"IMPACT OF MATERNAL ANAEMIA ON CORD BLOOD HAEMOGLOBIN"

Dissertation submitted

to

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

With partial fulfillment of the regulations
for the award of the degree of

FOR THE DEGREE OF DOCTOR OF MEDICINE (M.D)

BRANCH VII - PAEDIATRIC MEDICINE

GOVT. KILPAUK MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R MEDICAL UNIVERSITY

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APRIL 2017

BONAFIDE CERTIFICATE

This is to certify that dissertation named **"IMPACT OF MATERNAL ANAEMIA ON CORD BLOOD HAEMOGLOBIN"** is a bonafide original research work carried out by **Dr. DHANASEKARAN R** post graduate student, Department of Paediatrics, Govt. Kilpauk Medical College, Chennai-10 under our direct supervision and guidance in partial fulfilment of the requirements for the award of the degree of Doctor of Medicine (M.D Paediatrics) Branch VII Paediatric Medicine during the academic year 2014-2017.

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DECLARATION

I **Dr. DHANASEKARAN R,** hereby solemnly declare that this dissertation entitled **"IMPACT OF MATERNAL ANAEMIA ON CORD BLOOD HAEMOGLOBIN"** has been conducted by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **PROF. DR. K. SUGUNA M.D.,D.C.H**., Professor, Department of Paediatrics, Govt. Royapettah Hospital/Govt. Kilpauk Medical College & Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University rules and regulations for the award of the degree of **M.D. Branch VII (Paediatrics)**.

This has not previously been submitted by me for the award of any degree or diploma from any other university.

Dr. DHANASEKARAN R

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "IMPACT OF MATERNAL ANAEMIA ON CORD BLOOD HAEMOGLOBIN"- For Project Work submitted by Dr.R.Dhanasekaran, Post Graduate in MD (Paediatrics), Govt. Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

8/8/16

DEAN

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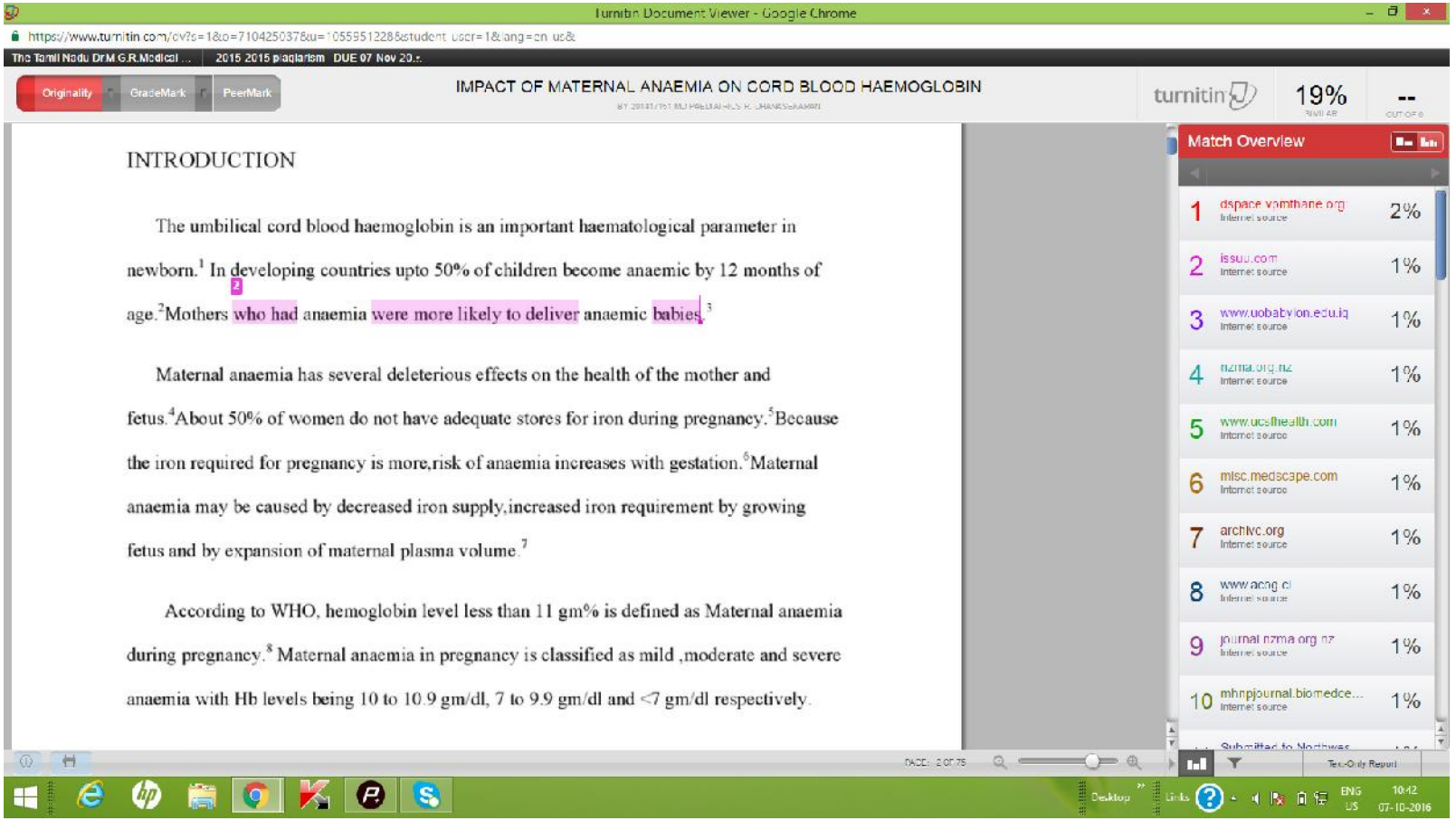
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The umbilical cord blood haemoglobin is an important haematological parameter in newborn.[1] In developing countries upto 50% of children become anaemic by 12 months of age.[2]Mothers who had anaemia were more likely to deliver anaemic babies.[3]
Maternal anaemia has several deleterious effects on the health of the mother and fetus.[4]About 50% of women do not have adequate stores for iron during pregnancy.[5]Because the iron required for pregnancy is more,risk of anaemia increases with gestation.[6]Maternal anaemia may be caused by decreased iron supply,increased iron requirement by growing fetus and by expansion of maternal plasma volume.[7]
According to WHO, hemoglobin level less than 11 gm% is defined as Maternal anaemia during pregnancy.[8] Maternal anaemia in pregnancy is classified as mild ,moderate and severe anaemia with Hb levels being 10 to 10.9 gm/dl, 7 to 9.9 gm/dl and <7 gm/dl respectively.
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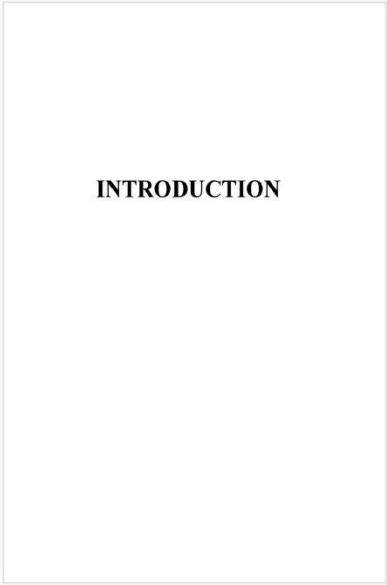

INTRODUCTION

ABBREVIATION

Hb	Haemoglobin
Hct	Haematocrit
WHO	World Health Organisation
LBW	Low Birth Weight
IUGR	Intra Uterine Growth Retardation
IUD	Intra Uterine Death
MCV	Mean Corpuscular Volume
GA	Gestational Age
ICH	Intra cranial haemorrhage
SGA	Small for gestational age
CLD	Chronic lung disease
BPD	Broncho pulmonary dysplasia
RSV	Respiratory synctial virus
HbsAg	Hepatitis B surface antigen
SD	Standard deviation
CMV	Cytomegalo virus
PPHN	Persistent pulmonary hypertension of newborn
PRBC	Packed red blood cells
TRALI	Transfusion associated acute lung injury
GVHD	Graft versus host disease
HSCT	Hematopoietic stem cell transplantation

CONTENTS

INTRODUCTION

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The birth weight of infants born to women with anaemia was low compared to infants born to non anaemic mothers.[9]Incidence of pre term delivery and birth of IUGR babies, IUD (Intra uterine death) were more in women with maternal anaemia.[10]

EPIDEMIOLOGY

Anaemia is a commonest medical disorder and around 50% of women become anaemic during pregnancy worldwide.Maternal anaemia during pregnancy is common in developing countries which affects 57% of pregnancies.[11,12]

Umbilical cord blood is the most valuable underutilized resource in the care of neonates. Utilization of umbilical cord blood for laboratory testing of neonates is a promising new practice which has shown to improve neonatal outcomes. Full implementation of this practice is therefore an important step in better utilization of umbilical cord blood in improving the outcomes of neonates.[13]

HAEMOGLOBIN STRUCTURE:

Haemoglobin is produced inside in the red bone marrow. It is a conjugated protein comprising of Haem and Globin. Iron and porphyrin compound in haem constitutes 4% and Globin compound constitutes 96%.[14]

Iron ion along with porphyrin forms haem compound. Porphyrin is an heterocyclic ring. Four pyrrole molecules with protoporphyrin linked together cyclically by methane bridges.[15] Metalloporphyrin is formed by combination of metal iron with protoporphyrin.

Metalloporphyrin consist of 0.34%. Iron (Fe) in ferrous state. Haemoglobin consist of tetramers with two polypeptide chains of one kind & two of another. Haem keeps iron in ferrous state by globin & it combines loosely & reversibly with Oxygen.[16]

In normal adult Hb (HbA-globin) chains are called α and β chains and in HbA2 –α and δ chain are present. In each chain there are about 150 aminoacids in sequence. The substitution of any one of these amino acids by another, results in formation of abnormal chain, In sickle cell anemia, Hb S is present in which glutamic acid is replaced by Valine.[17]

Synthesis of Haemoglobin :- Synthesis of Haemoglobin begins at the erythroblast stage and then through normoblast and reticulocyte stage . Formation of haemoglobin occurs in ribosome of endoplasmic reticulum. Haem portion of haemoglobin is synthesized in the following steps:

Formation of Haem

- In Krebs cycle, acetic acid changes to α- Keto glutaric acid which then combines with Glycine forming Pyrrole compound.
- Four Pyrrole compounds combine to form Protoporphyrin III compound.
- Protoporphyrin III + Fe ---------Haem (Metallopophyrin) Formation of Haemoglobin [18-20]

- four Haem molecules + Globin --------- Haemoglobin

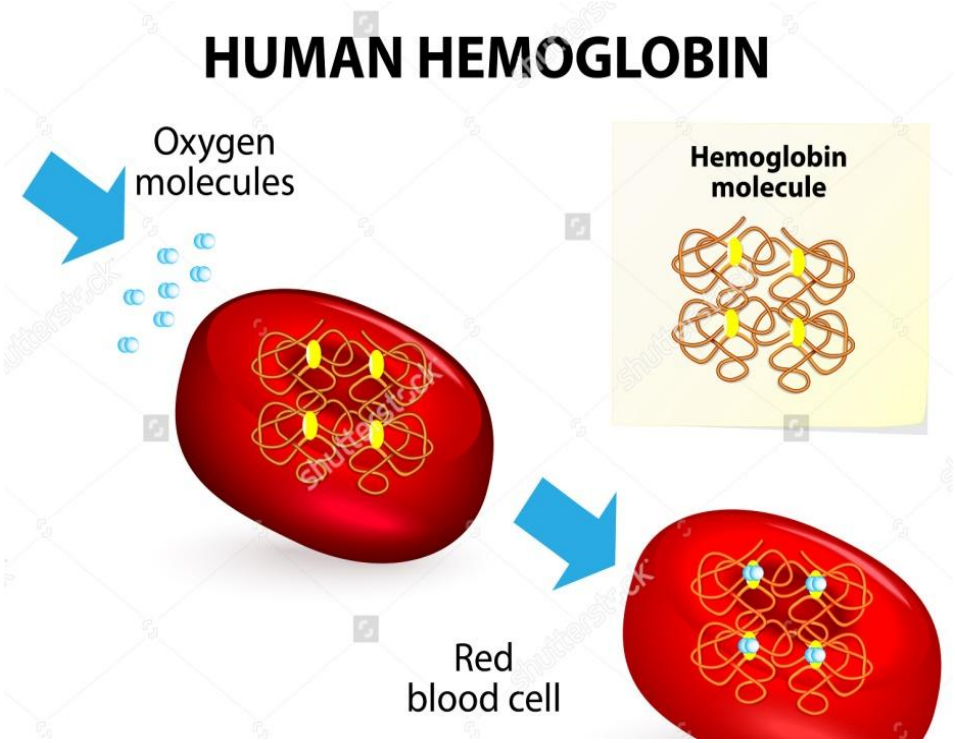
The most common haemoglobin type is a tetramer which is haemoglobin A in adults. Two α and two β polypeptide chains (α1 β1 and α2 β2) noncovalently bound, each made up of 141 and 146 amino acid residues respectively, in globin chain of Hb A. Abnormalities in the chain alter the physical characteristic of Hb.

In human fetus,Fetal Hb (α2,γ2) during the last seven months of development in the uterus persists in the newborn until roughly 6 months old is the major transport protein. Fetal hemoglobin is nearly completely replaced by adult hemoglobin by approximately 6 months postnatally in newborns .[21]

Fetal hemoglobin differs from adult hemoglobin in that it is able to bind oxygen with greater affinity than the adult form, so that it gives the developing fetus better access to oxygen from the mother's bloodstream. Level of foetal haemoglobin decreases after birth. The normal level of foetal haemoglobin in adults is usually less than 2%.

Haemoglobin A is the major haemoglobin in humans: Haemoglobin A2 first appears at 12 weeks after birth. Normal level of haemoglobinA2 is 3-6 percent.[22]

HAEMOGLOBIN	GLOBIN CHAIN
Haemoglobin A	α2 β2
Haemoglobin F	α2,γ2
Haemoglobin A2	α2δ2
Haemoglobin A1c	α β2-glucose



The oxyhemoglobin dissociation curve is useful in recognising how the haemoglobin delivers oxygen to the tissues. In oxy dissociation curve oxygen saturation (SO_2) and partial pressure of oxygen in the

blood (PO_2)are the two parameters involved. This is a S shaped curve which denotes the affinity of haemoglobin.

At partial pressure of oxygen of 60mm of hg, the oxygen dissociation curve is flat. Even the partial pressure of oxygen increases above 60mm 0f hg saturation of oxygen is not altered significantly.[23]

Factors influencing Standard Dissociation Curve[24]

Oxygen- hemoglobin binding can be influenced by several factors. These factors have an influence on shifting the oxyhemoglobin curve of an healthy person. Shift to right of the curve is caused by

- increase in temperature,
- increase in 2,3-DPG concentration
- increase in PCO_2
- decrease in pH.

The curve is shifted to the left by the following factors

- decrease in temparature
- decrease in 2,3-DPG concentration
- decrease in PCO2
- increase in pH

A rightward shift means there is decrease in the hemoglobin affinity for oxygen. Higher partial pressure is required to achieve the same oxygen saturation.When the oxygen dissociation curve is shifted to right , hemoglobin have reduced affinity of oxygen . Conversely, shift to the left increases the affinity of oxygen. So that it is easy for the hemoglobin to release the oxygen to the tissues.

Variation of the hydrogen ion concentration

A decline in pH shifts the standard curve to the right while an increase in pH shifts it to the left. This is known as the **Bohr effect**.

- **Effects of carbon dioxide**. The curve is affected by carbon dioxide in two ways:

1. carbon dioxide affects intracellular pH (the Bohr effect),
2. CO2 causes generation of carbamino compounds through various interactions.

Reduced levels of carbamino compounds have an effect of shifting the curve to the right, while higher levels cause a leftward shift.

Effects of 2,3-DPG. 2,3-diphosphoglycerate, or 2,3-DPG, is produced in erythrocytes during glycolysis. This compound is an organophosphate. The generation of 2,3-DPG is likely an important

protective mechanism, because the 2,3-DPG production increases for several conditions in the presence of reduced peripheral tissue oxygenation. High levels of 2,3-DPG shift the curve to the right. In the following conditions there is high levels of 2,3-DPG.

- hypoxemia,
- chronic lung disease,
- anemia,
- congestive heart failure

Low levels of 2,3-DPG cause a leftward shift, seen in states such as

- septic shock
- hypophosphatemia.[20]

Temperature:

- Temperature does not have dramatic effect as the previous factors, but hyperthermia causes shift of the curve to the right, while hypothermia causes a leftward shift.

- **Fetal Hemoglobin**. Fetal hemoglobin (HbF) is structurally different from normal hemoglobin (Hb). The fetal dissociation curve is shifted to the left relative to the curve for the normal adult. Typically, fetal arterial oxygen pressures are low, and hence the leftward shift enhances the placental uptake of oxygen.[20]

- **Carbon Monoxide**.

