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PREDICTION OF ADVERSE PERINATAL OUTCOME IN GROWTH RESTRICTED FETUSES WITH ANTENATAL DOPPLER STUDY

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CERTIFICATE

This is to certify that this dissertation in **"PREDICTION OF ADVERSE PERINATAL OUTCOME IN GROWTH RESTRICTED FETUSES WITH ANTENATAL DOPPLER STUDY"** is a work done by **Dr.ANITA .S,** under my guidance during the period 2004 - 2007. This has been submitted in partial fulfillment of the award of M.D. Degree in Radiodiagnosis (Branch - VIII) by the Tamil Nadu Dr.M.G.R. Medical University, Chennai - 600 032.

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DECLARATION

I declare that this dissertation titled **"PREDICTION OF ADVERSE PERINATAL OUTCOME IN GROWTH RESTRICTED FETUSES WITH ANTENATAL DOPPLER STUDY"** is a work done by me, under the guidance and supervision of **Prof.T.S.SWAMINATHAN, M.B., M.D., DMRD., FICR.,** Director, Barnard Institute of Radiology, Madras Medical College. It is submitted in partial fulfillment of the requirement for the award of the M.D., Radiodiagnosis, March 2007 examination to be held under Dr.M.G.R.Medical University, Chennai. This has not been submitted previously by me for the award of any degree or diploma from any other University.

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INTRODUCTION

Ante partum fetal surveillance is the corner stone of preventive obstetric management aimed at reducing maternal and perinatal mortality and morbidity. Ante partum detection of fetus at risk of death or compromise in utero remains the major challenge in modern obstetrics. Specific and accurate methods for detection of fetus at risk can result in early appropriate intervention and hence reduce fetal loss. Antenatal test of fetal well being depends indirectly on changes in fetal physiology, an aspect of fetus, which until recently, has been relatively inaccessible to study by the paucity of techniques to measure the placental function - the critical organ through which the transfer of nutrients occur. New technologies have now become available in the clinical assessment of placental function. Doppler measurement of the pulsatile blood velocity in umbilical artery gives direct information on feto-placental circulation and hence identifies placental circulatory failure.

Diagnostic ultrasound is the main stay in the evaluation and management of obstetric patients. Fetal growth and development rely on normal uteroplacental and fetoplacental circulation to supply oxygen and nutrients from the maternal circulation. Doppler sonography offers a unique tool for the noninvasive evaluation of physiological hemodynamic fetoplacental blood flow information. There are specific abnormalities in Doppler parameters in asymmetric intrauterine growth retardation.

Fetal Growth Restriction (FGR), otherwise known as Intrauterine Growth Restriction is defined as a pathologic decrease in the rate of fetal growth. Here the fetus does not achieve its inherent growth potential, thereby increasing perinatal morbidity and mortality. Small for gestational age (SGA) is conceptually not the same entity as FGR. It is defined as fetus which has failed to achieve specific and arbitrary anthropometric measurements or weight threshold by a specific gestational age, whereas in FGR, the infant has not achieved its genetic growth potential in utero (Rajan. R. 2001).

All FGR fetuses don't suffer from in utero compromise in terms of hypoxia or acidemia. Fetal growth restriction only means that the fetus has not grown appropriately for the corresponding gestational age, and does not necessarily mean it is a situation of uteroplacental respiratory insufficiency causing fetal hypoxia or acidemia. But many IUGR fetuses could sooner or later become hypoxemic, hypoxic and acidotic as a progressive event of the pathophysiology.

Diagnosis of IUGR is based on B-mode ultrasound. Estimation of fetal weight in utero using multiple ultrasound parameters remains the mainstay in screening for FGR. Use of various fetal morphometric ratios and/or measurements of other fetal parameters may provide additional useful information. Serial evaluation to assess interval growth may be necessary to clarify the diagnosis.

Doppler velocimetry has poor sensitivity in detecting IUGR, whereas it is helpful in assessing the hemodyanmic state. Doppler indices change if the fetus is compromised due to hypoxemia.

Doppler flow velocimetry, particularly of the middle cerebral and umbilical arteries is an earlier predictor of hypoxemia, when compared to BPP or NST. Ductus venosus flow study is an accurate predictor for acidemia.

The relationship between the size of fetal abdominal circumference and fetal head is used to characterize the pattern of FGR as being either symmetric or asymmetric. Symmetric IUGR refers to a growth pattern in which the growth of both the fetal abdomen and head are decreased proportionally. Asymmetric IUGR refers to the growth-retarded fetus in which a disproportionate decrease in the size of fetal abdomen with respect to the fetal head is seen.

Symmetric IUGR may result from an early insult such as genetic or infective pathology that impairs fetal cellular hyperplasia and therefore causes a proportionate decrease in size of all fetal organs. By contrast, asymmetric IUGR may be caused by a later insult that impairs cellular hypertrophy, causes a disproportionate decrease in the size of fetal abdomen in relation to that of the fetal head. Progressive uteroplacental insufficiency may be associated with this asymmetric growth pattern.

Nearly 70% of patients with IUGR may be classified as having an asymmetrical growth pattern. These cases may be at greater risk for perinatal hypoxia and neonatal hypoglycemia. However, their long-term prognosis with appropriate management is good.

Symmetric IUGR results from an early insult and is characterized by a long period of subnormal growth. These infants usually do not have perinatal hypoxia, but they are at risk of long-term neurodevelopmental dysfunction, resulting from a deficit in the total number of brain cells.

AIMS AND OBJECTIVES

1. To detect any abnormalities in fetoplacental unit and fetal circulation in IUGR.

2. To identify the hypoxemic fetus & timing delivery so as to precede acidemia.

3. To correlate the occurrence of adverse perinatal outcome with degree of abnormality in doppler indices.

REVIEW OF LITERATURE

To avoid undue alarm in patients, to whom the term "Retardation" implies abnormal mental function, the term "Fetal growth restriction" is now preferred. About 3-10% of infants are growth restricted (Divon and HSU, 1992)[1].

In 1961, Warkany and co-workers[2] reported normal values for infant weight, lengths and head circumferences which served to define fetal growth restriction. In 1963, Gruenwald[3] reported that approximately one third of low birth weight infants were mature and that their small size could be explained by "chronic placental insufficiency".

In 1963, Lubchenco and coworkers[5] from Denver published detailed comparisons of gestational ages to birth weights in an effort to derive norms for expected fetal size and therefore growth at a given gestational week. Battagalia and Lubchenco (1967)[6] classified small for gestational age infants as those whose weights were below the 10th percentile for their gestational age. The neonatal mortality rate of a small for gestational age infant born at 38 weeks was 1 percent compared with 0.2% in those with appropriate birth weights. Seeds and Peng (1998)[7] concluded that the threshold for impaired growth based upon the risk of fetal death should be set even higher at the 15th birth weight percentile.

Manning and Hohler (1991)[8] and Gardosi (1992)[9] concluded that 25-60% of infants conventionally diagnosed to be small for gestational age were in

fact appropriately grown when determinants of birth weight such as maternal ethnic group, parity & weight are considered.

A definition based upon birth weight below the fifth percentile was also proposed by Seeds (1984)[10]. Usher and McLean (1969)[11] proposed that fetal growth standards should be based on mean values with normal limits defined by $\pm$ 2 standard deviations because this definition would limit small for gestational age infants to 3% of births instead of 10% with use of the 10th percentile. From a clinical standpoint this definition appears to be most meaningful. This is because most poor outcomes are in those infants with birth weights below the third percentile.

In a study of 122,754 pregnancies delivered at Parkland Hospital, Mclntire and Colleagues (1999)[12] found that mortality and morbidity were significantly increased among infants born at term only when their birth weights were at or below the third percentile for their gestational age.

Relationship between birth weight percentile and perinatal mortality & morbidity is observed on 1560 SGA fetuses. A progressive increase in both mortality and morbidity is observed as birth weight percentile falls. (Manning 1995)[13].

Owen and Colleagues in 1997[14] and Owen and Khan in 1998[15] reported that reduction in the rate of velocity of fetal growth detected by serial ultrasonic fetal anthropometry is related to caesarean delivery for fetal distress and significant fetal growth restriction.

Fetal growth restriction is associated with substantial perinatal mortality and morbidity. Incidence of fetal demise, birth asphyxia, meconium aspiration, neonatal hypoglycemia and hypothermia are all increased, as is the prevalence of abnormal neurological development (Paz 1995[16], Piper 1996[17]). Postnatal growth and development of growth - restricted fetuses depends on the cause of restriction, nutrition in infancy and social environment (Kliegman 1997[18]). Infants with growth restriction due to congenital, viral infection, chromosomal or maternal constitutional factors remain small throughout life. Those infants with in utero growth restriction due to placental insufficiency will often have catch up growth after birth and approach their inherited growth potential when provided with an optimal environment.

Campbell and Thorns (1977)[19] described the use of the sonographic head to abdomen circumference ratio (HC/AC) to differentiate fetuses into subtypes - "Symmetrical" meaning proportionately small and "Asymmetrical" meaning those with disproportionately lagging abdominal growth. 70% of the fetuses with a HC/AC ratio above the 95th percentile were termed asymmetrical. Even though similar gene realization exists, those with aneuploidy typically had disproportionately large head size (Nicolaides 1991)[20].

Similarly most preterm infants with growth restriction due to preeclampsia and associated uteroplacental insufficiency demonstrate a symmetrical pattern of growth impairment rather than the hypothesized asymmetrical pattern (Salafia 1995)[21].

Dash and Colleagues (2000)[22] analyzed HC/AC ratio. Only 20% of growth restricted fetuses were at increased risk for intrapartum and neonatal

complications. Symmetrically growth restricted fetuses with birth weight less than 10th percentile were not at increased risk of adverse outcomes.

Early establishment of gestational age, attention to maternal weight gain, careful measurement of uterine fundal growth throughout pregnancy will serve to identify many cases of abnormal fetal growth in women without risk factors. Identification of risk factors, including a previously growth restricted fetus should raise the possibility of recurrence during the current pregnancy in women with significant risk factors, for whom serial sonography is considered. Definitive diagnosis, however, usually cannot be made until delivery.

Identification of the inappropriately growing fetus remains a challenge; such identification is not always possible even in the nursery. Carefully performed serial fundal height measurements throughout gestation are a simple, safe, inexpensive and reasonably accurate screening method to detect many small for gestational age fetuses (Gardosi and Francis 1999)[23] But its principal drawback is imprecision. Jenson and Larsen (1991)[24] and Walraven (1995)[25] found that symphysis to fundal measurements helped to correctly identify only 40% of such infants. Thus, small for age infants are either over-looked or over-diagnosed. Despite this, carefully measured fundal height is still considered as a simple screening method.

The method to measure fundal height by using a measuring tape was first reported by Jimenez and colleagues (1983)[26]. Between 18 and 30 weeks the uterine fundal height in centimeters coincides with the weeks of gestation. If the measurement is more or less than 2-3cm from the expected height, inappropriate fetal growth may be suspected.

Routine screening incorporates an ultrasound examination at 16-20 weeks to establish gestational age and rule out visible anomalies, and then follow-up imaging at 32-34 weeks to evaluate fetal growth .Combining head, abdomen and femur dimensions should in theory enhance the accuracy of predictions of fetal size. But any potential improvement is apparently lost by the cumulative error inherent in measurement of each individual fetal dimension. Thus most experts have accepted abdominal circumference measurements as the most reliable index of fetal size (Manning et al[13] 1995; Smith 1997[27], Snijders & Nicolaides 1994[28]). In these studies, estimated fetal weight calculated with abdominal circumference measurements was almost always within 10% of the actual birth weight.

The use of ultrasound for detection of fetal growth restriction does not preclude missed diagnoses. Dash and Colleagues (2000)[22] studied 8400 live births at Parkland hospital of those women who had ante partum ultrasound within 4 weeks of delivery. Although 70% of growth restricted fetuses were detected, 30% were missed.

Risk Factors of IUGR

Constitutionally small mothers

Small women typically have smaller babies. Data from a longitudinal study of all births during one week in 1958 in England, Wales and Scotland

indicate that there are intergenerational effects on birth weight that are transmitted through the maternal line (Emanuel 1992)[29]. Klebanoff (1997)[30] reported that reduced intrauterine growth of the mother is a risk factor for reduced intrauterine growth of her children. Brooks and co-authors (1995)[31] analyzed 62 births where the relative influence of the donor versus the recipient on birth weight, after ovum donation, was examined. They concluded that the environment provided by the mother was more important that her genetic contribution to birth weight.

Poor maternal weight gain and nutrition

In the women of average or low weight, lack of weight gain throughout pregnancy may be associated with fetal growth restriction (Simpson1975)[32]. Lack of weight gain in the second trimester strongly correlates with decreased birth weight (Abrams and Selvin 1995).

Social Deprivation

Williams (2001)[34] wrote "the social condition of the mother and the comforts by which she is surrounded also exert a marked influence upon child's weight, heavier children being more common in the upper walks of life".

Fetal Infections

Viral, bacterial, protozoan and spirochetal infections are implicated in upto 5% of cases of fetal growth restriction (Klein and Remington 1995)[35] -

Rubella, Cytomegalovirus virus, Hepatitis A, Hepatitis B, Listeriosis, Tuberculosis, Syphilis, Toxoplasmosis and congenital Malaria.

Congenital malformations

With major structural anomalies, 22% had accompanying growth restriction (Khoury 1988)[36]. More severe the malformation, the more likely the fetus is to be small for gestational age.

