EFFICACY OF TRANSCEREBELLAR DIAMETER / ABDOMINAL CIRCUMFERENCE RATIO VERSUS HEAD CIRCUMFERENCE/ ABDOMINAL CIRCUMFERENCE RATIO IN PREDICTION OF ASYMMETRICAL INTRAUTERINE GROWTH RETARDATION

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OBSTETRICS AND GYNAECOLOGY

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THANJAVUR MEDICAL COLLEGE

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

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CERTIFICATE

This is certify that the dissertation titled **"EFFICACY OF TRANSCEREBELLAR DIAMETER / ABDOMINAL CIRCUMFERENCE RATIO VERSUS HEAD CIRCUMFERENCE/ABDOMINAL CIRCUMFERENCE RATIO IN PREDICTION OF ASYMMETRICAL INTRAUTERINE GROWTH RETARDATION"** is a bonafide work done by Dr. C.SILVIN SOFHIA MARY in the Department of obstetrics and Gynaecology, Thanjavur Medical college, in partial fulfillment of the university rules and regulations for the award of MS degree in Obstetrics and Gynaecology under my guidance and supervision during the academic year 2017-2020.

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DECLARATION

I solemnly declare that this dissertation titled **"EFFICACY OF TRANSCEREBELLAR DIAMETER / ABDOMINAL CIRCUMFERENCE RATIO VERSUS HEAD CIRCUMFERENCE/ ABDOMINAL CIRCUMFERENCE RATIO IN PREDICTION OF ASYMMETRICAL INTRAUTERINE GROWTH RETARDATION"** was done by me at Dept. of Obstetrics and Gynaecology, Thanjavur Medical College during year 2017-2020 under guidance and supervision of **Prof. Dr. R.RAJARAJESWARI, MD.,DGO.,DNB OG.,.** This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University towards partial fulfillment of requirements for the award of MS degree in Obstetrics and Gynaecology (BRANCH II).

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CONTENTS

INTRODUCTION

The Process of birth is the most dangerous journey an individual undertakes. A healthy newborn is the goal of every expectant mother and her obstetrician.

A fetus with an estimated weight below the 10 th percentile for a given gestational age is considered to have fetal growth restriction (FGR) also called as intrauterine growth restriction (IUGR). It is estimated that the incidence of fetal growth restriction is 3-10%.

The growth potential of the fetus is dictated, on one hand by the fetal genome and on the other hand by the intrauterine environment. The intrauterine environment is under the influence of both maternal and placental factors.

Fetal growth restriction is linked to an increased risk perinatal morbidity and mortality. Growth restricted fetuses are more prone to intrauterine hypoxia / asphyxia. Still birth and hypoxic ischemic encephalopathy (HIE) are more likely to occur in growth restricted fetuses. In addition, it has been also found that these growth restricted infants have increased 1-year infant mortality rate and abnormal neurological development.

In order to prevent such mal occurrence during pregnancy clinicians has developed various methods for assessing the fetal growth in utero. Ideal and best investigation that is simple, reliable, accurate, non-invasive and safe is prenatal ultrasonography. an accurate determination of gestational age, identification of

congenital anomalies, evaluation of fetal growth and assessment of fetal wellbeing and maturity are all possible due to availability of ultrasound.

The most commonly used parameters to evaluate fetal growth are biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC) and femur length (FL). Out of all these parameters best predictor of fetal growth restriction is AC (abdominal circumference). But all these parameters can be correlated if the gestational age is accurately known. But uncertainity of the gestational age makes the differentiation between the appropriate for gestational age and the small for gestational age fetus difficult.

Transcerebellar diameter (TCD) is the maximum transverse diameter of the fetal cerebellum. The fetal cerebellar hemispheres are located in the posterior cranial fossa which is resistant to the external pressure and growth deviations, thus making it a better indicator for the determination of gestational age .Conversely , fetal abdominal circumference (AC) is the earliest affected parameter in the process of impaired fetal growth .Thus , a ratio of TCD/AC which is gestational age independent is very useful in predicting IUGR . Head circumference is another parameter which remain minimally affected by external pressure effects causing deformation of fetal head and by growth alterations. HC/AC ratio is another gestational age independent parameter which may be used in predicting IUGR.

Fetal cerebellum can be visualized as early as 10- 11 weeks by USG. From second trimester onwards, it grows with the linear correlation with gestational age.

This study was primarily planned to study the efficacy of TCD/ AC ratio and HC/ AC ratio in prediction of asymmetrical IUGR.

AIMS AND OBJECTIVES OF THE STUDY

To compare the accuracy of trans cerebellar diameter (TCD) / abdominal circumference ratio (AC) ratio with head circumference (HC) / abdominal circumference (AC) ratio in predicting asymmetrical IUGR.

MATERIALS AND METHODS

A prospective study consisting of 200 antenatal women was conducted in Government Raja Mirasudhar Hospital, Thanajvur medical college, Thanjavur during the period of January 2018 – December 2018 (12 months).

INCLUSION CRITERIA:

- Singleton intrauterine pregnancies > 30 weeks
- Cases with clinical suspicion of IUGR a discrepancy of 4 weeks in period of gestation and clinical examination was taken as evidence of IUGR.

EXCLUSION CRITERIA:

- Multiple pregnancies.
- Poly hydramnios
- Anomalies
- Irregular menstrual periods
- Symmetrical IUGR.

METHODOLOGY:

Two groups are chosen control group (contains 100 normal cases) and study group (100 clinically suspected IUGR) . TCD/AC ratio and HC/ AC ratio of normal group are calculated. mean and standard deviation are calculated for the normal group. Then the values of the study group is compared with the normal group. The values more than 2SD are labelled as IUGR (sonographically)

Then those clinically suspected IUGR cases are followed up to delivery and post-delivery new ballard score and CAN score (clinical assessment of nutritional status at birth) are calculated. Number of ultrasonographically detected IUGR compared with number of true IUGR and accuracy of both TCD/AC ratio and HC/AC ratio is compared.

- True positive values
- False positive values
- sensitivity
- specificity
- positive predictive value
- negative predictive value
- diagnostic accuracy

above mentioned are calculated and interpretation is done.

REVIEW OF LITERATURE

Fetal growth restriction can be defined as a condition where the fetus fails to achieve its genetic growth potential and consequently is at risk of increased prenatal morbidity and mortality.

Incidence of FGR is approximately 5% in general population. The expressions, retardation and restriction were previously used interchangeably for this phenomenon, but the term restriction describes the condition more appropriately, as IUGR indicates a limitation rather than a delay in growth.

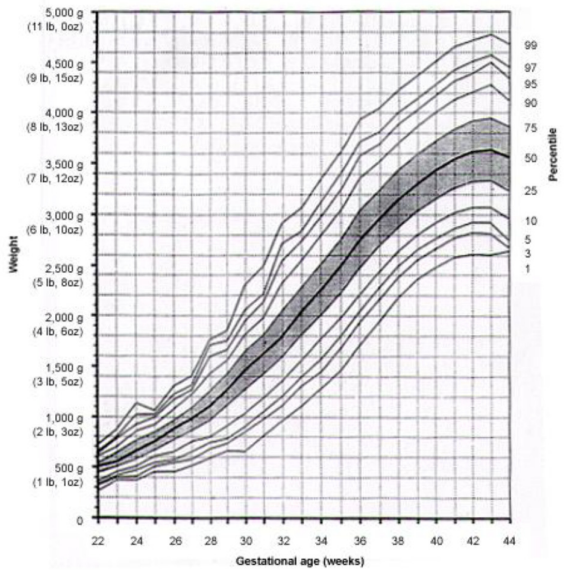
Birth weight is usually taken as the sole criterion to assess fetal growth and consequently fetuses with a birth weight less than the 10^{th} percentile of those born at the same gestational age, or two standard deviations below the population mean are considered growth restricted. However, this definition does not make a distinction among infants who are constitutionally small, growth-restricted and small, and not small but growth-restricted relative to their potential. Therefore, the term FGR refers to fetuses that are small for gestational age with features of chronic hypoxia or failure to thrive.

Moderate and severe FGR are defined as birth weight in the 3rd to 10th percentile and less than 3rd percentile, respectively.

The prenatal diagnosis of intrauterine growth restriction is defined as sonographically estimated fetal weight <10 th percentile of gestational age. The

incidence of IUGR varies depending on the population examined, from 4- 7 % in developed countries and up to 30 % in poor resource setting.

Growth percentiles for fetal weight versus gestational age



Normal term infants typically weigh more than 2500 g by 37 weeks gestation.

1. NORMAL FETAL GROWTH

The control of fetal growth is a complex process confounded by multiple variables such as maternal height, race, ethnicity, socio economic status and other factors. At the biological levels, fetal growth depends on two components: genetic potential and substrate supply.

The genetic potential is derived from both the parents and is mediated through growth factors such as insulin like growth factors. An adequate substrate supply is essential to achieve the genetic potential. This supply is derived from placenta which is dependent on uterine and placental vascularity.

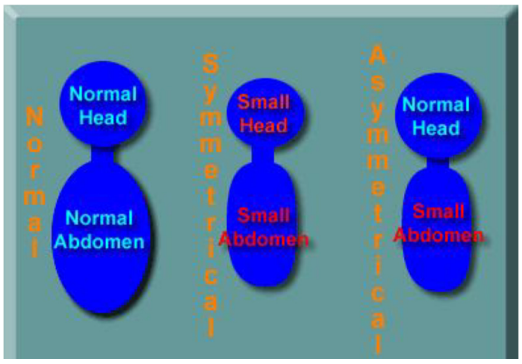
Fetal growth accelerates from 5gm per day at 14- 15 weeks of gestation to 10 gms per day at 20 weeks of gestation, peaks at 30-35gms per day at 32-34 weeks of gestation after which growth rate decreases. symphysiofundal height measured from upper border of the pubic symphysis to the level of uterine fundus increases by approximately 1cm per week between 14 to 32 weeks. abdominal girth increases on an average by 1 inch per week, after 30 weeks it is about 30 inches at 30 weeks in an averagely built woman.

The process of fetal growth comprises of three phases

FIRST PHASE	First 16 week of gestation	Cellular hyperplasia
SECOND PHASE	Between 16- 32 weeks of gestation	Concomitant cellular hyperplasia and hypertrophy
THIRD PHASE	Between 32 weeks and term	Rapid increase in cell size.

2. CLASSIFICATION OF FETAL GROWTH RESTRICTION

Campbell and Thoms (1997) described the use of head-to abdomen circumference ratio (HC/AC) to differentiate growth restricted fetuses. Those who were symmetrical were proportionally small, and those who were asymmetrical had disproportionally lagging abdominal growth.



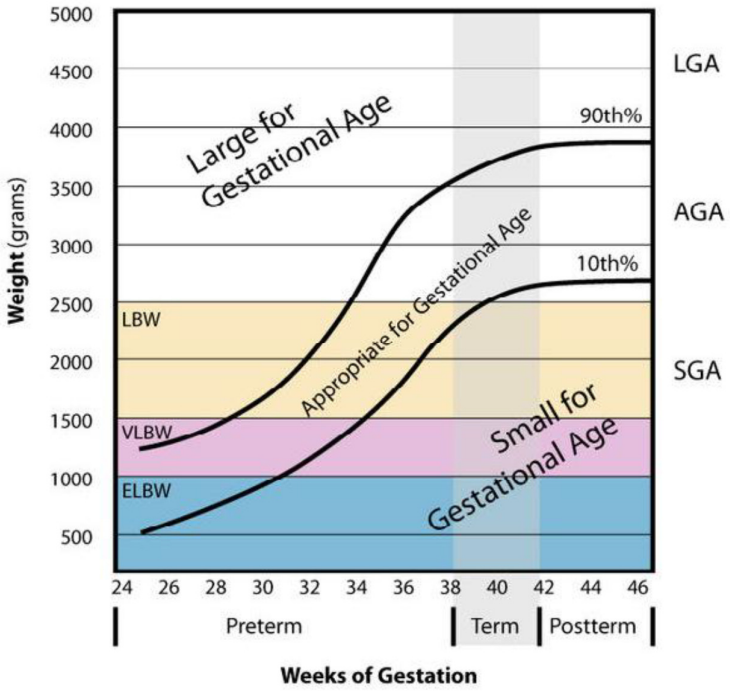
TYPE	TYPE1/ SYMMETRICAL / EARLY ONSET IUGR	TYPE2/ ASYMMETRICAL / LATE ONSET IUGR
ONSET	Early in utero	Late onset
ETIOLOGY	Congenital infections, genetic disorders	Uteroplacental insufficiency, maternal malnutrition, hypertension
PATHOPHYSIOLOGY	• Impaired cell division • Decreased cell number • Irreversible	• Impaired cellular hypertrophy • Decreased cell size • Reversible

CLINICAL FEATURES	• Inadequate growth of head and body • Head: abdomen ratio may be normal	• Brain is spared, therefore head: abdomen ratio increased
PROGNOSIS	Poor prognosis	More favourable prognosis

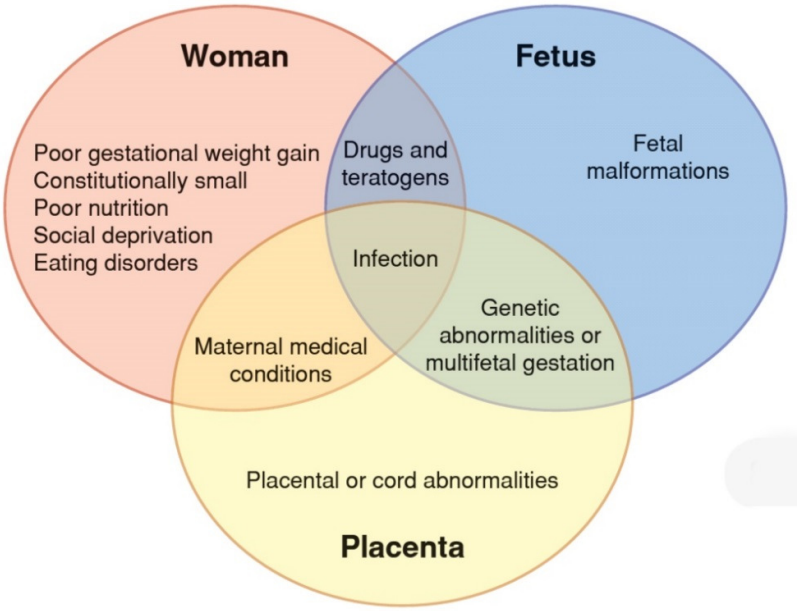
INTERMEDIATE IUGR:

It is a combination of type 1 and type 2 IUGR. As the term suggests, the insult to the fetal growth most probably occurs during the intermediate phase of fetal growth affecting both hyperplasia and hypertrophy, resulting in decrease of cell number as well as size. it approximately 5- 10 % of all growth restricted fetuses. Chronic hypertension, lupus nephritis and maternal vascular diseases that are severe and have onset in early second trimester, result in intermediate IUGR.

In utero Growth Status according to Birthweight percentile Fetal Growth



3. ETIOLOGY AND RISK FACTORS:



RISK FACTOR

FGR may be caused by maternal, placental, or fetal factors. Approximately one-third of FGRs are due to genetic causes, and twothirds are related to the fetal environment. However, no underlying etiology can be identified in at least 40 percent of SGA infants.

FETAL FACTORS

Genetic factors —

Population-based intergenerational studies of birth weight have found that genetic factors contribute 30 to 50 percent of the variation in birth weight [9]. Maternal genes influence birth weight more than paternal genes, but both have an effect. Specific allelic variants associated with birth weight include mutations in GCK and HNF1beta, which have been associated with low birth weight, and mutations in HNF4 alpha, which have been associated with high birth weight. Variants in ADCY5 and loci near CCNL1 also appear to lower birth weight [10]. The susceptibility to FGR is also heritable; in epidemiologic studies, women who were SGA themselves at birth have a two-fold increase in risk of FGR in their offspring [11,12]. Women who give birth to a growth restricted fetus are at high risk of recurrence, and the risk increases with increasing numbers of FGR deliveries.