Haemoglobin has very high affinity to carbon monoxide than with oxygen. Haemoglobin is able to bind 240 times more readily than with oxygen.The presence of carbon monoxide can interfere with the hemoglobin's acquisition of oxygen. In addition to lowering the potential for hemoglobin to bind to oxygen, carbon monoxide also has the effect of shifting the curve to the left. When carbon monoxide levels are increased a person can suffer from severe hypoxemia while maintaining a normal PO_2.[25,26]

- **Effects of Methemoglobinemia**

It is a form of abnormal hemoglobin. Methemoglobinemia causes a shift in the curve to the left.

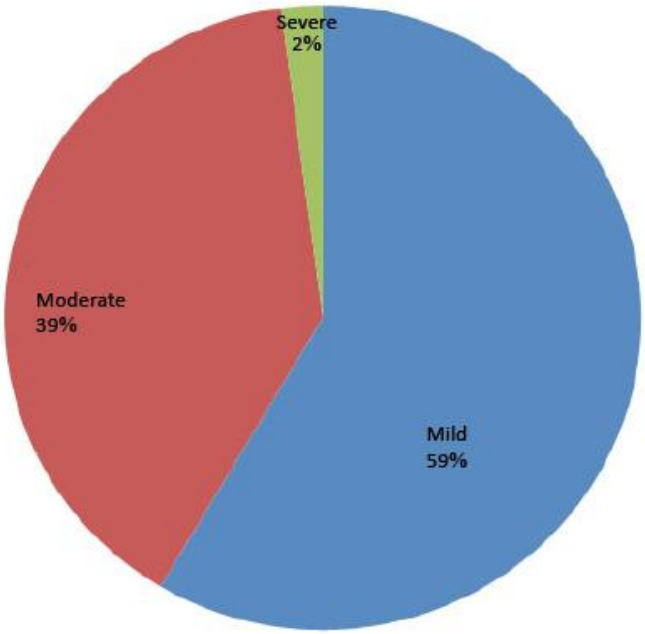
MATERNAL ANAEMIA IN PREGNANCY

Maternal anaemia during pregnancy is very common in developing countries which has several deleterious effect on the foetus.[27]It is considered as risk factor for poor pregnancy outcome and it can threaten life of both mother and foetus. Prevalance rate of anaemia during pregnancy are variable according to WHO because of differences in lifestyle,food habits and socio economic conditions.[28]

According to WHO, haemoglobin level below 11 gm% in pregnancy is considered as anaemia. Maternal anaemia in pregnancy is classified as :

Mild anaemia	-	Hb level (10 to 10.9 gm/dl)
Moderate anaemia	-	Hb level (7 to 9.9 gm/dl)
Severe anaemia	-	Hb level (<7 gm/dl)[29]

PREVALANCE OF MILD, MODERATE AND SEVERE ANAEMIA



In maternal anaemia,the number of red blood cells and their oxygen carrying capacity is insufficient to meet the physiological needs which occurs either due to defective RBC production,increased RBC

destruction and blood loss during pregnancy. Risk of anaemia increase by four fold from first to third trimester when monitored by centre of drug control(CDC) as part of pregnancy nutritional surveillance.[30]

Haemoglobin levels decrease progressively in pregnancy because of hemodilution and increasing needs of iron and other nutrients for both mother and fetus. While in third trimester, hemoconcentration occurs which results in higher haemoglobin levels.[31]

Many women undergo higher risk for anaemia because of increase in iron need during pregnancy.[13]When pregnancy progresses,iron requirement for fetal growth rise in proportion to weight of fetus.[32]

Anaemia during pregnancy ends up in impaired growth and mental development in children. There is a strong association between low maternal Hb levels during pregnancy and adverse fetal outcome.[33]

Classification of anaemia:

The anaemia may be classified in various ways. For all practical purposes,simplified classification is given which is helpful in the management of cases. Not uncommonly,an atypical form of anaemia may be met with and in such cases,the opinion of a haematologist should be sought for.

- **Physiological anaemia of pregnancy**
- **Pathological**

Deficiency anaemia(isolated or combined)

- iron deficiency
- folic acid deficiency
- vitamin B12 deficiency
- Protein deficiency [34]

Haemorrhagic

acute : following bleeding in early months or antepartum haemorrhage

chronic:hook worm infestations,bleeding piles

heriditary

thalassemias

sickle cell haemoglobinopathies

other haemoglobinopathies

Bone marrow insufficiency- hypoplasia or aplasia due to radiation ,drugs

Anaemia of infection(malaria, tuberculoosis)

chronic disease or neoplasm [34]

Concept of physiological anaemia

During pregnancy there is a dispropportionate increase in plasma volume, RBC volume and haemoglobin mass. In addition there is a marked demand of extra iron during pregnancy especially in the second half. Even an adequate diet cannot provide the extra demand of iron. Thus there always remains a physiological iron deficiency state during pregnancy. As a result there is not only fall in haemoglobin concentration but there is also associated low serum iron, increased iron binding capacity and increased rate of iron absorption. Thus, the fall in the haemoglobin concentration during pregnancy is due to combined effect of haemodilution and negative iron balance.[35,36]

CRITERIA OF PHYSIOLOGICAL ANAEMIA:

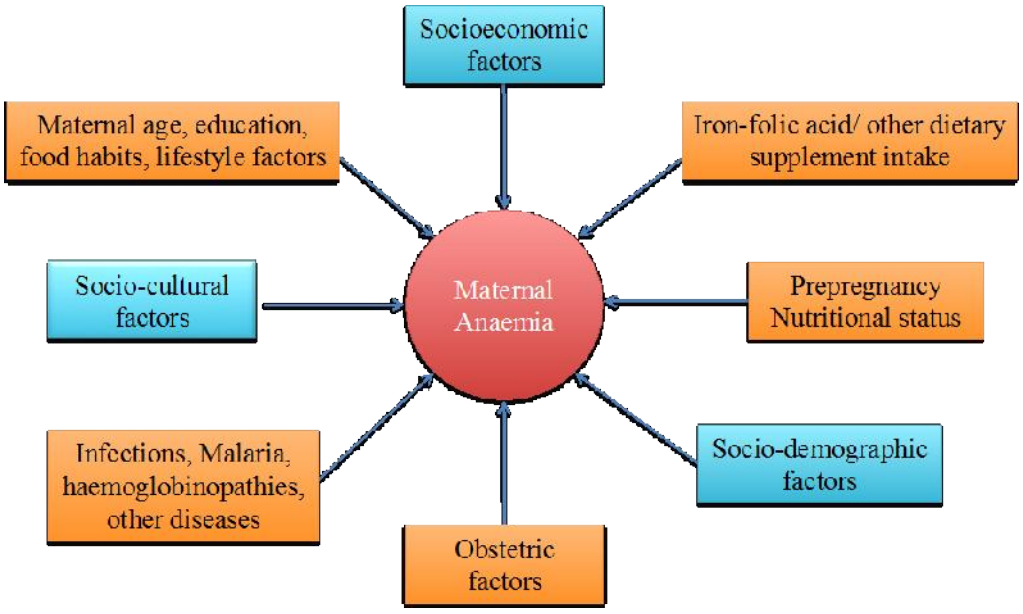
The lower limit of physiological anaemia during the second half of pregnancy should fulfill the following haematological values:

1. HB-10gm%
2. RBC-3.2 million/mm^3
3. PCV-30%
4. peripheral smear showing normal morphology of RBC with central pallor[37]

NORMAL BLOOD VALUES IN NON PREGNANT AND PREGNANT STATE

	Non pregnant	**Second half pregnancy**
Haemoglobin	14.8gm/100ml	11-148gm/100ml
Rbc	5 millioncu.mm	4-4.5 million cu.mm
Packed cell volume	39-42%	32-36%
Mch	27-32 pg	26-31pg
Mcv	75-100fl	75-95fl
Mchc	32-36%	30-35%
Serum iron	60-120 micro gm	Slightly lowered (65-75 micro gm)
Total iron binding capacity	300-350 µgm/100 ml	300-400 µgm/100 ml
Saturation percentage	30%	Less than 16%
Serum ferritin	20-30 µgm/l	15 µgm/l

CAUSES OF MATERNAL ANAEMIA



Factors which led to the development of anaemia during pregnancy:

- Increased demand of iron.
- Diminished intake of iron.
- Disturbed erythropoietic function of bone marrow.
- Pre pregnant health status.[38]

Increased demands for iron during pregnancy:

The daily requirement for iron during pregnancy is 14.5-14.7 mg/day. Therefore, 30-60 mg of elemental iron is given daily as supplementation along with folic acid.[39]

Diminished intake of iron:

Because of poor socio economic status, decreased appetite and vomiting during pregnancy are some of the contributory factors.

Infections, malaria, haemoglobinopathies like sickle cell anaemia, thalessemia.

Pre pregnant health status:

Majority of women start pregnancy on a pre existing anaemic state or at least with inadequate iron reserve.[40]

Multiple pregnancy:

Women with rapidly recurring pregnancy within 2 years ,following the last delivery need more iron to replenish deficient iron reserve.

Disturbed erythropoietic function of bone marrow:

Pregnancy depresses the erythropoietic function of bone marrow by competing for the available raw materials such as folic acid, vitamin B 12,proteins apart from iron.[39]

INVESTIGATIONS:

1. Estimation of Haemoglobin to assess severity of anaemia.

2. Peripheral blood smear estimation.Eg: in case of iron deficiency anaemia,microcytosis,anisocytosis and hypochromic vacuolated red cells are seen in abundance.

3. Blood indices estimation:

Mean corpuscular volume(MCV) and MCHC estimation.

MCV -below 80 cubic microns and

MCHC- less than 30% in case of iron deficiency anaemia.

4. Bone marrow examination: Done to differentiate the type of anaemia.

5. Stool examination:

Done which may reveal co-existing ankylostomiasis.[41,42]

COMPLICATIONS OF MATERNAL ANAEMIA DURING PREGNANCY:

There are several effects on new born baby because of maternal anaemia during pregnancy.[50]

Increased incidence of low birth weight (LBW) babies.

Intra uterine growth retardation of baby(IUGR)

Pre term birth of babies.

Intra uterine death of babies (IUD)

PRETERM BIRTH:

Preterm birth means when the neonate is born before the end of the 37weeks of gestation.[51]

A. **Incidence:**

Approximately 12.7% of all births are preterm. The distribution of this group is gradually shifting to a relatively older gestational age because of a 25% increase in late preterm infants (34 to 36 weeks) since 1990 to current rate of 9.1%

B. Etiology is unknown in most cases.

Important risk factors of preterm and lowbirth weight delivery are:

1. Poor socioeconomic class measured by family income,geographic area, social class, educational level and occupation.
2. Non-Hispanic black women are more than three times as likely to deliver an extremely preterm infant (1.9%) compared with non-Hispanic white and Hispanic women (0.6%).
3. Women younger than 16 or older than 35 are more likely to deliver pre-term or LBW infants; the association with age is more significant in whites than in African Americans.[52]

4. Prolonged periods of standing and increased physical cavity may be associated with IUGR and prematurity.

5. Multiple-gestation births frequently deliver preterm (60% of twins and 94% of triplets in 2005). In such births, higher rate of neonatal mortality is primarily due to prematurity.

6. Any maternal illness which may be acute or chronic is associated with early delivery.

7. Previous history of preterm birth.

8. Uterine malformations, trauma to the uterus, low lying placenta, premature separation of placenta, hypertensive disorders, preterm cervical shortening, previous cervical surgery, premature rupture of membranes, and chorio-amnionitis also contribute to prematurity.

9. Fetal conditions such as nonreassuring testing of fetal well-being, IUGR, or severe hydrops may require preterm delivery.

10. Inadvertent early delivery because of incorrect estimation of gestational age (GA) is increasingly uncommon.[52]

C. **Problems of preterm birth**:

They are related to difficulty in extrauterine adaptation due to immaturity of organ system including respiratory, cardiovascular, neurologic, gastrological, renal and metabolic system.[53]

1. **Respiratory**:

a. Perinatal depression in the delivery room due to poor transition to breathing.

b. Respiratory Distress Syndrome due to surfactant deficiency and pulmonary immaturity

c. Apnea due to immaturity in mechanisms controlling breathing.

d. Chronic lung disease (CLD) of prematurity formerly called broncho pulmonary dysplasia (BPD), Wilson-Mikity disease, and chronic pulmonary insufficiency of prematurity.[54]

2. **Neurologic**:

Preterm infants have a higher risk for neurologic problems, including the following:

a. Perinatal depression

b. Intracranial Hemorrhage (ICH)

3.Cardiovascular. Preterm infants may present with cardiovascular problems, including the following:

i. Hypovolemia.

ii. Cardiac dysfunction.

iii. Sepsis-induced vasodilation

b. **Patent ductus arteriosus** is common and may lead to pulmonary over-circulation and diastolic hypotension .

4. **Hematologic**. Conditions for which preterm infants are at higher risk in-clude the following:

a. Anaemia

b. Hyperbilirubinemia.

5. **Nutritional**.

Preterm infants require specific attention to the content, caloric density, volume and route of feeding.[52]

6. **Gastrointestinal**.

Necrotizing enterocolitis is an important complication of preterm infants. formula feeds may also precipitate necrotising enterocolitis.

7. **Metabolic**. In glucose and calcium metabolism, are more common in preterm infants.

8. **Renal**.

Immature kidneys are characterized by decreased glomerular filtration rate. preterm infants have decreased ability of the kidneys to excrete water and solutes and so the fluid and electrolyte administration should be monitored continuously.

9. **Temperature regulation**.

Preterm infants are especially susceptible to hypothermia and hyperthermia.[55]

10. **Immunologic**.

Because of deficiency in both humoral and cellular response, preterm infants are at greater risk for infection than are term infants.

11**. Ophthalmologic**.

Retinopathy of prematurity may develop in the immature retina of infants <32 weeks or with birth weight <1,500 g .[55]

INTRA UTERINE GROWTH RETARDATION (IUGR)

Definition:

Though many use the terms “small for gestational age” (SGA) and “intrauterine growth retardation” (IUGR) interchangeably, they refer to two subtly different populations. SGA describes a neonate whose birth weight or birth crown-heel length is <10th percentile for GA or <2 standard deviations (SD) below the mean for the infant’s GA (approximately the 3rd percentile for GA).[56]

IUGR describes diminished growth velocity in the fetus as documented by at least two intrauterine growth assessments. Babies who are constitutionally SGA are at overall lower risk compared to those who are IUGR due to some pathologic process.[57]

Potential complications related to IUGR:

i. Congenital anomalies.

ii. Perinatal depression.:Perinatal/neonatal depression is a clinical, descriptive term that pertains to the condition of the infant on physical examination in the immediate postnatal period (i.e., in the first hour after birth).

The clinical features of infants with this condition include depressed mental status, muscle hypotonia, and possibly distur-bances in spontaneous respiration and cardiovascular function. This term makes no association with the prenatal or later postnatal condition, physical exam, laboratory tests, imaging studies, or electroencephalograms (EEGs).

iii. Meconium aspiration: Acute or chronic hypoxia and/or infection can result in the passage of meconium in utero. In this setting, gasping by the fetus or newly born infant can cause aspiration of amniotic fluid contaminated by meconium. Meconium aspiration before or during birth can obstruct airways, interfere with gas exchange and cause severe respiratory distress .[58]

iv. Pulmonary hemorrhage.

v. Persistent pulmonary hypertension:

Persistent pulmonary hypertension of the newborn (PPHN) is defined as the failure of the normal circulatory transition that occurs after birth. It is a syndrome characterized by marked pulmonary hypertension that causes hypoxemia secondary to right-to-left shunting of blood.

PPHN is often associated with the following signs and symptoms of perinatal distress:

- Asphyxia,Tachypnea, respiratory distress,Loud, single second heart sound (S2) or a harsh systolic murmur (secondary to tricuspid regurgitation)
- Low Apgar scores,Meconium staining,Cyanosis; poor cardiac function and perfusion,
- Systemic hypotension and Symptoms of shock.[56]

vi. Hypotension.

vii. Hypoglycemia from depletion of glycogen stores.

viii. Hypocalcemia.

ix. Hypothermia from depletion of subcutaneous fat.

x. Dyslipidemia.

xi. Polycythemia.

xii. Neutropenia.

xiii. Thrombocytopenia.

xiv. Acute tubular necrosis/renal insufficiency.[58]

Management of maternal anaemia:

Preventive measures to prevent anaemia need to be considered when diagnosis is made.

1. Proper counselling to avoid frequent childbirth.Atleast 2 to 3 years gap is needed between pregnancies.

2. Prophylactic iron therapy is given during pregnancy.

It is recommended that 100 mg of iron and 500 µg of folic acid must be given to all pregnant women in second and in third trimester of pregnancy.[43]

Treatment:

Depends on the degree of anaemia and period of gestation at diagnosis.