Chromosomal abnormalities

Placental insufficiency, primary abnormal cellular growth and differentiation may contribute to the significant degree of fetal growth restriction associated with karyotype abnormalities.

Prominent postnatal growth failure occurs in trisomy 21, whereas fetal growth restriction is generally mild (Thelander, Pryor 1966)[37]. Trisomy 18 is associated with severe fetal growth restriction.

Trisomy 16 is the most common trisomy in spontaneous abortions and is lethal to the fetus in non-mosaic state (Lindor 1993)[38] Patches of trisomy 16 in the placenta called "confined placental mosaicism" leads to placental insufficiency & may account for many cases of previously unexplained fetal growth restriction (Kalousek 1993)[39]. Here chromosome abnormality is confined to placenta. Inherited syndromes such as osteogenesis imperfecta and various chondrodystrophies are associated with fetal growth restriction.

Chemical teratogens

Anticonvulsants such as phenytoin and trimethadione may produce specific and characteristic syndromes that include fetal growth restriction (Hanson 1976)[40]. Cigarette smoking causes growth restriction as well as preterm delivery in a direct relationship with the number of cigarettes smoked (Cliver 1995)[41]. Narcotics decrease maternal food intake and fetal cell number. Alcohol acts in a linear dose related fashion.

Vascular disease

Chronic vascular disease, especially when complicated by superimposed pre-eclampsia, commonly causes growth restriction. Pre-eclampsia, by itself may cause fetal growth failure, when the onset is before 37 weeks (Xiong 1999)[42].

Renal disease may be accompanied by restricted fetal growth (Stettlerand Cunningham 1992)[43].

Chronic hypoxia

Fetuses of women who reside at high altitude usually weigh less than those born to women who live at a lower altitude. Fetuses of women with cyanotic congenital heart diseases are frequently severely growth restricted (Patton 1990)[44].

Maternal anemia

Sickle cell anemia or other inherited anemias associated with serious maternal disease cause fetal growth restriction.

Deficient total maternal blood volume early in pregnancy is linked to fetal growth restriction (Duvekot 1995)[45].

Placental and cord abnormalities

Chronic partial placental separation, extensive infarction, chorioangioma, circumvallate placenta, placenta previa, marginal insertion of the cord and velamentous insertions are all accompanied by growth restricted fetuses. Many cases of IUGR occur in pregnancies with apparently normal fetuses with grossly normal placentas. Growth failure in these cases is presumed to be due to uteroplacental insufficiency. Women with otherwise unexplained FGR demonstrated a four-fold reduction in uteroplacental blood flow compared with normally grown fetuses (Lunell and Nylund 1992)[46]. Similar reductions were also seen in growth restricted fetuses with congenital malformations suggesting that maternal blood flow might in part be regulated by the fetus (Howard 1987[47]). Uteroplacental blood flow is also reduced in women with pre-eclampsia compared with normotensive women.

Multiple fetuses

Twin pregnancy is more likely to be complicated by diminished growth of one or both fetuses compared with normal singletons. Growth restriction has been reported in 10-50% of twins (Hill 1994)[48].

Antiphospholipid antibody syndrome

Two classes of antibodies, associated with fetal growth restriction, are anticardiolipin antibodies and lupus anticoagulant (Lockwood and Rand 1994)[49]. Pathophysiological mechanism in the fetus appears to be caused by maternal platelet aggregation and placental thrombosis.

DOPPLER VELOCIMETRY

Basic principles

Doppler shift is a physical principle that states that when a source of light or sound wave is moving relative to an observer, observer detects a shift in the wave frequency. Thus, when sound waves strike a moving target, the frequency of sound waves reflected back is shifted proportionate to the velocity and direction of the moving target. As the magnitude and direction of the frequency shift depend on the relative motion of the moving target, their velocity and direction can be determined (Williams, 2001)[34].

Doppler principle is used to determine the volume and rate of blood flow through maternal and fetal vessels. Here, the sound source is ultrasound transducer & moving targets are the red blood cells flowing through the circulation. When the distance between the source of sound and the reflector changes, there is a change in the frequency of the reflected echo-Doppler shift - which is converted by the machine to 3 different kinds of output, namely;

- Audio output on a loudspeaker-characteristic for each vessel-we can identify each vessel easily

- Spectral waveform-is also unique to each vessel. The pattern of the waveform depends largely on the distal vascular impedance and the compliance of the vessel.

- Colour doppler- used to code the blood flow in a portion of the vessel. Flow toward the transducer is shown as red and away from the transducer as blue.

Duplex ultrasound: B- mode, color flow and spectral waveform together are called triplex mode.

Doppler is generally used in two ways to estimate circulatory hemodynamics:

1. Direct measurement of volume of blood flow.

2. Indirect estimation of flow velocity using wave form analysis.

Blood flow measurement

Errors in direct measurement of the volume of blood flow have led to the development of several indirect indices of flow, which are independent of the angle of insonation and do not require measurement of the diameter of the vessel.

Colour Doppler is switched on to locate the vessel, then the pulsed Doppler sample volume is placed on the vessel and optimal spectral waveforms are obtained.

Specific measurements like peak systolic velocity (PSV) and the end diastolic velocity (EDV) are made from this optimal spectral waveform, and the various ratios are computed. If the peak systolic velocity is called "S" and the end diastolic velocity is called "D" the various ratios calculated are

(1) S/D ratio

(2) Resistance Index (RI) = S-D/S

(3) Pulsatility Index (PI) = S-D / Mean

Mean velocity is the average of all the velocities that are present during one cardiaccycle and is computed by the machine. Of these, the PI, according to Gosling and King (1975)[50] would seem to be best suited for practical purposes, as it increase linearly with increasing flow impedance.

All ultrasound equipment will have measurement facility and in most equipment, an automatic trace of the waveform is made as the waveform is being generated in real time.

Uterine and arcuate arteries

Uterine artery is a branch of the internal iliac artery,arising close to the bifurcation. Uterine blood flow increase from 50ml/mt shortly after conception to 500 to 750 ml/mt by term..Doppler waveforms of the uterine artery undergo gradual changes in their charecterestics with advancing gestation.

- A sharp systolic peak and forward flow in diastole

- Upto 16 wks of gestation an early diastolic notch can be noticed(represents the presence of elastic coat in the spiral arteries)

- Beyond this period, the notch disappears (18-23wks)(due to trophoblastic invasion of spiral arterioles).

- Diastolic velocity increases and thus the indices decrease as term approaches(SieroszewskiP 2005)[51]

- Failure of the pattern to appear or the presence of a notch in the waveform at end systole after mid trimester has been reported with fetal growth restriction(Schulman 1986)[52]

- Women with abnormal uterine artery Doppler indices and persistence of notch are at high risk of obstetric comlications (Papageorghiou, Nicolaides 2002)[53]

- Women with normal uterine artery doppler have low risk of developing obstetric complications related to placental dysfunction.

- Following a positive test the risk of developing preeclampsia is increased by 6 times and the risk of developing IUGR is increased by 3 times (Papageorghiou 2002)[53]

- However a meta analysis of observational studies on uterine artery Doppler in predicting IUGR&preeclampsia,showed that the likelihood ratios did not alter the pre test probabilities of IUGR and preeclampsia

in a great way-Hence the utility of doppler as a screening test for pre-eclampsia and IUGR is guarded(Chien 2000)[54]

UMBILICAL ARTERY

Anatomy

The umbilical arteries arise from the internal iliac arteries of the fetus and course along the umbilical cord to reach the placenta. Intraplacentally, they branch into primary, secondary and tertiary stem villous vessels, which form the placental vascular bed. As pregnancy advances, increase in tertiary stem villi leads to steady decrease in vascular bed resistance.

Normal umbilical artery waveforms

- In early weeks of gestation the umbilical artery reveals absent diastolic flow.
- Beyond 15 weeks of gestation, diastolic flow is consistently seen.
- As pregnancy advances, there is an increase in diastolic flow and the RI is low.

Abnormal umbilical artery waveforms

- Seen when 70% of the tertiary villi are affected (IUGR is seen even when 40% are affected).This is the reason why umbilical artery doppler cannot be used as a screening test for IUGR.

- 3 types of abnormalities are seen(indication of increasing resistance which correlates with fetal hypoxia)

a) Low diastolic flow (high resistance)

b) Absent diastolic flow

c) Reversed flow in diastole

A meta-analysis of all randomized studies of doppler ultrasound of umbilical artery in IUGR by Alfirevic and Neilson in 1995[55] concluded that all women with suspicion of IUGR should have access to Doppler of the umbilical artery.

Elevated PI, RI or S/D ratios in umbilical and uterine arteries have been correlated to morphological changes in placental vascular bed (Olofsson et al 1993)[56].

Gudmundsson and Marshal in 1991[57] showed that ARED in umbilical artery Doppler, 96% of these fetuses showed hypoxia or asphyxia at delivery. These results indicate that umbilical artery Doppler has the capacity to find those SGA fetuses that are truly at risk of developing asphyxia and therefore need surveillance. Additional information may be found in the other vascular beds.

Cerebral blood flow

- Doppler evaluation of blood flow through cerebral vessels allows detection of altered cerebral circulation before hypoxemia significant enough to change the fetal heart rate has occurred

- Middle cerebral artery is the most accessible vessel and has been reported to demonstrate reduction in the pulsatility index at the onset of hypoxemia (Wladimiroft 1991)[58].

- Chandran and colleagues (1993)[59] compared middle cerebral artery pulsatility indices with fetal heart rate analysis in 27 growth restricted fetuses, and found that Doppler was a more sensitive predictor of hypoxemia at birth than fetal heart rate testing but had lower specificity.

- Unlike the uterine and umbilical vascular beds which constantly change with advancing gestational age, the MCA vascular bed resistance is almost constant throughout pregnancy (the RI is 0.75-0.85).

- This is due to the fact that the intracerebral flow is controlled by an auto regulatory mechanism which responds to fetal oxygen saturation.

- Reduction in the oxygen saturation (hypoxemia) causes dilatation of the intracerebral vessels, leading to increased flow or decreased cerebral resistance (Vyas, Nicolaides et al 1990)[60].

- Cephalisation of flow is associated with redistribution of cardiac output in favour of the left ventricle preferentially perfusing the brain and myocardium with oxygenated blood (Al-ghazali et al 1987)[61]. This occurs during hypoxia and is reflected in MCA as increased diastolic flow with reduced RI.

- With increasing hypoxia, the fetus can become acidemic which results in cerebral edema. This causes increased cerebral resistance and diastolic flow in the MCA may be reduced or even absent. This indicates poor prognosis.

- Demonstration of cephalisation enhances the positive predictive value of an elevated umbilical artery Doppler index for hypoxemia.

- This brain sparing effect has been evaluated in animal studies and confirmed in human studies(Malcus et Al[62] 1991,Mari and Deter 1992)[63]

- Further it pre-dates the appearance of late decelerations on the CTG strip of IUGR fetus by an average of two weeks (Arduini et al 1992)[64].

Cerebroplacental ratio

- A ratio of the resistances in the umbilical artery and the MCA can be compared for better interpretation of fetal hypoxia.

- In normal fetuses, the placental vascular resistance decreases as pregnancy advances, whereas the MCA resistance is constant.

- The RI of MCA / RI of Umb. A is more than 1.

- In cerebral redistribution, the MCA RI decreases and the umbilical artery increases, leading to cerebroplacental ratio of less than 1, indicating fetal hypoxia.

- Clinical reports suggest that ratios between cerebral and umbilical arteries have the best predictive values for adverse outcome in growth restricted fetuses.

- Gramellini et al 1992[65] found the diagnostic accuracy for the cerebro placental ratio to be 90% for predicting adverse outcome in IUGR, compared to 83% and 79% for umbilical artery and MCA alone respectively in the same study.

Ductus venosus

In venous Doppler, the first and most important conduit in fetal circulation is the ductus venosus.

- It is a narrow vessel which arises from the transverse portion of the left portal vein and is connected to the IVC.
- It is funnel shaped and has a muscular coat.
- 45% of the blood from the umbilical vein enters the right atrium via the IVC through the ductus venosus, bypassing the liver.
- The ductus venosus waveform has a typical M pattern
- The audio signals of the ductus venosus have a typical rhythmic musical quality which is very characteristic.
- Doppler of the ductus is only indicated if the umbilical artery and MCA are abnormal.
- Cardiac deterioration is associated with acidemia. Here venous studies are informative (Baschat et al 2000)[66].
- Venous indices reflect ventricular function and to a certain extent cardiac overload. Prolonged hypoxemia leads to hypoxemic cardiomyopathy, ventricular dysfunction, and a fall in cardiac output. As cardiac output declines, central venous pressure rises causing increased reversed flow during atrial systole. As the severity intensifies, direction of blood flow in the ductus venous reverses during atrial contraction causing pulsatile umbilical venous flow. Its development is associated with decreased fetoplacental perfusion and intra uterine fetal

death (Gudmunsson et al 1993[76]). It is in these late stages, reversal of diastolic flow in umbilical artery is observed.

- An abnormal ductus waveform is recognized when there is a dip in the A wave, i.e. when the forward flow during atrial systole is less. This causes an increase in PI.

- Reversal of A wave indicates cardiac decompensation and acidemia.

- Growth restricted fetuses with abnormal venous flow have worse perinatal outcome compared to those where flow abnormality is confined to umbilical or MCA (Baschat et al 2000)[66].

- In a majority of severely growth restricted fetuses, sequential deterioration of arterial and venous flows precedes biophysical score deterioration.