Chromosome Abnormalities –

Karyotypic abnormalities account for up to 20 percent of all FGR [13,14]. The presence of a chromosomal abnormality often results in restriction of fetal growth early in pregnancy; as many as one-quarter of fetuses with early onset FGR have chromosomal abnormalities. Most cases are symmetric, but asymmetric early FGR also occurs [15]. Chromosomal abnormalities associated with FGR include [16]:

- Aneuploidy (e.g. trisomy 18 or 13, Turner 45 X, triploidy)
- Partial deletions (e.g. Cri du chat syndrome 5q, Wolf-Hirschhorn syndrome 4q)
- Ring chromosomes
- Uniparental disomy (e.g. for chromosomes 6, 14, and 16)
- Confined placental mosaicism
- Gene mutations (e.g. mutations in the gene for insulin-like growth factor)

Multiple gestation:

Fetal growth in multiple gestations has a direct relationship to the number of fetuses present; the type of placentation also plays a role (monochorionic versus dichorionic). Growth is similar to that of singletons until the third trimester and then slows.

The lower weight of fetuses from multiple gestations is thought to be due to an inability of the environment to meet the nutritional needs of multiple fetuses, as well as pregnancy complications more common in multiple gestation (eg, maternal undernutrition, preeclampsia, twin-twin transfusion, congenital anomalies). Placental and umbilical cord anomalies potentially associated with underperfusion (e.g. velamentous cord insertion) are also more common in multiple gestations.

Infection — Infections that develop early in pregnancy have the greatest effect on subsequent growth, but account for less than 5 percent of all cases of FGR. Viruses and parasites (e.g. rubella, toxoplasmosis, cytomegalovirus, varicella-zoster, malaria, syphilis, herpes) may gain access to the fetus transplacentally or across the intact fetal membranes and impair fetal growth by a variety of mechanisms (e.g. cell death, vascular insufficiency). Although uncommon, CMV (Cytomegalo Virus) is the most frequent viral etiology of FGR in developed countries [17].

There is less evidence implicating bacterial infection as an etiology for FGR, although maternal infection with listeria, tuberculosis, chlamydia, and mycoplasma has been reported to increase the risk to FGR.

PLACENTAL FACTORS

Many cases of FGR, particularly recurrent cases, are the result of ischemic placental disease. This term refers to a disease process of the placenta that clinically manifests as preeclampsia, FGR, abruption, or a combination of these disorders [18,19]. All of these disorders may be associated with preterm birth or fetal loss and represent late manifestations of abnormal placental development dating from the earliest stages of pregnancy.

Gross and histological lesions —

Any mismatch between fetal nutritional or respiratory demands and placental supply can result in impaired fetal growth. Studies have suggested that there is significant excess placental functional capacity. In sheep models, fetal growth is affected when one-half of the placenta is removed.

The human fetus may be more sensitive to a reduction in placental mass: placental weight is 24 percent smaller in growth restricted fetuses than in normally grown fetuses when adjustments are made for gestational age [20].

However, placental functional capacity cannot be accurately assessed from placental weight or dimensions alone. Abnormal development, narrowing or obstruction of placental vessels, and physical separation at the maternal interface all impair placental function. The types, distributions, and sizes of parenchymal and vascular lesions also play a role; moreover, some maternal disorders (eg, severe maternal malnutrition or alcohol abuse) can affect fetal nutrition without causing a recognizable histopathological lesion [21]

Identifiable placental histological abnormalities associated with fetal undernutrition include abnormalities of the uteroplacental vasculature (maldevelopment, obstruction, disruption), chronic abruption, chronic infectious and idiopathic inflammatory lesions (eg, infection related villitis, chronic villitis of unknown etiology), infarction, distal villous hypoplasia, massive perivillous fibrin deposition (i.e. maternal floor infarction), and thrombosis in the uteroplacental, intervillous and/or fetoplacental vasculature [22]. Diffuse chronic villitis of unknown etiology appears to be the most common placental finding in otherwise idiopathic FGR [17,22,23]. Gross placental structural anomalies possibly associated with FGR include single umbilical artery, velamentous umbilical cord insertion, bilobate placenta, circumvallate placenta, placental hemangioma, and, possibly, placenta previa.

Confined placental mosaicism — Confined placental mosaicism (CPM) refers to chromosomal mosaicism (usually involving a trisomy) found in the placenta, but not in the fetus. It occurs significantly more often in the placentas associated with FGR than in controls of normal weight. Approximately 10 percent of placentas associated with idiopathic FGR have been reported to have CPM [24,25]; the rate of CPM in controls undergoing CVS is about 1 percent. The extent of FGR depends upon the chromosomes involved, the proportion of mosaic cells, and the presence of uniparental disomy [26].

Placentas with CPM have a high ratio of placental infarcts and decidual vasculopathy, and one-third of placentas with these findings and FGR have CPM

MATERNAL FACTORS

Reduction in uteroplacental blood flow

Uteroplacental blood flow may be diminished by faulty development, acquired obstruction, or disruption of the uteroplacental vasculature. Maternal medical disorders (e.g. hypertension, renal insufficiency, diabetes, collagen vascular disease, systemic lupus erythematosus, antiphospholipid syndrome) and obstetrical complications (e.g. preeclampsia) associated with vasculopathy and/or reduced maternal blood volume or blood pressure diminish uteroplacental perfusion and result in FGR [27]. Preeclampsia, in particular, is characterized by primary failure of trophoblast invasion of the spiral arteries leading to failure of dilatation of these vessels, acute atherosis, occlusion, and infarction.

Constitutionally small mothers

If a women begins pregnancy weighing less than 100 pounds, the risk of delivering an SGA infant is increased at least twofold (simpson and colleagues, 1975). Moreover, intergenerational effects on birthweight are transmitted through the maternal line such that reduced intrauterine growth of the mother is the risk factor for reduced intrauterine growth of her offspring.

Diminished caloric intake —

Maternal weight at birth, prepregnancy weight, and weight gain during pregnancy are generally responsible for about 10 percent of the variance in fetal weight [28]. However, severe maternal starvation during pregnancy can have a major impact on fetal growth. As an example, the Dutch population suffered severe famine during the winter of 1944 to 1945; mean maternal caloric intake fell to 450 to 750 kcal a day. As one result of this deprivation, average infant birth weight during this period decreased by 250 grams. Similarly, in Leningrad during the World War II German siege, which resulted in a longer and more profound starvation period (down to 300 kcal of mostly carbohydrates and no protein), average birth weight fell by more than 500 grams.

Modest degrees of nutritional deficiency also have an effect on birth weight. Women who are underweight at the start of pregnancy or have poor weight gain during pregnancy are at higher risk of delivering an infant weighing less than 2500 grams.

Hypoxemia —

Chronic maternal hypoxemia due to pulmonary disease, cyanotic heart disease, or severe anemia is associated with diminished fetal growth. As an example, a study of 96 pregnancies in women with cyanotic congenital heart disease reported that the mean birth weight of full-term infants was only 2575 grams, which is significantly lower than the mean birth weight of 3500 grams in the general population [29]. Residing at high altitude also results in a chronic hypoxemic state and lower birth weight. A direct relationship between increasing altitude and lower birth weight has been demonstrated. Birth weight data from 15 areas in Peru located anywhere from sea level to 4575 meters showed birth weight declines an average of 65 grams for every additional 500 meters in altitude above 2000 meters [30]. The fetus can compensate for hypoxemia in a number of ways, including redistribution of circulation to vital organs and deferment of growth, decreased gross body movements, and increasing tissue oxygen extraction. The exact level and duration of fetal hypoxemia that exceed these compensatory mechanisms are not defined in humans.

Hematological and immunologic disorders —

Hematological disorders, such as sickle cell disease, may cause thrombosis of the intervillous space. Autoimmune and alloimmune disorders (e.g. antiphospholipid syndrome) may cause chronic villitis, as well as vasculopathy. Fetal undernutrition and hypoxia are possible sequelae.

Substance use and cigarette smoking — Maternal substance use, including cigarette smoking, alcohol consumption, and illicit drug use can cause FGR either by a direct cytotoxic effect or indirectly from related variables, such as inadequate nutrition. Smoking during the third trimester appears to have the greatest impact on birth weight; women who quit smoking by the third trimester have birth weights similar to those of nonsmokers [31].

Toxins — Toxic exposures, including various medications such as warfarin, anticonvulsants, antineoplastic agents, and folic acid antagonists, can produce FGR with specific dysmorphic features [32,33]. Fetal exposure to therapeutic, but not diagnostic, doses of radiation can cause permanent restriction of growth. Prepregnancy radiation therapy to the pelvis can result in anatomic changes in the pelvic vasculature that may lead to reduced fetoplacental perfusion and growth restriction.

Assisted reproductive technologies:

Singleton pregnancies conceived via assisted reproductive technologies have a higher prevalence of both low birth weight and SGA infants compared with naturally conceived pregnancies.

Others:

- FGR is more common among pregnancies at the extremes of reproductive life.
- Uterine malformations may affect uteroplacental perfusion and result in FGR

- A short interpregnancy interval has been associated with low birth weight and FGR, and this may be mediated through a relative depletion in folic acid.
- Chronic maternal stress may also be a factor. Chronic stress is associated with elevated corticotropin-releasing hormone (CRH) levels, which, in turn, may be associated with impaired fetal growth and preterm birth.

4. PATHOPHYSIOLOGY

Interference with placental nutrient supply can affect all aspects of placental function. The gestational age at onset, the magnitude of the injury, and the success of adaptive mechanisms determine the ultimate severity. Mild placental disease is more likely to affect organ function and maturation at the cellular level, with little perceivable growth delay perinatally, but may affect adult health (fetal programming), often through epigenetic modifications. With more severe placental disease, fetal growth delay and adaptive organ responses become evident in utero.

MECHANISMS OF PLACENTAL DYSFUNCTION

The efficiency of maternal to fetal exchange of nutrients, fluid, and waste can become suboptimal when there is a decrease in substrate transporters, an increase in the diffusion distance between maternal and the fetal compartments, a decrease in the exchange area or impedance to blood flow in the maternal and fetal compartments in the placenta. Typically, trophoblast invasion is confined to the decidual portion of myometrium, and the spiral and radial arteries do not transform into lowresistance vessels.

Altered expression of vasoactive substances increases vascular reactivity, and if hypoxia-stimulated angiogenesis is inadequate, placental autoregulation becomes deficient. Maternal placental floor infarcts and fetal villous obliteration and fibrosis increase placental blood flow resistance, producing a maternal-fetal placental perfusion mismatch that decreases the effective exchange area.

The severity of placental vascular dysfunction is clinically assessed in the maternal and fetal compartments of placenta with Doppler ultrasound. An early diastolic notch in the uterine arteries at 12-14 weeks suggests delayed trophoblast invasion, whereas persistence of "notching" beyond 24 weeks provides confirmatory evidence.

METABOLIC AND CELLULAR EFFECTS OF PLACENTAL DYSFUNCTION

Oxygen and glucose consumption by the placenta is unaffected when nutrient delivery to the uterus is only mildly restricted and the fetal demands can be met by increased fractional extraction. Fetal hypoglycemia occurs uterine oxygen delivery and likely substrate delivery is less than a critical value and fetal oxygen uptake is reduced. Insulin is an important fetal growth factor. Fetal pancreatic insulin responses are blunted by mild hypoglycemia, allowing gluconeogenesis from hepatic glycogen stores. At this stage, fetal glucose stores and lactate are preferentially diverted to the placenta to maintain placental metabolic, endocrine, and nutrient transfer function. Hypoglycemia, hyper lactic acidemia , and growing base deficit correlate with the degree of fetal hypoxemia and protein energy malnutrition. Down-regulation of

several cellular transporters and the Na/H+ pump affects placental cellular function. Simultaneously, the principle endocrine growth axis (insulin and insulin like growth factors) as well as leptin-coordinated fat deposition is down-regulated.

FETAL RESPONSE IN MAJOR ORGANS

Enhanced blood flow to the individual organs is documented in the myocardium, spleen, and liver. Conversely, blood flow resistance in the peripheral pulmonary arteries, celiac axis, mesenteric vessels, kidneys, and femoral and iliac arteries increases. The overall effect is an improved distribution of well-oxygenated blood to vital organs, with preferential streaming of descending aorta blood flow to the placenta for reoxygenation. There is progressive decrease in the amniotic fluid volume after long-standing redistribution.

A delay occurs in all aspects of central nervous system maturation in fetuses with chronic hypoxemia. There is also a progressive decline in global fetal activity. This results in higher baseline heart rate, with lower short- and long-term variation.

FETAL DECOMPENSATION

If placental dysfunction is progressive or sustained, the adaptive mechanisms become exhausted and decompensation begins. Multipleorgan failure as a result of placental dysfunction is caused by the metabolic milieu and the regulatory loss of cardiovascular hemostasis. Metabolic abnormalities are exaggerated, acidemia worsens, and the risk of intrauterine damage or perinatal death increase dramatically.

5. DIAGNOSIS OF FGR

Early establishment of gestational age, ascertainment of maternal weight gain, and careful measurement of uterine fundal growth throughout pregnancy will identify many cases of abnormal fetal growth in low-risk women. Risk factors, including a previous growth-restricted fetus, have an increased risk of recurrence. In women with risks, serial sonographic evaluation is considered. Although examination frequency varies depending on indications, an initial early dating examination followed by an examination at 32 to 34 weeks, or when otherwise clinically indicated, will identify many growth-restricted fetuses. Even so, definitive diagnosis frequently cannot be made until delivery.Identification of the inappropriately growing fetus remains a challenge. There are, however, both simple clinical techniques and more complex technologies that may prove useful.

Diagnosis of FGR is important because it has demonstrable effects on survival and development of fetus.

CLINICAL ASSESSMENT

Clinical assessment is a screening tool for FGR in low risk pregnancies. Clinical assessment is based on assessment of past and present risk factors, physical examination, and ultrasound studies.

Accurate assessment of gestational age —

Determination of gestational age is of utmost importance for the diagnosis of IUGR. Although this usually calculated from the date of last menstrual period, the

gestational age so determined is not always reliable. This may be because of irregular cycles, lactation or recent use of oral contraceptives. However, even in women with regular menstrual cycles, ultrasound dating before 20 weeks of pregnancy provides a more accurate estimate of gestational age than by menstrual history.

Symphysis-fundal height measurement —

Clinically the most common method for detecting IUGR is the serial measurement of the symphysiofundal height. It is measured from the upper border of the pubic symphysis to the top of the uterine fundus using simple tape.

Symphysiofundal height increases by 1cm per week between 14 to 32 weeks. A lag in the fundal height of 4 weeks is suggestive if moderate IUGR, a lag of 6 weeks suggests severe IUGR. however, this method has low sensitivity 44%.

The accuracy of fundal height measurements for screening and diagnosis of FGR is controversial; Observational studies using symphysis-fundal height measurements

have reported a wide range of sensitivities: 28 to 86 percent of small fetuses were detected.

Abdominal palpation — Clinical assessment of fetal size by abdominal palpation does not perform well as a test for detecting FGR: sensitivities range from 30 to 50 percent.

SONOGRAPHIC SCREENING AND DIAGNOSIS

An initial sonographic examination at 16-20weeks followed by a second examination at 32-34weeks serial sonography should serve to identify many cases of fetal growth restriction (Ewigman and colleagues,1993)

With sonography, the most common method for identifying poor fetal growth is estimation of weight using multiple fetal biometric measurements. Combining head, abdomen, and femur dimensions has been shown to optimize accuracy, whereas little incremental improvement is gained by adding other biometric measurements Of the dimensions, femur length measurement is technically the easiest and the most reproducible. Biparietal diameter and head circumference measurements are dependent on the plane of section and may also be affected by deformative pressures on the skull. Last, abdominal circumference measurements are more variable. However, these are most frequently abnormal with fetal-growth restriction because soft tissue predominates in this dimension.

Commonly used parameters include biparietal diameter, head circumference, abdominal circumference, femur length and various morphometric ratios like HC/AC, and FL/AC. Ultrasound results need to be interpreted in terms of pretest risk

of FGR and take into account whether the subject population was at low, moderate, or high risk of fetal growth abnormality.

The morphometric tests are more likely to overlook fetuses with symmetric FGR, but can be used as confirmatory tests of suspected asymmetric FGR. As discussed above, symmetric FGR comprises 20 to 30 percent of growth restricted fetuses and asymmetric FGR occurs in the remaining 70 to 80 percent of the FGR population.

Abdominal circumference — When fetal growth is compromised, the fetal abdominal circumference (AC) is smaller than expected because of depletion of abdominal adipose tissue and decreased hepatic size related to reduced glycogen storage in the liver. An abdominal circumference within the normal range for gestational age reliably excludes growth restriction, whereas a measurement less than 5th percentile is highly suggestive of growth restriction (American College of Obstetricians and Gynecologists, 2000b).