If pregnant women is severely anaemic and period of gestation is near term,she requires blood transfusion.In anaemic mothers blood transfusion is associated with risk of congestive heart failure and preterm labour.So it is better to transfuse packed cells slowly under cover of a diuretic.[44]

Oral treatment:

Ferrous salts are found to be better absorbed and more effective. Ferrous sulphate,ferrous carbonate,ferrous fumarate and gluconates are all used in dose of 200 mg three times a day.Response to treatment is observed by increase in reticulocytes within week of starting iron therapy and Hb levels begin to rise.[45,46]

Parenteral therapy:

This is indicated when pregnant mother is unable to take oral therapy because of side effects or is non compliant.It is also indicated when cases are seen for first time during last 8- 10 weeks with severe anaemia.[47]

Intra muscular therapy:

Iron dextran complex is reported to be least toxic when injected intra muscularly.Injection of iron is given by Z technique (pulling the skin and subcutaneous tissue to one side before inserting the needle).Oral iron is stopped at least 24 hours prior to injection to minimise reaction.[47]

Iron sorbitol citric acid complex is another preparation of iron for i.m.injection.

Procedure of injections:

After an initial test dose of 1 ml ,injections are given daily or on alternate days in doses of 2ml intramuscularly.To prevent the dark staining of the skin,over the injection sites and to minimise pain,the injections are given with a two inch needle deep in to the upper outer quadrant of the buttock using a Z technique(pulling the skin and the subcutaneous tissues to one side before inserting the needle).[48]An additional precaution is to inject small quantity of air or saline down the

needle before withdrawing it.These procedures prevent even a slight drop of solution to come beneath the skin surface so as to stain it.

Intravenous therapy:

The deficit is first calculated using formula:

2.4×weight(kg) ×(15-Hb of patient)+ 1000 gms

After calculating total deficit ,iron dextran is diluted in 500 ml of 5% glucose as slow intravenous infusion. When reactions like rigors, chest pain and palpitation are observed drip should be stopped immediately.[48]

Before starting infusion, immediate resuscitation should be ready. Administration of anti histaminic minimises the reaction.

Advantages of intravenous route:

1. It eliminates repeated and painful intramuscular injections.
2. The treatment is completed in a day and the patient may be discharged much earlier from the hospital.
3. It is less costly compared to the repeated muscular therapy.

LIMITATIONS OF INTRAVENOUS THERAPY:

1. as the maximum haemoglobin response does not appear before 4 to 9 weeks, the method is unsuitable if atleast 4 weeks time is not available to raise the haemoglobin to a safe level.
2. Contraindicated in cases of previous history of allergic reaction to parenteral preparations.[49]

BLOOD TRANSFUSION

Indications of blood transfusion:

1. To correct anaemia due to blood loss and to combat postpartm haemorrhage.
2. Patient with severe anaemia seen in later months of pregnancy
3. Refractory anaemia
4. Associated infection.[47]

Advantages of the blood transfusion:

1. Increase the oxygen carrying capacity of the blood.
2. Haemoglobin from the haemolysed red cells can be used for the formation of new red cells.

3. Supplies the natural constituents of blood like proteins, antibodies

4. Stimulates erythropoiesis.

5. Improvement is expected after 3 days.[49]

Cord blood hemoglobin of the newborn is an important indicator of anaemia in newborn at birth. It was thought previously what ever may be the haemoglobin level in the mother ,baby will not be anaemic. Recent studies are showing a linear relationship between cord blood haemoglobin and maternal haemoglobin.[59]

Studies conducted to assess the impact of maternal anaemia on cord blood haemoglobin in newborn were done with lesser sample size.Still further studies are needed to confirm positive correlation between maternal Hb levels and cord blood Hb levels in newborn.[60] So this study was aimed to assess the effect of maternal anemia on cord blood haemoglobin in newborn.

AIM OF THE STUDY

To study the impact of maternal anaemia on cord blood haemoglobin of neonates.

REVIEW OF LITERATURE

Neonatal survival outcome can be increased significantly by improvement in the care of high risk mothers and also in neonatal care and treatment. Better understanding of maternal antenatal factors including maternal anaemia is necessary to improve neonatal survival rate.

CORD BLOOD HAEMOGLOBIN

Normal value of mean cord blood haemoglobin is 16.8g/dl. Estimation of haemoglobin of neonates from cord blood is useful in diagnosing neonatal anaemia.[60]

Mamoury GH, Hamedy AB et al, in study in 2003 titled "Cord Hemoglobin in Newborns in Correlation with Maternal Hemoglobin in Northeastern Iran" concluded that no association was found between cord blood haemoglobin and maternal Hb levels.[60]

TABLE SHOWING NORMAL RED BLOOD CELL VALUES FROM 18 WEEKS OF GESTATION TO 14 WEEKS OF LIFE.[116]

AGE	HEMOGLOBIN (g/dL)	HEMATOCRIT (%)	MCV (μ^3)	RETICULOCYTES (%)
GESTATIONAL (wk)				
18-20*	11.5 ± 0.8	36 ± 3	134 ± 8.8	N/A
21-22*	12.3 ± 0.9	39 ± 3	130 ± 6.2	N/A
23-25*	12.4 ± 0.8	39 ± 2	126 ± 6.2	N/A
26-27	19.0 ± 2.5	62 ± 8	132 ± 14.4	9.6 ± 3.2
28-29	19.3 ± 1.8	60 ± 7	131 ± 13.5	7.5 ± 2.5
30-31	19.1 ± 2.2	60 ± 8	127 ± 12.7	5.8 ± 2.0
32-33	18.5 ± 2.0	60 ± 8	123 ± 15.7	5.0 ± 1.9
34-35	19.6 ± 2.1	61 ± 7	122 ± 10.0	3.9 ± 1.6
36-37	19.2 ± 1.7	64 ± 7	121 ± 12.5	4.2 ± 1.8
38-40	19.3 ± 2.2	61 ± 7	119 ± 9.4	3.2 ± 1.4
POSTNATAL (DAYS)				
1	19.0 ± 2.2	61 ± 7	119 ± 9.4	3.2 ± 1.4
2	19.0 ± 1.9	60 ± 6	115 ± 7.0	3.2 ± 1.3
3	18.7 ± 3.4	62 ± 9	116 ± 5.3	2.8 ± 1.7
4	18.6 ± 2.1	57 ± 8	114 ± 7.5	1.8 ± 1.1
5	17.6 ± 1.1	57 ± 7	114 ± 8.9	1.2 ± 0.2
6	17.4 ± 2.2	54 ± 7	113 ± 10.0	0.6 ± 0.2
7	17.9 ± 2.5	56 ± 9	118 ± 11.2	0.5 ± 0.4
POSTNATAL (wk)				
1-2	17.3 ± 2.3	54 ± 8	112 ± 19.0	0.5 ± 0.3
2-3	15.6 ± 2.6	46 ± 7	111 ± 8.2	0.8 ± 0.6
3-4	14.2 ± 2.1	43 ± 6	105 ± 7.5	0.6 ± 0.3
4-5	12.7 ± 1.6	36 ± 5	101 ± 8.1	0.9 ± 0.8
5-6	11.9 ± 1.5	36 ± 6	102 ± 10.2	1.0 ± 0.7
6-7	12.0 ± 1.5	36 ± 5	105 ± 12.0	1.2 ± 0.7
7-8	11.1 ± 1.1	33 ± 4	100 ± 13.0	1.5 ± 0.7
8-9	10.7 ± 0.9	31 ± 3	93 ± 12.0	1.8 ± 1.0
9-10	11.2 ± 0.9	32 ± 3	91 ± 9.3	1.2 ± 0.6
10-11	11.4 ± 0.9	34 ± 2	91 ± 7.7	1.2 ± 0.7
11-12	11.3 ± 0.9	33 ± 3	88 ± 7.9	0.7 ± 0.3
12-14	11.9	37	86.8	0.9

Sweet et al,in UK in 2001 in his study titled "Study of maternal influences on fetal iron status at term using cord blood transferrin receptors "had shown that mothers with iron deficiency anaemia during pregnancy gave birth to newborns with lower Hb levels.[59]

Najeeba C.M et al in their study have observed that 18 mothers(36%) had mild anaemia with haemoglobin value between 9-11g/dl,2 mothers(4%) had moderate anaemia with haemoglobin value between 4-7g/dl,and only one had very severe anaemia (<4g/dl).29 (58%)mothers had normal haemoglobin level.The cord blood haemoglobin values reduce as the haemoglobin level of the mother reduces,but the reduction is not statistically significant.The mean birth weight reduces in babies born to moderate anaemic mothers.

Nadia Mudher-Hilli et al, in 2010 in their study titled" The Effect of Maternal Anaemia on Cord Blood Haemoglobin & Newborn Birth Weight" done in Department of Gynaecology Obstetrics/ College of Medicine/ Babylon University stated that there is a positive correlation between maternal haemoglobin and fetal cord blood haemoglobin.[61]

Aarthi s,kusam M, Satnam S et al, in 2013 conducted a study in Gian Sagar Medical college and hospital,Rajpura found that cord blood haemoglobin appears to show a linear relationship with maternal

haemoglobin with cord blood haemoglobin being less in mothers who have anaemia.Mother who had severe anaemia had babies with lower cord blood haemoglobin.[62]

Debbarmarubi et al, 1n 2015 conducted study at regional institute of medical sciences, imphal, department of physiology in collabaration with obstetrics & gynaecology department titled "Effect of Maternal Anaemia on Cord Haemoglobin And Birth Weight of Newborns" found a linear relationship between maternal haemoglobin, cordblood haemoglobin and birth weight of the newborns.[63]

Lund and Nhonoli et al found that the newborn of iron deficient mothers had significantly lower levels of haemoglobin and iron in the cord blood.[64,65] Singla et al found that maternal haemoglobin had a linear correlation with haemoglobin and iron levels in the cord blood.[66] Fenton et al and Milman et al found that maternal ferritin correlated with cord and newborn ferritin levels.[67]

However Cantwell et al showed that mothers who were given less than adequate iron therapy during pregnancy had babies with similar cord-blood haemoglobin levels.[68]Turkay et al found no correlation between maternal haemoglobin at 16 and 34 weeks gestation and newborn haemoglobin parameters.[69]

Bhargava et al found maternal iron depletion did not adversely affect newborn haemoglobin levels.[70] Sturgeon demonstrated that fetal-cord-bloodhaemoglobin levels were similar in anaemic and non-anaemic mothers.[71]

Rusia et al found that maternal haemoglobin did not correlate with cord blood haemoglobin, but maternal haemoglobin were found to correlate positively with cord ferritin.[72]

NEONATAL ANAEMIA

BACKGROUND

Haemoglobin levels increases with advancing gestational age: At term, Cord blood hemoglobin is 16.8 g/dL (14-20 g/dL); hemoglobin levels in very low birthweight (VLBW) infants are 1-2 g/dL less than those in term infants. Haemoglobin value less than the normal level of haemoglobin for birthweight and postnatal age is defined as anemia. Physiologic decrease in haemoglobin content is noticed at 8-12 wks in term infants (haemoglobin, 11 g/dL) and at about 6 wk in premature infants (7-10 g/dL).[73]

I. HEMATOLOGIC PHYSIOLOGY OF THE NEWBORN

Significant changes occur in the red blood cell (RBC) mass of an infant during the neonatal period and ensuing months. The evaluation of

anemia must take into account this developmental process, as well as the infant's physiologic needs.[74]

A. Normal development: The physiologic anemia of infancy

1. In utero, the fetal aortic oxygen saturation is 45%, the erythropoietin levels are high, and the RBC production is rapid. The fetal liver is the major site of erythropoietin production

2. After birth, the oxygen saturation is 95%, and the erythropoietin is undetectable. RBC production by day 7 is 1/10th the level in utero. Reticulocyte counts are low, and the hemoglobin level falls .

3. Despite dropping haemoglobin levels, the ratio of haemoglobin A to haemoglobin F increases and the levels of 2,3-diphosphoglycerate (2,3-DPG) (which inter-acts with hemoglobin A to decrease its affnity for oxygen, thereby enhancing oxygen release to the tissues) are high. As a result, oxygen delivery to the tissues actually increases. This physiologic "anaemia" is not a functional anaemia in that oxygen delivery to the tissues is adequate. Iron from degraded RBCs is stored.[75]

4. At 8 to 12 weeks, hemoglobin levels reach their nadir, oxygen delivery to the tissues is impaired, renal erythropoietin production is stimu-lated, and RBC production increases.

5. Infants who have received transfusions in the neonatal period have lower nadirs than normal because of their higher percentage of hemoglobin A.[76]

6. During this period of active erythropoiesis, iron stores are rapidly utilized. The reticuloendothelial system has adequate iron for 15 to 20 weeks in term infants. After this time, the hemoglobin level decreases if iron is not supplied.[77]

B. **Anaemia of prematurity** is an exaggeration of the normal physiologic anaemia.

1. RBC mass and iron stores are decreased because of low birth weight; however, hemoglobin concentrations are similar in preterm and term infants.

2. The hemoglobin nadir is reached earlier than in the term infant because of the following:

   a. RBC survival is decreased in comparison with the term infant.

   b. There is a relatively more rapid rate of growth in premature babies than in term infants. For example, a premature infant gaining 150 g/week requires approximately a 12 mL/week increase in total blood volume.[78]

c. Many preterm infants have reduced red cell mass and iron stores because of iatrogenic phlebotomy for laboratory tests. This has been somewhat amelio-rated with the use of microtechniques.

d. Vitamin E deciency is common in small premature infants, unless the vitamin is supplied exogenously

3. The hemoglobin nadir in premature babies is lower than in term infants, because erythropoietin is produced by the term infant at a hemoglobin level of 10 to 11 g/dLand is produced by the premature infant at a hemoglobin level of 7 to 9 g/dL.

4. Iron administration before the age of 10 to 14 weeks does not increase the nadir of the hemoglobin level or diminish its rate of reduction. However, this iron is stored for later use.[78]

5. Once the nadir is reached, RBC production is stimulated, and iron stores are rapidly depleted because less iron is stored in the premature infant than in the term infant.

Causes of neonatal anaemia

1. **Obstetric causes** of blood loss, including the following malformations of placenta and cord:

a. Abruptio placentae

b. Placenta previa.

c.Incision of placenta at cesarean section

d. Rupture of anomalous vessels (e.g., vasa previa, velamentous insertion of cord,

e. Hematoma of cord caused by varices or aneurysm.

f. Rupture of cord (more common in short cords)[79]

2. **Occult blood loss**:

a. **Fetomaternal bleeding** may be chronic or acute. It occurs in 8% of all pregnancies;

The diagnosis of this problem is by Kleihauer-Betke stain of maternal smear for fetal cells. Chronic fetal-to-maternal transfusion is suggested by a reticulocyte count >10%.[80]

Many conditions may predispose to this type of bleeding

i. Placental malformations—chorioangioma or choriocarcinoma

ii. Obstetric procedures—traumatic amniocentesis, external cephalic version, internal cephalic version, breech delivery.

iii. Spontaneous fetomaternal bleeding

b. **Fetoplacental bleeding:**

i. Chorioangioma or choriocarcinoma with placental hematoma

ii. Tight nuchal cord or occult cord prolapse.

c. **Twin-to-twin transfusion**

3. Bleeding in the neonatal period may be due to the following causes:[80]

a. Intracranial bleeding associated with:

i. Prematurity

ii. Second twin

iii. Breech delivery

iv. Rapid delivery

v. Hypoxia

b. Massive cephalhematoma, subgaleal hemorrhage.

c. Retroperitoneal bleeding.

d. Ruptured liver or spleen.

e. Adrenal or renal hemorrhage

f. Gastrointestinal bleeding (maternal blood swallowed from delivery or breast should be ruled out by the Apt test) :

i. Peptic ulcer ii. Necrotizing enterocolitis iii. Nasogastric catheter

g. Bleeding from umbilicus.

4. Iatrogenic causes. Excessive blood loss may result from blood sampling with inadequate replacement.

B. **Hemolysis**

1. **Immune hemolysis** :

a. Rh incompatibility

b. ABO incompatibility

c. Minor blood group incompatibility (e.g., c, E, Kell, Duffy)

d. Maternal disease (e.g., lupus), autoimmune hemolytic disease, rheumatoid arthritis (positive direct Coombs test in mother and newborn, no antibody to common red cell antigen Rh, AB, etc.), or drugs.[78]

2. **Hereditary RBC disorders:**

a. **RBC membrane defects** such as spherocytosis, elliptocytosis, or stomatocytosis.

b. **Metabolic defects**.

Glucose-6-phosphate dehydrogenase (G6PD) deficiency ,

pyruvate kinase deficiency,

5' nucleotidase deficiency, and glucose-phosphate isomerase deficiency.

c. **Hemoglobinopathies** :

i. α- and γ-Thalassemia syndromes

ii. α - and γ -Chain structural abnormalities.