- Thus, the typical progression begins with increased resistance in the umbilical artery followed by decreased resistance in the middle cerebral artery and is completed with the development of abnormal venous waveforms as cardiac function deteriorates.

- Adding serial doppler of umbilical artery MCA and DV to IUGR surveillance will enhance the performance of biophysical score in detecting fetal compromise and optimizing the time of intervention (Baschat et al 2001)[67].

MATERIALS AND METHODS

This study was conducted jointly at the Institute of Obstetrics and Gynecology and Barnard Institute of Radiology, Chennai both coming under the Madras Medical College, Chennai. Two hundred documented IUGR cases confirmed by clinical evaluation and serial ultrasound biometry were selected for the study and it was done on singleton pregnant women with well-documented period of gestation beyond 34 weeks. Known congenital anomalies were excluded from the study.

The machine used for Doppler was an Aloka 3500 color Doppler machine with a 3.5 to 5 MHz curvilinear probe.

Name, Age, Unit, Registration number and Address of the patients were noted. Detailed obstetric history including the history of pregnancy induced hypertension; gestational diabetes and chronic hypertension were obtained. History of previous pregnancies including birth weight of previous babies, perinatal deaths, and mode of delivery were elicited. Details of present pregnancy were asked, including the date of last menstrual period, details of scan in the first trimester and clinical examination noting, if available, were scrutinized.

A note was made of the maternal weight, blood pressure and obstetric examination findings of fundal height and various laboratory investigation results. Those with uterine fundal height less than 3cms from the expected height were clinically diagnosed as IUGR and ultra sound examination was done with special emphasis on morphometric measurements. Abdominal

circumference less than 5th percentile and estimated foetal weight less than 10th percentile for that gestational age were selected for study. In cases with risk factors, serial sonography was done to identify fetal growth restriction. Initial dating scan followed by second ultra-sound examination was done at around 34 to 36 weeks.

Patients with irregular cycles, unknown dates, those with restricted growth from the 1st trimester onwards by ultrasound and pelvic examination were excluded from the study group as were those with history of viral exanthematous fever, intake of drugs like antiepileptics, antipsychotics & anticoagulants.

All these cases were kept under surveillance till confinement. A careful search for causes of IUGR like Smoking, Alcoholism and Hypertension were made. Anemia, if present, was corrected and PIH, if detected, was managed appropriately. The cases were monitored by Fetal Kick Count, Cardiotocography, Serial measurements of fetometry AFI and Doppler studies. Doppler studies were done on Umbilical artery, Middle Cerebral Artery and Ductus venosus with a real time color Doppler ultra sound machine. Umbilical cord was located in the pool of amniotic fluid and values were taken at mid cord or placental insertion. Middle cerebral artery was localized in transverse section of fetal skull, at the level of thalamus in the Sylvian fissure. The ductus venosus was sampled in the abdominal circumference section, where it joins the umbilical vein to IVC. The Doppler transducer was placed on the abdominal wall over the uterus and carefully manipulated till Doppler signals appropriate for those particular vessels were identified.

The signals were recorded for a minimum of 5 to 8 cycles with blood flow velocity waveforms of equal shape and amplitude and of satisfactory quality were obtained. The image was frozen and measurements taken. Doppler was considered as abnormal when there was absent or reverse diastolic flow in umbilical artery or PI values were above the 95th percentile for that gestational age. Cerebro placental ratio less than one was also taken as abnormal.

Those cases where fetal assessment was normal were monitored fortnightly till delivery. Those with absent and reverse flow were taken up for termination of pregnancy. In those cases with low diastolic flow in umbilical artery, where fetal maturity adequate for survival was present, the pregnancy was terminated. In cases where fetal maturity was not reached monitoring was done with NST and BPP daily or twice weekly depending upon the severity of abnormality and associated complications. Pregnancy was terminated when there were abnormal readings from CTG or a low score on the bio-physical profile. In those cases where differential shunting of blood flow to fetal brain was present, termination was done even before NST or BPP were found to be abnormal. Mode of delivery was planned depending on the weight and gestational age and amount of liquor present. Outcome of pregnancy was recorded in detail including intrauterine demise, neonatal death, birth weight, Apgar score, development of neonatal complications and presence of congenital anomalies, placental weight and pathology. These details were entered in a proforma and the data was statistically analyzed and evaluated.

Procedure of Obstetric ultrasound examination and Doppler evaluation performed are given below.

FETOMETRY

Biparietal Diameter

Measurement was performed from the outer edge of skull on the proximal surface, to the inner edge of skull on the distal surface in a section that included the midline echo with the cavum septum pellucidum in the anterior third and the thalami on either side. During the study, care was taken to apply minimal pressure to the maternal abdomen with the transducer as the fetal head compression is associated with alterations of intra-cranial arterial flow velocity waveforms (Vyas et al 198987).

Head Circumference

It was measured at the same level as the BPD using the method of expanding ellipse.

Femur Length

A section showing both ends of the femur clearly was obtained and measurement of diaphysis was performed.

Abdominal circumference

A cross sectional view of the fetal abdomen showing the intrahepatic portion of umbilical vein in the anterior third of the abdominal circumference was used for measurement of abdominal circumference by the expanding ellipse method.

DOPPLER EVALUATION

Umbilical Artery

A loop of umbilical cord close to the placenta was located. The segment of umbilical cord was elongated so that the two umbilical arteries and one umbilical vein could be distinguished. Angle of insonation was adjusted to less than 60 degrees. An optimum Doppler signal was obtained and the pulsatility index was measured.

	PI
28 Weeks	1.2
40 Weeks	1.1

Foetal Middle Cerebral Artery

Section of foetal head used for BPD measurement was obtained and then the transducer was angled caudally till the middle cerebral artery was seen coursing along the sphenoid wings. Sample volume size and angle of insonation were adjusted after placing the cursor in the artery and appropriate signals obtained. The pulsatility index was measured.

	PI
28 Weeks	2.1
40 Weeks	1.5

In FGR, the expected abnormal findings would be an increased diastolic flow due to the cephalisation of blood flow and brain sparing effect. This would reflect as a decrease in PI values.

Foetal ductus venosus

A transverse section of the fetal abdomen was obtained with the transducer angled slightly cephalad, color flow switched on and the aliasing signal in the ductus venosus identified where it connects the umbilical vein to the IVC.

The sample volume and the angle of insonation were set and the waveform obtained. The PI was then measured.

	PI
28 Weeks	1.1
40 Weeks	0.8

OBSERVATIONS

The study was conducted on 200 third trimester women with ultrasonologically confirmed IUGR cases and the following observations were made.

Among the 200 cases that were confirmed to be IUGR by B-Mode Ultrasound, 179 cases showed abnormalities in the Doppler wave forms. 21 cases revealed normal Doppler wave forms.

TABLE NO.1

Total No of IUGR Cases	**200**	**%**
Normal Doppler	21	10.5
Abnormal Doppler	179	89.5

GRADING OF DOPPLER ABNORMALITIES

According to the increasing severity of altered Doppler indices in the 200 IUGR cases, we categorized the cases into six from grade 0 (normal Doppler) to grade 5.

Table showing details of Doppler abnormalities.

TABLE NO. 2

GRADES		No	%
0	Normal doppler	21	10.5
1	Increased UA PI alone	38	19
2	CPR reversal	93	46.5
3	Absent/reversed EDF in UA with decreased MCA PI	19	9.5
4	Absent/reversed EDF in UA with increased MCA PI	23	11.5
5	Ductus venosus alteration	6	3

Out of the 179 cases, 48 cases showed absent/reversed diastolic flow in umbilical artery, out of which 19 had compensated MCA flow while 23 had gone in for decompensated MCA flow (hypoxic and decompensated fetus), 38 cases showed only low diastolic flow in umbilical artery, 93 cases showed low diastolic flow in umbilical artery and increased diastolic flow in middle cerebral artery (hypoxic and compensated fetus). 6 cases showed increased PI in the ductus (acidotic fetus).

Representative normal and abnormal tracings from the study are shown on plates attached.

AGE DISTRIBUTION

The age distribution of the patients in our study is shown in the following table.

TABLE NO.3

Age Group	No of patients	Percentage (%)
18-22	61	30.5
23-27	93	46.5
28-31	40	20
32-36	5	2.5
> 36	1	0.5

Most of the patients were in the age group of 23-27 yrs. There was only 1 patient more than 36 years of age.

GRAVIDITY

This table shows the birth order of the patients in our study group.

TABLE NO.4

Gravidity	No. of Patients	Percentage (%)
PRIMI	108	54
SECOND	51	21.5
THIRD	34	17
FOURTH AND MORE	7	3.5

Majority of our patients were primigravidae (54%).Only 7 patients were of the birth order 4 or more.

GESTATIONAL AGE

The distribution of gestational age at which Doppler analysis was done in the study group is shown in the table below.

TABLE NO. 5

GESTATIONAL AGE(WEEKS)	NUMBER OF PATIENTS GRADE						
34-36	0	0	0	0	0	5	5 [2 %]
36-37	2	5	17	11	13	1	49 [24%]
37-38	1	3	26	6	6	0	42 [21%]
38-39	7	16	25	2	3	0	53 [26 %]
39-40	10	12	24	0	0	0	46 [23%]
> 40	1	2	2	0	0	0	5 [2%]

Most of our patients with mild Doppler abnormalities were adequately monitored till term or even beyond. Patients with higher grades of Doppler abnormalities were induced earlier based on their biophysical profile, non stress test and liquor status for best fetal outcome.

RISK FACTORS

This table shows the distribution of risk factors in the study group. 67(33.5%) of them had pregnancy induced hypertension as the risk factor. In 5cases (2.5%) gestational diabetes was the identified risk factor.6 cases(3%) had heart disease complicating pregnancy. 48 cases (24 %) had one of the other risk factors like breech, postdates, bronchial asthma, anaemia, hypothyroidism or chronic hepatitis. 102 patients(54%) had no risk factors.

TABLE NO. 6

Risk Factor	No. of Patients	Percentage (%)
Preeclampsia	67	33.5
Gest. Diabetes	5	2.5
Heart disease	6	3
Epilepsy	5	2.5
Others	48	24
None	102	51

Table showing mode of delivery

TABLE NO.7

Mode of delivery	**Doppler grade**						**Total**
	0	**1**	**2**	**3**	**4**	**5**	
Spontaneous labour	9	18	32	0	0	0	59 [29.5%)
Induction of labour	8	7	18	8	11	5	57 [28. 5%]
Cesarean section	4	13	43	11	12	1	84 [42%]

The table shows that mothers of small for gestational age babies with abnormal umbilical artery Doppler studies needed a cesarean section for fetal distress more than the normal Doppler group. In those cases with absent and reverse end diastolic flow in umbilical artery, the labour was induced after counseling the parents regarding prognosis of the baby and in some cases labour was induced for maternal indications like severe preeclampsia.

Outcome

Out of the 200 cases, 169 were live born and 24 were neonatal deaths. There were 5 cases of intrauterine deaths of the fetuses and 2 were still born. Out of the live borns, 32 had increased perinatal morbidity characterized by poor apgar scores, development of necrotizing enterocolitis, hypoxic ischemic encephalopathy (HIE), meconium aspiration syndrome (MAS), hyperbilirubinemia and prolonged admission in Neonatal Intensive Care Unit for reasons like sepsis or birth asphyxia.

Outcome data was correlated with Doppler finding in the table given below.

TABLE NO. 8

Perinatal Outcome	Number of Patients Grade						Total
	0	1	2	3	4	5	
IUD	0	0	0	0	2 [9%]	3 [50%]	5 [3%]
Stillborn	0	0	0	0	2 [9%]	0	2 [1%]
NND	0	1 [3%]	0	3 [16%]	17 [74%]	3 [50%]	24 [12%]
Increased Perinatal Morbidity	0	0	15 [16%]	15 [79%]	2 [9%]	0	32 [16%]
No significant adverse outcome	21 [100%]	37 [97%]	78 [84%]	1 [5 %]	0	0	137 [68%]

Table showing distribution of birth weight in the study group

TABLE NO. 9

Birth Weight	Number of Babies Grade						Total
	0	1	2	3	4	5	
Less than	0	0	0	0	1[20%]	4[80%]	5[2.5%]
1-1.4	0	0	2[18%]	2[18%]	7[63%]	0	11[5.5%]
1.5-1.9	7[10.5%]	5[7.5%]	28[42%]	12[18%]	13[19%]	2[19%]	67 [33%]
2.0-2.4	13[12.6%]	29[29%]	58[54%]	5[4.7%]	2[1.9%]	0	107 [53.5%]
2.5 and more	1[10%]	4[40%]	5[50%]	0	0	0	10[5%]

Most of the babies in the group 0, 1, 2 had a birth weight of 2 to 2.4 kgs.

Most of the babies in group 3 and 4 had birth weights ranging from 1.5 to 1.9 kgs.

In group5, 4 babies weighed less than 1 Kg and 2 babies weighed between 1.5 to 1.9kgs.

Perinatal mortality and morbidity was more in cases with low birth weights more so if birth weight was less than 1.5 kgs.

DISCUSSION

Fetometry by B-Mode Ultrasound is a reliable method of investigation to distinguish between IUGR and normal fetuses. This is probably because in IUGR fetuses, the earliest feature is reduced growth that is readily assessed by a measurement of abdominal circumference that will show consistently lower values than those expected for the particular gestational age. However the B-Mode ultrasound did not reliably detect the adverse perinatal outcome.

Predictive capability of Doppler of adverse outcome in USG confirmed IUGR cases, was analyzed.

