM	yes	rds/pt/lbw	no	31	24	20
122	435381	2.1	38	f	1	lscs	rmh	9.6	pih	4,6	im	yes	downs	no	96	84	90
123	413438	3.1	39	m	1	nvd	pvt hos	20.3	nil	8,8	EM	yes	resp distress of nb	no	74	56	50
124	412043	3	38	f	1	outlet forceps	rmh	11.3	nil	8,8	im	yes	msaf/resp dist	no	130	122	86
125	412619	3	38	f	6	nvd	pvt hos	10.3	nil	8,8	EM	yes	nnh	no	89	88	64
126	412682	2.5	40	f	1	nvd	rmh	15.3	nil	8,8	im	yes	resp distress of nb	no	144	132	88
127	434934	3	38	m	2	lscs	tiruvarur gh	5.6	gdm	4,6	EM	no	birth asphyxia	no	98	110	110
128	435335	2	36	f	1	nvd	rmh	9.6	nil	4,6	im	no	birth asphyxia	no	132	136	128
129	433724	1.2	28	m	1	nvd	rmh	28.5	pih	3,5	im	no	birth asphyxia	no	106	94	86
130	433723	1.2	30	m	1	nvd	rmh	28,5	pih	3,5	im	no	birth asphyxia	no	76	64	58

131	434748	4	38	f	1	lscs	rmh	3,7	pih	5,7	im	yes	resp distress of nb		108	102	94
132	413438	3.1	39	m	2	nvd	pvt hos	20.3	nil	8,8	EM	yes	resp distress of nb	no	127	94	93
133	434521	2.3	38	f	1	nvd	rmh	6.6	nil	8,8	im	yes	msaf/resp dist	no	78	76	70
134	434818	2.4	34	f	1	lscs	rmh	6.6	pih	4,6	im	yes	rds/pt/lbw	no	36	28	26
135	412761	1.2	28	m	1	nvd	rmh	16.3	anemia	6,7	im	yes	rds/pt/lbw	no	312	262	302
136	412655	1.2	34	m	1	nvd	rmh	15.3	nil	8,8	im	yes	rds/pt/lbw	no	84	72	70
137	412475	3	38	m	7	lscs	rmh	14.3	nil	8,8	im	yes	nnh	no	70	77	58
138	434067	3.3	38	m	2	lscs	rmh	30,5	hypothyroid	8,8	im	yes	sepsis	no	56	64	51
139	435243	2.4	34	m	1	lscs	rmh	9.6	hypothyroid	7,8	im	yes	rds/pt/lbw	no	78	68	64
140	434691	2.25	38	f	24	lscs	kumbakonam gh	11.5	nil	4,6	EM	yes	sepsis	no	68	40	56
141	412627	1.2	30	m	2	nvd	pvt hos	15.3	hypothyroid	7,8	EM	yes	rds/pt/lbw	no	20	24	26
142	412679	1.8	39	m	2	nvd	rmh	17.3	nil	6,7	im	yes	anomalous baby	no	63	32	37
143	437546	2.2	38	f	6	lscs	rmh	2.6	nil	4,6	im	yes	birth asphyxia	no	68	56	58
144	434940	2.7	38	m	2	nvd	rmh	6,6	nil	4,6	im	yes	msaf/resp dist	no	56	72	60
145	425215	2.2	39	f	12	nvd	pvt hos	28.5	nil	na,na	EM	yes	sepsis	no	72	58	60
146	434723	2.3	34	m	3	lscs	rmh	5.6	pih	4,6	im	no	birth asphyxia	no	62	56	50
147	434629	2.5	38	f	22	nvd	ariyalur gh	15.5	nil	5,7	EM	yes	msaf/resp dist	no	78	64	68
148	433277	1.3	32	m	5	nvd	rmh	24.5	pih	3,5	im	no	birth asphyxia	no	60	56	48
149	434814	2.7	39	m	2	nvd	rmh	5,6	pih	na,na	im	yes	hypoglycemia	no	28	28	20
150	431907	2.5	37	m	2	lscs	rmh	6.6	nil	7,8	im	yes	sepsis	no	88	77	77
151	434929	1.5	36	f	1	nvd	rmh	6.6	nil	6,6	im	no	birth asphyxia	yes	68	74	68
152	434697	2.8	38	f	23	nvd	pvt hos	12.5	gdm	4,6	EM	yes	hypoglycemia	no	38	36	33