Studies report that reduced AC is the most sensitive single morphometric indicator of FGR [40-43]. The performance of AC measurement was illustrated by a study of 3616 pregnancies over 25 weeks of gestation that had a single ultrasound examination performed within two weeks of delivery [45]. AC measurement predicted small for gestational age (SGA) infants (i.e., birth weight below the 10th percentile for GA) with sensitivity, specificity, positive and negative predictive values of 61, 95, 86, and 83 percent, respectively.

Measurement of AC was more predictive of FGR than measurement of either head circumference (HC) or biparietal diameter (BPD) or the combination of AC with either one of these two variables. The optimal time to screen for FGR was at approximately 34 weeks of gestation.

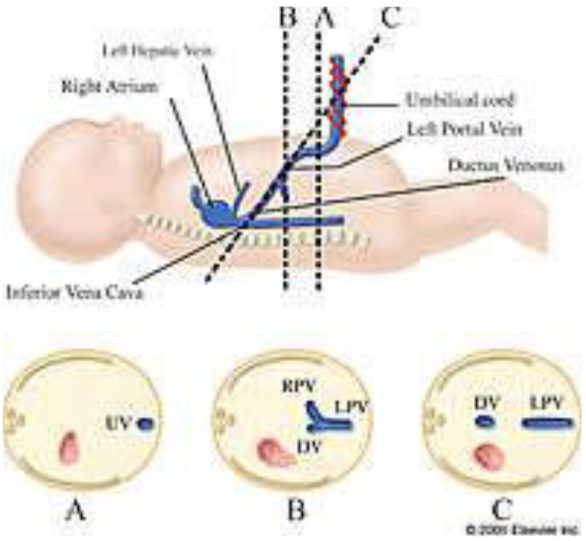
The following factors affect the sensitivity of the AC measurement:

- Symmetric versus asymmetric growth abnormality. AC is more sensitive in asymmetric FGR. [46].
- Gestational age. AC is more sensitive later in gestation. [47].
- Time interval between AC measurements. AC is more sensitive when the interval between measurements is more than two weeks [48].

MEASUREMENT OF ABDOMINAL CIRCUMFERENCE:

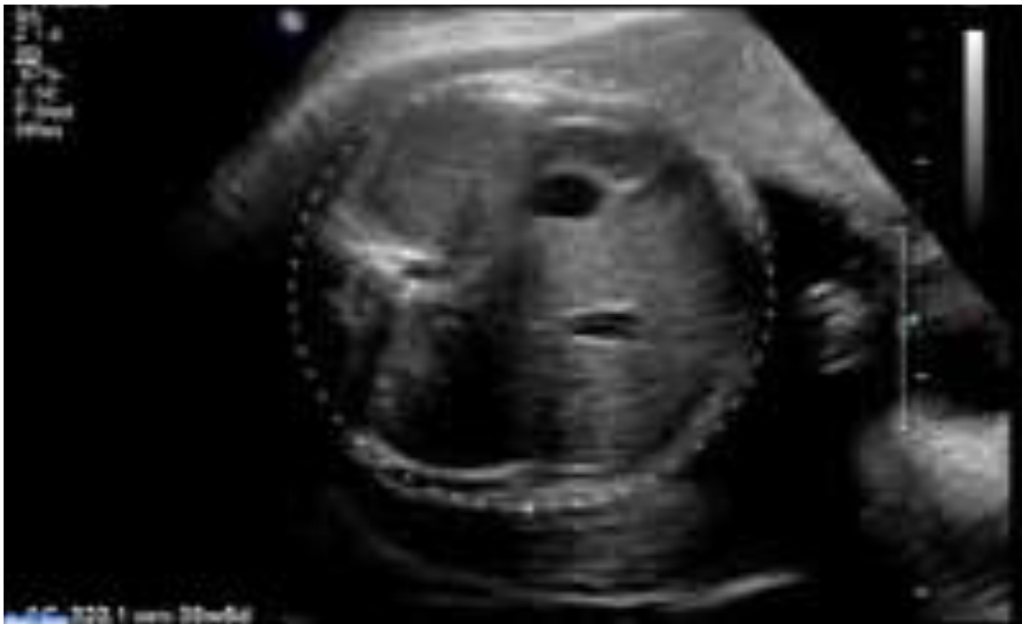
The abdominal circumference is obtained in the transaxial view of the fetal abdomen, at the level of fetal liver, using umbilical portion of the left portal vein as a landmark. The fetal stomach is at the same level, which is slightly caudal to the fetal heart and cephalad to the kidneys.

ABDOMINAL CIRCUMFERENCE



Umbilical venous circulation through the fetal liver. A. Plane of section depicting the umbilical vein (UV) in short axis.This plane is too caudal for abdominal circumference measurement. B. Plane of section through the junction of the left (LPV) and right (RPV) portal veins.This is the correct level for AC measurement (DV, ductus venosus). C. Plane of section aligned along the course of the LPV. Note that this plane is too inclined in a craniocaudal axis. (Illustration by James A. Cooper, MD, San Diego, CA.)

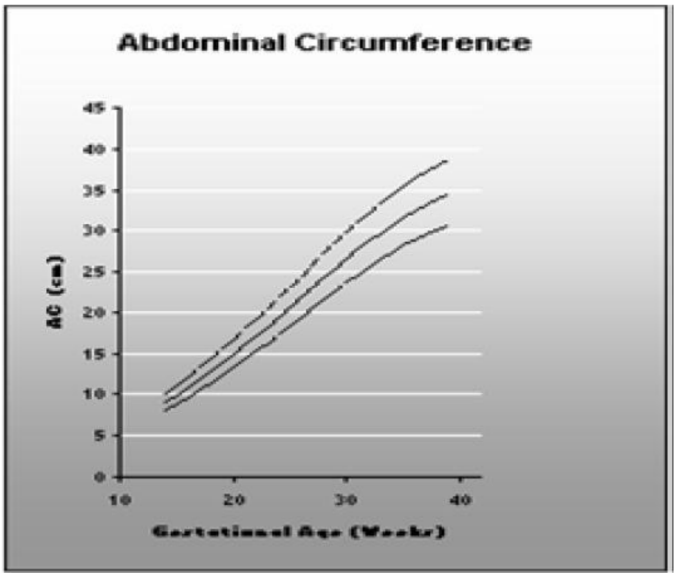
Plane of measuring abdominal circumference



Normal Range for Abdominal Circumference

Gestational age (mm) range (weeks + days)	Abdominal circumference (mm)		
	5th centile	median	95th centile
14+0-14+6	80	90	102
15+0-15+6	88	99	112
16+0-16+6	96	108	122
17+0-17+6	105	118	133
18+0-18+6	114	128	144
19+0-19+6	123	139	156
20+0-20+6	133	149	168
21+0-21+6	143	161	181
22+0-22+6	153	172	193
23+0-23+6	163	183	206
24+0-24+6	174	195	219
25+0-25+6	184	207	233
26+0-26+6	195	219	246
27+0-27+6	205	231	259
28+0-28+6	216	243	272
29+0-29+6	226	254	285
30+0-30+6	237	266	298

31+0-31+6	**246**	**277**	**310**
32+0-32+6	**256**	**287**	**322**
33+0-33+6	**265**	**297**	**334**
34+0-34+6	**274**	**307**	**345**
35+0-35+6	**282**	**316**	**355**
36+0-36+6	**289**	**324**	**364**
37+0-37+6	**295**	**332**	**372**
38+0-38+6	**302**	**339**	**380**
39+0-39+6	**307**	**345**	**387**



Estimated fetal weight (EFW):

Fetal weight estimation has become one of the most common methods of identifying the growth-restricted fetus. Equations that incorporate AC, BPD, and FL seem to provide the most accurate estimates of fetal weight[49]. In general, estimated fetal weight measurements are within 10 percent of the actual birthweight in 75 percent of patients in whom there is a clinical suspicion of FGR.

The average sensitivity, specificity, positive and negative predictive values for FGR using these parameter are approximately 90, 85, 80, and 90 percent, respectively [55-58]. The sensitivity is generally higher for infants with severe growth restriction (birth weight less than the 3rd percentile). But this can diagnose FGR only when the gestational age is known.

BIRTH WEIGHT PERCENTILE

Age (wk)	5th	10th	50th	90th	95th
20	249	275	412	772	912
21	280	314	433	790	957
22	330	376	496	826	1023
23	385	440	582	882	1107
24	435	498	674	977	1223
25	480	558	779	1138	1397
26	529	625	899	1362	1640
27	591	702	1035	1635	1927
28	670	798	1196	1977	2237
29	772	925	1394	2361	2553
30	910	1085	1637	2710	2847
31	1088	1278	1918	2986	3108
32	1294	1495	2203	3200	3338
33	1513	1725	2458	3370	3536
34	1735	1950	2667	3502	3697
35	1950	2159	2831	3596	3812
36	2156	2354	2974	3668	3888
37	2357	2541	3117	3755	3956
38	2543	2714	3263	3867	4027
39	2685	2852	3400	3980	4107
40	2761	2929	3495	4060	4185
41	2777	2948	3527	4094	4217
42	2764	2935	3522	4098	4213
43	2741	2907	3505	4096	4178
44	2724	2885	3491	4096	4122

Source : Alexander and associates (1996).

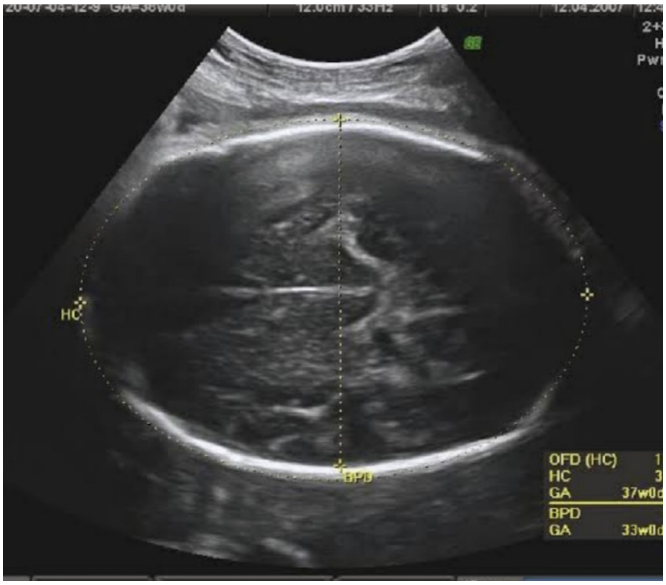
Growth velocity — As discussed above, the use of any parameter (eg, AC, EFW) in the prediction of FGR is based upon accurate assessment of GA. If dates are unknown, serial sonographic examinations at two-week intervals should be performed to evaluate the rate of interval growth (ie, growth velocity). Irrespective of GA, there is a significantly lower rate of change over time of AC or EFW in FGR fetuses compared with those fetuses whose growth is appropriate for GA. In one

study, as an example, a change in fetal AC of less than 10 mm over a two-week period had a sensitivity of 85 percent and specificity of 74 percent for identifying FGR [50]. Fetuses with normal growth velocity are at low risk of complications associated with FGR.

HEAD CIRCUMFERENCE:

It is a better measurement than BPD in predicting IUGR as it is not subjected to variability as is BPD. The cephalic index which is the ratio of BPD to occipito frontal diameter, is age independent and helps in identifying dolicocephaly and brachycephaly.

HC is measured on an axial plane traversing thalami and cavum septum pellucidum with the transducer perpendicular to the central axis of the head. The cerebral hemispheres and calvaria should appear symmetric and the cerebellar hemispheres should not be visible on this plane. The ellipse must be drawn with calipers around the outer aspects of the calvarium.



Body proportions — The HC/AC ratio, FL/AC ratio, and ponderal index have also been used to identify the growth restricted fetus, particularly in the setting of asymmetric FGR.

HC/AC ratio —

The HC/AC ratio has been proposed for evaluating fetuses with asymmetric FGR. In these infants, the size of the liver tends to be disproportionately small compared to the circumference of the head or length of the femur, which are initially spared from the effects of nutritional deficiency.

The HC/AC ratio decreases linearly throughout pregnancy and a ratio greater than 2 standard deviations (SD) above the mean for GA is considered abnormal. The sensitivity, specificity, positive and negative predictive values of an abnormal HC/AC in a population with FGR of mixed etiologies were 36, 90, 67, and 72 percent, respectively [51]. These findings demonstrate that an abnormal HC/AC ratio is more accurate in predicting FGR related to uteroplacental insufficiency (often asymmetric) than FGR from other etiologies (often symmetric). However, not all fetuses with an elevated HC/AC ratio have FGR. As an example, macrocephaly could also be associated with an abnormal HC/AC, which would be unrelated to FGR.

FL/AC ratio — The FL/AC ratio uses sonographic elements that relate to both weight and length in the prediction of FGR. An FL/AC ratio greater than 23.5 percent has a sensitivity of 56 to 64 percent and specificity of 74 to 90 percent for identification of asymmetric FGR[52]. This ratio is independent of GA in normally grown fetuses in the last half of pregnancy. However, an abnormal FL/AC ratio does

not accurately diagnose symmetric FGR. The sensitivity, specificity, positive and negative predictive values of the 90th percentile of FL/AC ratio in a mixed population of FGR fetuses were 30, 91,14, and 96 percent, respectively [53].

Therefore, the FL/AC ratio is unsuitable for screening for FGR in the general population.

Ponderal index:

PI is often used as an index (ie, PI = [weight (in g) x 100] ÷ [length (in cm)](3) to define growth restriction(54). A fetal PI has been calculated based upon a sonographically derived EFW and measurement of the FL. One study reported sensitivity, specificity, and positive predictive value of the fetal PI for FGR of 77, 82, and 36 percent, respectively; however, there was a poor correlation between fetal and neonatal PI [55). With normal growth, the PI increases gradually from 30 to 37 weeks gestation and then remains constant. Decreased growth of adipose tissue and skeletal muscle, the major contributors to body weight, results in a reduced PI. Reductions in PI or other indices, such as the ratio of mid-arm to occipito-frontal circumference, can identify growth restriction in newborns whose weight is greater than the 10th percentile. PI of less than 10th percentile reflects fetal malnutrition; PI of less than third percentile indicates severe wasting.

Amniotic fluid volume —

An association between pathological fetal-growth restriction and oligohydramnios has long been recognized (Chap. 11, p. 236). Chauhan and colleagues (2007) found oligohydramnios in nearly 10 percent of pregnancies suspected of growth restriction. This group of women was two times more likely to undergo cesarean delivery for nonreassuring fetal heart rate patterns. Petrozella and associates (2011) reported that decreased amnionic fluid volume between 24- and 34-weeks' gestation was significantly associated with malformations. In the absence of malformations, a birthweight < 3rd percentile was seen in 37 percent of pregnancies with oligohydramnios, in 21 percent with borderline amnionic fluid volume, but in only 4 percent with normal volumes. Hypoxia and diminished renal blood flow has been hypothesized as an explanation for oligohydramnios.

However, Magann and coworkers (2011) reviewed the literature and determined that the etiology of oligohydramnios is likely more complex and possibly involves altered intramembranous absorption as well.

Oligohydramnios refers to amniotic fluid volume that is less than expected for gestational age. It is typically diagnosed by ultrasound examination and may be described qualitatively or quantitatively by various methods. Oligohydramnios is one of the sequelae of FGR. The proposed mechanism is diminished fetal urine production due to hypoxia-induced redistribution of blood flow to vital organs at the expense of less vital organs, such as the kidney [56]. Oligohydramnios commonly occurs with complications of pregnancy other than FGR. In addition, a significant

proportion (approximately 15 to 80 percent) of fetuses with FGR do not have decreased amniotic fluid volume. Therefore, oligohydramnios is a poor screening modality for suboptimal growth [43,57]. However, if it is present in the absence of ruptured membranes, congenital genitourinary anomalies, or prolonged pregnancy, FGR is the most likely etiology.

Soft tissue measurements — FGR results in a decrease in both adipose tissue and muscle mass. Measurement of fetal soft tissue is probably predictive of FGR; however, there are inadequate data for defining the best site for measurement or the sensitivity and specificity of this parameter.

Doppler velocimetry doppler flow studies are an important adjunct to fetal biometry in identifying the IUGR fetus at risk of adverse outcome. the most widely used arterial idices are

- PULSATALITY INDEX (PI): systolic and diastolic peak velocity / time averaged maximum velocity
- RESISTANCE INDEX (RI): systolic and diastolic peak velocity / systolic peak velocity
- SYSTOLIC TO DIASTOLIC RATIO (S/D): systolic peak velocity / diastolic peak velocity.