TABLE NO.10

	Perinatal Outcome		
	Adverse	**Good**	**Total**
Doppler Abnormal	63	116	179
Doppler Normal	0	21	21

Sensitivity of Doppler in predicting adverse perinatal outcome: 100 %

Specificity of Doppler in predicting adverse perinatal outcome: 15.3%

Predictive value of an abnormal Doppler study: 35.19%

Predictive value of a normal Doppler study: 100 %

But on grading the abnormalities from 0 to 5 based on increasing severity of altered Doppler indices, we got the following statistics:

Grade 0 – had a negative predictive value of 100%

Grade 1 - also had a negative predictive value of 100% and that one neonatal death in this grade was due to an unrelated cause namely hand prolapse

Grade 2 – negative predictive value of 84%

Grade 3 – 95% positive predictive value for adverse outcome

Grade 4 – 100% positive predictive value for adverse outcome

Grade 5 -- 100% positive predictive value for adverse outcome 100% mortality rate

We also found that the patients who had mild abnormalities on Doppler (Grade 0, 1 and 2), did not have any mortality related to severity of IUGR, nor did they have any significant morbidity.

* There was a significant increase in occurrence of adverse perinatal outcome with increasing severity of Doppler abnormalities (P value 0.001).

* 97.91% of the patients with marked Doppler abnormalities (Grade 3 or more), had adverse perinatal outcome, compared to 10% of those with mild Doppler abnormalities (Grade 0, 1 and 2).This again was significant (P value 0.001).

* There was a significant increase in perinatal mortality with increasing grades of Doppler (including intrauterine demise, neonatal deaths and stillbirths), with a P value of 0.001.All the cases with intra- uterine demise and stillbirths as also 23 out of 24 neonatal deaths all had grade 3 or more Doppler abnormalities.

* There was also a significant rise in perinatal morbidity with increasing grades of Doppler (P value 0.001).

These observations show us that Doppler can accurately prognosticate IUGR cases and can help in optimizing the time of intervention in a hypoxic fetus. Among the 200 USG confirmed IUGR cases, 179 cases revealed Doppler abnormalities (89.5%).21 cases revealed normal Doppler findings. No adverse perinatal outcome was observed in these cases. It means that if the Doppler is normal, in an IUGR case, the possibility of an abnormal perinatal outcome is very rare. The normal Doppler result has more importance than an abnormal Doppler result.

Newham et al evaluated the efficacy of Doppler flow velocity waveform analysis as a screening test in pregnancy. They found that prediction of fetal hypoxia by Doppler analysis was enhanced in IUGR fetuses, where it was weak when umbilical artery PI values were evaluated as primary screening tests for fetal hypoxia. These findings very much conform to our observations.

The explanation for these observations is probably that fetal growth retardation can be either due to low intrinsic growth potential or due to defective placental nutritive and circulatory functions, of which Doppler can investigate only the circulatory component.

GESTATIONAL AGE

All the IUGR cases in our study were 34 weeks or more by gestational age. This is explained by 2 reasons:

1. Patients who were diagnosed to have IUGR at peripheral hospitals were referred late for delivery and NICU (Neonatal Intensive Care Unit)care, by which time they had an advanced gestational age, and sometimes marked and severe Doppler abnormalities, which had a poor fetal outcome.

2. Patients with preterm IUGR were not included in the study. This was done to eliminate the added risk of prematurity on the perinatal outcome of these IUGR babies. (i.e.) A baby which may have only a mild growth restriction as evidenced by Doppler, if delivered prematurely due to causes unrelated

to the growth restriction, may have an adverse perinatal outcome by virtue of its extreme prematurity, which can actually lead to false negative results for the doppler test.

3. In a patient who has been booked and monitored regularly, we find that each baby has its own growth profile which becomes established only around 24-26 weeks. Thus, a deviation from this growth profile resulting in growth retardation is effectively detectable only after 28 weeks. This was in agreement with the report of Harold Schulman et al in which the most reliable time to screen for IUGR is described as after 24-26 weeks. These patients are also delivered before the Doppler indices worsen, to ensure that the baby has the best chances of survival.

RISK FACTORS

In our study, among the cases that had identified risk factors, the majority had hypertension as the risk factor (33.5%). This is explained by the absence of physiological modification of spiral arteries that causes IUGR in hypertensive cases. This observation conformed to the study of Martin et al, which reported hypertension as the risk factor of particular importance in IUGR. Out of the 200 USG confirmed IUGR cases, 21 cases had normal results on Doppler evaluation. All these cases had no increased perinatal complications except for a low birth weight. This might be due to the fact that these cases were only minimally involved and did not progress beyond the

phase of decreased growth in the sequence of events of IUGR. The absence of significant redistribution of fetal circulation and hypoxia, as evidenced by normal Doppler evaluation would explain the better perinatal performance of these fetuses. Our observations were in agreement with the study of Benson et al in which all the IUGR fetuses with normal Doppler waveforms were not compromised in utero and were healthy although the biometric measurements were less than normal.

Most of the studies (Fleischer et al, Benson et al, Trudinger, Ardunini et al, Arbeille Pet al) have assessed the predictability of IUGR by Doppler analysis, and have reported the sensitivity varying from 17% to 78%. But we have studied the predictability of adverse perinatal outcome by Doppler analysis. Our study result was comparable to the findings of Chambers et al (Sensitivity 100% & Specificity 77%).

All the 179 cases with abnormal Doppler waveforms were subsequently analyzed for their outcome. Only 63 of these cases showed some form of increased perinatal complications. The remaining cases showed only low birth weight. Out of the 63 cases, 31 were deaths and 32 had neonatal morbidity.

From these observations, it is clear that regarding outcome of an IUGR case, a negative Doppler evaluation (normal Doppler findings) has a predictive value of 100% i.e., all cases with a negative Doppler evaluation will subsequently have better outcome without grave perinatal complications. A

positive Doppler evaluation as such without any grading of the abnormality on the other hand has a predictive value of only 35% i.e., about 35 of 100 cases with an abnormal Doppler test will subsequently have a grave outcome. This study by Frusca et al reported the predictive value of 37% for an abnormal Doppler evaluation in perinatal complications. The predictive value of 35% in our study is comparable.

This shows the necessity for grading of Doppler abnormality to prognosticate IUGR cases accurately.

TABLE NO.11

Grade of Doppler	**0**	**1**	**2**	**3**	**4**	**5**
No adverse perinatal outcome	21	37	78	1	0	0
Adverse perinatal outcome	0	1	15	18	23	6

P Value-0.001

In our study, when the perinatal outcome was analyzed according to the grades of Doppler abnormality as in the above table, it was obvious that there was a significant increase in occurrence of adverse perinatal outcome with increasing severity of Doppler abnormalities (P value 0.001).

To facilitate comparison, we grouped the patients into 2 groups-Those with mild Doppler abnormalities (Grade 0, 1 and 2) and those with marked Doppler abnormalities (Grade 3, 4 and 5).Perinatal outcome was compared in both groups.

TABLE NO.12

	Mild Doppler Abnormalities	**Marked Doppler Abnormalities**	**Total No. of patients**
Number of patients	152	48	200
Number of patients with adverse perinatal	16 [10.5%]	47 [97.91%]	63 [31.5%]
IUD	0	5	5
STILL BIRTH	0	2	2
NND	1	23	24
Perinatal morbidity	15	17	32

From this table, we see that 97.91% of the patients with marked Doppler abnormalities (Grade 3 or more) had adverse perinatal outcome, compared to 10% of those with mild Doppler abnormalities (Grade 0, 1 and 2).This again was statistically significant (P value 0.001).

This is reinforced by the fact that out of 63 patients who had adverse perinatal outcome, 47(74.6%) were from the group with marked abnormalities on Doppler and 25.39% were from the mild abnormalities group (i.e.) a statistically significant group of those with adverse perinatal outcome had higher grades of Doppler abnormalities.X2-15.88 (P Value-0.001).

The perinatal mortality rate in our study was 38%which is comparable to a study by Trudinger et al who reported perinatal mortality of 20% in cases with abnormal Doppler analysis.

When we analyze the neonatal deaths, 23 out of 24 deaths occurred in the group with marked Doppler abnormalities (95.83%). The one death that occurred in the group with mild Doppler abnormalities was due to a cause unrelated to the growth restriction-namely hand prolapse with birth asphyxia. This dramatic increase in neonatal deaths with increase in Doppler abnormalities was found to be statistically significant (X2-110.6 P Value-0.001).

The contributing factors to the neonatal deaths include:

- Hypoxic Ischemic Encephalopathy (50% babies)
- Meconium Aspiration Syndrome (33.3%)
- Necrotizing Enterocolitis (29.16%)
- Neonatal Sepsis (16%)
- Hyperbilirubinemia (4%)

More than one cause was responsible in 15 babies.

All the patients, who had intrauterine demise or stillbirths, had marked Doppler abnormalities and this was found to be statistically significant(X2 – 15.55 (P Value-0.001).

When we analyzed the perinatal morbidity rates, 17 patients out of 32 (53.12%) had marked Doppler abnormalities and 15 (46.87%) had mild Doppler abnormalities. This rise of perinatal morbidity with increasing grades of Doppler abnormalities was found to be statistically significant(X2 –26.56 P Value –0.001)

TABLE NO.13

Perinatal morbidity	Doppler abnormalities		Total no. of patients
	Mild	Moderate	
APGAR< 7 AT 5 MINUTES	4	6	10
HIE	0	3	3
MAS	5	1	6
HYPER	1	2	3
Prolonged NICU admission			
NEC	1	2	3

* 10 babies had low APGAR at 5 minutes

* 9 babies had prolonged NICU admissions due to reasons like sepsis, severe birth asphyxia

* 6 babies had Meconium Aspiration Syndrome

* 3 babies had Hypoxic Ischaemic Encephalopathy

* 3 babies had Necrotizing enterocolitis

* 3 babies had hyperbilirubinemia

Some babies had more than one of these factors. All these babies had some morbidity which could be reliably predicted by the grade of Doppler abnormalities (P value 0.001).

In our study, when the Doppler abnormalities were analyzed for their severity, it was obvious that all cases with a severe grade of abnormality had a worse outcome, as compared to the rest of cases with a lesser grade of abnormality. The grades 0 to 5, according to increasing severity were of accurate prognostic significance.

Grade 0 – had a negative predictive value of 100%

Grade 1 – also had a negative predictive value of 100% and that one neonatal death was due to an unrelated cause, namely hand prolapse

Grade 2 – negative predictive value of 84%

Grade 3 – 95% positive predictive value for adverse outcome

Grade 4 – 100% positive predictive value for adverse outcome

Grade 5 - 100% positive predictive value for adverse outcome 100% mortality rate

This clearly shows the importance of grading- while grades 0 to 2 have a high negative predictive value for adverse outcome, grades 3, 4 & 5 have a

high positive predictive value for adverse outcome and grade 5 actually had a 100% mortality rate.

Some of the other observations made in our study were

* Small for gestational age babies with abnormal umbilical artery wave forms were at an increased risk for oligohydramnios than those with normal Doppler and they also had a higher incidence of non reassuring non stress tests.

* Small for gestational age babies with abnormal umbilical artery velocity wave forms had a shorter diagnosis to delivery interval than those with normal Doppler cases.

* Small for gestational age babies with abnormal Doppler were more frequently delivered by cesarean section because of fetal distress.

SUMMARY AND CONCLUSIONS

* Diagnosis of IUGR was done by clinical assessment and serial sonography.

* The routine use of SFH measurement together with the use of serial ultrasound examinations in the 3rd trimester of high risk pregnancies detected majority of IUGR cases.

* With the use of Doppler of umbilical and middle cerebral arteries, it is possible to predict that an IUGR fetus is not hypoxic.

* With ductus venosus evaluation, detection of fetal acidemia is possible.

* Predictive value of normal Doppler is 100%. It means that if the Doppler is normal in an IUGR fetus the possibility of an abnormal perinatal outcome is very rare. So, unnecessary intervention can be reduced in those pregnancies with normal Doppler and normal amniotic fluid volume.

* There is a strict co-relation between abnormal umbilical Doppler velocimetry and an increased incidence of perinatal complications in an IUGR fetus.

* Days of admission to NICU and incidence of perinatal mortality are increased with the worsening of Doppler velocimetry.

* In cases with absent and reversed diastolic flow in umbilical artery the perinatal morbidity is nearly100%.

* The perinatal mortality in cases of ductus venosus alteration is 100%.