3. **Acquired hemolysis**

a. Infection: bacterial or viral

b. Disseminated intravascular coagulation

c. Vitamin E deficiency and other nutritional anemias

d. Microangiopathic hemolytic anemia,

cavernous hemangioma, renal artery stenosis and severe coarctation of the aorta

c. Diminished RBC production

Is manifested by a decreased Hct, decreased reticulocyte count, and normal bilirubin level.[76]

1. Diamond-Blackfan syndrome

2. Congenital leukemia or other tumor

3. Infections, especially rubella and parvovirus

4. Osteopetrosis, leading to inadequate erythropoiesis

5. Drug-induced suppression of RBC production

6. Physiologic anaemia or anaemia of prematurity[79]

PATHOPHYSIOLOGY OF NEONATAL ANAEMIA

Gradual and progressive decline in Hb concentration is noted in the eight to ten weeks post natal age in the neonates.

The decline of haemoglobin in term infants reaches a nadir of approximately 11 to 12 g/dL and is considered as early anemia. This is a normal physiological process and does not need any treatment.It is a physiological condition believed to be due to several factors. The most important factor is increase in tissue oxygenation experience. There is a switching from placental dependency to dependency on the lungs for oxygen exchange.[73]

This transition occurs due to decreased responsiveness to hypoxia in fetus relative to adults. There is a lower set point of tissue oxygenation for fetuses compared to to adults. Other factors contributing to the postnatal decrease in haemoglobin levels include increased rate of body growth, low blood erythropoietin (EPO) levels and shortened RBC lifespan. The cause of the anemia can be identified by history and physical examination.[74]

Hemoglobin Changes in Babies in the First Year of Life[117]

Week	Hemoglobin level		
	Term babies	Premature babies (1,200–2,500 g)	Small premature babies (<1,200 g)
0	17.0	16.4	16.0
1	18.8	16.0	14.8
3	15.9	13.5	13.4
6	12.7	10.7	9.7
10	11.4	9.8	8.5
20	12.0	10.4	9.0
50	12.0	11.5	11.0

Source: Glader B, Naiman JL. Erythrocyte disorders in infancy. In: Taeusch HW, Ballard RA, Avery ME, eds. *Diseases of the Newborn*. Philadelphia: WB Saunders; 1991.

Hemoglobin Nadir in Babies in the First Year of Life[117]

Maturity of baby at birth	Hemoglobin level at nadir	Time of nadir (wk)
Term babies	9.5–11.0	6–12
Premature babies (1,200–2,500 g)	8.0–10.0	5–10
Small premature babies (<1,200 g)	6.5–9.0	4–8

Source: Glader B, Naiman JL. Erythrocyte disorders in infancy. In: Taeusch HW, Ballard RA, Avery ME, eds. *Diseases of the Newborn*. Philadelphia: WB Saunders; 1991.

CLASSIFICATION OF ANAEMIA IN THE NEWBORN

Reticulocytes	Bilirubin	Coombs test	RBC morphology	Diagnostic possibilities
Normal or ↓	Normal	Negative	Normal	Physiologic anemia of infancy or prematurity; congenital hypoplastic anemia; other causes of decreased production
Normal or ↑	Normal	Negative	Normal	Acute hemorrhage (fetomaternal, placental, umbilical cord, or internal hemorrhage)
↑	Normal	Negative	Hypochromic microcytes	Chronic fetomaternal hemorrhage
↑	↑	Positive	Spherocytes Nucleated RBC	Immune hemolysis (blood group incompatibility or maternal autoantibody)
Normal or ↑	↑	Negative	Spherocytes	Hereditary spherocytosis
Normal or ↑	↑	Negative	Elliptocytes	Hereditary elliptocytosis
Normal or ↑	↑	Negative	Hypochromic microcytes	α- or γ-Thalassemia syndrome
↑	↑	Negative	Spiculated RBCs	Pyruvate kinase deficiency
Normal or ↑	Normal or ↑	Negative	Schistocytes and RBC fragments	Disseminated intravascular coagulation; other microangiopathic processes
↑	↑	Negative	Bite cells (Heinz bodies with supravital stain)	Glucose-6-phosphate dehydrogenase deficiency
Normal, ↑ or ↓	↑	Negative	Normal	Infections; enclosed hemorrhage (cephalhematoma)

↓ = decreased; ↑ = increased; RBC = red blood cell.
Source: Adapted from the work of Dr. Glader B. *Director of Division of hematology-oncology*. California: Children's Hospital at Stanford, 1991.[3]

CLINICAL FINDINGS

In case of mild anaemia,there may be no signs.With more severe anemia, findings include:[75]

CLINICAL FEATURES OF ANAEMIA

- Pallor
- Tachycardia
- Tachypnoea
- Apnoea
- Increased oxygen requirements
- Lethargy
- Poor feeding
- Hepatosplenomegaly
- Jaundice
- Wide pulse pressure
- Hypotension
- Metabolic acidosis with severe anaemia

III. DIAGNOSTIC APPROACH TO ANAEMIA IN THE NEWBORN

The family history should include questions about anemia, jaundice, gallstones, and splenectomy.

B. The obstetric history should be evaluated.

C. The physical examination may reveal an associated abnormality and provide clues to the origin of the anemia.

1. Acute blood loss leads to shock, with cyanosis, poor perfusion, and acidosis.

2. Chronic blood loss produces pallor, but the infant may exhibit only mild symptoms of respiratory distress or irritability.

3. Chronic hemolysis is associated with pallor, jaundice, and hepatosplenomegaly.[77]

D. Complete blood cell count.

Capillary blood Hct is 2.7% to 3.9% higher than venous Hct. Warming the foot reduced the difference from 3.9% to 1.9% .

E. Reticulocyte count

Elevated with chronic blood loss and hemolysis, depressed with infection and production defect.

F. Blood smear

G. Coombs test and bilirubin level [78]

H. Apt test on gastrointestinal blood of uncertain origin.

I. Kleihauer-Betke preparation of the mother's blood. A 50-mL loss of fetal blood into the maternal circulation will show up as 1% fetal cells in the maternal circulation.

J. Ultrasound of abdomen and head.

K. Parental testing. Complete blood cell count, smear, and RBC indices are useful screening studies. Osmotic fragility testing and RBC enzyme levels (e.g., G6PD, pyruvate kinase) may be helpful in selected cases.

L. Studies for infection. Toxoplasmosis, rubella, cytomegalovirus (CMV), and herpes simplex

M. Bone marrow examination is rarely used, except in cases of bone marrow failure from hypoplasia or tumor.[79-80]

MANAGEMENT OF NEONATAL ANAEMIA

Severity of anemia. should be assessed as the management dffers.

1. Prenatal causes : significant fetal anaemia occurs in hemolytic disease and parvovirusB19, where as other conditions have mild anaemia.[81]

2. Postnatal:

A. Anaemia of prematurity:

The important methods management are:

•frequent blood samling for laboratory tests should be avoided

•Treatment with recombinant human erythropoietin should be considered

• packed red blood cells transfusion (PRBCs) for severe anaemia.

In case of Severe anaemia: With severe, symptomatic anaemia, the infant's cardiovascular system may not be able to tolerate the increased blood volume from simple transfusion of packed red blood cells. In such cases, performing a partial exchange transfusion with Packed red blood cells (PRBCs) is done.[82]

Volume of PRBCs to exchange, use the following formula:

Volume of PRBCs = (Desired haematocrit – Patient's Haematocrit)x weight (kg) x 90 cc/kg

for exchange (cc) (PRBC Hct – Patient's Hct)

Indications for transfusion

The decision to transfuse must be made in consideration of the infant's condition and physiologic needs.[83]

Transfusion with adult RBCs provides the added benefit of lowered haemoglobin oxygen affinity, which augments oxygen delivery to tissues. Blood should be fresh (3–7 days old) to ensure adequate 2,3-DPG levels.

Isovolemic transfusion with high hematocrit (Hct)-packed RBCs may be required for severely anemic infants, when routine transfusion of the volume of packed RBCs necessary to correct the anemia would result in circulatory overload .[84]

TRANSFUSION GUIDELINES

1. Asymptomatic infants with Hct <21% and reticulocytes <100,000/UL (2%)
2. Infants with Hct <31% and hood O_2 <36% or mean airway pressure <6 cm H_2O by CPAP or IMV or >9 apneic and bradycardic episodes per 12 h or 2/24 h requiring bag-and-mask ventilation while on adequate methylxanthine therapy or HR >180/min or RR >80/min sustained for 24 h or weight gain of <10 g/d for 4 d on 100 Kcal/kg/d or having surgery
3. Infants with Hct <36% and requiring >35% O_2 or mean airway pressure of 6–8 cm H_2O by CPAP or IMV
CPAP = continuous positive airway pressure by nasal or endotracheal route; HR = heart rate; Hct = hematocrit; IMV = intermittent mandatory ventilation; RR = respiratory rate. From the multicenter trial of recombinant human erythropoietin for preterm infants. *Source*: Data from Strauss RG. Erythropoietin and neonatal anemia. *N Engl J Med* 1994;330(17):1227–1228.

General Principles of packed red blood cells transfusion

RBCs provide oxygen carrying capacity for patients whose blood lacks sufficient oxygen carrying capacity due to anemia, hemorrhage or hemoglobinopathySeveral types of RBC units are available that vary in the preservatives added.[85-87]

Chemical additives delay storage damage to RBCs allowing for extended storage times. a. Anticoagulant-preservative solution units:

These units contain approximately 250 mL of a concentrated solution of RBCs. The average hematocrit of these units is 70% to 80%. In addition, these units contain 62 mg of sodium, 222 mg of citrate, and 46 mg of phosphate.[88]

Three types of units are currently approved for use. These are

i. CPD. This contains 773 mg of dextrose and has a 21-day shelf life.

ii. CP2D. This contains 1,546 mg of dextrose and has a 21-day shelf life.

iii. CPDA-1. This contains 965 mg of dextrose and 8.2 mg of adenine and has a 35-day shelf life. This is the most widely used of the anticoagulant preservative solution units.

b. Additive solution units. Most RBC units used are additive units. Three additive solutions are currently approved for use. Each of these units contains approximately 350 mL, has an average hematocrit of 50% to 60%, and has a 42-day shelf life. [90]

Several changes occur in Packed RBCs during storage:

a. The pH decreases from 7.4–7.55 to pH 6.5–6.6 at the time of expiration.

b. Potassium is released from the RBC. The initial plasma K+ concentration is 4.2 mM and increases to 78.5 mM in CPDA-1 units at

day 35 and 45 to 50 mM in additive solution units on day 42. CPDA-1 units contain about one-third the plasma volume as additive units so the total amount of extracellular potassium is similar in all units of the same age.[91]

c. 2,3-diphosphoglycerol (2,3-DPG) levels drop rapidly during the first 2 weeks of storage. This increases the affinity of the hemoglobin for oxygen and decreases its efficiency in delivering oxygen to tissue. The 2,3-DPG levels replenish over several hours after being transfused.

3. Toxicity.

Although there are theoretical concerns that mannitol may cause a rapid diuresis and adenine may be a nephrotoxin in the premature infant, case reports and case series have found no risk associated with additive solution units. Hence, some hospitals transfuse additive solution units to neonates. [92]

Nonadditive solution units or washed additive solution units for larger transfusions such as exchange transfusions are used.

Dosing and administration

The dose for a simple transfusion is 5 to 15 mL/kg which is transfused at a rate of 5 mL/kg/hour. This may be adjusted depending on

the severity of the anaemia and the patient's ability to tolerate increase in intra-vascular volume.

Side effects of transfusion of Packed red blood cells:

1. **Acute transfusion reactions:**

a. Acute hemolytic transfusion reactions.

These reactions are usually due to incompatibility of donor RBCs with antibodies in the patient's plasma. The antibodies usually responsible for acute hemolytic transfusion reactions are iso-hemagglutinins (anti-A, anti-B). These reactions are rare in neonates because they do not make isohemagglutinins until they are 4 to 6 months old. However, maternal isohemagglutinins can be present in the neonatal circulation.[92-93]

i. Symptoms. Possible symptoms include hypotension, fever, tachycardia, infusion site pain, and hematuria.

ii. Treatment.

Administer fluids and furosemide to protect kidneys. If necessary, treatment for hypotension with pressors and hemostatic agents for bleeding are used. Transfusion with compatible Packed RBCs may be needed.[94]

b. **Allergic transfusion reactions**.

These are unusual in neonates. Allergic reactions are due to antibodies in the patient's plasma that react with proteins in donor plasma.[93]

i. Symptoms. Mild allergic reactions are characterized by hives and possibly wheezing. More severe reactions can present such as anaphylaxis.

ii. Treatment. These reactions can be treated with antihistamines, bronchodilators and corticosteroids as needed. These reactions are usually specific to individual donors. If they are serious, RBCs and platelets can be washed.[94]

c. **Volume overload**.

Blood components have high oncotic pressure and rapid infusion can cause excessive intravascular volume. This can cause a sudden deterioration of vital signs. Chronically anemic neonates can be especially susceptible to volume overload from transfusions.[95]

d. **Hypocalcemia**.

Rapid infusion of components especially Fresh frozen plasma, can cause transient hypocalcemia.

e. **Hypothermia.** Cool blood can cause hypothermia. Transfusion through blood warmers can prevent this.

f. **Transfusion-associated acute lung injury (TRALI**).

This is often due to antibodies in donor plasma that react with the patient's histocompatibility (HLA) antigens. These reactions present as respiratory compromise and are more likely to occur with blood components containing significant amounts of plasma such as platelets or FFP.[96]

g**. Hyperkalemia**.

Extracellular potassium dosage is not significant for simple transfusions of 5 to 20 mL/kg. However, hyperkalemia can be important in large transfusions such as exchange transfusions or transfusions for major surgery. Ideally, fresher PRBC units can be provided for these transfusions. If fresh RBCs are unavailable, washing blood will temporarily reduce the extracellular potassium.[96]

h. **Febrile Nonhemolytic transfusion reactions**

These reactions are usually due to cytokines released from leukocytes in the donor unit. These occur less frequently if the unit is leukoreduced.

i. **Bacterial contamination** can occur but is relatively rare with RBC transfusions.

j. **Transfusion-associated graft-versus-host disease** (TA-GVHD).

Lymphocytes from donor blood components can mount an immune response against the patient. Patients are at risk if they are unable to mount immune responses against the transfused lymphocytes. Such patients include premature infants, infants with congenital immune deficienciesTransfusion associated-GVHD can be prevented by irradiation.[97]

B. Prophylaxis

1. Term infants should be sent home from the hospital on iron-fortified formula (2 mg/kg/day) if they are not breastfeeding .
2. Premature infants (preventing or ameliorating the anemia of prematurity)

Iron supplementation in the preterm infant prevents late iron deficiency. Supplementation of iron in premature infants at a dose of 2 to 4 mg of elemental iron/kg/day can be given once full enteral feeding is achieved

Vitamin E (15 to 25 IU of water-soluble form) is given daily upto 38 to 40 weeks postconceptional age.This is usually stopped at discharge from the hospitalThese infants should be followed up carefully and additional iron supplementation may be required.[98]

Recombinant human erythropoietin (rh-EPO) has been evaluated as a promising measure in ameliorating anemia of prematurity. Studies have shown that rh-EPO stimulates RBC production and decreases the frequency and volume of RBC transfusions administered to premature infants. [98]

UMBILICAL CORD BLOOD

History of Umbilical Cord Blood

- First successful related cord blood transplant conducted in France, on a six-year-old male patient suffering from Fanconi's Anemia done in 1988.
- First public bank for umbilical cord blood established in 1992 by Dr.Pablo at New York blood center.
- First unrelated cord blood transplant in the world is performed in 1993 by Dr. Joanne Kurtzberg at Duke University's Pediatric Blood and Marrow Program.[99]

- National Marrow Donor Program (NMDP) launched cord blood program in 1998
- The Stem Cell Research and Therapeutic Act of 2005, to create a national inventory of 150,000 diverse, high-quality cord blood samples done in 2005.
- Over 30,000 unrelated cord blood transplants performed in 2012.[100]

UMBILICAL CORD

The umbilical cord contains two umbilical arteries and one umbilical vein. Umbilical vessels are surrounded by connective tissue known as "Wharton's jelly". Length of the cord less than 35 cm is called as short cord and the length of the cord measuring more than 80 cm is considered as long cord .The average umbilical cord circumference is 3.6 cm at 40 weeks. In a term baby, area of the umbilical cord is 1.3 cm square.[101]

The length of the cord is an important parameter that affects the foetal activity. It is dependent on the tension caused by the freely moving foetus, mainly during the second trimester. The short cord is associated with decreased foetal activity. short cord may also be associated with maldevelopment of the central nervous system.[102] Certain developmental

abnormalities including Down syndrome .may be associated with short cord.