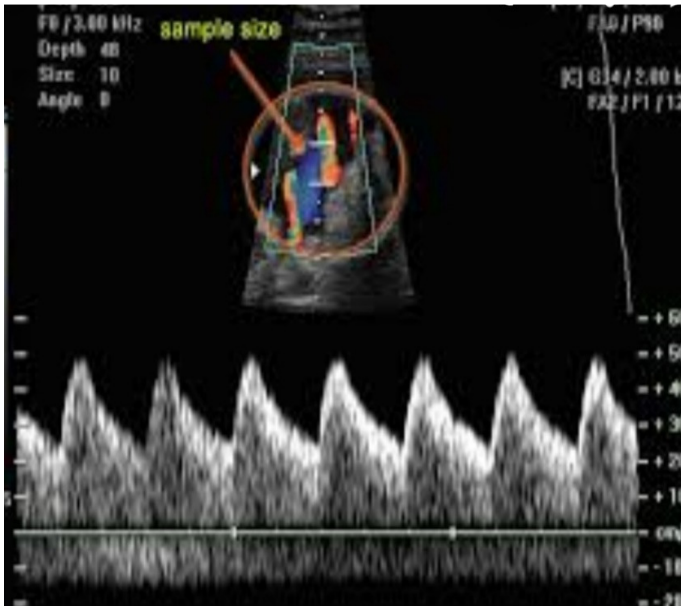
The essential vessels to be examined include the umbilical artery and middle cerebral arteries. As the vascular impedance in the placenta increases, fetal protective mechanisms are triggered which are reflected in the doppler studies. normal

pregnancy is characterized by a low resistance fetoplacental system with continuous flow through the cardiac cycle . where there is under perfusion of the placenta, the tertiary villi capillary bed is damaged resulting in increased placental resistance. This leads to decreased umbilical artery blood flow and systolic/ diastolic flow ratio.

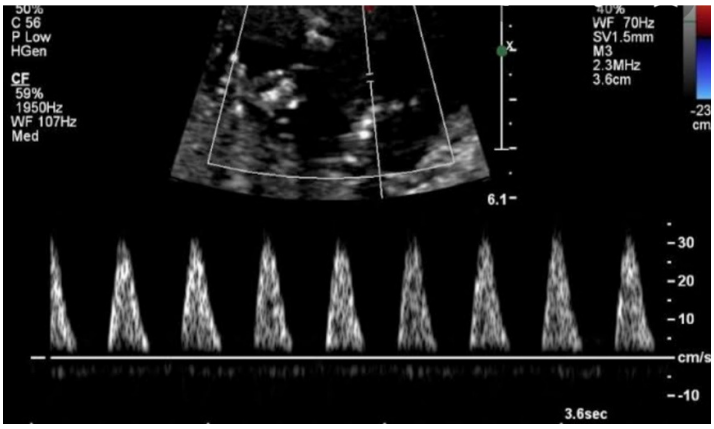
UMBILICAL ARTERY:

In IUGR there is a chronological process characterised by increased umbilical artery resistance , (increased S/D ratio), absent end diastolic flow . perinatal mortality rate increases significantly in fetuses with absent end diastolic flow (9-41%) and reversed end diastolic flow (33- 73%) in umbilical artery .

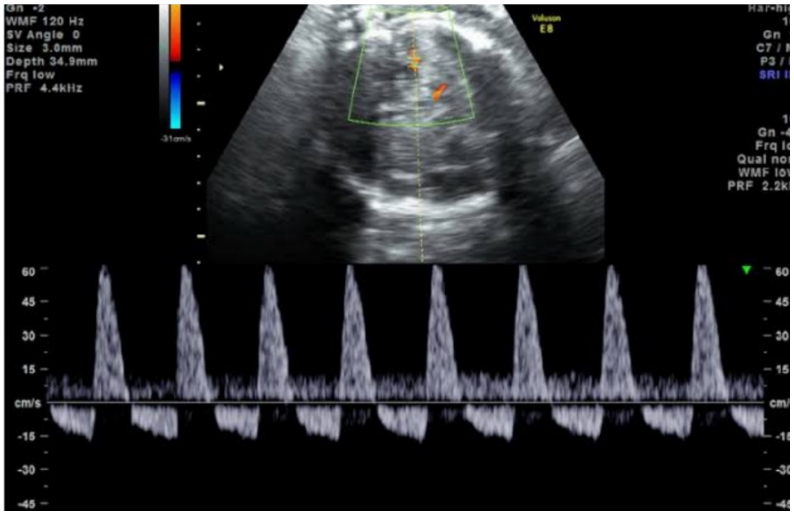
Umbilical artery can be used to distinguish between high risk small fetus that is truly growth restricted who needs increased monitoring and low risk small fetus



NORMAL BLOOD FLOW IN UMBILICAL ARTERY



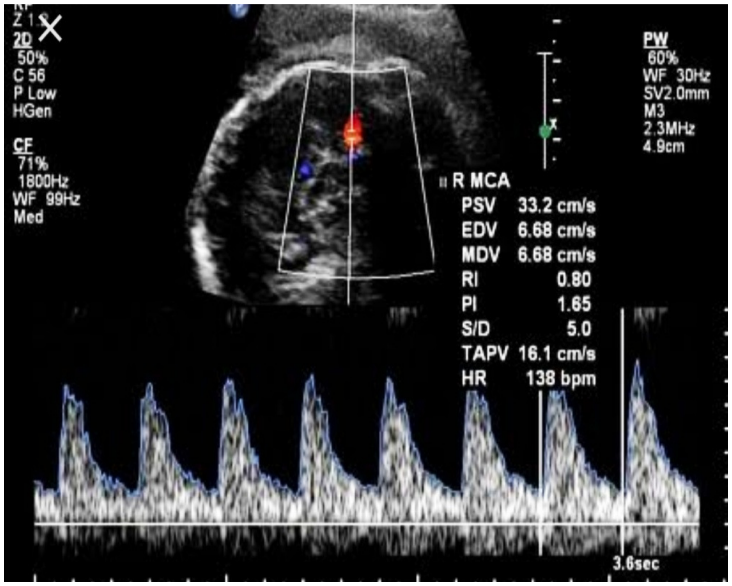
ABSENT DIASTOLIC FLOW IN UMBILICAL ARTERY



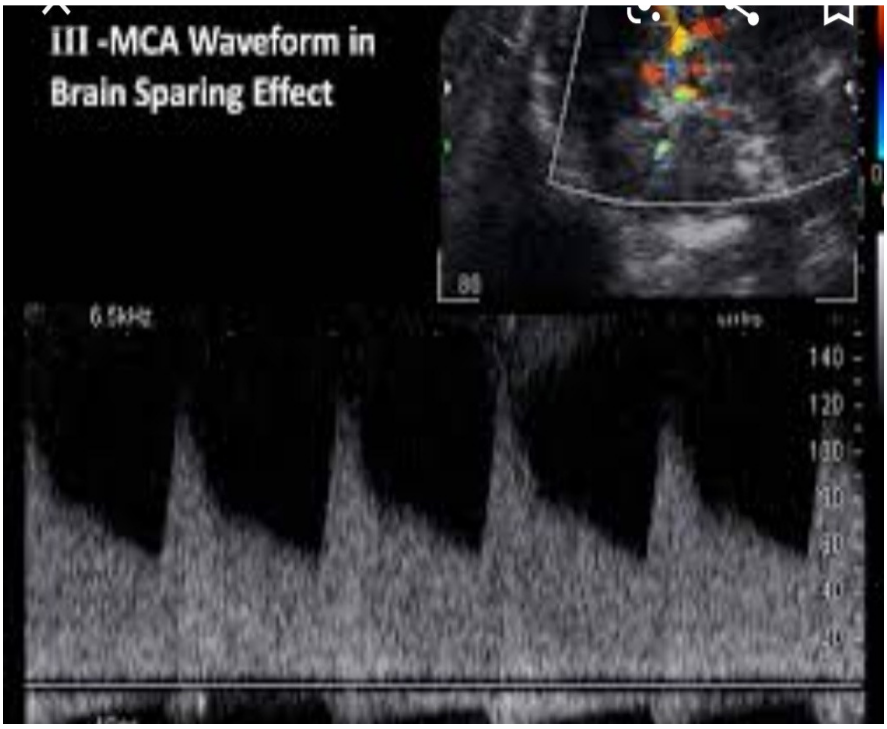
REVERSAL OF DIASTOLIC FLOW IN UMBILICAL ARTERY

MIDDLE CEREBRAL ARTERY

The middle cerebral artery doppler in normal fetus has relatively little flow during diastole. Increased resistance to blood flow in the placenta results in the redistribution of the cardiac output to favour cardiac and cerebral circulations. This results in an increased flow in the diastolic phase with reduced S/D ratio.



NORMAL MIDDLE CEREBRAL ARTERY FLOW



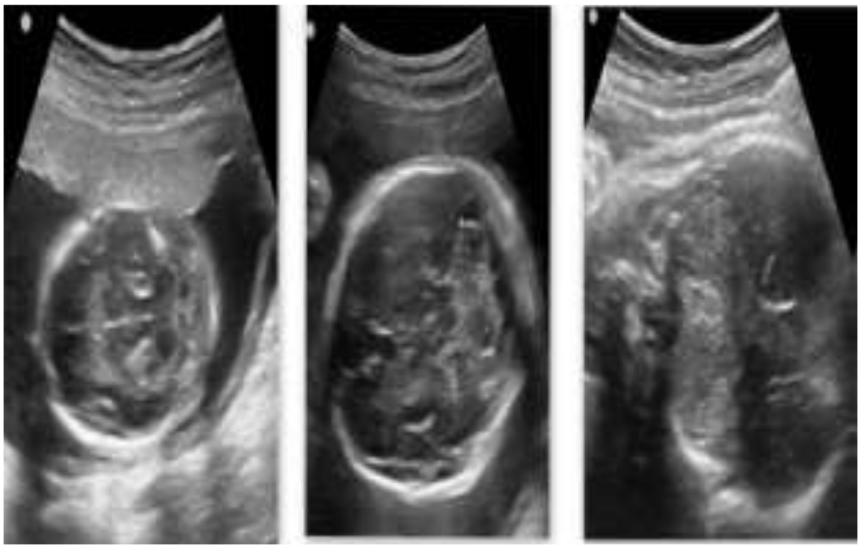
MIDDLE CEREBRAL ARTERY BRAIN SPARING EFFECT

6. THE NORMAL CEREBELLUM [63,64]

Cerebellum is in the posterior fossa and consists of two hemispheres connected by the vermis. Cerebellum is peanut shaped with central constriction denoting the vermis and flared ends representing two hemispheres. Its location in the posterior fossa (surrounded by the dense petrous ridges and occipital bone) makes it more resistant to deformation by extrinsic pressure. It has therefore been proposed that the transverse cerebellar diameter is a better predictor predictor of gestational age than the BPD when there are variations in the shape of the fetal head (dolichocephaly or brachycephaly).

On ultrasound, the cerebellar hemispheres are normally echo-poor to moderately echogenic, bounded superiorly by the echogenic tentorium cerebella. Cistern magna is a fluid collection posterior to the cerebellum. The vermis separates the cisterna magna from the fourth ventricle. Can be sonographically visualized as early as 9-10 weeks. It grows rapidly in the second trimester having a linear relationship with gestational age. Measurement in mm approximately equals the gestational age in weeks.

In prenatal ultrasound, an axial plane 15 to 30 degrees from the canthomeatal line visualizes both the cerebellum and cistern magna. This plane is usually reached by starting with the level where the standard BPD is obtained, then exaggerating the posterior tilt of the transducer to include the cerebellum. Measurement of the nuchal skin can also perform at this level in the early second trimester. The “banana sign” in fetuses with chiari 2 malformations is also seen at this level. Where the cerebellar hemispheres become oriented anteriorly and appear to wrap around the cerebral peduncles giving rise to the elongated crescentic “banana”.



Spot US images of posterior fossa with Gr I (A), Gr II (B), and Gr III (C) cerebellum with advancement from a fluid filled cystic eyeglass appearance to dumbbell configuration and final homogenous echogenic solid cerebellar tissue.

GRADES OF CEREBELLUM

Grade 1:

- Seen predominantly upto 27 weeks of gestation.
- Cerebellar hemisphere is rounded and lacks echogenicity.
- Vermis poorly developed giving the cerebellum the appearance of an "eyeglass".

Grade 2:

- Seen predominantly from 28-32 weeks of gestation.

- Vermis more prominent and appears as an echogenic rectangular tissue connecting both hemispheres.
- Cerebellar hemisphere is oval and the central portion is more echogenic than the peduncles but less echogenic than the circumferential margin of the hemisphere.
- Cerebellum has “dumbbell” appearance

Grade 3:

- Seen predominantly after 32 weeks of gestation.
- Hemispheres become triangular or “fan-shaped”.
- Echo pattern from the central portion of the hemisphere is now similar to the margin of the vermis.
- Cerebellum now looks more solid than cystic

Transverse Cerebellar Diameter

The cerebellum can be measured in an axial plane using the transverse outer-to-outer margins. There is high degree of correlation between TCD and gestational age. Prior to 24 weeks the transverse cerebellar diameter in millimetres is equivalent to the gestational age in weeks following which there is a flattening of the growth curve [65]. Cerebellum is the last organ affected by decrease in the blood flow. In acute asphyxia, cerebellar blood flow remains unchanged as a consequence of redistribution of cardiac output [66]. To assess the fetal growth TCD has been one of

the most reliable parameters in assessing the growth and gestational age estimation [67]. Thus, TCD may serve as an independent indicator of GA against which other potential deviations of growth may be compared.

MEASUREMENT OF TRANSVERSE CEREBELLAR DIAMETER:

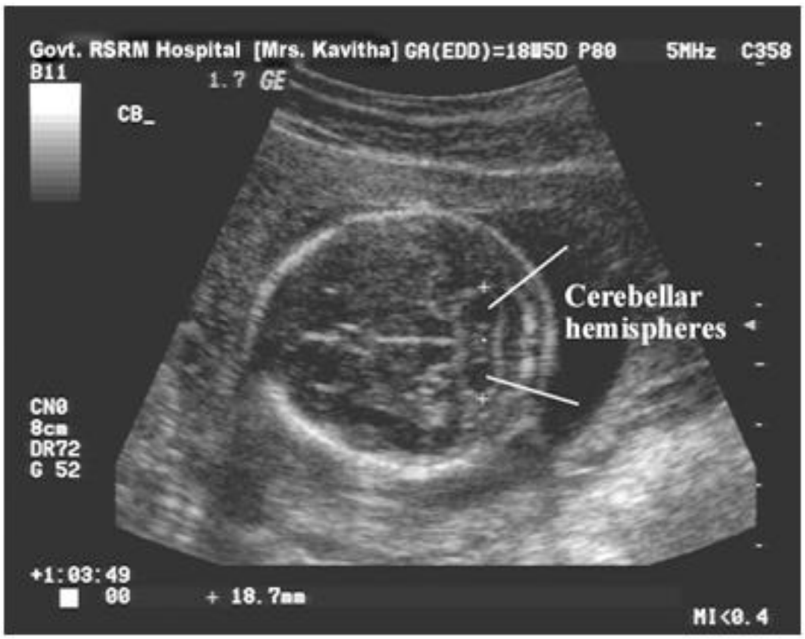
McLeasy et al (1984) and Goldenstein et al (1987) described the technique for measuring TCD, in which the usual thalamic plane used for BPD is obtained, the transducer is then rotated about 300 from reids baseline demonstrated the contents of posterior fossa. In all cases, the widest diameter of the cerebellum was measured. The vermis of the cerebellum, cerebellar hemispheres, cisterna magna and the nuchal translucency are seen in this plane. The cerebral peduncles, the falx cerebri and the cavum septum pellucidi are imaged in the midline.