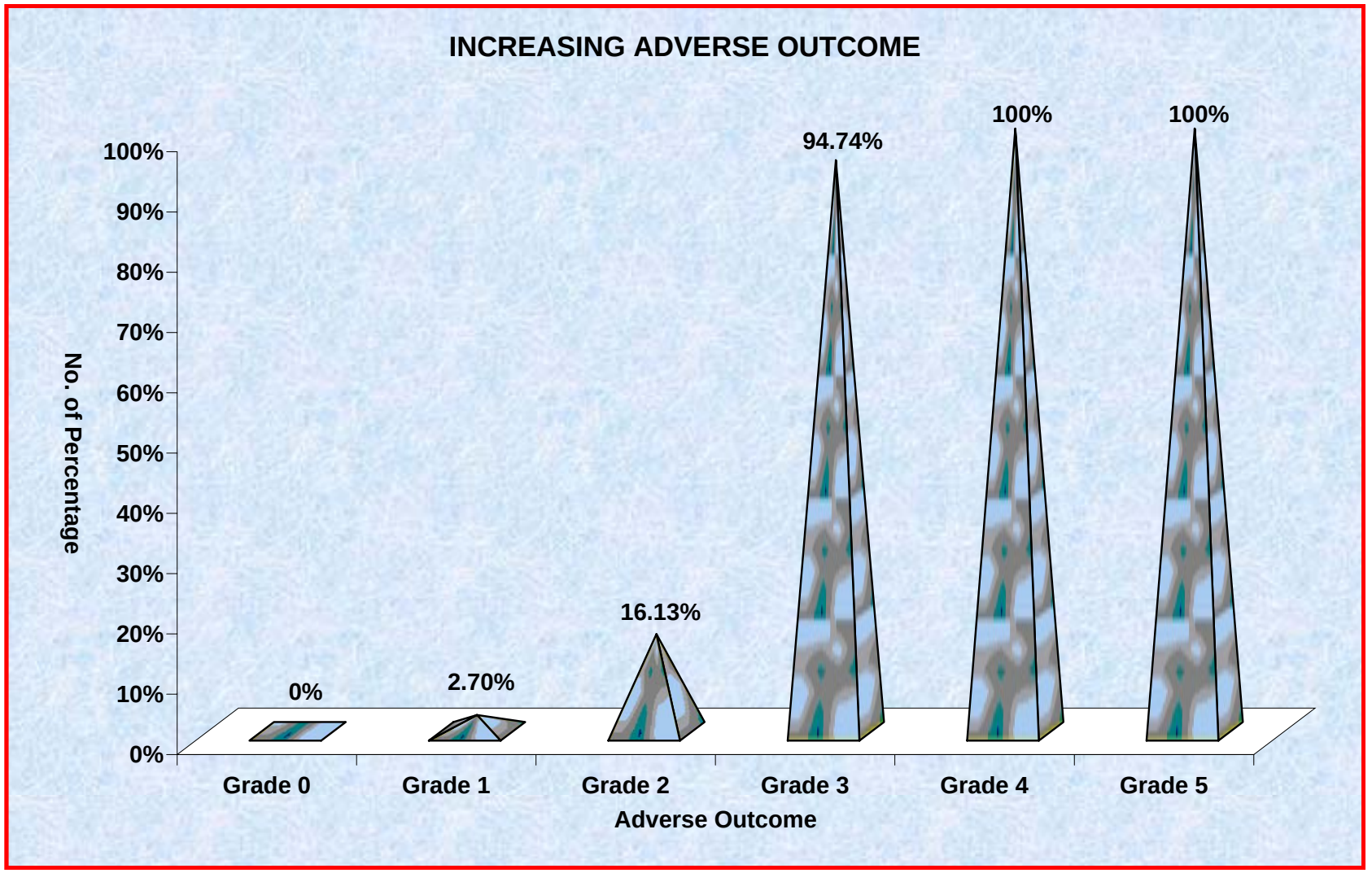
* In cases with differential shunting of blood flow to the fetal brain, frequent monitoring and early delivery should be done.

* The Doppler ultrasound finding of increased resistance of umbilical artery and decreased resistance of middle cerebral artery, detects the fetus at risk of complications 2 weeks earlier than the conventional methods like NST.

* After identifying those fetuses at risk of complications, close monitoring is done by non stress test and bio-physical scoring for planning the delivery so as to improve the perinatal outcome.

* Since ductus venosus has been shown to cause irreversible fetal compromise and inevitably leads to fetal demise, close monitoring should be done so as to deliver before the fetus becomes acidotic which is shown by the increase in ductus PI values

* Thus Doppler can be used as a prognostic tool in IUGR fetus as it gives an accurate prediction of the potentially compromised IUGR fetus.


INCREASING ADVERSE OUTCOME
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
No. of Percentage
0%
2.70%
16.13%
94.74%
100%
100%
Grade 0
Grade 1
Grade 2
Grade 3
Grade 4
Grade 5
Adverse Outcome