Fetal metabolic condition can be determined by the umbilical cord gas sampling at the moment of birth. Both arterial and venous umbilical cord blood samples for analysis are preferred.[103]

Normal Cord Blood Gas and pH (during and post labour)

At term	pH	Base Excess mmol/L	pO_2 mm Hg	pCO_2 mm Hg
UA	**7.10 – 7.38**	**-9.0 – 1.8**	**4.1 – 31.7**	**39.1 – 73.5**
UV	**7.20 – 7.44**	**- 7.7 – 1.9**	**30.4 – 57.2**	**14.1 – 43.3**

Equipment Required for cord blood sampling:[104]

. Gloves

. Heparinised syringes x 2

. Needles 21g x 2

. Face shield

. 5 clamps

. Optional (ice and water)

METHOD OF CORD BLOOD COLLECTION

Instructions for Collection of cord blood Prior to Expulsion of Placenta:

Double clamping of the umbilical cord is done after delivery of the infant and also prior to expulsion of the placenta.

⇩

Proposed needle insertion site of the cord is sterilised with the antiseptic prep pad.

⇩

Needle insertion site should be just above the clamp that remains on the cord.

⇩

Insertion of the needle of Collection Bag is done into the cord.

⇩

Collection of the blood is done as much as possible via gravity-flow, aiming to fill a minimum of 80 mL.

⇩

Cord will appear empty and pale when collection is completed.

⇩

Two tight knots are done in the tubing as close to the needle as possible.

⇩

Gentle rotation of the bag several times to thoroughly mix the cord blood with the anticoagulant is finally done.[105]

Instructions for Collection of cord blood following Expulsion of Placenta or After Caesarean Delivery:

Double-clamping and cutting of the umbilical cord is being done.

↓

Placenta and umbilical cord is placed in a sterile tray in such a way that fetal side is down.

↓

The placenta is placed on an elevated surface and umbilical cord is draped out of the tray.

↓

Proposed needle insertion site of the cord is sterilised with the antiseptic prep pad

↓

Needle insertion site should be just above the clamp that remains on the cord.

↓

Collection of the blood is done as much as possible via gravity-flow, aiming to fill a minimum of 80 mL.

↓

Two tight knots are done in the tubing as close to the needle as possible.

↓

Gentle rotation of the bag several times to thoroughly mix the cord blood with the anticoagulant is finally done.[106]

Procedure for labelling Cord Blood Sample :

- Collection Kit case ID is affixed to the Collection Bag and the Collection Bag is placed into Specimen Transport Bag. A cord blood sample in a heparinised syringe is stable for up to 60 minutes at room temperature.[107]
- The Collection Form is then filled
- Blood samples are labelled with Unique Number of mothers as the results are part of the maternal obstetric history.

CORD BLOOD USES

Umbilical cord blood was not stored before till the discovery of stem cells in the cord blood. Now cord blood of neonates are stored in the cord blood banks. There is no risk in collecting cord blood and it is safely preserved in the cord blood bank.Cord blood contains stem cells and these cells are haematopoietic stem cells capable of differentiating into cells of any organ.[108]

Since 1989, cord blood is being used to treat children with blood diseases and also in research used to treat adult patients. Cord blood cells has capacity to grow and they can develop into various types of cells throughout the body. They can be harvested after birth and stored for

future transplantation in patients with blood disorders and also in many types of cancer.

Haematopoietic stem cells (HSCs) are maintaining blood production throughout our lives since they can make every type of cell in the blood like RBC,WBCs and platelets.Cord blood is being used for years in bone marrow transplants for treating blood diseases.[109] Umbilical cord blood used as Hematopoietic Stem Cell Transplantation (HSCT) source for conditions transplanted with Peripheral Blood Progenitor Cell (PBPC).

Stem cells of the cord blood can produce specialised cells such as nerve cells. cord blood samples are used to treat children with cancerous blood disorders and also genetic blood diseases like Fanconi anaemia.[110] These include:

UMBILICAL CORD BLOOD USES IN DISEASES

- Hematological and Other Malignancies

Leukemias:

Chronic myelogenous leukemia(CML)

Acute myelogenous leukemia (AML)

Acute lymphoblastic leukemia (ALL)

Chronic lymphoblastic leukemia (CLL)

Lymphoma:

Hodgkin's disease

Non hodgkin's lymphoma

Histiocytosis X

Auto immune lympho proliferative disease

Myelodysplastic syndrome(MDS)

Myelo fibrosis

- Immune deficiencies
- Bone Marrow Failure Diseases
- Platelet Diseases:

Congenital thrombocytopenia

Glanzmann's thrombasthenia

Haemoglobinopathies

- Sickle Cell Disorders
- Thalassemia Disorders

Metabolic/ Storage disorders

- Hurler's disease
- Morquio syndrome
- Amyloidosis
- Gaucher's disease
- Krabbe's disease
- Niemann pick disease
- Lesch-Nyhan Syndrome
- Metachromatic Leukodystrophy

Red Blood Cell Diseases

White Blood Cell Diseases:

- Chronic granulomatous disease(CGD)
- Chediak Higashi syndrome
- Severe Combined Immune Deficiency (SCID)
- Wiskott-Aldrich Syndrome
- X-linked Hyper-IgM Syndrome
- X-linked Immune Dysregulation

Inherited Transfusion Dependent Anemias[111]

Cord blood is being transplanted into the patient, where the hematopoietic stem cells make new healthy blood cells to replace those damaged by medical treatment such as cancer chemotherapy.[110]

Cord blood offers alternative for bone marrow transplants for few patients. It can be stored frozen till it is needed. Umbilical cord blood is less likely than bone marrow to cause immune rejection such as Graft versus Host Disease than bone marrow. This means that cord blood need not to be perfectly matched as bone marrow to the patient.[112]

Stem cells derived from cord blood has therapeutic potential for neuroregeneration and also for improved functional outcome. Cord blood also finds its use in treatment of hypoxic brain injury.

Likely mechanism

Participation in blood vessel regeneration, suppression of the release of inflammatory cells from the spleen, improvement of survival of intrinsic cells via neurotropic factors.

Most important issue being timing of administration of cord blood with respect to time of injury: earlier the better.[113]

Umbilical cord blood transplants have some limitations. Two units of cord blood is needed for treatment of one adult. Many studies has shown cord blood uses to treat blood diseases only. For non-blood-related

diseases no therapies have been developed using hematopoietic cells from cord blood.

Several phase1 and phase 2 studies are in progress in evaluating the use of cord stem cells in the treatment of cerebral palsy,Type I diabetes, in traumatic brain injury and in neonatal encephalopathy.

These are the following tests performed on the cord blood:

- Complete blood count: CBC is an important paerameter to know the evidence of infection.
- Platelet count: Measure of blood's clotting ability
- Blood type: blood grouping typing
- Blood cultures : To test for aerobic and anaerobic bacteria ,in order to rule out infection.
- Positive blood culture may be useful in detecting abnormal bacteria in the mother or infant.
- Cord blood pH (acid level)-useful indicator of acidosis and this is useful in the diagnosis of perinatal hypoxia.[112]
- Low pH (< 7.25) indicates acidosis in the infant's bloodstream. Acidosis occurs if mother or baby does not get enough oxygen during delivery.

- Blood gas analysis : in order to evaluate the levels of oxygen and carbon dioxide levels in newborns.[113]

CORD BLOOD BANKING:

Prospective parents may seek information regarding umbilical cord blood banking. Balanced and accurate information regarding the advantages and disadvantages of public versus private banking should be provided. [114]

Health care providers should dispense the following information

1. There is clinical potential of hematopoietic stem cells found in cord blood.
2. Where logistically possible, collection and support of umbilical cord blood for public banking is encouraged.
3. The indications for autologous (self) transplantation are limited.
4. Private cord blood banking should be encouraged when there is knowledge of a family member, particularly a full sibling, with a current or potential medical condition (malignant or genetic) that could potentially benefit from cord blood transplantation.

5. Storing cord blood as “biologic insurance” should be discouraged because there is currently no scientific data to support autologous transplantation.[115]

MATERIALS AND METHODS

This study is a cross sectional type of study done at Govt.kilpauk medical college and hospital in the Department of paediatrics. The study protocol was approved by Ethical committee for research studies of kilpauk medical college and hospital.

Study design:

Cross sectional study

Study period :

6 months (March 2016-August 2016)

Study population:

Pregnant mothers attending the labour room in kilpauk medical college hospital and their babies delivered were included in the study.

Sample size:

400 pregnant mothers attending the labour room in kilpauk medical college and their babies delivered were included in this study.Sample size was calculated depending upon the prevalence of anaemia in antenatal mothers in previous studies by using the formula 4p/L^2.prevalance of anaemia in the previous study was around 50%.

INCLUSION CRITERIA

- Fullterm neonates [37-41weeks]
- Preterm neonates > 34 weeks
- Women with singleton pregnancies
- Primi/multiparity
- Babies born to normal vaginal deliveries/caesarean section
- Babies born with birth weight of 2-4 kg

EXCLUSION CRITERIA:

- Newborns with congenital malformations
- Birth asphyxia
- Twins
- Rh incompatability
- Maternal risk factors like Gestational diabetes mellitus,Pregnancy induced hypertension, placenta praevia and abruptio placenta.

METHODOLOGY:

400 pregnant mothers attending the labour room in kilpauk medical college and their babies delivered were included in this study. After the delivery of the baby, cord blood haemoglobin was collected. Double

clamping of the umbilical cord is done after delivery of the infant and also prior to expulsion of the placenta.Umbilical vein was identified and the needle insertion site was sterlised with antiseptic preparation pad. Cord blood sample was collected from the placental end of severed cord. About 2 ml of blood was aspirated from the umbilical vein using a sterile syringe and blood was transferred to a test tube containing EDTA.Collected blood samples were analysed in the pathology lab in automated analyser for haemoglobin estimation.

Predelivery maternal haemoglobin was estimated. Both cord blood and the maternal haemoglobin were determined. Based on the maternal haemoglobin values mothers were classified in to two groups,namely anaemic and non anaemic. Those mothers with haemogobin values less than 11g/dl were considered as anaemic and those mothers with haemoglobin more than 11g/dl were considered as non anaemic. Anaemic mothers with haemoglobin less than 11g/dl were classified into 3 groups,namely mild ,moderate and severe anaemia.

Cord blood haemoglobin values of anaemic and non anaemic group was compared. Among the anaemic mothers the mean cord blood haemoglobin in the three groups (mild, moderate, severe) were determined. The mean cord blood haemoglobin of each group was compared with the mean cord blood haemoglobin of the neonates born to the non anaemic mothers.

STATISTICAL ANALYSIS

Statistical analysis was performed using SPSS 21. Student t test was used to determine whether there was any significant difference between the two groups.

P value of lessthan 0.05 was taken as significant.

AUTOMATED ANALYSER

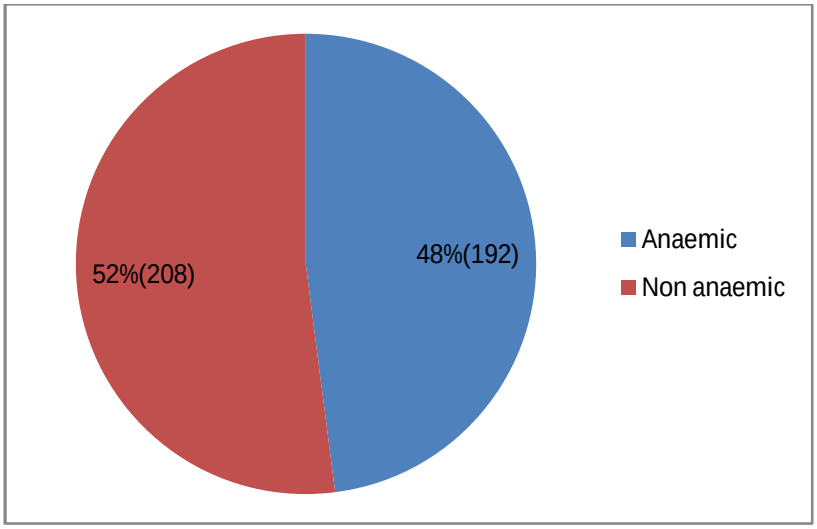
CORD BLOOD COLLECTION

RESULTS

TABLE SHOWING NUMBER AND PERCENTAGE OF ANAEMIC AND NON ANAEMIC MOTHERS

GROUP	Number of subjects	Percentage of subjects (%)
Anaemic mothers	192	48
Non anaemic mothers	208	52

PIE CHART SHOWING NUMBER OF ANAEMIC AND NON ANAEMIC MOTHERS



48 % of anaemic mothers and 52% of non anaemic mothers were included in our study.

TABLE SHOWING NUMBER OF ANAEMIC MOTHERS BASED ON ITS SEVERITY

Classification	No of anaemic mothers	Percentage(%)
Mild- 10-10.9 gm/dl	94	48.9
Moderate- 9.9-7 gm/dl	82	42.8
Severe- <7 gm/dl	16	8.3

Showing categorisation of anaemic mothers based on its severity:

BAR DIAGRAM SHOWING NUMBER OF ANAEMIC MOTHERS BASED ON ITS SEVERITY

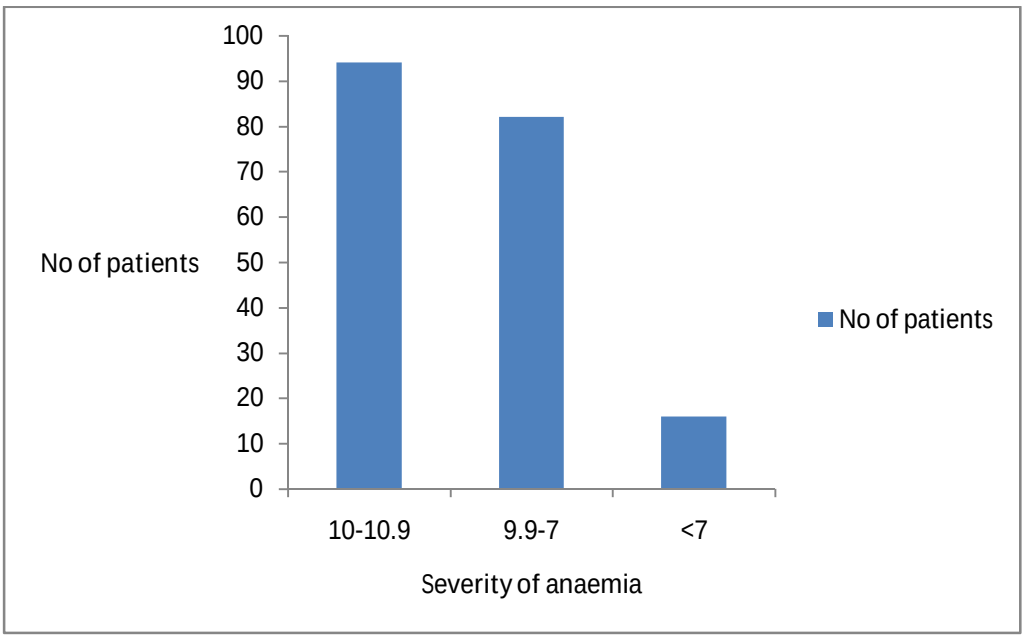


TABLE SHOWING NUMBER AND PERCENTAGE OF PRE TERM AND TERM BABIES:

Gestation	Number of babies	Percentage of babies
Pre term babies	52	13
Term babies	348	87

PIE CHART SHOWING NUMBER OF PRE TERM AND TERM BABIES

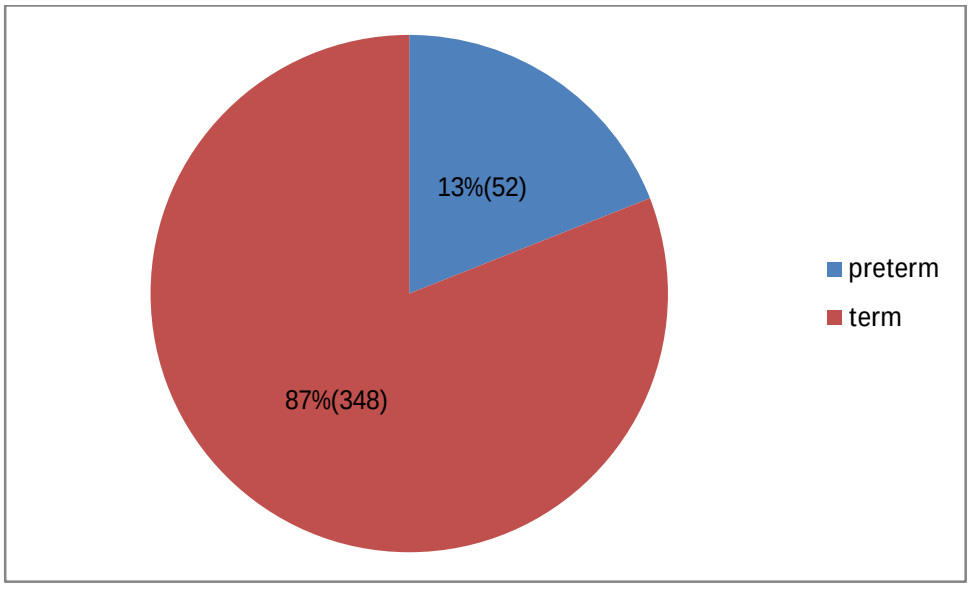


TABLE SHOWING NUMBER AND PERCENTAGE OF ANAEMIC MOTHERS BASED ON THEIR RURAL /URBAN AREA

Rural/urban area	Number of anaemic mothers	Percentage of anaemic mothers (%)
Rural	122	63.6
Urban	70	36.4