PREDICTED MENSTRUAL AGE ACCORDING TO

TRANSVERSE CEREBELLAR DIAMETER MEASUREMENTS

Cerebellum (mm)	Menstrual Age (Week)	Cerebellum (mm)	Menstrual Age (Week)
14	15.2	35	29.4
15	15.8	36	30.0
16	16.5	37	30.6
17	17.2	38	31.2
18	17.9	39	31.8
19	18.6	40	32.3
20	19.3	41	32.8
21	20.0	42	33.4
22	20.7	43	33.9
23	21.4	44	34.4
24	22.1	45	34.8
25	22.8	46	35.3
26	23.5	47	35.7
27	24.2	48	36.1
28	24.9	49	36.5
29	25.5	50	36.8
30	26.2	51	37.2
31	26.9	52	37.5
32	27.5	53	38.0
33	28.1	54	38.3
34	28.8	55	38.5

Plane of measuring transverse cerebellar diameter



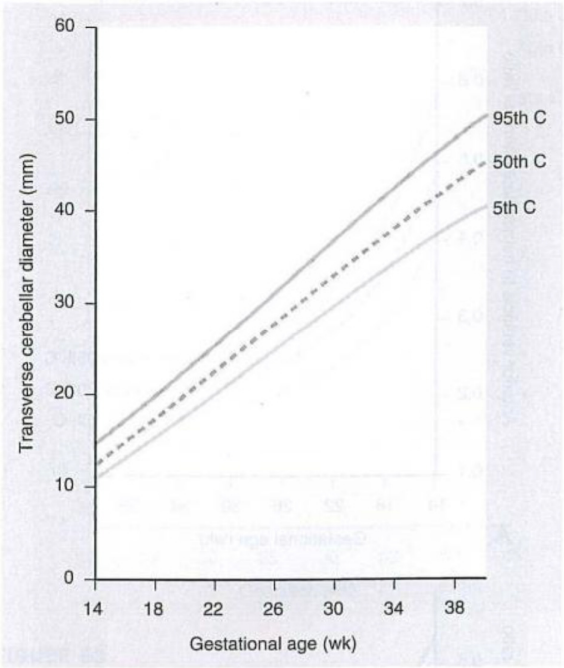
TCD/AC RATIO

This ratio compares the most preserved organ in the malnourished fetus, the cerebellum with the most compromised organ, liver, represented by fetal AC. In normally grown fetuses, there is a strong linear correlation with TCD measurement and AC. The TCD/AC ratio remains constant throughout gestation. A value exceeding 2 SD of the mean was significantly associated with birth of small-for-gestational age infant, being abnormal in 98% and 71% of asymmetrically and symmetrically growth-retarded infants respectively [69].

CALCULATION OF THE TCD/AC RATIO%:

TCD/ AC ratio% = TCD in cm /AC in cm x 100

Centile Chart for TCD



Relationship between Birth weight percentile and perinatal mortality and morbidity in SGA

COMPLICATIONS:

Fetal: (a) Antenatal—Chronic fetal distress, fetal death (b) Intranatal—Hypoxia and acidosis (c) After birth:

***Immediate*:** (1) Asphyxia, bronchopulmonary dysplasia and RDS

(2) Hypoglycaemia due to shortage of glycogen reserve in the liver

(3) Meconium aspiration syndrome

(4) Micro coagulation leading to DIC

(5) Hypothermia

(6) Pulmonary haemorrhage

(7) Polycythemia, anemia, thrombocytopenia

(8) Hyperviscositythrombosis

(9) Necrotizing enterocolitis due to reduced intestinal blood flow (10) Intraventricular hemorrhage

(11) Electrolyte abnormalities, hyper phosphatemia, hypokalemia due to impaired renal function

(12) Multiorgan failure

(13) Increased perinatal morbidity and mortality.

***Late*:** Asymmetrical IUGR babies tend to catch up growth in early infancy. The fetuses are likely to have:

(1) retarded neurological and intellectual development in infancy. The worst prognosis is for IUGR caused by congenital infection, congenital abnormalities and chromosomal defects.

Other long-term complications are: (2) Increased risk of metabolic syndrome in adult life: obesity, hypertension, diabetes and coronary heart disease (CHD). (3) LBW infants have an altered orexigenic mechanism that causes increased appetite

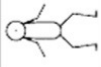
and reduced satiety. (4) Reduced number of nephrons—causes renal vascular hypertension.

Maternal: Per se fetal growth restriction does not cause any harm to the mother. But underlying disease process like pre-eclampsia, heart disease, malnutrition may be life threatening. Unfortunately for a woman with a growth retarded infant, risk of having another is two fold.

MORTALITY: The immediate neonatal mortality is about 6 times more than the normal newborn. However, it is lower than premature AGA infants of the same birth weight. Most of the babies die within 24 hours. The morbidity rate rises about 50 %. They are at higher risk for poor postnatal growth and adverse outcome.

The New Ballard Score

NEUROMUSCULAR MATURITY

SIGN	SCORE							SIGN SCORE
	-1	0	1	2	3	4	5	
Posture								
Square Window	 >90°	 90°	 60°	 45°	 30°	0°		
Arm Recoil		 180°	 140°-180°	 110°-140°	 90°-110°	 <90°		
Popliteal Angle	 180°	 160°	 140°	 120°	 100°	 90°	 <90°	
Scarf Sign								
Heel To Ear								
TOTAL NEUROMUSCULAR SCORE								

SIGN	SCORE							SIGN SCOR
	-1	0	1	2	3	4	5	
Skin	Sticky, friable, transparent	gelatinous, red, translucent	smooth pink, visible veins	superficial peeling &/or rash, few veins	cracking, pale areas, rare veins	parchment, deep cracking, no vessels	leathery, cracked, wrinkled	
Lanugo	none	sparse	abundant	thinning	bald areas	mostly bald		
Plantar Surface	heel-toe 40-50mm: -1 <40mm: -2	>50 mm no crease	faint red marks	anterior transverse crease only	creases ant. 2/3	creases over entire sole		
Breast	imperceptable	barely perceptable	flat areola no bud	stippled areola 1-2 mm bud	raised areola 3-4 mm bud	full areola 5-10 mm bud		
Eye / Ear	lids fused loosely: -1 tightly: -2	lids open pinna flat stays folded	sl. curved pinna; soft; slow recoil	well-curved pinna; soft but ready recoil	formed & firm instant recoil	thick cartilage ear stiff		
Genitals (Male)	scrotum flat, smooth	scrotum empty, faint rugae	testes in upper canal, rare rugae	testes descending, few rugae	testes down, good rugae	testes pendulous, deep rugae		
Genitals (Female)	clitoris prominent & labia flat	prominent clitoris & small labia minora	prominent clitoris & enlarging minora	majora & minora equally prominent	majora large, minora small	majora cover clitoris & minora		
TOTAL PHYSICAL MATURITY SCORE								

Signature of Examiner

MATURITY RATING

TOTAL SCORE	WEEKS
-10	20
-5	22
0	24
5	26
10	28
15	30
20	32
25	34
30	36
35	38
40	40
45	42
50	44

WHO FETAL GROWTH CHART

Gestational Age (Weeks)	Estimated Fetal Weight (g) by Percentile								
	2.5	5	10	25	50	75	90	95	97.5
14	70	73	78	83	90	98	104	109	113
15	89	93	99	106	114	124	132	138	144
16	113	117	124	133	144	155	166	174	181
17	141	146	155	166	179	193	207	217	225
18	174	181	192	206	222	239	255	268	278
19	214	223	235	252	272	292	313	328	340
20	260	271	286	307	330	355	380	399	413
21	314	327	345	370	398	428	458	481	497
22	375	392	412	443	476	512	548	575	595
23	445	465	489	525	565	608	650	682	705
24	523	548	576	618	665	715	765	803	830
25	611	641	673	723	778	836	894	938	970
26	707	743	780	838	902	971	1,038	1,087	1,125
27	813	855	898	964	1,039	1,118	1,196	1,251	1,295
28	929	977	1,026	1,102	1,189	1,279	1,368	1,429	1,481
29	1,053	1,108	1,165	1,251	1,350	1,453	1,554	1,622	1,682
30	1,185	1,247	1,313	1,410	1,523	1,640	1,753	1,828	1,897
31	1,326	1,394	1,470	1,579	1,707	1,838	1,964	2,046	2,126
32	1,473	1,548	1,635	1,757	1,901	2,047	2,187	2,276	2,367
33	1,626	1,708	1,807	1,942	2,103	2,266	2,419	2,516	2,619
34	1,785	1,872	1,985	2,134	2,312	2,492	2,659	2,764	2,880
35	1,948	2,038	2,167	2,330	2,527	2,723	2,904	3,018	3,148
36	2,113	2,205	2,352	2,531	2,745	2,959	3,153	3,277	3,422
37	2,280	2,372	2,537	2,733	2,966	3,195	3,403	3,538	3,697
38	2,446	2,536	2,723	2,935	3,186	3,432	3,652	3,799	3,973
39	2,612	2,696	2,905	3,135	3,403	3,664	3,897	4,058	4,247
40	2,775	2,849	3,084	3,333	3,617	3,892	4,135	4,312	4,515

doi:10.1371/journal.pmed.1002220.t011

CAN SCORE

Table 1. The Nine signs for CAN Status in the Newborn[4]

Hair	Large amount, smooth, silky, easily groomed (4). Thinner, some straight, 'staring' hair (3). Still thinner, more straight, 'staring' hair which does not respond to brushing (2). Straight 'staring' hair with depigmented strip (flag sign) (1).
Cheeks	Progression from full buccal pads and round face (4); to significantly reduced buccal fat with narrow, flat face (1)
Neck and Chin	Double or triple chin fat fold, neck not evident (4); to thin chin. No fat fold, neck with loose, wrinkled skin, very evident (1).
Arms	Full, round, cannot elicit 'accordion' folds or lift folds of skin from elbow or tricep area (4); to a striking 'accordion' folding of lower arm, elicited when examiner's thumb and fingers of the left hand grasps the arm just below the elbow of the baby and thumb and fingers of the examiners right hand circling the wrist of the baby are moved towards each other; skin is loose and easily grasped and pulled away from the elbow.
Legs	Like arms.
Back	Difficult to grasp and lift skin in the interscapular are (4); to skin loose, easily lifted in a thin fold from the interscapular area (1).
Buttocks	Full round gluteal fat pads (4); to virtually no evident gluteal fat and skin of the buttocks and upper posterior high loose and deeply wrinkled (1).
Chest	Full, round, ribs not seen (4); to progressively prominence of the ribs with obvious loss of intercostal tissues (1).
Abdomen	Full, round, no loose skin (4); to distended or scaphoid, but with very loose skin, easily lifted, wrinkled and 'accordion' folds demonstrable.

CAN score4 has nine superficial readily detectable signs, which are rated from 1 (worst-severe FM) to 4 (best well-nourished). The highest possible score is 36and lowest possible score is.9 A CAN score of ≤24 was taken as malnourished fetus.

RESULTS AND STATISTICAL ANALYSIS

In total, 200 patients participated in the present study. They were divided into two groups of 100 patients each. One group was controls (Group A) and another group is the test group (Group B).

TABLE 1: GROUP DISTRIBUTION

Group	Group A	Group B
No. of patients (n)	100	100
Type of patients	control	test

Demographic Data:

The age, weight and height of patients were noted. The data was analyzed statistically using the Student 't' test.

Distribution of age:

Both groups comprised of 100 patients each between 19 to 36 years of age with mean age of 26.27 years in Group A and 26.07 in Group B. There was no statistically significant difference in the age between the two groups (p= 0.711). (Table 2)

Distribution of BMI:

The mean BMI in the group B was 28.97 in patients with normal neonatal growth and 28.01 in patients with IUGR. There was significant difference between the two groups in BMI distribution patients with IUGR babies had lower BMI (p= 0.0002). (Table 2)

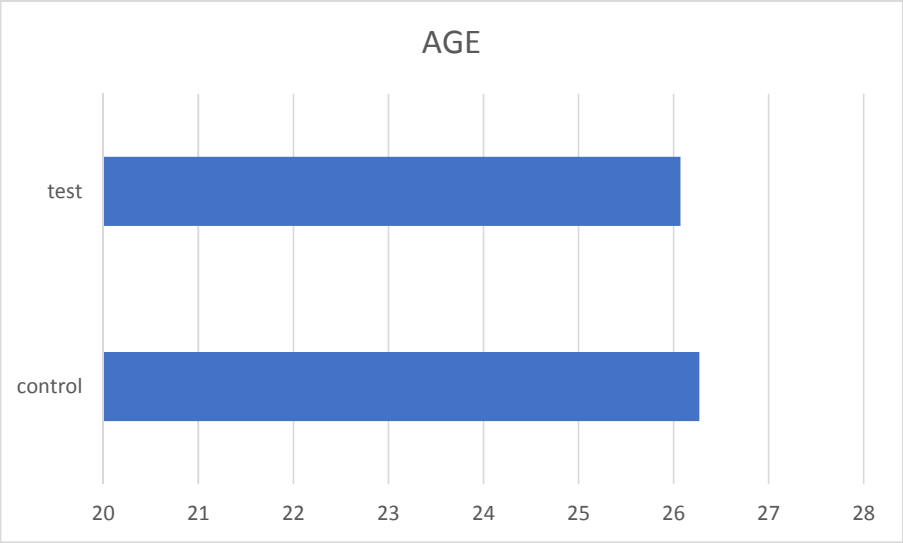
TABLE 2A: DISTRIBUTION OF AGE

	GROUP A		GROUP B		P-VALUE
	Mean	**SD**	**Mean**	**SD**	
AGE	26.27	3.581	26.070	4.051	0.711

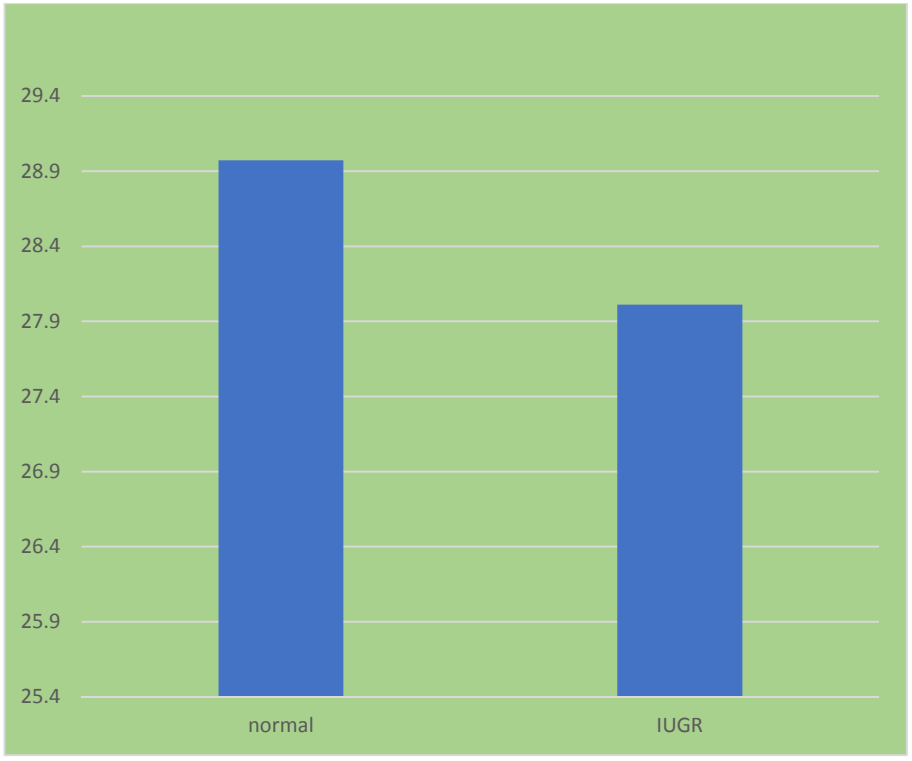
TABLE 2B: DISTRIBUTION OF BMI

GROUP B	**Normal growth**		**IUGR**		**P-VALUE**
	Mean	**SD**	**Mean**	**SD**	
AGE	28.97	1.539	28.01	2.007	0.0002

GRAPH 1 – MEAN AGE



GRAPH 2 – BMI



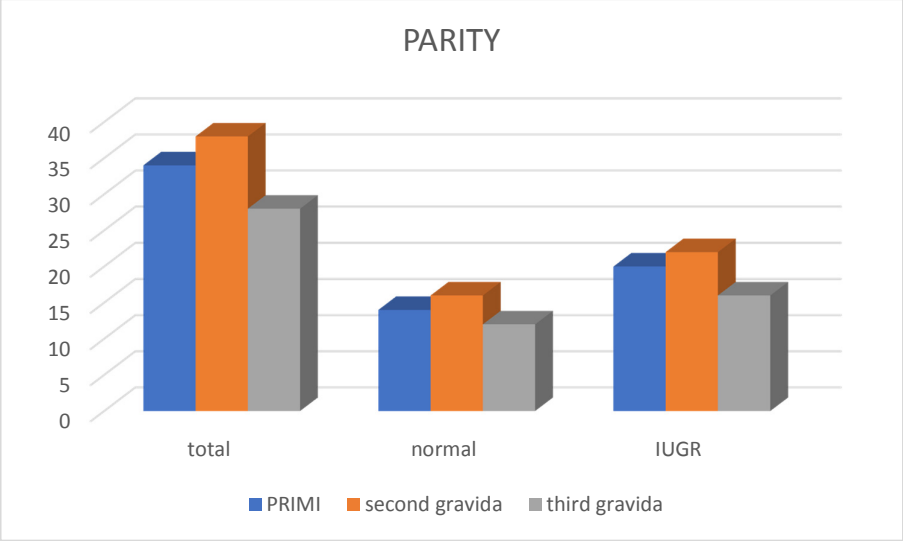
Distribution of Parity

Out of 100 patients in group B, there were no statistically significant difference between the two subgroups of patients delivering normal growth baby and IUGR baby, with respect to parity (table 4).