FETOMETRY

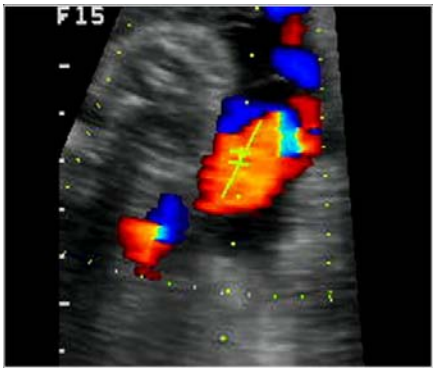
ALOKA 3500

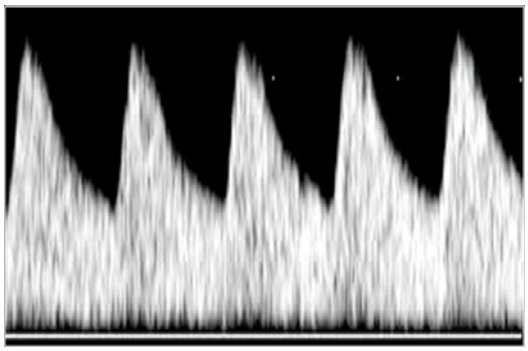
BPD

AC

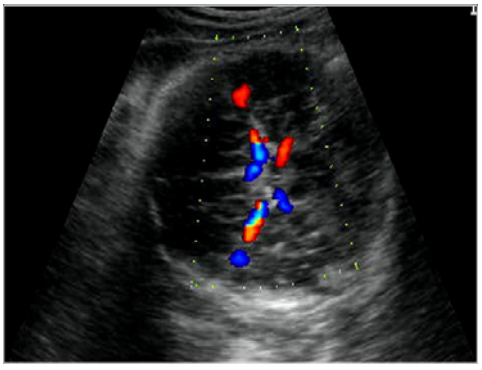
FL

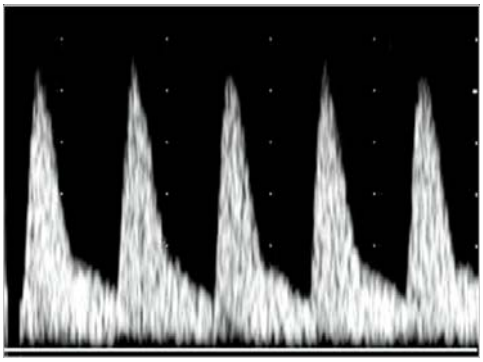
GRADE 0 - NORMAL DOPPLER



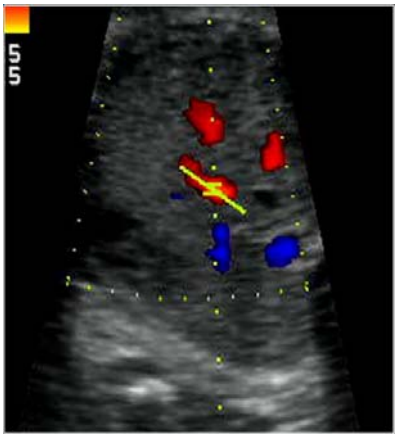


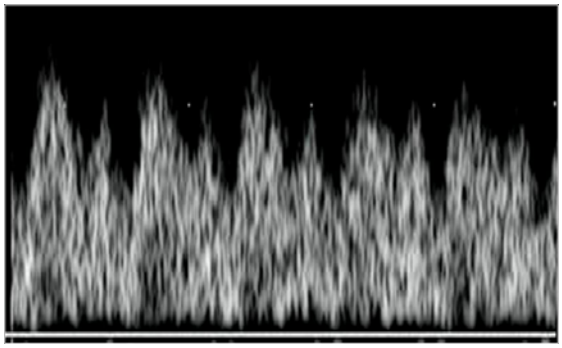
UMBILICAL ARTERY





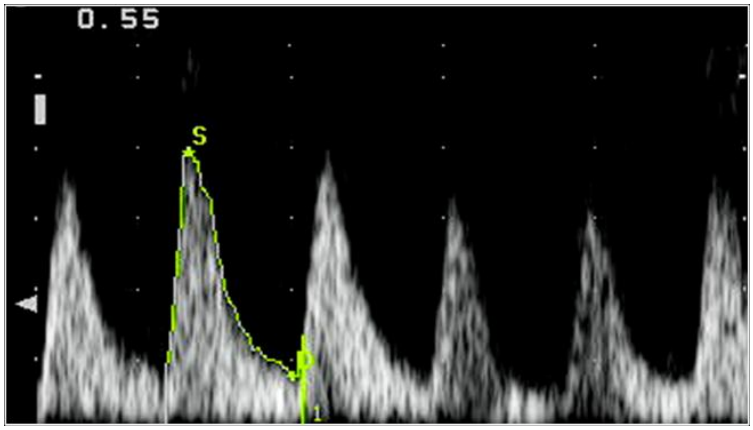
MIDDLE CEREBRAL ARTERY



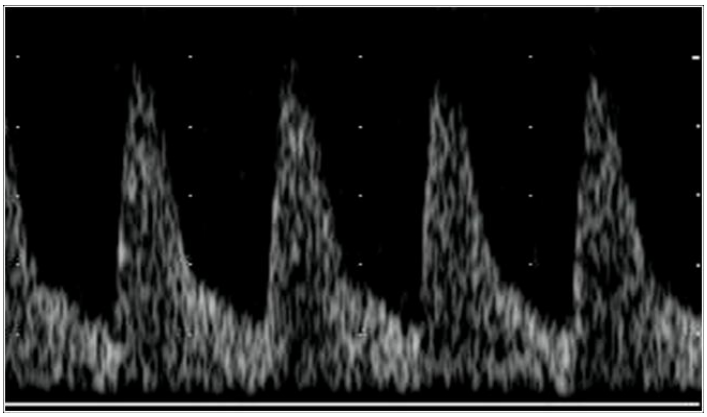


DUCTUS VENOSUS

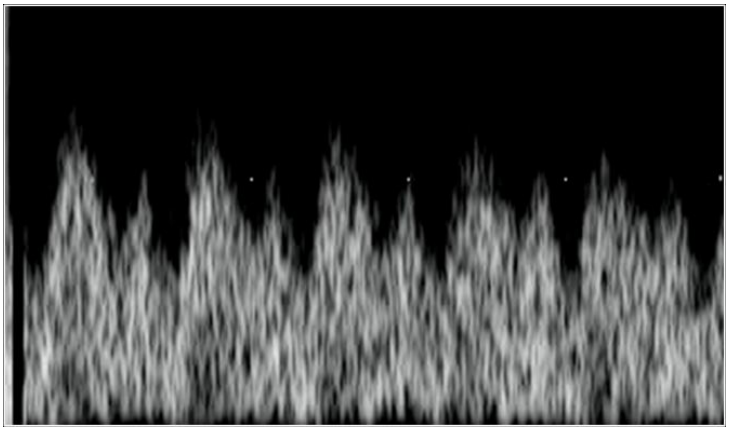
GRADE 1- INCREASED UA PI ALONE



UMBILICAL ARTERY

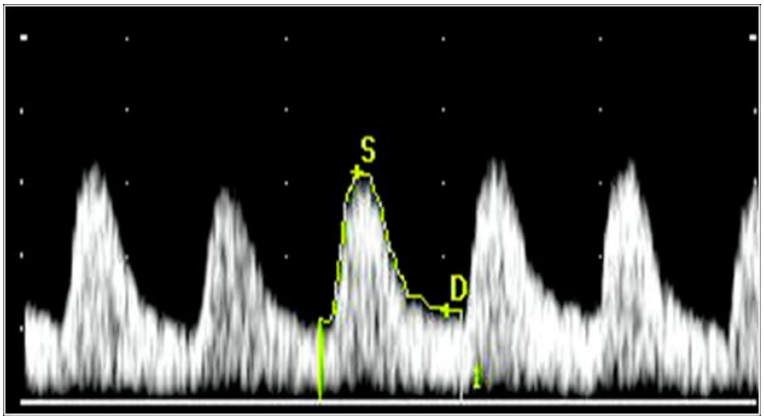


MIDDLE CEREBRAL ARTERY

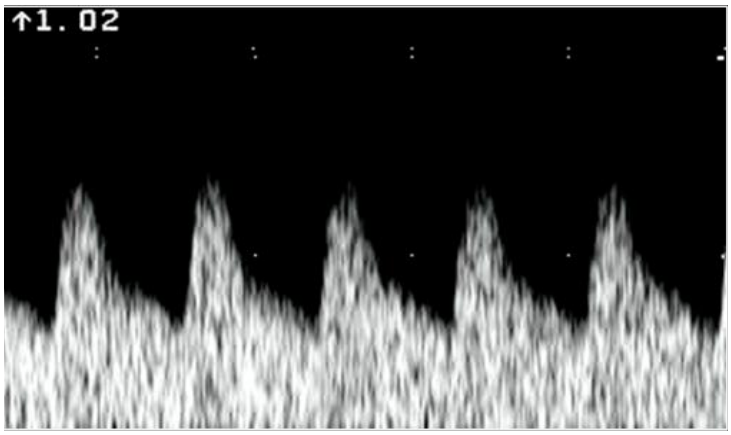


DUCTUS VENOSUS

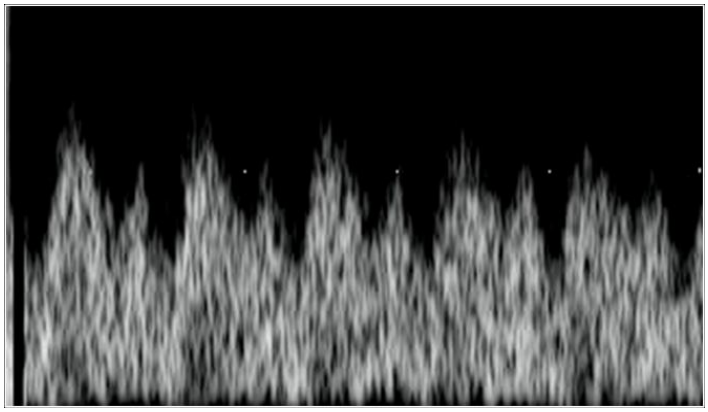
GRADE -2: CEREBRO PLACENTAL RATIO REVERSAL



UMBILICAL ARTERY

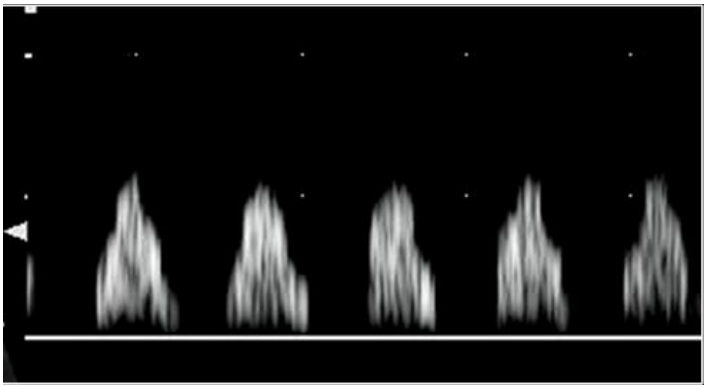


MIDDLE CEREBRAL ARTERY



DUCTUS VENOSUS

GRADE - 3: ABSENT / REVERSED EDF IN UA WITH DECREASED MCA PI



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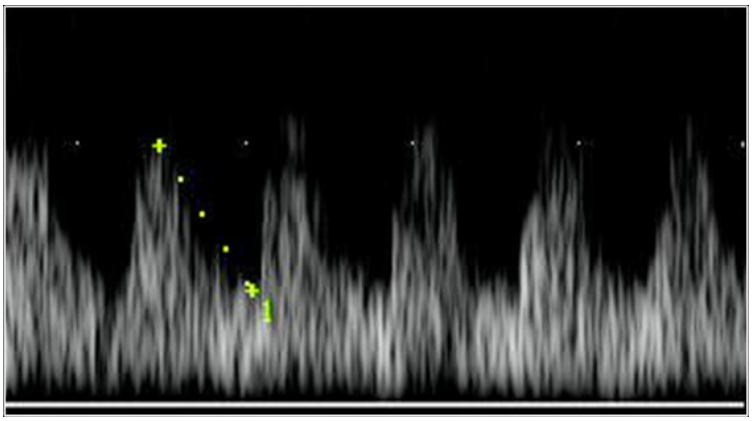
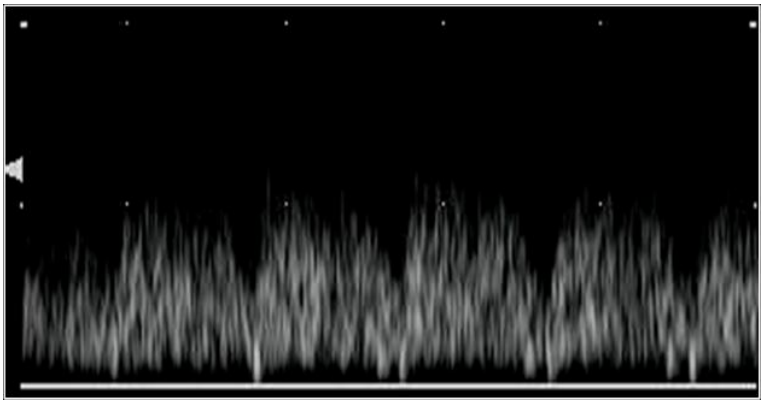
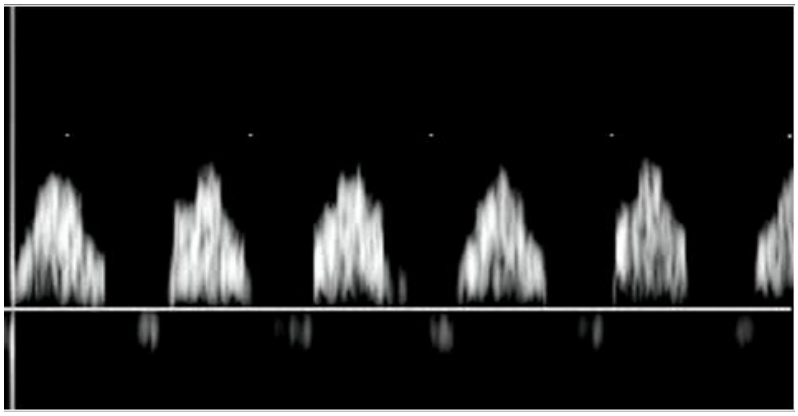


MIDDLE CEREBRAL ARTERY



DUCTUS VENOSUS

GRADE - 4: ABSENT / REVERSED EDF IN UA WITH INCREASED MCA PI



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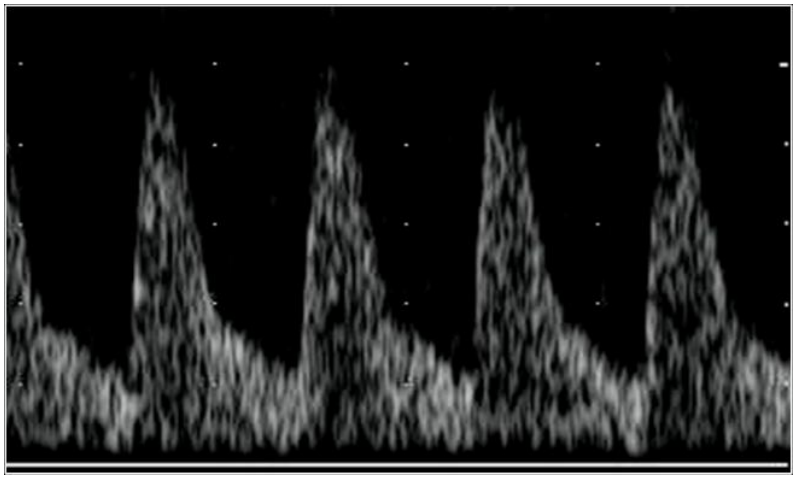

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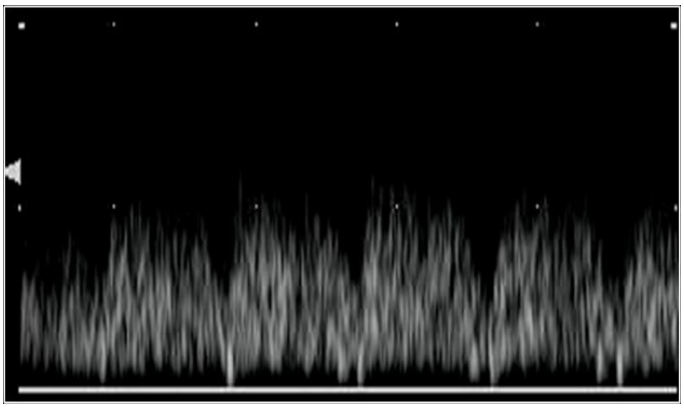
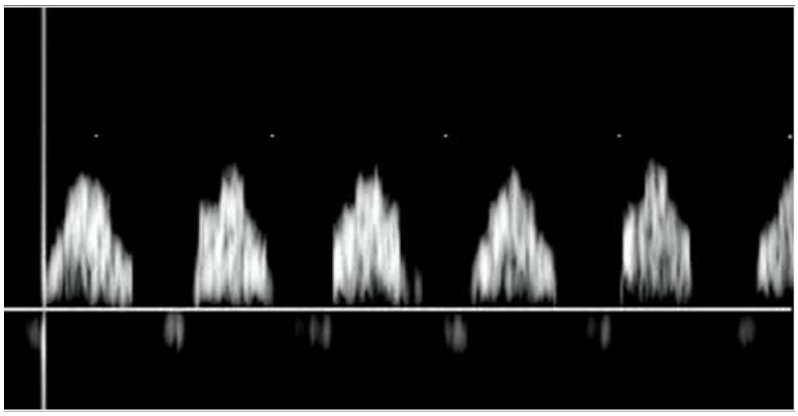


DUCTUS VENOSUS

GRADE - 5: DUCTUS VENOSUS ALTERATION



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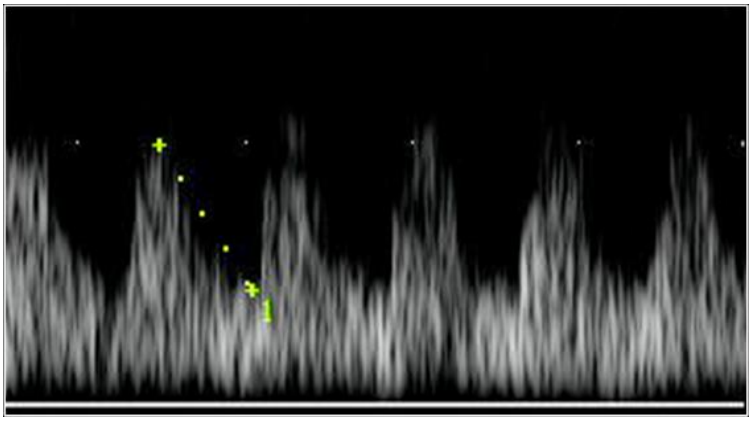

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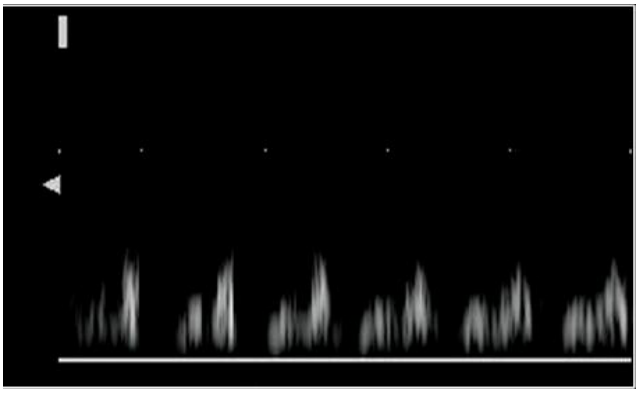

DUCTUS VENOSUS

S.No	Name/Age	Clinical	USG	PI			Grade	NST	Mode of Delivery	Birth weight	Perinatal outcome
				Um.A	MCA	DV					
1	Venkatammal 28	Primi 38wks	36 wks AFI 11	0.8	1.8	0.6	0	R	Vaginal induced	2.4 kg	Good
2	Meena /30	$G_2P_1L_1$ 39	35 wks	0.7	1.6	0.7	0	R	Vaginal induced	1.9	Good
3	Ansar beevi/24	G3P2L2	24-25 wks	1.9	1.8	1.5	5	NR	Vaginal induced	0.8	IUD
4	Ramani/28	G3P2L2	33-34 wks	1.0	0.8	0.7	2	R	LSCS	1.7	Good
5	Lashmi/20	Primi 38-39	34-35 wks	0.9	1.7	0.6	0	R	Vaginal	2.1	Good
6	Subashini/29	Primi 36-37	34 wks	1.1	0.9	0.7	2	R	LSCS	1.6	Good
7	Narsamma/30	G2A1 36-37	31-32WKS	1.3	1.4	0.8	4	NR	LSCS	1.4	NND
8	Vimla/20	Primi 38-	30-31wks	0.8	1.9	0.6	0	R	Vaginal induced	1.8	Good
9	Parvathi/24	G2P1L0 37-	34-35wks	1.6	1.4	0.7	2	R	LSCS	2.5	Good
10	Anita/21	G2P1L1 40-	34wks	1.8	1.4	0.6	2	R	Vaginal induced	2	Good
11	Rajeswari/26	Primi39-	34-35wks	1.7	1.5	0.7	2	R	LSCS	2.5	Good
12	Vijayalaxmi/35	G2P1L0 36-	32-33wks	1.1	1.9	0.6	0	R	LSCS	2.3	Good
13	Valli/25	G3P2L238w	32-33wks	1.7	1.2	0.7	2	R	Vaginal induced	1.6	Good
14	Regina/24	Primi38-	36-37wks	1.8	1.8	0.8	4	NR	LSCS	1.75	NND
15	Dhanalaxmi/21	Primi 37wks	33-34 wks	1.9	1.4	0.7	3	R	LSCS	1.65	NND
16	Dhana/29	Primi38wks	33wks AFI4	1.6	1.8	0.7	4	R	LSCS	1.5	NND
17	Kalaivani/24	Primi 38-	34wks	1.7	1.4	0.6	2	R	Vaginal induced	2	Good
18	Vijayaalaxmi	Primi 36-	34 wks	1.5	1.9	0.6	1	R	Vaginal induced	2.1	Good
19	Kamatchi/24	Primi 36-	32-33wks	1.7	1.9	0.8	4	R	Vaginal induced	1.5	STILLBORN
20	Sharmila/21	Primi 38-	35-36 wks	1.6	1.3	0.7	2	R	Vaginal	2.25	Good
21	Vasuki/30	G3P2L1 38-	30-31wks	1.7	1.9	0.8	4	NR	Vaginal induced	1.65	NND
22	Geetha/31	G2P1L1	28-30wks	1.8	2.0	1.2	5	NR	Vaginal induced	0.9	IUD
23	Prema/27	Primi40-	36wks	1.7	1.5	0.7	2	R	Vaginal	2.75	Good
24	Shobana/27	G3P1L1A1	33-34wks	1.4	1.3	0.6	2	R	Vaginal induced	2.2	Good
25	Selvi/23	G5P2L0A3	30-32wks	1.5	1.8	0.7	1	R	LSCS	1.75	NND
26	Parvathy/27	Primi	33-34 wks	1.5	1.4	0.6	2	NR	LSCS	2.25	Good
27	Saradha/27	G5P3L2 36-	30wks	1.8	2.0	0.8	4	NR	Vaginal induced	1	IUD