153	434769	2.5	39	f	8	nvd	kumbakonam gh	29.5	nil	8,8	EM	yes	sepsis	no	80	76	76
154	433259	2.3	34	m	21	nvd	kumbakonam gh	3.5	nil	na,na	EM	yes	sepsis	no	66	58	54
155	434675	1.7	35	f	1	nvd	rmh	4.6	pih	na,na	im	yes	rds/pt/lbw	no	128	112	97
156	434028	1.5	32	f	10	nvd	rmh	29.5	nil	8,8	im	yes	rds/pt/lbw	no	90	94	72
157	435716	2	30	m	1	nvd	rmh	18.6	pih	4,5	im	no	birth asphyxia	no	48	24	35
158	435342	1.7	34	m	1	lscs	rmh	10.8	pih	4,8	im	yes	hypoglycemia	no	43	36	20
159	436160	1.6	37	m	1	lscs	16.6	r	pih	5,8	im	yes	rds/pt/lbw	no	40	36	27
160	435841	1.9	32	m	1	lscs	rmh	14.6	pih	4,7	im	yes	rds/pt/lbw	no	77	65	62
161	435840	2.14	32	f	1	lscs	rmh	14.8	pih	3,8	im	yes	rds/pt/lbw	no	90	56	68
162	435714	1.75	30	m	1	nvd	rmh	13.6	pih	3,8	im	yes	rds/pt/lbw	yes	76	58	54
163	436882	2.3	36	f	1	lscs	rmh	21.6	gdm	5,7	im	yes	rds/pt/lbw	no	34	28	23
164	436216	2.2	32	m	4	lscs	rmh	13.6	abrubtion	4,8	im	yes	rds/pt/lbw	yes	66	54	56
165	436006	2.2	34	f	1	nvd	rmh	16.6	prom	3,6	im	yes	sepsis	no	43	37	31
166	436842	3	38	f	1	lscs	rmh	21.6	hypothyroid	5,6	im	yes	meningomyeocee	no	68	76	64
167	436155	2.5	36	f	1	nvd	pvt hos	16.6	nil	2,3	EM	no	birth asphyxia	yes	54	58	52
168	435683	2.6	39	f	7	nvd	rmh	13.6	pih	5,8	im	yes	nnh	no	66	64	50
169	436679	3.5	38	f	1	nvd	ariyalur gh	19,6	pih	4,6	EM	no	birth asphyxia	yes	28	26	26
170	436062	3.5	40	f	1	nvd	mannarkudi gh	15.6	anemia	5,8	EM	yes	hdn	no	76	78	72
171	436844	2.8	39	f	1	nvd	pvt hos	20.6	nil	5,8	EM	yes	sepsis	no	58	62	59
172	436200	2	39	f	2	lscs	rmh	17.6	hypothyroid	4,8	im	yes	sepsis	no	86	77	75
173	436983	3.1	38	m	2	nvd	rmh	15.6	prom	5,7	im	yes	hypoglycemia	no	33	32	36
174	436179	3.7	38	m	1	lscs	mannarkudi gh	16.6	hypothyroid	5,8	EM	yes	chd	no	67	68	59

175	436463	3	38	m	9	nvd	kumbakonam gh	9.6	hypothyroid	4,7	EM	yes	sepsis	no	64	78	67
176	436069	4	38	m	13	nvd	ariyalur gh	3,6	gdm	6,8	EM	yes	chd	no	64	56	56
177	435363	2.7	39	m	3	nvd	rmh	9.6	pih	6.8	im	yes	nnh	no	112	102	90
178	409046	2.75	40	m	2	nvd	rmh	14.6	prom	4,7	im	yes	sepsis	yes	132	64	62
179	436972	2	34	f	20	nvd	kumbakonam gh	31.5	pih	4,6	EM	yes	rds/pt/lbw	no	154	128	115
180	436467	3	39	m	1	nvd	rmh	17.6	polyhydraminos	2,5	im	no	birth asphyxia	yes	88	76	75
181	436493	0.75	26	m	1	nvd	rmh	18.6	pih	3,6	im	yes	rds/pt/lbw	no	137	145	126
182	436760	0.99	28	m	1	nvd	rmh	20.6	pih	4,8	im	yes	rds/pt/lbw	yes	23	20	31
183	436894	2.4	36	m	1	lscs	ariyalur gh	21.6	pih	5,8	EM	yes	rds/pt/lbw	no	77	56	54
184	427761	2.8	38	f	1	nvd	rmh	16.6	nil	3,7	im	yes	sepsis	no	118	124	104
185	411682	2.6	38	f	2	outlet forceps	rmh	8.3	nil	8,8	im	yes	resp distress of nb	no	76	115	67
186	418983	2.7	39	m	3	lscs	rmh	1.5	nil	8,8	im	yes	sepsis	no	26	28	33
187	421624	2.1	37	m	1	lscs	rmh	21,5	idm	8,8	im	yes	sepsis	no	38	39	30
188	435335	2	36	f	1	nvd	rmh	9.6	nil	4,6	im	no	birth asphyxia	no	132	136	126
189	417539	2.3	38	f	1	nvd	rmh	22.4	nil	na,na	im	yes	resp distress of nb	no	82	74	56
190	436006	2.2	34	f	1	nvd	rmh	16.6	prom	3,6	im	yes	sepsis	no	77	88	87
191	434691	2.25	38	f	24	lscs	kumbakonam gh	11.5	nil	4,6	EM	yes	sepsis	no	38	33	24
192	416927	2.8	40	m	3	nvd	rmh	22.6.16	nil	8,8	im	yes	sepsis	no	85	88	98
193	411681	4.1	39	m	2	lscs	rmh	8.3	nil	8,8	im	yes	resp distress of nb	no	98	81	90
194	427077	1.2	32	f	1	nvd	kumbakonam gh	4.6	nil	na,na	EM	yes	rds/pt/lbw	yes	52	44	46
195	424717	1.5	34	m	23	nvd	mannarkudi gh	17.5	nil	na,na	EM	yes	rds/pt/lbw	yes	146	140	120

196	435714	1.75	30	m	1	nvd	rmh	13.6	pih	3,8	im	yes	rds/pt/lbw	yes	53	48	48
197	427497	1	28	f	8	nvd	rmh	15,6	nil	4,7	im	yes	rds/pt/lbw	no	116	113	112
198	421567	1.7	32	m	3	nvd	rmh	17.6.16	nil	8,9	im	yes	sepsis	no	84	78	56
199	436160	1.6	34	m	1	lscs	16.6	r	pih	5,8	im	yes	rds/pt/lbw	no	23	36	32
200	435840	2.14	32	f	1	lscs	rmh	14.8	pih	3,8	im	yes	rds/pt/lbw	no	76	65	59