TABLE 3: PARITY

PARITY IN GROUP B	NORMAL BABY	IUGR BABY	TOTAL	SIGNIFICANCE
PRIMI	14	20	34	P= 0.511
SECOND GRAVIDA	16	22	38	P= 0.456
THIRD GRAVIDA	12	16	28	P=0.574

GRAPH 3: PARITY



Distribution of mode of delivery:

In the 100 patients of group B, there were 58 natural labour and 42 caesarean sections

TABLE 4; MODE OF DELIVERY

GROUP B	NATURAL LABOUR	LSCS
NUMBER	58	42

GRAPH 4: MODE OF DELIVERY

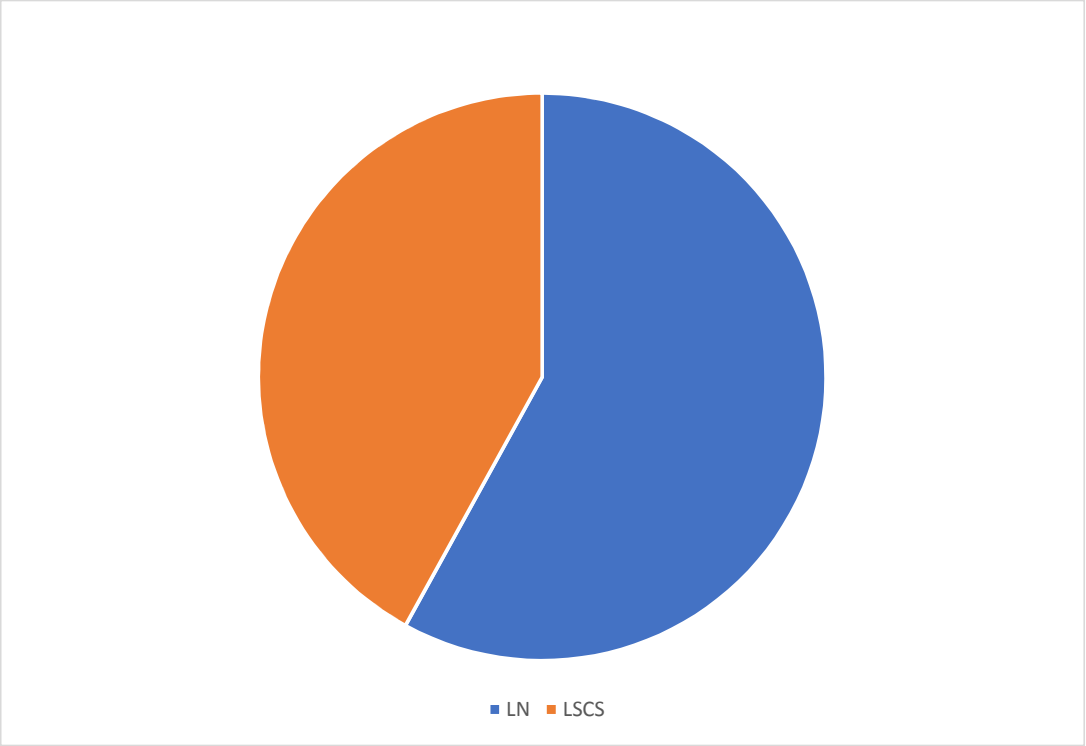


TABLE 5: APGAR

In our test group mean APGAR score was 7 at 1 minute and 8 at 5th minute in normal growth group, and 6.6 at 1 minute and 7.8 at 5th minute in IUGR group

APGAR	NORMAL		IUGR	
	1 MIN	5 MIN	1 MIN	5 MIN
MEAN	7	8	6.6	7.8

GRAPH 5: APGAR

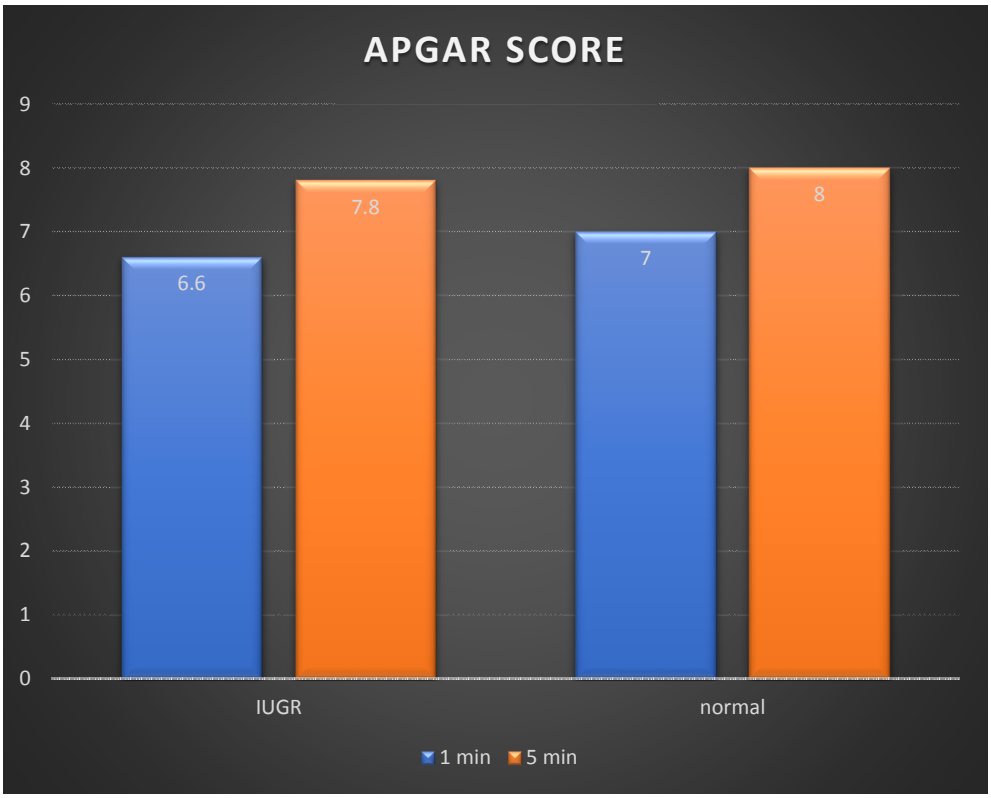


TABLE 6: NICU STAY

GROUP B NICU STAY	NORMAL (N= 44)	TRUE IUGR (N= 56)	SIGNIFICANCE
NUMBER	1	45	
PERCENTAGE	2.27%	80.36%	P<0.001

GRAPH 6: NICU STAY

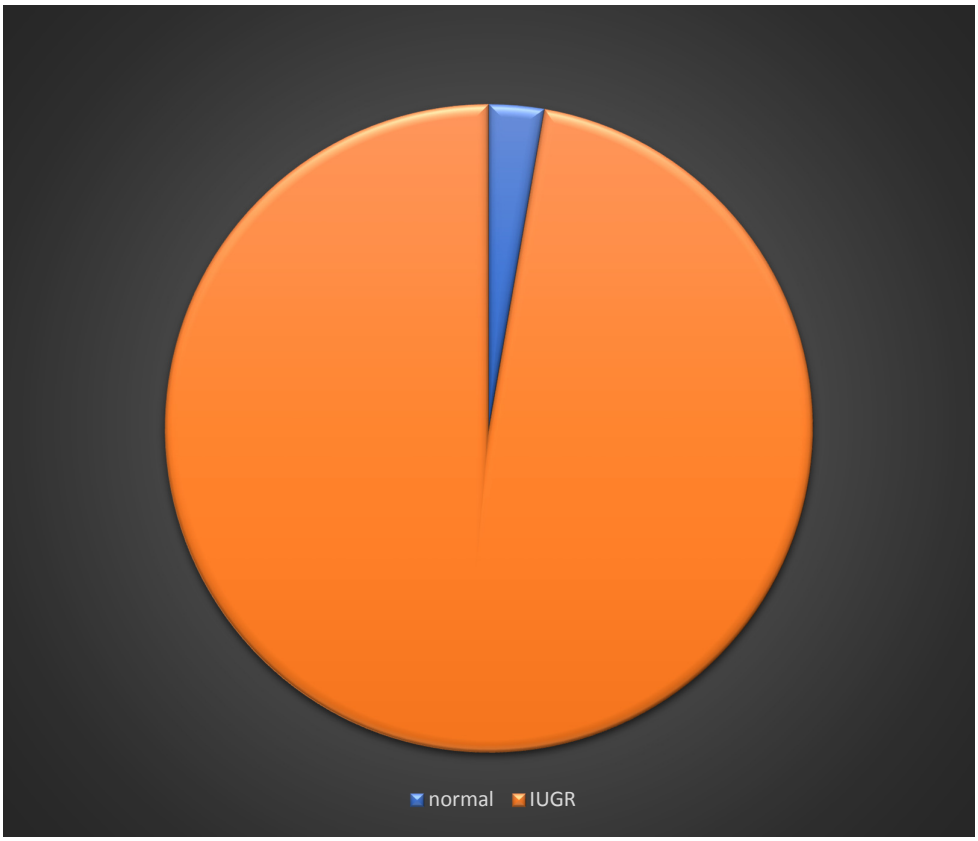


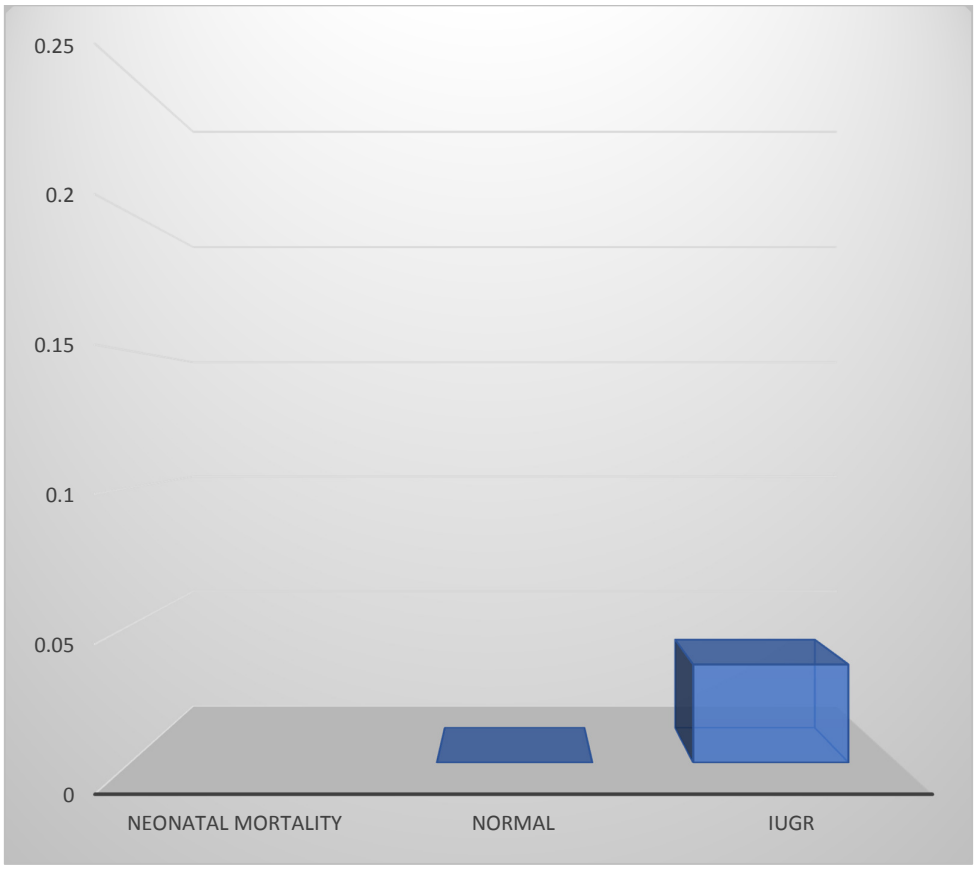
TABLE 7 NEONATAL MORTALITY:

Distribution of neonatal mortality:

None of the normal growth subgroup had neonatal mortality, whereas IUGR subgroup had 2 mortality out of 56 neonates.

Neonatal Mortality	**Normal**	**IUGR**
Number	0	2
Percentage	0%	3.57%

GRAPH 7 NEONATAL MORTALITY:



Sensitivity and specificity of TCD/AC:

In our study group, 56 had true FGR and 44 had AGA.

Table 8 true FGR and AGA:

Group B	True FGR	AGA
N=	56	44

GRAPH 8

true FGR and AGA:

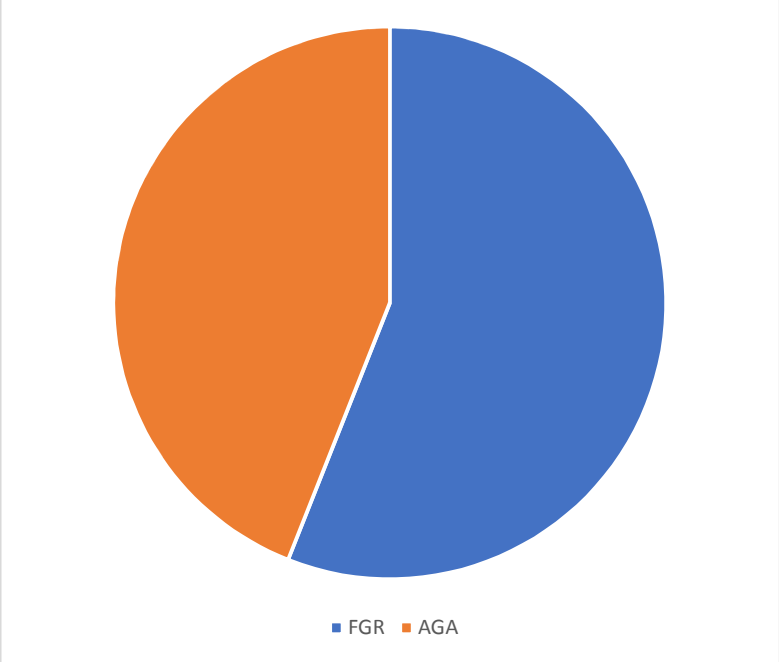


Table 9 TCD/AC:

In our study TCD/AC detected 47 out of 56 FGR, and 15 had false positive values

TCD/AC	**Positives**	**Negatives**	**Total**
FGR	47	9	56
AGA	15	29	44
Total	62	38	100

TRUE POSITIVE = 47

FALSE POSITIVE = 15

TRUE NEGATIVE = 29

FALSE NEGATIVE = 9

Graph 9 TCD/AC

Table 9B TCD/AC sensitivity and specificty:

Deriving sensitivity and specificity of TCD/AC, we get

Statistic variable	Value TCD/AC	CI interval
Sensitivity	83.93	71.67% to92.38%
Specificity	65.91	50.08% to 79.51%
Positive likelihood ratio	2.46	1.61 to 3.77
Negative likelihood ratio	0.24	0.13 to 0.46
Disease prevalence	56%	45.72% to 65.92%
Positive predictive value	75.81%	67.16% to 82.76%
Negative predictive value	76.32%	63.06% to 85.88%
Accuracy	76%	66.43% to 83.98%

Sensitivity and specificity of HC/AC:

In our study HC/AC detected 41 out of 56 FGR, and 26 had false positive values

Table 10 HC/AC:

HC/AC	Positives	Negatives	Total
FGR	41	15	56
AGA	26	18	44
Total	67	33	100

TRUE POSITIVE = 41

FALSE POSITIVE = 26

TRUE NEGATIVE = 18

FALSE NEGATIVE= 15

Graph 10 TCD/AC

Table 10B HC/AC sensitivity and specificty:

Deriving sensitivity and specificity of HC/AC, we get

Statistic variable	**Value HC/AC**	**CI interval**
Sensitivity	73.21%	59.70% to 84.17%
Specificity	40.91%	26.34% to 56.75%
Positive likelihood ratio	1.24	0.92 to 1.66
Negative likelihood ratio	0.65	0.37 to 1.15
Disease prevalence	56%	45.72% to 65.92%
Positive predictive value	61.19%	54.07% to 67.87%
Negative predictive value	54.55%	40.67% to 67.75%
Accuracy	59%	48.71% to 68.74%

Predictors of NICU admission:

Out of 46 NICU admissions, 40 had been detected as IUGR by TCD/AC ratio. And among all the fetus detected as IUGR by TCD/AC (n= 62), 40 neonates required NICU stay.