28	Pushpalatha/22	G2P1L0 38-	36 wks	1.7	1.6	0.7	2	R	LSCS	2.2	Good
29	Kirupa/25	G2P1L139-	36-37wks	1.6	1.4	0.6	2	R	LSCS	2.2	Good
30	Sumathi/26	G3P2L0 35-	24 wks	1.9	1.9	1.2	5	R	Vaginal induced	1.75	NND
31	Shanthi/26	G2P1L137-	33-34wks	1.5	1.4	0.6	2	R	Vaginal	1.9	Good
32	Shajitha/24	G3P1L1A1	34-35wks	1.4	1.8	0.6	1	R	LSCS	2.3	Good
33	Shanthi/26	Primi 39-	33-34wks	1.7	1.5	0.7	2	R	Vaginal	2.0	Good
34	Asha/20	Primi36wks	33-34 wks	1.5	1.9	0.6	1	R	Vaginal	2.0	Good
35	Rachel/35	G4P3L035-	26 wks	1.9	1.8	1.3	5	NR	Vaginal induced	0.9	IUD
36	Nithyapriya/27	G3P1L1A13	33wks	1.5	1.9	0.5	1	R	LSCS	2.5	Good
37	Jeyasudha/25	G2P1L137-	30wks	1.7	1.9	0.8	4	NR	Vaginal induced	1.9	NND
38	Anandhi/22	G3A238-	34wks	1.6	1.5	0.7	2	R	LSCS	1.75	Good
39	Malathy/22	Primi39-	36wks	1.5	1.8	0.6	1	R	Vaginal induced	2.2	Good
40	Dowlath/21	Primi36wks	31 wks	1.7	1.5	0.8	3	R	LSCS	1.25	NND
41	Swarnalatha/25	G2P1L139-	34-35wks	1.5	1.2	0.7	2	R	Vaginal	2.4	Good
42	Selvi/26	G2A1 39-40	36-37wks	1.4	1.3	0.6	2	R	LSCS	2.25	Good
43	Balasaraswathi/2	G3P2L137-	37wks	1.5	1.3	0.7	2	R	LSCS	2	Good
44	Malini/25	G3P1L1A13	37wks	1.4	1.9	0.6	1	NR	LSCS	2.4	Good
45	Sathya/20	Primi 36-	28wks	1.8	1.9	0.8	4	NR	Vaginal induced	1.1	STILLBORN
46	Valli/25	Primi36-	32wks	1.5	1.3	0.7	2	R	Vaginal	1.8	morbidity
47	Bhavani/27	Primi36-	32 wks	1.8	1.4	0.7	3	NR	LSCS	1.75	morbidity
48	Arokiyastella/	Primi38-	33 WKS	1.4	1.9	0.6	1	R	Vaginal	2	Good
49	Jaya/24	G3P2L1 36-	28wks	1.5	1.3	0.7	2	R	Vaginal	1.5	Good
50	Latha/26	Primi38-	32-33wks	1.4	1.3	0.7	2	R	Vaginal	1.9	Good
51	Prabavathy/21	Primi39-	34 wks	1.0	1.9	0.6	0	R	Vaginal	2.1	Good
52	Saraswathi/18	Primi 36-	36WKS	1.7	1.5	0.8	2	R	Vaginal	1.25	Good
53	Jagatha/21	Primi38-	36wks	1.6	1.5	0.8	2	R	Vaginal induced	2	Good
54	Nandhini/22	G3A2 36-	34wks	1.4	1.3	0.7	2	R	Vaginal	1.75	Good
55	Meenatchi/20	Primi39-	33-34wks	0.9	1.8	0.6	0	R	Vaginal	2	Good
56	Anitadevi/24	Primi38-	34 wks	1.2	1.9	0.8	1	R	Vaginal	1.9	Good

57	Selvi/20	G1P1L1 37-	32-33wks	1.5	1.4	0.7	2	R	Vaginal	1.8	Good
58	Selvikrishnan/	Primi39-	32 wks	1.6	1.5	0.6	2	R	LSCS	2.2	Good
59	Dhanalaxmi/22	Primi39-	34 wks	1.3	1.9	0.7	1	R	LSCS	2.5	Good
60	Sudha Iyappan	Primi37-	32-34wks	1.8	1.9	0.8	4	NR	Vaginal Induced	2	NND
61	Malliga/30	G2P1L1 38-	34wks	1.3	1.9	0.6	1	R	Vaginal	2	Good
62	Shanthi/23	Primi36-	32wks	1.5	1.4	0.7	2	R	Vaginal induced	1.75	morbidity
63	Thilaga/24	Primi39-	36wks	1.6	1.5	0.6	2	R	Vaginal	2.2	morbidity
64	Mala/26	G2P1L138-	36-37wks	1.7	1.6	0.7	2	R	LSCS	2.3	morbidity
65	Sampangi/30	G3P2L136-	32 wks	1.8	1.8	0.7	4	NR	Vaginal induced	1.8	NND
66	Jyothi /20	Primi38-39	36wks	1.4	1.9	0.7	1	R	Vaginal	2.25	Good
67	Punithavathy/	Primi39-	36-37WKS	1.3	1.8	0.8	1	R	LSCS	2.3	Good
68	Nagalaxmi/22	Primi38-	32 wks	1.6	1.5	0.7	2	R	Vaginal	1.9	Good
69	Mariammal/23	Primi37-	36 wks	1.9	2.0	0.9	4	NR	LSCS	2.3	morbidity
70	Umarani /24	Primi 38-	36 WKS	1.4	1.7	0.8	1	R	Vaginal	2.25	Good
71	Malathi/18	Primi39-	36wks	1.5	1.8	0.7	1	R	Vaginal	2.2	Good
72	Ambika/25	Primi38-	37 wks	1.3	1.8	0.8	1	R	Vaginal	2.3	Good
73	Gomathi/27	G3P1L1A1	34 wks	1.8	1.5	0.8	3	R	Vaginal induced	2.05	morbidity
74	Venila/26	G2P1L1 38-	36 wks	1.6	1.4	0.7	2	R	LSCS	2.4	Good
75	Lakshmi/28	G2P1L1 37-	35 wks	1.5	1.4	0.7	2	R	Vaginal	2	Good
76	Shanthi/25	Primi 38-	30 wks	1.7	1.5	0.8	3	R	Vaginal induced	2.3	morbidity
77	Jayanthi/30	G3P1L1A1	36wks	1.3	1.9	0.7	1	R	LSCS	2.3	Good
78	Rajeswari/25	G3P1L1A1	34-35wks	1.5	1.4	0.8	2	R	LSCS	2.25	Good
79	Arifa/28	G2A1 37-	34 wks	1.4	1.3	0.7	2	R	LSCS	2.2	Good
80	Vimala/28	Primi 36-	36wks	1.4	1.2	0.7	2	R	Vaginal	2	Good
81	Tharangini/30	G2P1L1 37-	37wks	1.3	1.8	0.8	1	R	Vaginal	2	Good
82	Eswari/25	Primi38-	36wks	1.4	1.2	0.8	2	R	LSCS	2.3	Good
83	Ponnammal/21	G2P1L139-	37wks	1.5	1.4	0.7	2	R	LSCS	2.4	Good
84	Bhavani/27	Primi 36-37	30wks	1.8	1.5	0.8	3	R	LSCS	1.75	morbidity
85	Laxmi/21	Primi 38-	34wks	1.4	1.3	0.8	2	R	Vaginal	2.25	Good

86	Malar/30	G3P2L1 39-	35-36wks	1.3	1.9	0.7	1	R	Vaginal	2.25	Good
87	Hasanamma/22	Primi 38-	34wks	1.4	1.7	0.6	1	R	Vaginal	2	Good
88	Nalini/31	Primi 37-	32-34wks	1.5	1.3	0.7	2	R	Vaginal induced	1.8	Good
89	Sangeetha/20	Primi38-	36-37wks	1.4	1.7	0.7	1	R	Vaginal	2.3	Good
90	Siromani/26	G2P1L1 38-	35-36wks	1.5	1.4	0.8	2	R	LSCS	2.2	Good
91	Santhanamary	G2P1LO 36-	33-34 wks	1.8	1.5	0.9	3	R	Vaginal induced	1.5	morbidity
92	Vasantha/21	Primi 40-	35-36wks	1.3	1.7	0.7	1	R	LSCS	2.4	Good
93	Shaheeda banu	G2P1L1 39-	36wks	1.5	1.3	0.6	2	R	LSCS	2.3	Good
94	Menaka/24	Primi 36-	32wks	1.8	1.9	1.3	5	NR	Vaginal induced	0.9	NND
95	Kanchana/26	G5P1L0A3	34 wks	1.7	1.5	.0.8	3	R	LSCS	1.8	NND
96	Sridevi/25	G3A239-	36 wks	1.4	1.2	0.8	2	R	Vaginal	2.25	Good
97	Kamatchi/23	G2P1L1 37-	36 wks	1.3	1.2	0.7	2	R	Vaginal	2	Good
98	Usha 26	Primi 39-	37wks	1.5	1.4	0.7	2	NR	Vaginal induced	2.4	Good
99	Josephine 27	Primi 36wks	32-33 wks	1.9	1.9	1.2	5	NR	LSCS	1.5	NND
100	Deepa 22	Primi 36-	30wks	1.8	2.0	1.3	4	NR	Vaginal induced	1.0	IUD
101	Malliga/26	G2P1L038-	36wks	1.3	1.9	0.7	1	R	LSCS	2.1	Good
102	Prasanthini/30	Primi38-	30wks	1.8	1.8	0.9	4	NR	Vaginal induced	0.95	NND
103	Neelavathi /23	G2P1L037-	36wks	1.4	1.3	0.7	2	R	Vaginal	2	Good
104	Devi/21	Primi38-	35-36wks	1.5	1.3	0.8	2	NR	LSCS	2	Good
105	Saradha/27	Primi37-	36wks	1.8	1.5	0.8	3	R	LSCS	1.9	morbidity
106	Rekha/26	Primi39-	36 wks	1.3	1.9	0.7	1	R	Vaginal	2.2	Good
107	Lakshmi/21	Primi37-	34wks	1.6	1.5	0.7	2	NR	Vaginal	1.75	Good
108	Sivagami/28	Primi36-	36wks	1.4	1.3	0.8	2	R	Vaginal	2	Good
109	Rathi/21	Primi36-	36-37WKS	1.4	1.2	0.7	2	R	Vaginal	2.1	Good
110	Zeenath/23	Primi36-	34wks	1.9	1.8	0.9	4	R	LSCS	1.7	NND
111	Selvi/22	Primi39-	36-37wks	1.4	1.3	0.7	2	NR	LSCS	2.1	Good
112	Thilaga/26	Primi38-	34-35wks	1.5	1.4	0.7	2	NR	LSCS	1.9	Good
113	Chitra/19	Primi39-	36wks	1.4	1.2	0.8	2	NR	Vaginal induced	2.4	Good
114	Thilagavathi/22	Primi38-	36wks	1.3	1.2	0.7	2	R	Vaginal	2.2	morbidity