PIE CHART SHOWING NUMBER OF ANAEMIC MOTHERS BASED ON THEIR RURAL / URBAN AREA

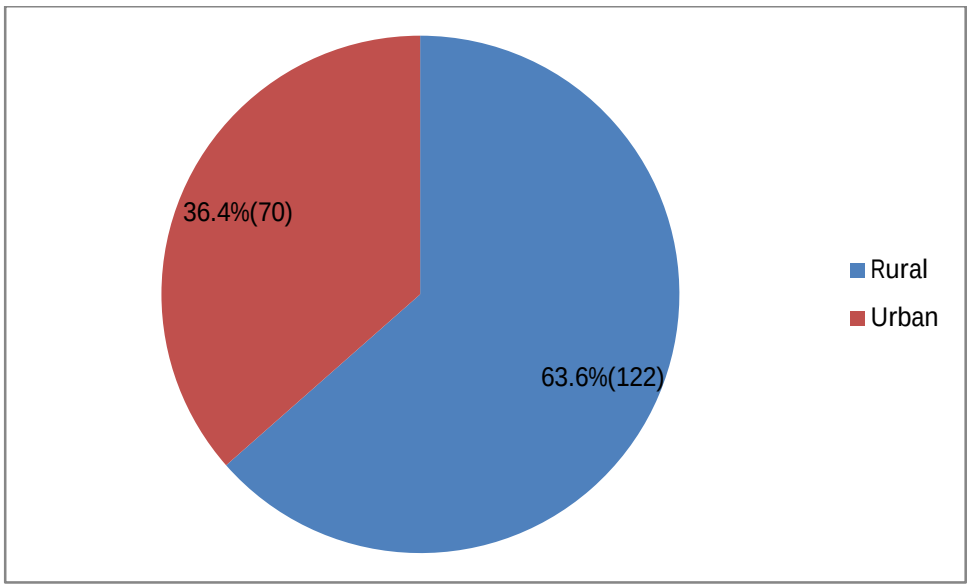


TABLE SHOWING MEAN AGE,MEAN MATERNAL HAEMOGLOBIN AND MEAN CORD HAEMOGLOBIN AMONG ANAEMIC AND NON ANAEMIC MOTHERS

Group	Mean age	Mean maternal haemoglobin	Mean cord haemoglobin	P value
Anaemic mothers	25.79±2.22	9.38±1.08	15.03±1.04	P<0.05
Non anaemic mothers	25.72± 2.46	11.78 ± 0.52	16.37 ± 0.85	

Mean cord haemoglobin between anaemic and non anaemic mothers were compared and the difference between them was statistically significant with p<0.05

TABLE SHOWING MEAN HAEMOGLOBIN IN ANAEMIC MOTHERS BASED ON THE SEVERITY OF ANAEMIA

Maternal haemoglobin	Mean haemoglobin gm/dl	No. of patients
10-10.9 gm/dl	10.43±0.23	94
9.9-7gm/dl	9.03±0.85	82
<7gm/dl	6.6 ±0.31	16

TABLE SHOWING MEAN MATERNAL AND CORD BLOOD HAEMOGLOBIN

Maternal Haemoglobin	Mean haemoglobin gm/dl	No. of patients	Mean Cord blood hamoglobin gm/dl	P value
10-10.9 gm/dl	10.43±0.23	94	15.54±0.77	P<0.05
9.9-7gm/dl	9.03±0.85	82	14.7±0.93	P<0.01
<7gm/dl	6.6 ±0.31	16	14.08±0.88	P<0.05

The mean cord hemoglobin among the three groups (mild moderate severe) were compared with mean cord hemoglobin of the non anaemic group. The difference between the groups were statistically significant with P values <0.05,0.01 and 0.05 respectively.

DISCUSSION

In our study we compared the maternal haemoglobin with cord haemoglobin in order to find whether there is any relationship between the two parameters. We enrolled 400 mothers in the study and their predelivery haemoglobin level was determined. Out of 400 mothers,192 mothers had haemoglobin less than 11g/dl and 208 mothers had haemoglobin more than 11g/dl. Among the anaemic mothers 94 had mild anaemia (haemoglobin between 10-10.9g/dl). 82 mothers had moderate anaemia(haemoglobin between 7-9.9g/dl).16 mothers had severe anaemia with haemoglobin less than 7g/dl.

We observed about 48% of the mothers were anaemic and 52% were non anaemic. Among the anaemic mothers 48.9% of them had mild anaemia, 42.8% of them had moderate anaemia and 8.3% of them had severe anaemia.

Among the 400 babies delivered 348 were term babies(87%) and 52 were preterm babies(13%).we observed that among the 192 anaemic mothers , 122 of them were from rural area which accounts to about 63.6% and 70 of them were from urban area which accounts to about 36.4%.

We found that mean age of anaemic mothers were 25.79± 2.22 and mean age of non anemic mothers were 25.72±2.46. The mean maternal haemoglobin among non anaemic mothers were 11.78 ± 0.52 and among the anaemic mothers was found to be 9.38±1.08.

The mean cord haemoglobin among non anaemic mothers was 16.37 ± 0.85 and among the anemic mothers it was 15.03±1.04. The mean maternal haemoglobin in mothers with mild anaemia was 10.43± 0.23 and the mean haemoglobin in mothers with moderate anaemia was 9.03± 0.85 and the mean haemoglobin in mothers with severe anaemia was 6.6 ± 0.23.

The mean cord hemoglobin among the three groups (mild, moderate, severe) was 15.54± 0.77,14.7± 0.93 and 14.08± 0.88 respectively. The mean cord hemoglobin between the anaemic and non anemic group was compared and the difference between the two groups was statistically significant with p value < 0.05.

The mean cord hemoglobin among the three groups(mild moderate severe) were compared with mean cord hemoglobin of the non anaemic group and the difference was statistically significant with P value less than 0.05,0.01 and 0.05 respectively.

On comparing the cord haemoglobin with maternal haemoglobin we found that there was a linear relationship between the two parameters. It was observed as that as mean maternal haemoglobin decreases, there was a decrease in the cord haemoglobin. This denotes that there is an impact of maternal anaemia on cord haemoglobin. This observation was similar to the study done by Nadia et al in babylon university which showed a linear relationship between maternal haemoglobin and cord haemoglobin.

Debbarmarubi et al also showed a linear relationship between the cord and maternal haemoglobin similar to our study. Marmoury GH et al study differs from our study as they reported that there was no association between cord haemoglobin and maternal haemoglobin levels.[63]

In our study we found that mothers with anaemia were more likely to deliver babies with lower hemoglobin levels.This observtion made us to rethink the belief that fetus continues to extract iron from the mother regardless of her iron status. Sweet et al in their study had shown that mothers with iron deficiency anaemia gave birth to newborn with lower hemoglobin level.Previous studies also suggest that iron supply to the placenta and the fetus is affected in maternal anaemia and the fetus takes iron in direct proportion to the levels available in the mother.

Terefe B et al in their study titled "Effect of Maternal Iron Deficiency Anemia on the Iron Store of Newborns "enrolled 21 anaemic mothers and 78 non anaemic mothers and found that lower level of ferritin in newborns delivered from Iron deficiency anaemia (IDA) mothers compared to non anaemic mothers suggests reduced iron stores in these newborns. And also found that newborns delivered from IDA mothers had a significantly lower concentration of hemoglobin than newborns from non anaemic mother, which is correlating with the results of our study.

Iron is actively transported from mother to fetus. In order to ensure an adequate iron supply to the growing fetus even in the anaemic mother, there is up regulation of iron transport proteins in the placenta in the iron deficiency state.Our study also demonstrated that the cord haemoglobin is lower in anaemic mothers and that the decrease in cord haemoglobin appears to be proportional to the degree of anaemia. This suggests that placental iron transport mechanisms may not work at higher degrees of anaemia and thereby it leads to a fall in cord haemoglobin.

There are some limitations in our study, iron status of the mother was not determined and maternal haemoglobin level was not determined in the first and second trimester. However, it is likely that mothers who were anaemic in the third trimester had poor iron intake throughout their pregnancy and this may lead to decreased cord haemoglobin level.

CONCLUSION

In our study we observed that maternal anaemia affects the cord haemoglobin of neonates. Our study infers that anaemic mothers deliver babies with lower haemoglobin compared to non anaemic mothers. We have found a linear relationship between maternal haemoglobin and cord blood haemoglobin of the newborns.Anaemia during pregnancy is a commmon complication that can be detected by simple screening test. Anaemia can lead to complications in both mother and foetus.

In developing countries like India, prophylaxis during pregnancy can prevent anaemia and this may decrease the incidence of foetal and maternal complications.

Overall neonatal survival outcome may also be increased. Further studies are needed to determine the relation of iron stores of the mother to the fetal iron and ferritin levels.

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PROFORMA

Serial number:

Name :

Mother details:

Age: LMP:

Weight: EDD:

Height: Gravida: Para:

BMI:

Socio economic status:

Rural/urban status:

Haemoglobin:

Treatment:

Iron supplements:

Oral iron :

Parentral iron:

Blood transfusion:

Baby details:

Gestational age: Preterm / Term / post term

Cried immediately after birth: yes / no

Birth weight:

Cord blood haemoglobin:

PARTICIPANTS INFORMATION SHEET

Investigator : - Dr. DHANASEKARAN R.

Name of the participant : -

Title: . **IMPACT OF MATERNAL ANAEMIA ON CORD BLOOD HAEMOGLOBIN**

You are invited to take part in this research study. We have got approval from the IEC. You are asked to participate because you satisfy the eligibility criteria.

What is the purpose of this research?

In this study, the effect of maternal anaemia on the cord blood haemoglobin of the neonates is evaluated, since maternal anaemia is associated with neonatal anaemia and poor survival rate

BENEFITS:

This study is helpful to know the impact of maternal anaemia over the neonates.maternal anaemia has several deleterious effects on the health of the foetus.By decreasing the incidence of maternal anaemia ,survival rate of the neonates can be improved.

CONFIDENTIALITY:

Patients who participated in the study and their details will be maintained confidentially and at any cost, those details will not be let out.

RIGHT TO WITHDRAW :

Patients will not be forced to complete the study. At any cost, in such circumstances the treatment will not be compromised.

Signature of the investigator: -

Place :

Signature/Thumb impression of participant

Sl.NO	Name	Age	Weight	Height	BMI	Gravida/	Rural/	Maternal	Gestation	Birth	cord blood
		yrs	kg	cm	kg/m2	para	urban	Hb (gm/dl)		weight(kg)	Hb (gm/dl)
1	b/o malliga	24	66	158	26.43	g2p1	rural	8.6	term	2.6	14.2
2	b/o kanaga	26	75	160	29.33	primi	urban	10.8	term	2.5	16.8
3	b/o sangeetha	27	73	159	29.24	primi	urban	7.2	term	2.9	13.6
4	devi	24	76	162	28.95	g2p1	urban	12.2	term	3.1	17.1
5	srilakshmi	28	68	165	24.97	g3p1	rural	11.4	term	2.63	15.4
6	b/o priya	22	75	157	30.42	g2p1	urban	9.8	term	2.76	14.9
7	b/o geetha	25	76	154	31.62	primi	urban	10.6	preterm	2.24	15.4
8	b/o deepa	28	65	149	29.27	primi	rural	10.3	term	3.1	14.6
9	b/o harini	26	68	152	29.43	primi	rural	11.2	term	3.3	16.4
10	b/o sujatha	27	72	160	28.12	primi	rural	11.6	term	2.6	15.6
11	b/o kavitha	30	75	162	28.57	g3p1	rural	11.4	term	2.8	16.2
12	b/oradha	27	74	160	28.19	g2p1	urban	11.1	term	2.9	15.8
13	b/o kumari	23	68	163	25.91	primi	rural	10.7	term	2.6	16.1
14	b/osankari	28	72	159	28.48	primi	urban	9.6	term	2.2	14.6
15	b/oammu	26	78	158	30.85	g3p1	rural	10.1	term	2.5	15.3
16	b/0 sundari	28	65	145	30.91	primi	rural	10.2	term	3.1	15.6
17	b/o muthulakshmi	23	62	156	25.47	primi	urban	8.6	term	2.8	13.1
18	b/o aiswarya	25	61	150	27.55	g2p1	rural	7.8	PRETERM	2.18	13.9
19	b/o vasugi	28	65	152	28.13	g2p1	rural	11.8	term	3.4	16.8
20	durga	24	74	160	28.9	primi	urban	9.6	term	3.3	17.2
21	mobeen	26	75	158	29.64	g2p1	urban	7.8	preterm	2.12	17.6

22	b/o christina	27	68	154	28.67	g2p1	rural	11.8	term	2.5	15.4
23	b/o rajamma	29	64	156	26.29	g2p1	urban	11.6	term	2.5	16.2
24	b/o kamala	23	74	160	28.9	primi	rural	12.1	term	2.7	17.8
25	b/o vaishnavi	30	73	165	26.81	primi	rural	11	term	2.9	16
26	b/o aarthi	32	65	156	28.65	g2p1	urban	9.2	term	3.2	14.7
27	b/o maragatham	26	61	159	24.12	g291	rural	10.4	term	3.5	14.8
28	b/o suseela	25	70	148	31.95	g2p1	rural	7.8	term	2.8	16.5
29	b/o rajeswari	27	60	160	23.43	g3p1	urban	10.2	preterm	2.1	13.9
30	b/o varsha	28	73	164	27.14	primi	urban	6.2	term	2.6	13.4
31	b/o kalyani	31	76	166	27.58	primi	rural	10.8	term	2.1	15.4
32	b/o ramya	29	74	158	29.64	g2p1	deepa	11.8	term	3.23	16.5
33	b/o ananthi	25	72	159	29.32	g2p2	urban	9.9	term	2.55	15
34	b/o fathima .m	27	70	163	26.34	primi	urban	11.5	term	2.65	16.8
35	b/o adhilakshmi	25	69	158	27.64	primi	rural	11.2	term	2.76	15.1
36	b/o savithri	30	72	154	30.35	primi	rural	10.1	term	2.8	14.6
37	b/o divya	28	75	153	32.03	g2p1	urban	6.6	term	2.9	15.1
38	b/o vani	27	68	150	30.22	g3p2	urban	10.4	term	2.6	15.1
39	b/o sridivya	26	69	156	28.35	g2p1	urban	12.1	term	2.7	15.4
40	b/o lalitha	23	66	154	27.83	g2p1	rural	7.6	term	2.7	16.4
41	b/o ramadevi	26	68	156	27.94	primi	rural	9.1	term	2.8	16.2
42	b/o tulasi	27	72	160	28.12	primi	urban	11.8	preterm	2.2	15.8
43	b/o rani	28	75	154	31.62	g3p2	urban	11.1	term	2.7	15.6
44	b/o dhanalakshmi	29	71	152	31.13	g2p1	rural	9.9	term	2.8	15.4
45	b/o indra	23	65	160	25.39	primi	urban	10.3	term	2.5	15.3
46	b/o nirmala	25	63	148	28.76	g2p1	rural	11.8	term	2.9	15.1
47	b/o leela	26	73	153	31.18	g2p1	rural	10.2	term	3.1	15.6
48	b/o vidya	30	67	154	28.25	primi	rural	9.8	preterm	2.2	15.5

49	b/o kanimozhi	28	72	157	29.21	primi	urban	11.1	term	2.56	16
50	b/o kamala	26	65	158	26.03	G3P2	rural	10.1	term	3.01	15.8
51	b/o uma	24	72	152	31.2	g2p1	rural	10.4	term	2.9	16
52	b/o banu	23	71	149	32	g2p1	urban	9.6	term	3.1	16.2
53	b/o dharani	25	68	152	29.4	primi	urban	9.8	term	2.9	15.8
54	b/o elavarasi	26	74	160	28.9	primi	urban	10.2	term	3.2	15.4
55	b/o harshini	24	65	151	28.5	g2p1	rural	11.4	preterm	2.1	16.1
56	b/o rohini	26	68	153	28.6	g2p1	urban	12.1	term	3.3	17.2
57	b/o vijaya	27	72	156	29.2	primi	urban	11.6	term	3.2	16.2
58	b/o yogeswari	28	71	158	29.1	primi	rural	11.7	term	3.1	16.8
59	b/o parameswari	24	65	152	28.1	primi	urban	12.6	term	2.6	17.4
60	b/o fathima	30	74	159	29.3	g3p2	urban	11.5	term	3.3	15.7
61	b/o oviya	24	72	163	27.5	primi	urban	11.7	term	3.2	16.1
62	rukmani	26	74	162	27.4	primi	urban	10.8	preterm	2.1	15.1
63	b/okirthika	24	72	156	29.6	g2p1	rural	12.6	term	2.4	17.4
64	b/o indhumathy	22	65	156	26.7	primi	rural	9.3	term	2.54	14.8
65	b/o sumathi	31	76	154	32	g2p1	urban	11.5	term	2.69	15.2
66	b/o roja	30	73	162	27.8	g3p2	urban	11.6	term	2.8	15.4
67	b/o latha	28	71	158	29.2	primi	urban	11.2	term	3.1	14.8
68	b/o revathi	24	74	165	26.8	primi	rural	10.6	term	2.5	15
69	b/o saraswathi	25	68	167	24.4	g2p1	urban	12.4	term	2.6	16.2
70	b/o krishnaveni	23	64	148	31	primi	urban	11.5	term	2.456	15.4
71	b/o nancy	26	66	163	24.8	g2p1	rural	10.3	term	2.35	15.3
72	b/o urmila	22	71	159	28.1	primi	urban	11.1	term	2.56	15.4
73	b/o suguna	27	58	146	26.5	g2p1	urban	12.1	term	3.32	17.4
74	b/o BRINDA	26	72	154	31.2	primi	urban	9.3	term	3.4	15.1
75	b/o isaiyoli	31	67	158	26.8	g3p2	urban	11.8	term	3.09	16.2
77	b/o soundaruj	23	63	165	24.6	primi	rural	11.2	term	2.582	15.6