Table 11 Predictors of NICU admission:

TCD/AC	no NICU stay	NICU stay	Total
IUGR	22	40	62
no IUGR	32	6	38
Total	54	46	100

Hence, low TCD/AC ratio has relative risk of 4.08 for NICU admission of the neonate (P = 0.0003)

COMPARISION

Statistic variable	Value TCD/AC	Value HC/AC
Sensitivity	83.93	73.21%
Specificity	65.91	40.91%
Positive likelihood ratio	2.46	1.24
Negative likelihood ratio	0.24	0.65
Disease prevalence	56%	56%
Positive predictive value	75.81%	61.19%
Negative predictive value	76.32%	54.55%
Accuracy	76%	59%

DISCUSSION

In our study two groups of patients were selected 100 in control group and 100 in study group.

Group A contains 100 normal antenatal cases of gestational age > 30 weeks and group B contains 100 clinically detected IUGR cases of gestational age > 30 weeks with single intrauterine pregnancies.

Group B was chosen based on clinical suspicion of IUGR a discrepancy of 4 weeks in period of gestation on clinical examination.

Multiple pregnancies, polyhydramnios, anomalies, irregular menstrual cycles and symmetrical IUGR are excluded.

TCD/AC ratio and HC/ AC ratio of normal group are calculated. mean and standard deviation are calculated for the normal group. Then the values of the study group are compared with the normal group. The values more than 2SD are labelled as IUGR (sonographically).

Then those clinically suspected IUGR cases are followed upto delivery and post-delivery new ballard score and CAN score (clinical assessment of nutritional status at birth) are calculated.

Number of ultrasonographically detected IUGR compared with number of true IUGR and accuracy of both TCD/AC ratio and HC/AC ratio is compared.

- True positive values
- False positive values
- sensitivity
- specificity
- positive predictive value
- negative predictive value
- diagnostic accuracy

above mentioned are calculated and interpretation is done.

COMPARISION OF VARIOUS PARAMETERS OF TCD/AC IN DIAGNOSING ASYMMETRICAL IUGR

AUTHORS	GESTATIONAL AGE	CUT OFF	SENSITIVITY	SPECIFICITY	PPV	NPV	P VALUE
Vinkenstein et al	-		82	-	-	-	-
Campbell et al	15- 38	>15.9	71	77	79	68	<0.0001
Dhumale et al	18-34	13.56	-	-	-	-	-
Meyer et al	14-42	13.68	83	96.2	94.5	88	<0.0001
Hill et al	14-42	-	52	-	-	-	-

COMPARISION OF VARIOUS PARAMETERS OF HC/AC IN DIAGNOSING ASYMMETRICAL IUGR

AUTHORS	GESTATIONAL AGE	CUT OFF	SENSITIVITY	SPECIFICITY	PPV	NPV	P VALUE
Benson et al	-	Elevated	82	94	62	98	-
Divon et al	16-40	>2SD	36	90	67	72	-
Meyer et al	14-42	>2SD	49.3	87.6	75.6	69	<0.0001
Kurjak et al	16-40	>2SD	-	-	80	-	-

VALUES OF OUR STUDY

Statistic variable	**Value TCD/AC**	**Value HC/AC**
sensitivity	83.93	73.21%
Specificity	65.91	40.91%
Positive likelihood ratio	2.46	1.24
Negative likelihood ratio	0.24	0.65
Disease prevalence	56%	56%
Positive predictive value	75.81%	61.19%
Negative predictive value	76.32%	54.55%
accuracy	76%	59%

CONCLUSION

1. The TCD and AC measurements correlates well with gestational age.

2. The TCD and AC has strong linear relationship, hence the TCD/AC ratio is fairly constant throughout pregnancy.

3. TCD unlike AC is not affected in FGR, because of brain sparing.

4. Hence, TCD/AC ratio is increased in FGR.

5. As the TCD/AC ratio is constant throughout the pregnancy, it is a gestational age independent parameter, can diagnose FGR in antenatal women with unknown gestational age.

6. Hence, TCD/AC ratio can be a screening test to diagnose FGR in the antenatal period. So, that early intervention could be attempted to improve the perinatal outcome.

7.However TCD/AC ratio had a better diagnostic validity and accuracy compared to HC/ AC ratio in predicting asymmetrical IUGR.

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PROFORMA

Name

Age

Menstrual History

Obstetric Code

LMP

EDD

Dating Ultrasonogram : done / not done

Risk Factors

Preeclampsia Yes / No

Chronic Hypertension Yes / No

Oligohydramnios Yes / No

Gestational Diabetes Mellitus Yes / No

Chronic Renal Disease Yes / No

Vasculopathy Yes/No

Others Yes / No

GENERAL PHYSICAL EXAMINATION

Built & nourishment

Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy / Edema

VITAL SIGNS : T: PR: RR: BP:

Breast :

Thyroid :

Spine :

HT :

WT :

SYSTEMIC EXAMINATION

CVS :

RS :

CNS :

OBSTETRIC EXAMINATION

Date **WT** **BP**

P/A **SFH** **P/V**

INVESTIGATIONS

Routine:

Hb% **HIV**

Urine Routine

Blood Group and RH Typing

Blood Urea **Serum Creatinine**

LFT:

Total protein

Urine:

Albumin Sugar: Deposits

Cardiotocography:

Ultrasonogram :

USG FINDINGS

BPD

HC

FL

AC

TCD

FH

Liquor

TCD/AC

FH

FOLLOW UP (STUDY GROUP)

Gestational Age at Delivery :

Mode of Delivery: Vaginal / LSCS :

New born Details:

Birth weight :

In-utero growth status :

APGAR at one minute :

APGAR at 5 minutes :

NEW BALLARD SCORE:

CAN SCORE:

NICU Admission :

Perinatal Morbidity : **Perinatal mortality:**

KEY WORDS

AC- Abdominal Circumference

APGAR - Appearance, Pulse, Grimace, Activity, Respiration

AGA - Appropriate for Gestational Age

BPD - Biparietal Diameter

CHT - Chronic Hypertension

FGR - Fetal Growth Restriction

FL - Femoral Length

GA - Gestational Age

GDM - Gestational Diabetes Mellitus

HC - Head Circumference

LGA - Large for Gestational Age

LSCS - Lower segment caesarean section

NICU - Neonatal Intensive Care Unit

SGA - Small for Gestational Age

TCD - Transverse Cerebellar Diameter

GCK - Glucokinase

CPM - Confined Placental Mosaicism

CRH - Corticotrophin Releasing Hormone

SD - Standard Deviation

IUGR - Intrauterine Growth Restriction

PPV - Positive Predictive Value

NPV - Negative Predictive Value

S.NO	NAME	AGE	IP NO / OP NO	GA	TCD mm	AC mm	HC mm	TCD/AC	HC/AC	MEAN		SD		2SD	
										TCD/AC	HC/AC	TCD/AC	HC/AC	TCD/AC	HC/AC
1	Indrani	30	563081	32	38	265	298	13.9	1.1	14.2	1.06	0.28	0.02	14.7	1.1
2	Manjnula	22	601852	32	38	280	295	14.6	1.05						
3	lakshmi	23	601630	32	36	270	280	14.09	1.03						
4	padma	26	603622	32	38	275	298	14.4	1.08						
5	srimani	27	603919	32	38	275	285	14.4	1.04						
6	Ranjani	23	608458	32	36	280	290	13.9	1.09						
7	Radhika	24	611112	33	42	280	306	15	1.09	14.4	1.08	0.37	0.02	15.14	1.12
8	Selvi	25	229496	33	40	270	308	14.8	1.14						
9	Vinodhini	26	225351	33	41	275	304	14.9	1.1						
10	Rani	28	635558	33	42	285	304	14.7	1.06						
11	Mani	29	347709	33	38	270	308	14	1.1						
12	Menammal	21	635969	33	39	275	306	14.1	1.1						
13	Yazhini	22	636976	33	41	280	312	14.6	1.1						
14	Vaishnavi	24	157017	33	40	275	298	14.5	1.08						
15	Jhenni	23	150007	33	40	280	303	14.2	1.08						
16	Beevi	26	161859	33	40	280	306	14.2	1.09						
17	Bhuvaneshwari	28	161851	33	40	280	310	14.2	1.1						
18	Aarthi	27	702866	33	42	285	308	14.7	1.08						
19	Malini	22	699783	33	42	290	308	14.4	1.06						
20	Vanathi	32	902881	33	40	290	310	13.7	1.06						
21	Revathi	25	702744	34	40	285	310	14	1.08	14.5	1.05	0.63	0.02	15.9	1.09
22	Shalini	26	112311	34	42	290	312	14.4	1.07						
23	Shanthi	22	702836	34	42	295	310	14.2	1.05						
24	Sandhiya	31	702718	34	41	285	308	14.3	1.08						
25	Alamelu	34	704340	34	42	280	310	15	1.1						
26	Seetha	23	127457	34	40	295	316	13.5	1.07						
27	Deepa	24	229188	34	40	290	318	13.7	1.09						
28	Eswari	25	603351	34	45	300	310	15	1.03						
29	Fanitha	22	705079	34	46	310	316	15.3	1.01						
30	Hamuiya	23	704690	34	44	308	320	14.2	1.03						

S.NO	NAME	AGE	IP NO / OP NO	GA	TCD mm	AC mm	HC mm	TCD/AC	HC/AC	MEAN		SD		2SD	
										TCD/AC	HC/AC	TCD/AC	HC/AC	TCD/AC	HC/AC
31	Geetha	21	705062	34	4	304	318	15.7	1.04						
32	Indra	24	713985	34	45	306	316	14.7	1.03						
33	Janani	26	708166	34	46	308	318	14.9	1.03						
34	Kowsalya	27	707729	35	46	306	320	15	1.04						
35	Lavanya	28	708085	35	45	308	316	14.6	1.02						
36	Sheela	21	708221	35	45	308	312	14.6	1.01						
37	Stella	22	708031	35	47	306	314	15.3	1.02						
38	Shamira	23	707936	35	46	313	318	14.6	1.01						
39	Fanitha	24	713079	35	44	310	320	14.1	1.03						
40	Kuppaye	27	713000	35	46	308	320	14.9	1.03	14.4	1.02	0.54	0.01	15.4	1.12
41	Balamani	28	713120	35	45	308	321	14.6	1.04						
42	LAkshmi	29	714149	35	42	312	323	13.5	1.03						
43	Mohana	30	714032	35	43	311	324	13.8	1.04						
44	Nikitha	21	714157	35	44	31	321	14.1	1.03						
45	Neela	34	713544	35	45	308	326	14.6	1.05						
46	Oviya	32	713866	36	42	310	330	13.5	1.06						
47	Pavithra	35	713867	36	43	310	320	13.8	1.03						
48	quinhes	23	713835	36	42	310	320	13.5	1.03						
49	Ramya	26	713924	36	46	316	324	14.5	1.02						
50	Sophia	27	713944	36	43	318	326	13.5	1.02						
51	Sabeena	28	713870	36	42	320	330	13.1	1.03	13.6	1.03	0.41	0.01	14.4	1.11
52	Alamelu	22	713907	36	44	318	328	13.8	1.03						
53	Murshidhe	23	223516	36	42	314	329	13.3	1.04						
54	Madhavi	24	719222	36	43	311	326	13.3	1.04						
55	Vetriselvi	25	719213	36	44	312	326	14.1	1.04						
56	Neha	21	718831	37	45	314	330	14.3	1.05						
57	Manohari	23	718833	37	44	316	330	13.9	1.04						
58	Muthulakshmi	25	718834	37	44	318	320	13.8	1						
59	Alamelu	26	718835	37	45	320	330	14	1.03						
60	Keerthana	28	718830	37	44	312	334	14.1	1.07						

S.NO	NAME	AGE	IP NO / OP NO	GA	TCD mm	AC mm	HC mm	TCD/AC	HC/AC	MEAN		SD		2SD	
										TCD/AC	HC/AC	TCD/AC	HC/AC	TCD/AC	HC/AC
61	Brindha	29	563082	37	44	316	335	13.9	1.06	13.9	1.04	0.8	0.02	14.8	1.12
62	Dharini	26	601854	37	44	314	340	14	1.08						
63	Elavarasi	27	601632	37	45	315	330	14.2	1.04						
64	Farshed banu	28	603625	37	44	316	330	13.9	1.04						
65	Banumathi	29	608459	37	43	318	328	13.5	1.03						
66	Isabella	30	229497	37	44	316	322	13.9	1.03						
67	Cecilia	32	225361	37	44	318	330	13.8	1.03						
68	Gomathi	21	635589	37	44	316	332	13.9	1.05						
69	Harini	24	547710	38	45	320	333	14	1.04	14.1	1.08	0.3	0.03	14.7	1.1
70	Janvi	25	635610	38	45	322	334	13.9	1.03						
71	Kalaiselvi	26	636975	38	46	323	330	14.2	1.02						
72	loordhu	28	157019	38	47	320	330	14.6	1.03						
73	Neela	29	161850	38	45	318	340	14.5	1.06						
74	Pandiselvi	30	702868	38	46	316	340	14.5	1.04						
75	Veeraselvi	31	702882	38	44	320	340	13.7	1.06						
76	Bharathi	21	702747	38	46	320	340	14.3	1.06						
77	Aarthi	26	112316	38	45	322	342	13.9	1.06						
78	Senthamilselvi	31	702836	38	44	323	342	13.69	1.05						
79	Devi	24	704350	39	44	330	340	13.3	1.03	13.5	1.08	0.8	0.02	15.1	1.1
80	Kalaivani	23	127457	39	45	330	338	13.3	1.02						
81	Karthiga	23	603361	39	44	330	340	13.3	1.03						
82	Keerthika	26	70580	39	45	325	345	13.8	1.06						
83	Deepalakshmi	27	704692	39	45	327	346	13.7	1.05						
84	vinodhini	28	708065	39	44	320	344	13.7	1.07						
85	Susila	29	713989	39	46	324	346	14.1	1.06						
86	manammal	30	708168	39	44	325	346	13.5	1.06						
87	pavithra	31	707727	40	46	330	344	13.9	1.04						
88	parvathi	31	708087	40	44	332	344	13.2	1.03						
89	porselvi	28	7080225	40	45	333	346	13.5	1.03						
90	Dharini	27	707934	40	46	336	346	13.6	1.02						

S.NO	NAME	AGE	IP NO / OP NO	GA	TCD mm	AC mm	HC mm	TCD/AC	HC/AC	MEAN		SD		2SD	
										TCD/AC	HC/AC	TCD/AC	HC/AC	TCD/AC	HC/AC
91	Anbuselvi	21	718837	40	44	335	346	13.1	1.03	14	1.08	0.82	0.02	14.8	1.12
92	Bharathi	31	702747	40	45	332	344	13.5	1.03						
93	Pandeeswari	22	112344	40	46	328	346	14	1.05						
94	porselvi	32	702720	40	46	332	350	13.5	1.05						
95	Bhuvaneshwari	23	704370	40	44	333	346	13.2	1.03						
96	meena	33	705080	40	46	330	348	13.9	1.05						
97	Neelavathi	24	704698	40	46	330	348	13.9	1.05						
98	Sundari	34	705066	40	47	330	346	14.2	1.04						
99	Kannagi	25	708178	40	46	330	348	13.9	1.05						
100	Kannama	26	70942	40	47	332	346	13.8	1.04						