78	b/osrilakshmi	24	67	158	26.8	g2p1	urban	11.4	term	3.2	16
79	b/o karthika	25	64	165	23.5	g2p1	rural	10.6	preterm	2.1	15.8
80	b/o devi	24	73	160	28.5	primi	urban	11.5	term	2.4	16
81	b/o harshini	26	75	157	29.6	primi	urban	11.2	term	3.2	16.4
82	laila	27	68	158	27.6	primi	rural	9.9	term	3.3	14.9
83	b/o shalini	22	76	153	32.5	g2p1	urban	12	term	2.9	17.1
84	b/o surekha	28	73	162	29	primi	urban	11.1	term	2.6	15.1
85	b/o deepa	22	69	149	31.1	primi	urban	11.8	term	3.1	15.4
86	b/o vanitha	28	65	158	26	g2p1	urban	11.4	term	2.11	16
87	b/o indra	25	73	160	28.5	primi	urban	11.2	term	3.1	15.3
88	b/o stella	24	71	161	27.7	primi	rural	10.5	preterm	2.1	14.9
89	b/o mala	28	64	165	24.2	primi	urban	11.4	term	2.8	15
90	b/o nirmala	26	72	160	26.8	g2p1	urban	6.6	term	2.5	13.4
91	b/o chitra	24	75	154	30.4	g2p1	urban	11.1	preterm	2.11	15.4
92	b/o jeeva	26	72	157	29.2	primi	urban	12.3	term	2.7	16
93	b/o dhanam	30	66	152	26.8	g3p2	rural	7.4	term	2.5	14.2
94	b/o ragini	25	68	160	25.8	primi	urban	11.6	term	3.1	14.7
95	b/o revathy	27	62	148	31	g2p1	urban	12.2	term	2.75	16.8
96	b/o sundari	26	72	157	29.2	primi	urban	11.1	term	2.58	16.2
97	b/o rathy	24	65	153	27.8	primi	urban	11.2	term	2.5	15
98	b/o geethanjali	25	69	160	27	primi	urban	11.6	term	2.8	16.4
99	b/o rasiga	23	72	159	28.1	primi	rural	11.2	term	2.53	15.8
100	b/o aarth priya	25	75	165	26.4	g2p1	urban	11.4	term	2.54	16
101	b/o nisha	24	71	162	27.4	primi	rural	12.5	term	2.53	17.6
102	b/o lavanya	26	65	159	25.7	g2p1	urban	10.5	PRETERM	2.1	15.6
103	b/o mahalakshmi	28	68	156	27.9	g2p1	urban	9.4	term	2.52	13.7

104	b/o chandra	21	62	158	24.8	primi	rural	9.9	term	2.65	14.8
105	b/o bargavi	24	74	161	28.5	g2p1	urban	11.3	term	2.54	15.8
106	b/o manjula	28	68	153	33.3	g3p1	urban	12.1	term	2.8	16.3
107	b/o kirthika	23	73	148	31.3	primi	rural	11.4	term	3.12	16.2
108	b/o latha	30	59	162	22.5	g2p1	urban	11.3	PRETERM	2.18	15.8
109	b/o janani	24	62	149	27.9	primi	urban	12.2	term	3.1	15.6
110	b/o meenakshi	26	68	158	27.2	g2p1	urban	11.4	term	2.45	16.4
111	b/o gandhimathi	25	67	161	21.9	primi	rural	10.2	term	2.56	15.2
112	b/o padmini	27	69	158	27.6	g2p1	urban	10.1	term	2.84	15
113	b/o govindammal	24	72	152	31.2	primi	rural	11.5	term	2.35	16.5
114	b/o nithya	25	74	156	30.4	g2p1	urban	9.5	term	2.47	15.4
115	b/o vasugi	28	76	163	32.5	g2p1	rural	10.6	PRETERM	2.15	13.2
116	b/o radha	24	69	145	32.8	primi	urban	6	term	2.57	14.4
117	b/o suhasini	28	68	159	27.3	g3p2	rural	7.4	term	2.57	14.3
118	b/o fathima	25	72	158.5	28.5	g2p1	urban	7.8	term	2.56	14.1
119	b/o mary	25	60	167	24.3	g2p1	rural	7.9	term	2.72	15.4
120	b/o renuka	24	66	154	27.8	primi	rural	8.6	term	2.67	14.8
121	b/o rathdevi	26	66	154	27.8	g2p1	rural	8.9	term	2.65	15.3
122	b/o keerthana	24	78	161	30.1	primi	urban	9	term	3.15	15.1
123	b/o jothi	21	74	156	30.2	primi	urban	9.2	term	3.6	14.2
124	b/o rupa	20	70	159	27.7	primi	rural	9.3	term	2.43	15.1
125	b/o chinnaponnu	26	71	159.5	27.8	primi	urban	9.4	term	2.67	14.1
126	b/o parimalam	28	61	166	22.1	g3p2	rural	9.5	term	2.76	14.6
127	b/o abirami	24	70	161	27	primi	urban	9.5	preterm	2.156	14.9
128	b/o pushpa	26	65	154	25.7	g2p1	rural	9.5	term	2.56	15.7

129	b/o banu	23	74	154	31.2	primi	rural	9.6	preterm	2.145	14.2
130	b/o mala	24	73	158	29.6	primi	rural	9.6	term	2.65	14.8
131	b/o shenbagam	25	73	164	27.1	g2p1	urban	9.7	term	2.52	13.6
132	b/o manimegalai	26	74	160	28.9	primi	rural	9.7	term	2.67	15.1
133	b/o kanmani	24	69	148	31.5	primi	urban	9.9	term	2.78	14.9
134	b/o sindhu	25	64	165	24.4	g2p1	rural	10.1	term	2.56	14.8
135	b/o lalitha	25	70	153	29.9	primi	rural	10.1	term	2.31	15.3
136	b/o uma	29	70	154	29.5	g2p1	urban	10.1	preterm	2.154	16.1
137	b/o rani	21	77	158	30.8	primi	urban	11.1	term	2.54	16.8
138	b/o revathy	23	72	154.5	30	primi	rural	10.1	term	3.22	14.9
139	b/o selvi	24	71	152.5	30.7	primi	rural	10.2	preterm	2.24	16.4
140	b/o parvathy	24	67	157	27.2	primi	rural	10.2	term	3.23	15.5
141	b/o shanthi	27	58	165	21.3	g2p1	rural	10.2	term	2.67	15.2
142	b/o vidya	24	62	168	22	g2p1	urban	9.2	term	2.73	15.2
143	b/o kanchana	31	64	157	26	g2p1	urban	10.2	preterm	2.35	15.4
144	b/o sandhya	26	67	157.5	27.2	g2p1	rural	10.2	term	2.45	15.4
145	b/o ponnammaal	27	71	154.5	29.9	g2p1	urban	10.2	term	2.69	15.4
146	b/o maniammai	26	70	149	31.5	primi	rural	10.3	term	2.75	15.6
147	b/o revathy	24	60	167	21.5	primi	urban	10.3	term	2.54	15.6
148	b/o jamuna	27	63	156.5	25.9	g3p2	rural	10.3	term	2.67	15.6
149	b/o kanthimathi	27	76	157.5	30.8	g2p1	urban	10.3	term	2.6	16.4
150	b/o kanaga valli	24	70	160	27.3	primi	urban	10.4	preterm	2.16	16.4
151	b/o punitha	25	66	160	25.8	g2p1	urban	11.2	term	3.36	16.2
152	b/o gowri	30	64	156	26.3	g3p1A1	rural	10.4	term	3.21	15.2
153	b/o poovizhi	26	60	163	22.6	g2p1	rural	10.4	term	2.87	15.4
154	b/o uma	21	59	165	21.7	primi	rural	10.4	term	2.75	15.3
155	b/o sumathi	26	68	157	27.6	g2p1	rural	8.4	term	2.56	13.5
156	b/o	24	71	160	27.7	primi	rural	10.4	term	2.78	16.4

	bakkialakshmi										
157	b/o poovazhagi	28	71	159	28.1	g3p2	rural	10.4	term	2.78	15.5
158	b/o nandhini	28	76	153	32.5	g2p1	urban	11.4	term	2.78	16.2
159	b/o jaya	28	65	153	27.8	g2p1	urban	10.5	term	2.87	14.7
160	b/o swathy	28	72	156	29.6	g3p1A1	rural	6.5	term	2.45	13.1
161	b/o vasugi	28	71	153	30.3	g2p1	urban	10.5	term	2.79	15.8
162	b/o valarmathy	21	66	152	28.6	primi	rural	10.5	term	2.54	15.4
163	b/o muthulakshmi	26	78	158	31.2	g2p1	urban	10.5	term	2.89	16.4
164	b/o sembarutthi	26	71	157	28.8	g2p1	urban	10.5	term	2.57	15.5
165	b/o radha	28	61	164	22.7	g3p2	urban	10.6	preterm	2.25	16.9
166	b/o girija	25	68	159	26.9	primi	urban	10.6	term	2.87	15.4
167	b/o janaki	24	68	154	28.7	primi	rural	10.6	term	2.9	15.4
168	b/o saroja	27	70	153	29.9	g2p1	urban	10.6	term	2.57	15.8
169	B/O Thenmoli	25	65	156	26.7	Primi	rural	11.2	Term	2.3	16
170	b/o malathy	24	60	158	24.03	g2p1	urban	11	term	3.14	16.7
171	b/0 rajalakshmi	27	64	160	25	primi	urban	10.8	term	2.86	16.2
172	b/o malarvizhi	25	68	154	28.6	primi	urban	9.8	term	2.89	15.2
173	b/o sandhya	20	68	160	26.5	primi	urban	9.6	term	2.04	14
174	b/o vanniyamalar	26	58	162	22.6	primi	urban	8.8	term	2	14.8
175	b/o jayakumari	19	66	160	25.7	primi	rural	9.1	term	2.73	17.6
176	b/o mohana	30	65	164	24.1	g2p1	rural	10.1	Term	2.78	13.2
177	b/o jemila	24	59	166	23.04	g2p1	urban	12	Term	3.12	21
178	b/o sumitha	30	70	167	25	Primi	urban	11.2	Term	2.63	17.2
179	b/o nagabramma	27	52	160	20.3	g3p2	rural	6.4	Term	3.58	13.2
180	b/o meenakshi	26	58	156	23.8	g3p2	urban	11.6	Term	3.24	16.4
181	b/o anitha	25	70	168	24.8	g2p1	rural	11.2	Term	2.9	17

182	b/o sathya	23	62	162	23.6	g2p1	urban	12	Term	3.76	16.8
183	b/o kalaichelvy	28	64	168	22.6	g2p1	rural	12	Term	2.59	13.9
184	b/o veeralakshmi	26	62	164	23.05	Primi	rural	11.8	Term	3.06	17.4
185	b/o kavitha	30	95	160	37.1	g3p2l2	rural	10.1	Term	2.5	14.9
186	b/o geetha	28	66	158	26.4	g2p1	rural	10.8	term	2.45	16.6
187	b/o seetha	26	68	164	24.8	g3p2	rural	10.4	Term	2.46	15.2
188	b/o lalitha krishno 22	22	64	158	25.6	Primi	urban	11.1	term	3.1	15.1
189	b/o radha	30	70	168	24.8	g2p1	urban	12.2	Term	2.8	17.2
190	b/o kavya	25	68	156	27.9	primi	urban	11.8	Term	2,8	16.4
191	b/o sundari	23	72	166	26.1	g2p1	rural	11	term	2.6	16
192	b/o vijayalakshmi	24	65	168	23	g2p1	rural	10.6	Term	2.81	16.2
193	b/o varalakshmi	21	62	158	24.83	g2p1	rural	8	Term	2.2	14.1
194	b/o ambika	24	68	166	24.67	g3p2	urban	11.2	Term	2.89	16.2
195	b/o yashoda	26	66	170	22.83	G3P2	rural	7.6	Term	2.78	14.8
196	b/o anjali	24	70	168	24.8	primi	rural	11.2	Term	2.6	16.8
197	b/o chitra	24	68	156	27.94	primi	rural	11.2	preterm	2.2	15
198	b/o banu	25	72	167	25.81	G2P1	rural	13.2	Term	2.7	17
199	b/o kalaivani	27	58	160	22.65	G2P1	urban	12.2	Term	2.89	16.8
200	b/o kanaga	22	60	156	24.65	G3P1	rural	11.2	Preterm	1.89	16.2
201	b/o saroja	26	58	164	21.56	primi	urban	9.8	Term	2.7	14.4
202	b/o vijaya	30	68	169	23.8	G3P2	rural	9.8	Term	3.2	14.4
203	b/okalaiarasi	22	66	158	26.43	G3P1A1	rural	7.1	Term	2.67	14.2
204	b/o malathy	26	70	168	24.8	G2P1	rural	13	Term	3.4	19
205	b/o vennila	26	62	157	25.15	primi	urban	12.4	Term	3.3	18.2
206	b/o kavitha	24	66	150	29.33	G2P1	urban	12.6	Term	3.2	17.2

207	b/o sivapriya	28	70	158	28.04	Primi	urban	6.8	Term	2.2	12.2
208	b/o sridivya	22	74	160	28.9	G2P1	urban	11.2	Term	2.7	16.2
209	b/o malathy	27	76	164	28.25	G2P1	urban	11.4	Term	2.8	17.2
210	b/o malarkodi	22	76	167	27.25	Primi	rural	7.2	Term	2.8	14.8
211	b/o kalaimani	28	78	168	27.63	G2P1	urban	11.8	Term	3.6	16
212	b/o vasanthi	29	76	168	26.92	G2P1	rural	13.2	Term	3.2	17.8
213	b/o varalakshmi	22	68	160	26.56	primi	rural	12.6	Term	3.1	16.4
214	b/o sandhya	26	75	162	28.57	G2P1	rural	12.6	Term	2.6	16.4
215	b/o kaveri	21	66	158	26.43	G2P1	urban	9.6	Term	2.6	14.2
216	b/o manju	26	76	168	26.92	G3P2	rural	9.6	Term	2.1	14.8
217	b/o nagammal	27	68	156	27.94	G2P1	rural	5.8	Term	2.4	13.1
218	b/o nandhini	22	70	158	28.04	G2P1	rural	10.2	Term	3.1	15.6
219	b/o vijaya	28	72	165	26.44	G3P1A1	urban	11.4	Term	2.6	16.8
220	b/o savithri	30	74	162	28.19	G2P1	urban	11.6	Term	2.6	15.2
221	b/o lalitha krishno 22	26	74	160	28.9	G2P1	urban	12.8	Term	3.1	18.6
222	b/o lakshmi	22	76	156	31.22	primi	rural	12.6	Term	2.4	16.2
223	b/o malathy	26	74	162	28.19	G2P1	urban	9.5	Term	2.5	15
224	b/o vijaya	28	64	158	25.63	G4P2A1	rural	8	Term	2.1	14.1
225	B/O Thenmoli	22	62	156	25.47	G2P1	urban	11.2	Term	2.6	15.8
226	b/o maragatham	23	66	152	28.56	G2P1	rural	11.2	Term	2.6	15.6
227	b/o mala	26	74	160	28.9	G2P1	urban	11.2	Term	2.4	16.2
228	b/o karpagam	26	70	160	27.34	primi	urban	11.2	Term	3.1	16.8
229	b/o manju	27	76	165	27.91	G2P1	urban	7.8	Term	3.2	13.4
230	b/o sarala	26	74	160	29.68	primi	urban	12.1	Term	3.1	17.2
231	b/o vanitha	26	68	152	29.43	primi	urban	6.2	Term	2.16	13.8
232	b/o kavitha	21	62	152	26.83	primi	urban	7.4	Term	2.1	13.3
233	b/o saranya	26	66	158	28.56	G2P1	rural	11.6	Term	2.3	16.2

234	b/o sudha	30	76	164	28.25	G3P1A1	urban	12.2	Term	3.1	17.2
235	b/o savitri	24	72	158	28.84	primi	urban	11.6	Term	3.1	16.4
236	b/o malar	22	68	158	27.23	G2P1	urban	11.8	Term	3.1	16.4
237	b/o kalaivani	30	72	161	27.77	G3P2	urban	11.4	Term	2.6	15.8
238	b/o jayanthi	23	68	154	28.67	G2P1	rural	10.2	Preterm	1.96	16.2
239	b/o sandhya	22	72	160	28.12	primi	urban	9.3	Term	2.7	14.8
240	b/o indrani	23	68	160	26.56	G2P1	rural	7.1	Term	2.4	14.2
241	b/o pramila	26	67	156	27.94	G2P1	rural	12.1	Term	3.1	17.4
242	b/o vennila	26	72	168	25.51	G3P2	rural	11.2	Term	2.16	16.2
243	b/o muthulakshmi	28	67	170	23.18	primi	urban	9.1	Term	3.1	14.8
244	b/o mary	30	64	168	22.67	primi	urban	11.2	Term	2.7	15.4
245	b/o sangeetha	28	68	164	25.28	G2P1	urban	7.4	Term	2.8	12.4
246	b/o devi	22	68	152	29.43	G3P2	rural	11.6	preterm	2	16.2
247	b/o manonmani	26	76	168	26.92	G2P1	urban	9.2	Term	2.9	14.2
248	b/o bagya	28	76	152	32.89	primi	urban	11.8	Term	2.4	16.7
249	b/o kousalya	22	64	168	22.67	G2P1	urban	6.8	Term	3.2	14
250	b/o kannamma	28	66	168	22.67	G2P1	urban	10.7	Term	3	15.2
251	b/o jabakanni	26	66	152	28.56	G2P1	urban	9.8	Term	2.8	14.1
252	b/o suryakala	23	64	150	29.33	G2P1	urban	11.6	Term	3.1	15.8
253	b/o selvi	26	68	160	26.56	primi	urban	12.1	Term	3.16	18.1
254	b/o jaya	22	68	160	26.56	primi	urban	6.9	Term	2.2	14.2
255	b/o rajalakshmi	24	66	150	29.33	G2P1	rural	6.1	Term	2.8	11.8
256	b/o kalai	27	76	169	26.61	G2P1	urban	9.8	Term	2.6	15.1
257	b/o suguna	26	68	152	29.43	G2P1	rural	11.6	Term	2.6	16.8
258	b/o ashtalakshmi	27	67	152	29.43	G2P1	urban	9.2	preterm	2.1	13.6
259	b/o lakshmi	25	72	162	27.4	G2P1	rural	9.6	Term	2.3	14.2

260	b/o kantha	27	75	163	28.6	primi	urban	11.8	Term	2.8	16.4
261	b/o ramani	28	68	156	27.94	G2P1	rural	9.1	Term	3.1	14.1
262	b/o manju	26	72	156	29.58	primi	rural	10.2	Term	2.9	12.2
263	b/o shenbagam	26	72	165	26.4	G2P1	urban	6.4	Term	2.7	13.4
264	b/o visalatchi	23	74	162	28.1	G2P1	urban	11.4	Term	2.6	15.1
265	b/o manjula	27	65	172	21.9	G3P2	rural	11.2	Preterm	2.8	15.8
266	b/o megala	26	76	162	28.9	primi	urban	11.6	Term	2.9	16.2
267	b/o manju	22	70	165	25.7	primi	urban	11.8	Term	2.8	15.4
268	b/o mercy	27	62	156	25.47	G2P1	rural	10.8	Term	2.7	16.2
269	b/o gomathi	27	76	152	32.89	primi	rural	6.2	Term	2.7	12.8
270	b/o sangeetha	28	74	162	28.18	G2P1	urban	14.8	Preterm	2.9	16.6
271	b/o rajalakshmi	24	70	165	25.71	primi	urban	11.6	Term	2.7	16.1
272	b/o sumathi	26	70	163	26.34	G2P1	rural	12.2	Term	2.8	16.4
273	b/o sakthi	22	68	159	26.56	primi	rural	11.8	Term	2.8	16.2
274	b/o shanthi	28	72	165	26.44	G2P1	rural	10.8	Term	2.6	15.8
275	b/o swetha	28	72	158	28.84	G2P1	rural	11.6	Term	2.34	17.8
276	b/o kaveri	30	76	156	30.44	G3P2	urban	11.8	Term	3.1	16.4
277	b/o kasthuri	27	76	152	32.89	G2P1	rural	9.6	Term	2.68	15.4
278	b/o revathi	24	68	168	24.09	G2P1	rural	9.8	preterm	2.9	16.1
279	b/o sundari	25	64	160	25	G2P1	rural	10.6	Term	2.9	16.2
280	b/o mythili	26	68	152	29.43	G2P1	urban	9.6	Term	2.86	14.8
281	b/o gayatri	26	66	152	28.56	G2P1	urban	11.2	Term	2.8	17.2
282	b/o philomena	26	72	152	31.1	G2P1	urban	11.2	Term	2.8	15.8
283	b/o kumari	30	68	152	29.43	primi	urban	9.6	Term	3.2	14.2
284	b/o gowri	24	72	156	29.58	primi	rural	10.8	Term	2.9	16.6
285	b/o madhavi	24	68	156	27.9	G2P1	rural	11.8	Term	3.1	16.6
286	b/o chitra	23	77	165	28.28	primi	rural	10.2	Term	2.4	15.2
287	b/o sheela	30	74	165	27.18	G3P2	urban	11.6	Term	3.2	17.2

288	b/o swapna	24	66	162	25.14	G2P1	urban	11.2	Term	2.6	17.2
289	b/o praba	22	77	168	27.28	primi	rural	10.2	Term	2.4	14.9
290	b/o sridevi	26	66	162	25.14	g2p1	urban	9.8	Term	3.1	15.2
291	b/o usha	24	77	160	30.07	primi	urban	10.8	Term	2.8	16.2
292	b/o vanitha	26	76	154	31.22	G2P1	urban	10.6	Term	3.1	16.4
293	b/o gajalakshmi	27	72	156	29.28	G3P1A1	urban	10.6	Term	2.75	16.8
294	b/o malathi	28	72	163	26.44	G3P2	urban	12.2	Term	2.7	18.1
295	b/o preethi	26	70	165	25.71	G3P1A1	rural	10.8	preterm	2.65	16.4
296	b/o ganga	28	75	165	27.54	G2P1	urban	10.4	Term	2.8	17.1
297	b/o mohana	26	76	150	33.73	G2P1	rural	11.2	Term	2.4	16.4
298	b/o deepa	25	66	150	29.33	primi	rural	12.1	Term	3.2	17.4
299	b/o manjari	26	68	162	25.91	G3P2	urban	9.8	term	2.5	14.8
300	b/o alamelu	24	58	152	25.1	G2P1	rural	6.6	term	2.4	12.6
301	b/o padma	23	70	156	28.76	primi	urban	11.6	term	2.8	16.2
302	b/o varalakshmi	28	74	166	26.85	G2P1	urban	11.6	term	2.85	15.8
303	b/o panjali	30	72	` 165	26.44	G3P2	rural	10.2	term	2.5	15.6
304	b/o mageshwari	23	75	163	28.22	primi	rural	11.6	term	3.2	16.1
305	b/o brinda	24	62	150	27.55	primi	rural	8.8	term	2.5	14.6
306	b/o manonmani	28	75	158	30.04	G3P1A1	urban	10.6	term	3.1	15.8
307	b/o chitra	25	67	152	28.99	G3P2	rural	11.6	term	3.2	16.1
308	b/o savitha	30	75	160	29.2	G2P1	urban	10.1	term	2.4	15.1
309	b/o karthika	22	70	164	26.02	primi	rural	10.2	term	2.6	15.2
310	b/o madhumila	27	68	158	27.2	G2P1	urban	11.8	term	2.2	15.6
311	b/o mageshwari	25	66	148	30.1	primi	rural	10.6	term	2.9	15.4
312	b/o uma	26	64	162	24	G2P1	urban	9.2	term	2.4	14.4
313	b/o ambika	26	63	160	24.6	primi	rural	9.6	term	2.1	16.1
314	b/o sasi	26	72	164	24	G3P2	urban	11.6	term	2.6	17.2

315	b/o meenakshi	24	70	156	28.8	primi	urban	9.6	term	2.6	14.2
316	b/o sita	26	68	162	25.9	G2P1	rural	10.4	preterm	2.58	15.8
317	b/o vino	28	74	155	30.8	primi	rural	10.1	term	2.8	16.1
318	b/o jeya	26	70	165	25.7	primi	rural	9.8	term	2.8	15.8
319	b/o radha	24	66	151	29.3	primi	urban	12	term	3	17.2
320	b/o usha	28	73	166	26.8	G2P1	urban	10.2	term	3.1	16.4
321	b/o anusiya	24	73	160	28.5	primi	rural	6.6	term	3.12	13.6
322	b/o radha	24	62	162	23.6	primi	rural	10.6	term	2.56	15.3
323	b/o nadhiya	29	66	146	31	g3p1	rural	10.6	term	2.63	16
324	b/o darshini	30	67	155.4	27.9	g3p2	rural	10.6	term	2.43	15.8
325	b/o priya	27	74	154.5	31.2	g2p1	urban	10.6	term	2.56	16.8
326	b/o madhumathy	30	64	159	25.3	g3p2	urban	10.6	term	2.53	15.6
327	b/o subitha	26	72	157	29.2	g2p1	urban	8.6	term	2.54	13.5
328	b/o thenmozhi	25	69	157	28	g2p1	urban	9.6	term	2.65	15.5
329	b/o santhi	27	63	158	25.2	g2p1	urban	10.7	preterm	2.26	15.8
330	b/o jayanthi	25	75	149	33.8	g2p1	rural	10.7	term	2.42	15.7
331	b/o juli	27	69	160	27	primi	urban	10.7	term	2.37	15.9
332	b/o nagammal	28	70	156.6	28.8	g3p1A1	urban	9.7	term	2.75	15.6
333	b/o dharani	23	72	156	29.6	primi	rural	10.8	term	2.64	15.1
334	b/o navya	21	69	157	28	primi	rural	10.8	term	2.64	15.5
335	b/o sundari	27	71	160	27.7	g2p1	rural	11	term	2.95	16.3
336	b/o kavitha	24	64	156	26.3	primi	rural	11	term	2.98	16.1
337	b/o firoza	26	75	158	30	g2p1	rural	11.1	term	3.12	15.3
338	b/o saraswathi	26	69	160	27	g3p2	rural	11.1	term	2.65	16.3
339	b/o reshma	28	73	160	28.5	g2p1	urban	11.1	term	2.45	17.6
340	b/o anu	22	68	158	27.2	primi	urban	11.1	term	2.78	16.8
341	b/o lekha	22	66	159	26.1	primi	rural	11.1	term	2.13	16.3

342	b/o velvizhi	26	74	160	28.9	primi	urban	11.1	term	2.65	16.6
343	b/o kalaiarasi	28	72	161	27.8	g2p1	rural	11.1	term	2.65	16.1
344	b/o stella	26	69	164	25.7	primi	rural	11.2	term	3.08	16
345	b/o sangeetha	27	65	155.5	27.1	primi	urban	11.2	term	2.65	16
346	b/o amulu	21	72	154	30.4	primi	rural	11.2	term	2.98	17.4
347	b/o santhoshi	25	75	160	29.3	primi	urban	11.2	term	2.56	16.4
348	b/o sakthi	27	75	157	30.4	g2p1	urban	11.3	term	2.54	15.6
349	b/o monisha	31	76	156	31.2	g2p1	urban	11.3	term	2.67	16.4
350	b/o devi bala	26	68	156	27.9	g2p1	urban	11.4	term	2.45	16.2
351	b/o dhanalakshmi	26	67	159	26.5	primi	rural	11.4	term	2.87	16.2
352	b/o chitra	26	68	160	26.6	primi	rural	11.4	term	2.95	15.7
353	b/o divya	23	67	156	27.5	primi	rural	11.4	term	2.51	15.5
354	b/o nirosha	26	77	154	32.5	g2p1	urban	11.4	term	2.54	16.9
355	b/o gayathri	27	62	161	23.9	g2p1	urban	11.4	preterm	2.21	17
356	b/o lavanya	24	71	154	29.9	primi	urban	11.4	term	2.56	16.1
357	b/o malarkodi	21	67	156	27.5	primi	urban	11.4	preterm	2.12	16.3
358	b/o gudiya	27	73	159	28.9	g2p1	urban	11.5	term	2.89	15.6
359	b/o ponmani	24	68	156	27.9	primi	rural	11.5	term	2.45	16.2
360	b/o viji	19	72	158	28.8	primi	urban	11.5	term	2.34	16.6
361	b/o tamilarasi	25	68	156	27.9	primi	urban	11.5	term	2.65	15.6
362	b/o sathya	25	69	158	27.6	primi	urban	11.5	term	2.54	16.4
363	b/o durgadevi	29	65	156	26	primi	urban	11.5	term	2.54	16.5
364	b/o veeralakshmi	25	72	158	28.8	g2p1	urban	11.5	term	3.2	15.8
365	b/o deepika	25	67	154	28.3	g2p1	urban	11.5	term	2.87	16.4
366	b/o dilshad begum	21	67	154	28.3	primi	rural	11.5	term	2.89	15.7

367	b/o bhuvana	26	74	156	26.3	g2p1	urban	11.5	preterm	2.14	15.1
368	b/o vinothini	25	68	157	27.9	g2p1	urban	11.6	term	3.35	17.8
369	b/o naveena	21	63	156.5	25.6	primi	rural	11.6	term	2.64	16.5
370	b/o ranjani	26	63	158	25.2	g2p1	urban	11.6	preterm	2.14	16.8
371	b/o sarveswari	28	67	164	28.3	g3p2	rural	11.7	term	2.61	16.6
372	b/o anjalai	30	70	159	27.7	g3p2	rural	11.7	term	3.34	15.4
373	b/o vani	26	72	156	29.6	g2p1	urban	11.7	term	2.52	16.9
374	b/o jayadevi	24	68	152	29.4	primi	rural	11.7	term	3.13	15.7
375	b/o kanchanamala	28	72	160.5	28.1	g2p1	urban	11.7	term	2.75	15.7
376	b/o pavithra	21	71	164	26.4	primi	urban	11.8	term	2.56	16.5
377	b/o jaya kumari	21	64	162	24.4	primi	rural	11.8	term	2.74	16.1
378	b/o leelavathi	25	70	159	27.7	g2p1	urban	11.8	term	2.84	16.4
379	b/o vasanthi	25	73	156.5	29.6	primi	urban	11.8	term	2.63	17
380	b/o padmavathy	29	74	157.5	30	g3p1A1	urban	11.8	term	2.65	16.7
381	b/o anjali	24	69	157	28	primi	urban	11.8	term	2.78	16.2
382	b/o akila	27	71	155	29.6	g2p1	urban	11.8	term	2.76	16.8
383	b/o kanaga valli	25	74	154	31.2	g2p1	urban	11.9	term	2.45	16.2
384	b/o manjari	26	66	155	27.1	g2p1	rural	12.1	term	2.53	15.8
385	b/o radhika	24	75	154	31.6	primi	rural	12.1	term	2.57	17.9
386	b/o devi	26	72	157	29.2	g2p1	urban	12.1	term	2.65	17.5
387	b/o latha	27	76	162	29	primi	urban	12.2	term	3.12	17.8
388	b/o karthika	25	70	154	29.5	primi	urban	12.2	term	2.98	17.4
389	b/o nivedha	28	68	160	26.6	g2p1	urban	12.2	term	2.67	17.5
390	b/o hemavathy	23	65	156	26.7	primi	rural	12.2	term	3.14	16.1
391	b/o swetha	26	70	159	27.7	g2p1	urban	12.4	term	2.54	16.4
392	b/o ganga	27	73	154	30.8	primi	urban	12.5	term	2.59	16.7
393	b/o durga	24	64	154	27	g2p1	urban	12.6	term	2.86	15.6

394	b/o mahalakshmi	24	75	158	30	primi	urban	12.7	term	2.45	17.9
395	b/o vanisree	25	75	160	29.3	primi	urban	13.1	term	2.34	18.1
396	b/o yogeswari	26	76	150	33.8	primi	urban	13.2	term	2.65	17.8
397	b/o poongothai	26	63	148	28.8	g2p1	urban	12.5	term	2.85	15.9
398	b/opraveena	30	63	156	25.9	g3p2	urban	11.5	term	2.85	15.7
399	b/o kalaichelvi	29	70	158	28	g3p2	urban	11.6	term	2.65	16.2
400	b/o santhi	22	66	156	27.1	primi	urban	11	term	2.8	16.2