S.NO	NAME	AGE	IP NO / OP NO	BMI	GA	PARITY	TCD mm	AC mm	HC mm	TCD/AC	HC/AC	IUGR BY TCD/AC	IUGR BY HC/AC	GA at BIRTH	mode of delivery	NBS	CAN SCORE	BIRTH Wt	TRUE IUGR	APGAR 1	APGAR 5	NICU	MORTALITY
1	Abirami	23	229497	26.5	32	PRIMI	38	233	285	16.3	1.2	YES	YES	38	LSCS	35	22	2.4	YES	7	8		
2	Rosy	26	161427	28	32	G3P1L1A1	36	240	290	15.0	1.2	YES	YES	38	LSCS	35	22	2.2	YES	7	8		
3	ramya	31	231480	27	32	G3P3L3	38	250	295	15.2	112	YES	NO	39	LN	35	22	2.2	YES	7	8		
4	Vinodhini	32	211420	30.5	32	G2P1L1	36	233	285	15.4	1.08	YES	NO	38	LN	35	26	3	NO	7	8		
5	Sharmila	21	231412	29	32	PRIMI	36	255	290	14.9	1.2	YES	YES	38	LN	35	22	2.2	YES	7	8		
6	Shanthi	22	718834	29.5	32	G2A1	35	236	285	14.6	1.08	NO	NO	39	LN	35	22	2.8	NO	7	8		
7	Padma	33	718830	28.9	33	G3P2L2	40	235	300	17.0	1.2	YES	YES	39	LSCS	35	23	2.2	YES	7	8	A	
8	Janaki	24	563082	31	33	PRIMI	41	240	304	17.0	1.1	YES	NO	39	LN	35	23	2.1	YES	7	8	A	
9	Manjula	35	704670	30.8	33	PRIMI	39	245	306	15.9	1.2	YES	YES	38	LN	35	23	1.9	YES	4	7	A	
10	Lavanya	25	702748	29.8	33	PRIMI	41	250	306	16.4	1.1	YES	NO	38	LN	40	26	3	NO	7	8	A	
11	Radhika	21	718838	18.9	33	PRIMI	40	235	304	14.9	1.11	NO	NO	39	LSCS	35	21	2.1	YES	7	8		
12	Thulasi	22	702719	27.5	33	G3P1L1A1	39	245	304	14.8	1.2	NO	YES	39	LSCS	36	23	3	NO	7	8		
13	Uma	24	112313	28.7	33	G3P3L3	40	245	302	16.3	1.24	YES	YES	39	LSCS	37	23	2.1	YES	7	8	A	
14	yazhini	26	718835	29.6	33	G2P1L1	41	250	306	16.4	1.24	YES	YES	39	LSCS	40	22	2.2	YES	7	8		
15	Divya	25	718831	30	33	PRIMI	42	244	302	17.2	1.09	YES	YES	39	LN	36	27	2.7	NO	7	8		
16	Devi	24	563080	30.6	33	G2A1	41	246	300	16.6	1.2	YES	YES	39	LN	37	28	2.7	NO	7	8		
17	Eshwari	23	161222	31	33	G3P2L2	41	240	310	14.9	1.2	NO	YES	39	LN	38	26	3	NO	7	8		
18	Mehana	22	162142	26	33	PRIMI	40	245	304	14.8	1.24	NO	YES	39	LN	35	22	2.6	NO	7	8		
19	Rajakumari	21	124143	27	34	PRIMI	42	265	310	15.8	0.8	NO	NO	38	LN	35	22	2.1	YES	7	8	A	
20	Sreelakshmi	22	121146	28	34	PRIMI	45	270	312	16.6	1.15	YES	YES	38	LN	36	23	2	YES	7	8	A	
21	Menammal	23	563192	29	34	G2P1L1	46	260	318	17.6	1.2	YES	YES	39	LN	37	23	2	YES	7	8	A	
22	Karupayee	27	718142	30	34	G3P1L1A1	48	265	310	18.1	1.16	YES	YES	40	LSCS	38	26	3.1	NO	7	8		
23	Muniammal	26	211410	28.2	34	PRIMI	46	270	310	17.0	1.2	YES	YES	39	LSCS	40	22	1.7	YES	4	6	A	
24	Muthurathinam	25	721843	27.3	34	G2A1	45	265	308	15.7	1.08	NO	NO	40	LN	35	26	2.7	NO	7	8		
25	Aarthi	24	214142	29.53	34	G3A2	46	245	306	18.7	1.24	YES	YES	39	LSCS	38	26	2.7	NO	7	8		
26	Anandhi	23	161444	27	34	G2P1L1	48	270	300	17.7	1.1	YES	YES	39	LN	37	26	2.7	NO	7	8		
27	Ambika	22	718133	28	34	PRIMI	46	280	304	16.4	1.08	YES	NO	39	LSCS	40	22	2.1	YES	7	8	A	
28	Ambujam	21	702700	29	34	G2P1L1	45	280	304	16.0	1.06	YES	NO	39	LN	39	26	1.8	YES	6	8	A	
29	Sindhiya	20	704700	30	34	PRIMI	44	285	304	15.4	1.06	NO	NO	40	LN	39	23	2.8	NO	7	8		
30	Vivekapriya	19	563111	31	34	PRIMI	42	275	302	15.2	1.09	NO	YES	40	LN	36	23	2.1	YES	7	8	A	
31	Vishnupriya	22	213222	30.9	34	PRIMI	46	270	306	17.0	1.13	YES	YES	40	LN	36	22	1.9	YES	4	5	A	D
32	Keerthika	26	161142	29.8	34	G2P1L1	46	270	300	17.0	1.11	YES	YES	40	LN	36	23	2.7	NO	7	8		
33	Anushiya	28	702714	28.7	35	G2P1L1	42	280	320	15.0	1.1	NO	YES	39	LN	35	26	2.7	NO	7	8		
34	Meena	27	112341	26.5	35	G2P1L1	41	290	316	14.1	1.08	NO	NO	39	LSCS	36	27	2.6	NO	7	8		
35	Neela	26	702818	30	35	G3P1L1A1	43	280	318	15.3	1.13	NO	YES	38	LSCS	37	26	2.5	NO	7	8		
36	Sophie	25	127141	29	35	G3A2	44	300	322	14.6	1.07	NO	NO	39	LSCS	38	27	2.5	NO	7	8		
37	Jasmin	24	704162	28	35	G2A1	46	275	324	16.7	1.17	YES	YES	38	LN	39	22	1.9	YES	7	8	A	
38	Banu	23	713942	27	35	G2P1L1	42	270	326	15.5	1.2	YES	YES	39	LN	40	22	1.9	YES	6	7	A	
39	Lakshmi	22	708144	28	35	G3P1L1A1	46	270	320	17.0	1.1	YES	YES	38	LN	35	21	2	YES	7	8	A	
40	Bhavani	21	707146	29	35	PRIMI	46	275	316	16.7	1.1	YES	YES	39	LSCS	40	22	2	YES	7	8	A	
41	Beevi	20	713221	28.7	35	G2A1	45	280	304	16.0	1.08	YES	NO	38	LSCS	39	22	2	YES	7	8	A	
42	Renuka	19	713420	29.3	35	G3A2	45	285	326	15.3	1.08	NO	NO	39	LSCS	38	22	2.1	YES	7	8	A	
43	Radha	31	714249	26.8	35	G2P1L1	46	275	324	16.7	1.2	YES	YES	38	LN	37	26	3.2	NO	7	8		
44	Abirami	28	714232	31	35	PRIMI	45	290	326	15.5	1.1	YES	YES	39	LN	36	26	2.9	NO	7	8		
45	akilandeswari	25	714127	31	35	G2P1L1	46	290	320	15.8	1.1	YES	YES	38	LSCS	35	22	2.1	YES	7	8	A	
46	sreelakshmi	26	713166	30.3	36	PRIMI	43	290	318	14.8	1.09	YES	NO	38	LSCS	35	22	2.1	YES	7	8	A	
47	soumiya	21	713421	29.4	36	PRIMI	41	295	318	13.8	1.07	NO	NO	38	LSCS	35	23	1.9	YES	7	8	A	

48	sathya	24	223216	28.4	36	PRIMI	42	300	320	14.0	1.06	NO	NO	38	LN	35	24	1.8	YES	7	8	A	
49	padma	28	712421	30	36	G2P1L1	42	302	324	13.9	1.07	NO	NO	38	LN	38	27	3.2	NO	7	8		
50	preethi	29	718233	30.5	36	G2P1L1	42	290	326	14.4	1.1	YES	YES	38	LN	38	22	2.1	YES	7	8	A	
51	shanthi	31	702144	28.8	36	G2P1L1	43	295	320	14.5	1.08	YES	NO	38	LN	38	23	2.3	YES	7	8		
52	deepa	33	121611	26	36	G3P1L1A1	44	280	320	15.7	1.1	YES	YES	38	LN	37	23	2.1	YES	7	8	A	
53	deepika	31	702636	27	36	G3A2	45	280	318	16.0	1.1	YES	YES	38	LN	37	23	2.1	YES	7	8	A	
54	rani	35	702618	28	36	G2A1	42	275	318	15.2	1.1	YES	YES	39	LN	37	26	2.8	NO	7	8		
55	swathi	26	704640	29	36	G3P1L1A1	42	270	316	15.0	1.17	YES	YES	39	LN	36	26	2.8	NO	7	8		
56	rosy	24	127657	30	36	G3P3L3	42	290	312	14.3	1.07	NO	NO	39	LN	36	26	2.6	NO	7	8		
57	mary	29	229688	31	36	G2P1L1	41	280	320	14.4	1.14	YES	YES	40	LN	36	22	1.8	YES	4	6	A	D
58	vijayakumari	36	705679	31	36	PRIMI	41	290	320	14.1	1.1	NO	YES	40	LN	36	26	2.6	NO	7	8		
59	rakammal	32	707624	30	37	G2A1	42	290	322	14.4	1.1	NO	YES	39	LSCS	36	26	2.7	NO	7	8		
60	muthulakshmi	30	707636	29	37	G3P2L2	43	285	326	15.0	1.1	YES	YES	38	LSCS	35	22	2.2	YES	7	8		
61	rathna	23	702764	28	37	PRIMI	42	280	320	15.0	1.08	YES	NO	38	LN	35	22	2.1	YES	7	8	A	
62	ramya	24	113611	27	37	PRIMI	43	295	324	14.6	1.09	NO	NO	37	LSCS	36	25	2.6	NO	7	8		
63	bharathi	26	702636	26	37	PRIMI	44	300	326	14.6	1.08	NO	NO	38	LN	37	26	2.6	NO	7	8		
64	madhu	31	704640	30.9	37	G2P1L1	42	290	330	14.4	1.1	NO	YES	39	LN	35	26	2.7	NO	7	8		
65	Manju	32	127657	29.2	37	G3P1L1A1	43	285	320	15.0	1.1	YES	YES	40	LSCS	35	22	2.1	YES	7	8	A	
66	pradiksha	33	705659	28.6	37	PRIMI	44	285	322	15.4	1.1	YES	YES	38	LSCS	36	21	2	YES	7	8	A	
67	lakshmi	24	223655	27.9	37	G2A1	42	285	324	14.7	1.1	NO	YES	39	LSCS	37	22	2.1	YES	7	8		
68	nisha	25	708655	30	37	G3A2	43	290	326	14.8	1.1	YES	YES	40	LSCS	36	23	2	YES	7	8	A	
69	Anu	26	713620	29	37	G2P1L1	44	285	328	15.4	1.5	YES	YES	39	LN	35	24	2	YES	7	8	A	
70	rajathi	27	714149	28	38	PRIMI	44	300	330	14.6	1.1	NO	YES	38	LN	35	25	2.8	NO	7	8		
71	arokiamarry	22	713666	27	38	G2P1L1	45	300	332	15.0	1.1	YES	YES	39	LN	36	22	2	YES	7	8	A	
72	fathima	22	713624	26	38	PRIMI	43	285	330	14.5	1.1	NO	YES	38	LN	35	25	2.8	NO	7	8		
73	senthamarai	19	705262	26	38	PRIMI	44	289	330	14.7	1.1	YES	YES	39	LSCS	36	22	2.1	YES	7	8		
74	chitra	21	713785	27	38	PRIMI	44	300	332	14.6	1.1	NO	YES	38	LSCS	37	26	2.8	NO	7	8		
75	deviga	27	708966	28	38	G2P1L1	46	290	328	15.8	1.1	YES	YES	39	LSCS	36	22	2	YES	7	8	A	
76	bhuvana	26	707624	29	38	G2P1L1	45	295	330	12.2	1.08	YES	NO	39	LSCS	35	23	2.1	YES	6	7	A	
77	megala	28	716831	30	38	G2P1L1	44	300	330	14.6	1.09	NO	NO	39	LN	37	26	2.8	NO	7	8		
78	sagunthala	27	716883	31	38	G3P1L1A1	45	302	326	14.9	1.1	YES	YES	38	LN	36	22	1.9	YES	7	8	A	
79	rose	28	223616	26	38	G3A2	46	300	324	15.3	1.1	YES	YES	38	LN	35	23	2	YES	7	8	A	
80	parameshwari	31	713635	27	38	G2A1	44	302	326	14.5	1.08	NO	NO	37	LN	37	26	2.5	NO	7	8		
81	chinnaponnu	30	713646	28	38	G2P1L1	45	295	324	15.2	1.09	YES	NO	39	LN	36	24	2	YES	7	8	A	
82	rabeka	29	714649	29	38	G3P1L1A1	46	300	326	15.3	1.1	YES	YES	39	LN	37	23	2.1	YES	7	8	A	
83	jayarani	28	713620	30	39	PRIMI	44	300	330	14.6	1.1	NO	YES	39	LSCS	38	26	2.8	NO	7	8		
84	meena	27	707924	31	39	G2A1	44	299	332	14.9	1.1	YES	YES	38	LSCS	38	22	2.1	YES	7	8	A	
85	raji	26	708041	30	39	G3A2	45	298	330	15.1	1.1	YES	YES	39	LSCS	37	23	2	YES	6	8	A	
86	amudha	25	787377	29	39	G2P1L1	46	300	330	15.3	1.1	YES	YES	37	LN	38	22	2.1	YES	7	8	A	
87	sujatha	24	87389	30	39	PRIMI	45	300	332	15.0	1.1	YES	YES	38	LSCS	38	26	2.8	NO	7	8		
88	saranya	23	127467	31	39	G2P1L1	43	302	328	14.2	1.08	NO	NO	39	LN	38	26	3.3	NO	7	8		
89	devi	26	718434	29	39	PRIMI	43	304	330	14.4	1.08	NO	NO	39	LN	38	26	2.7	NO	7	8		
90	rajathi	27	718635	28	39	PRIMI	44	302	328	14.5	1.08	NO	NO	38	LSCS	36	26	2.7	NO	7	8		
91	preethi	28	563682	27	39	PRIMI	45	300	335	15.0	1.1	YES	YES	39	LSCS	37	22	2	YES	5	8	A	
92	neela	29	603725	26	39	G2P1L1	46	298	336	15.4	1.1	YES	YES	38	LSCS	35	23	1.7	YES	7	8	A	
93	mani	30	225461	26	40	G2P1L1	43	295	334	14.5	1.13	NO	YES	38	LSCS	35	22	1.8	YES	7	8	A	
94	kavitha	24	635910	27	40	G2P1L1	44	388	332	15.2	1.15	YES	YES	39	LN	36	23	2.1	YES	6	7	A	
95	vellimalar	27	702968	28	40	G3P1L1A1	45	290	330	15.5	1.1	YES	YES	39	LSCS	37	26	2.7	NO	7	8		
96	padmini	23	112416	29	40	G3A2	46	30	336	15.3	1.1	YES	YES	38	LN	38	27	3.2	NO	7	8		
97	rekha	31	704650	30	40	G2A1	44	302	334	14.5	1.1	NO	YES	39	LSCS	35	26	2.9	NO	7	8		
98	tamiarasi	32	708162	31	40	G3P1L1A1	44	302	335	14.5	1.1	NO	YES	38	LN	36	22	2.2	YES	7	8		
99	abirami	36	708968	29.9	40	G3P2L2	44	304	330	14.4	1.09	NO	NO	38	LSCS	37	26	3	NO	7	8		
100	saranya	36	768912	30.4	40	G3P2L2	44	304	330	14.4	1.09	NO	NO	38	LN	37	26	2.9	NO	7	8		