115	Majeswari /26	G3P2L2 36-	36wks	1.4	1.3	0.6	2	R	Vaginal	1.5	Good
116	Vijayalakshmi	Primi 36-	36 wks	1.3	1.2	0.7	2	NR	LSCS	1.9	morbidity
117	Vijayakumari	Primi37-	34-35wks	1.7	1.5	0.8	3	NR	LSCS	1.75	morbidity
118	Jayamani/22	G2P1L1 39-	37wks	1.0	1.7	0.7	0	R	LSCS	2.5	Good
119	Sathya/19	Primi 40-	37-38 wks	0.9	1.8	0.6	0	R	Vaginal	2.1	Good
120	Kala/30	G4P2L0A1	37 wks	1.3	1.7	0.8	1	R	LSCS	2.5	Good
121	Maheswari/22	Primi37-	34wks	1.8	1.8	0.9	4	R	LSCS	1.7	NND
122	Rani/24	G3P2L2 38-	36-37 wks	1.4	1.3	0.7	2	NR	Vaginal	2	morbidity
123	Shanthi/29	Primi37-	36wks	1.5	1.3	0.8	2	NR	LSCS	1.9	morbidity
124	Poongodi/30	G2P1L0 36-	32wks	1.4	1.3	0.7	2	R	LSCS	1.6	morbidity
125	Vimala/24	Primi36-	36wks	1.0	1.9	0.7	0	R	Vaginal	1.8	Good
126	Selvi/25	Primi39-	36 wks	0.9	1.8	0.6	0	R	Vaginal induced	2.25	Good
127	Dhanalaxmi/27	Primi36-	34-35wks	1.9	1.8	0.8	4	NR	LSCS	1.65	morbidity
128	Gajalaxmi/26	Primi37-	34wks	1.4	1.3	0.7	2	R	LSCS	2.0	Good
129	Vasantha/33	Primi38-	35wks	1.8	1.5	0.8	3	NR	LSCS	2.0	Good
130	Shanthi/23	Primi 36-	34 WKS	1.9	2.0	0.9	4	R	Vaginal induced	1.5	NND
131	Sharmila/20	Primi39-	36-37wks	1.1	1.8	0.6	0	NR	Vaginal	2.25	Good
132	Dhanalaxmi/28	G3A236-	30-32wks	1.3	1.2	0.6	2	NR	Vaginal induced	1.25	morbidity
133	Laxmi/31	G2P1L138-	36-37wks	1.4	1.3	0.7	2	R	LSCS	2.1	Good
134	Jayalaxmi/23	Primi37-	36-37wks	1.8	1.4	0.9	3	NR	Vaginal induced	2	morbidity
135	Vimala/26	Primi 39-	34wks	1.3	1.8	0.7	1	R	Vaginal	1.8	Good
136	Pachiammal/22	G2P1L1 39-	36wks	1.0	1.8	0.7	0	R	Vaginal	2	Good
137	Shabana /25	G3P1L1A1	33wks	1.8	1.6	0.8	3	R	Vaginal induced	1.5	morbidity
138	Rajalaxmi/21	Primi38-	36wks	1.4	1.3	0.7	2	NR	LSCS	2	morbidity
139	Poornima/28	G2P1L1 38-	36wks	1.3	1.7	0.7	1	R	Vaginal	2	Good
140	Geetha/26	G2P1L1 37-	34wks	1.8	1.4	0.8	3	NR	LSCS	1.75	morbidity
141	Anbarasi/24	Primi 37-	36wks	1.5	1.4	0.8	2	R	LSCS	2	Good
142	Padma/20	Primi 38-	36-37wks	1.3	1.7	0.7	1	R	Vaginal induced	2.25	Good
143	Suguna/26	G3P2L2 38-	36-37wks	1.4	1.8	0.8	1	R	Vaginal induced	2.2	Good

144	Sarojini/21	Primi39-40	37wks	1.5	1.4	0.8	2	R	LSCS	2.4	Good
145	Ponni/25	Primi38-	36 WKS	1.4	1.3	0.7	2	R	Vaginal induced	2.3	Good
146	Sarumathi/30	G2P1L1 37-	36-37wks	1.5	1.3	0.7	2	R	LSCS	2.5	Good
147	Parimala/20	Primi 36-	32-34 wks	1.4	1.3	0.8	2	R	Vaginal induced	1.5	Good
148	Srikumari/27	G2A1 36-37	32 wks	1.8	1.5	0.9	3	NR	Vaginal induced	1.55	morbidity
149	Sheela/25	G2P1L 38-	36 wks	1.4	1.2	0.8	2	R	LSCS	2.2	Good
150	Kirupa rani/33	G3A2 36-37	34 wks	1.8	1.9	0.9	4	NR	LSCS	1.8	NND
151	Prabavathi/19	G3P2L1 36-	34-35 wks	1.5	1.3	0.8	2	R	Vaginal induced	1.75	Good
152	Indrakumari/31	G2P1L1 39-	36-37 wks	1.4	1.8	0.7	1	R	LSCS	2.5	Good
153	Parvathi/20	Primi39-40	37 wks	1.5	1.4	0.8	2	R	LSCS	2.2	Good
154	Devi/26	G5P4L1 38-	36wks	1.4	1.3	0.8	2	R	Vaginal induced	2.2	Good
155	Ramani/31	Primi39-	36 wks	1.5	1.3	0.7	2	R	LSCS	2.5	Good
156	Geethalaxmi/	G3P2L0 37-	34 wks	1.8	1.5	0.9	3	R	LSCS	2	morbidity
157	Kannamma/25	Primi 37-	34 wks	1.4	1.3	0.8	2	NR	LSCS	1.7	Good
158	Gandhi/26	G2P1L1 36-	34wks	1.8	1.4	0.9	3	R	Vaginal induced	1.6	morbidity
159	Subashini/18	Primi 38-	36wks	1.0	1.8	0.7	0	R	Vaginal	2	Good
160	Hemalatha/23	G2P1L1 37-	32-34 wks	1.8	1.9	0.9	4	NR	LSCS	1.4	NND
161	Amsavalli/22	Primi 36-	32-34wks	1.9	1.9	0.8	4	NR	LSCS	1.5	NND
162	Dhanalakshmi/2	Primi 36-	33 wks	1.8	1.9	0.8	4	NR	LSCS	1.6	NND
163	Ambika/22	G2P1L1 38-	36-37wks	1.4	1.2	0.7	2	R	LSCS	2.3	Good
164	Alamelu/26	G3P1L1A1	36wks	1.5	1.4	0.8	2	R	LSCS	2.1	Good
165	Jesintha/22	Primi 37-	34wks	1.3	1.2	0.7	2	NR	LSCS	1.9	Good
166	Meena/26	G2A1 39-	36wkAFI9	1.4	1.3	0.8	2	R	LSCS	2.25	Good
167	Noorie/22	G2P1L139-	36-37wks	1.3	1.8	0.8	1	R	LSCS	2.4	Good
168	Kala/23	G2P1L1 38-	36-37wks	1.4	1.7	0.7	1	R	LSCS	2.25	Good
169	Epsiba/28	G2P1L1 37-	36wks	1.3	1.7	0.8	1	NR	LSCS	2.3	Good
170	Devi/28	Primi39-	34wks	1.5	1.4	0.7	2	R	Vaginal induced	2.4	Good
171	Laxmi/26	Primi37-	32wks	1.4	1.3	0.8	2	R	Vaginal	1.75	morbidity
172	Malarvizhi/25	G3P1L1A13	36wks	1.4	1.8	0.8	1	R	Vaginal	2.1	Good

173	Nagamani/25	Primi 37-	34wks	1.3	1.9	0.7	1	R	Vaginal	1.9	Good
174	Ramadevi/26	Primi36-	30wks	1.9	1.9	0.9	4	NR	Vaginal induced	1	NND
175	Jyothi/30	G3A2 38-	34-35wks	0.9	1.7	0.6	0	R	LSCS	1.8	Good
176	Pushpalatha/28	G2P1L1 39-	32 wks	0.8	1.8	0.7	0	R	Vaginal	2.1	Good
177	Anandalaxmi /24	G3P1L1A1	36wks	1.3	1.2	0.8	2	R	Vaginal induced	2.1	Good
178	Devi/26	Primi39-	36 wks	1.5	1.3	0.7	2	R	Vaginal	2	Good
179	Philomina/23	Primi 39-	35-36wks	1.4	1.3	0.8	2	R	Vaginal	2.2	Good
180	Nagajothi/21	Primi38-	36wks	1.5	1.4	0.7	2	R	Vaginal	2.2	Good
181	Shobana/22	G2A1 37-38	32WKS	1.4	1.2	0.8	2	R	LSCS	1.9	Good
182	Kokila/24	Primi 36-	36WKS	1.4	1.3	0.8	2	R	Vaginal induced	1.8	morbidity
183	Kowsalya/26	Primi 37-	34-35wks	1.5	1.3	0.7	2	R	Vaginal induced	2.3	Good
184	Chinamma/26	G2P1L1 37-	36 wks	1.9	1.8	0.9	4	NR	LSCS	1.4	NND
185	Vijaya/28	G3P2L239-	36 wks	1.4	1.2	0.8	2	R	Vaginal	2	Good
186	Bharathi/18	Primi 37-	34wks	1.5	1.3	0.7	2	R	Vaginal induced	1.8	morbidity
187	Saroja /28	G3A2 39-	32 wks	1.5	1.4	0.8	2	R	LSCS	2.5	Good
188	Dhatchayini/20	Primi39-	36-37wks	1.4	1.3	0.7	2	R	LSCS	2.4	Good
189	Kalavathy/25	G2P1L1 39-	36 wks	1.0	1.8	0.8	0	R	LSCS	2.25	Good
190	Mariammal/38	G5P1L1A3	34 wks	1.6	1.5	0.8	2	NR	LSCS	2	Good
191	Bakiya/30	G2P1L1 39-	35-36wks	1.1	1.5	0.7	0	R	Vaginal induced	1.9	Good
192	Sarawathi/30	Primi39-	36wks	1.0	1.6	0.7	0	R	Vaginal induced	2.25	Good
193	Sumalatha/16	Primi38-	34-35wks	1.0	1.7	0.6	0	R	Vaginal induced	1.8	Good
194	Swarnalatha/24	G2P1L1 38-	34wks	0.9	1.7	0.7	0	R	Vaginal induced	1.8	Good
195	Umamaheswari/	G3P2L1 37-	32-34wks	1.5	1.3	0.9	2	R	Vaginal induced	1.75	morbidity
196	Krishnaveni/20	G2P1L1 39-	36 wks	1.3	1.8	0.8	1	R	Vaginal	2.0	Good
197	Aarthi /25	Primi38-	35wks	1.3	1.8	0.8	1	R	Vaginal induced	1.9	Good
198	Saradha/32	Primi39-	36wks	1.4	1.9	0.7	1	R	Vaginal induced	2.2	Good
199	Seetha/25	Primi36-	32 wks	1.8	1.5	0.8	3	NR	Vaginal induced	1.4	morbidity
200	Thilagavathy/	Primi 36-37	35 wks	1.9	1.5	0.9	3	NR	LSCS	1.9	morbidity

PROFORMA

Name : Age :

Husband's Name : IP No :

Address : Date of Admission:

Date of Delivery:

Date of Discharge:

Socio economic Status : Education:

Obstetric formula : LMP : EDD :

Gynec. History : Age at Menarche :

Menstrual History :

Marital life :

Obstetric History :

Past History:

H/o Hypertension:

H/o Diabetes mellitus:

H/o Bronchial Asthma

H/o Tuberculosis:

H/o Heart Disease:

H/o Renal Disease :

H/o Epilepsy:

H/o Connective Tissue disease:

Abnormalities

Family History	Twinning	:		
	PIH	:		
Personal History:	Smoking	:		
	Alcoholism	:		
Drug History:				
General condition:	Build	:	Nourishment	:
	Height	:	Weight	:
	Pallor	:	Clubbing	:
	Jaundice	:	Pedal Edema	:
	Cyanosis	:	Lymphadenopathy:	
Vitals:	Temperature:		Respiratory rate:	
	Pulse Rate		Blood Pressure	:
Cardiovascular System:				
Respiratory System	:			
Thyroid	:	Breast:		
Per Abdomen	:			
Investigations:				
HB%	:		PCV	:
Platelets	:		Urine: Alb	:
B1 Sugar	:		Sugar	:
B1. Urea	:		Micro	:
S. Creatinine	:		C & S	:
S. Uric Acid	:			
VDRL	:		Stool: OVA:	
B1. Group and Type	:		Cysts	:
Ultra Sound	:			
Early Scan	:		Serial Scan	:

Presentation			
BPD			
AC			
FL			
HC/AC			
EF.Wt			
CGA			
Placental Position & Grade			
AFI			
Cong. Anomalies			

Provisional Diagnosis :

Colour Doppler :

Umbilical artery PI :

Middle cerebral artery PI :

Ductus venosus PI :

Impression :

Mode of Delivery :

Apgar score :

Birth Weight :

Placental weight and abnormalities:

Neonatal Complications:

BIBLIOGRAPHY

1. Divon and HSU. 1992, Maternal and fetal blood flow velocity waveforms in intrauterine growth retardation. Clin. Obste. Gynecol. 35 : 156, 1992.

2. Warkany et al., 1961. Intrauterine growth retardation. Am. J. Dis. Child, 102 : 24, 1961.

3. Gruenwald, 1963. Chronic fetal distress and placental insufficiency. Biol. Neonate, 5 : 215, 1963.

4. Rajan R. 2001. IUGR, Ultrasound in human reproduction 2nd Edition, 10-23.

5. Lubchenco, et al., 1963. Intrauterine growth as estimated from liveborn birth - weight data at 24 to 42 weeks of gestation Pediatrics, 32 : 793, 1963.

6. Battaglia et al., 1967. A practical classification of newborn infants by weight and gestational age, J. Pediatr. 17 : 159, 1967.

7. Seeds et al., 1998. Impaired fetal growth - Definition and Clinical daignosis Obstet. Gynecol. 64 : 303.

8. Manning et al., 1991. Intrauterine growth retardation. Diagnosis, Prognostication, and Management based on Ultrasound methods. Principles and Practices of Ultrasonography in Obstetrics and Gynaecology, 4th ed. 1991.

9. Gardosi, et al., 1992. Customized antenatal growth charts, Lancet, 339 : 283, 1992.

10. Seeds 1984, Impaired fetal growth. Definition and Clinical Diagnosis. Obstet. Gynecol, 64 : 303, 1984.

11. Usher and McLean - 1969. Intrauterine growth of live born caucasian infants at sea level. J. Pediatr. 74 : 901, 1969.

12. McIntrie et al., 1999. Birthweight in relation to morbidity and mortality among newborn infants. N. Engl. J. Med. 340 : 1234, 1999.

13. Manning 1995. Intrauterine growth retardation. In fetal Medicine Principles and Practice, 1995, p.317.

14. Owen et al., 1997. Fetal size and growth velocity in the prediction of intrapartum cesarean section for fetal distress. Br. J. Obstet. Gynecol. 1904 : 445, 1997.

15. Owen et al., 1998. Fetal growth velocity in the prediction of intrauterine growth restriction in a low risk population. Br. J. Obstet. Gynaecol. 105 : 536, 1998.

16. Paz et al., 1995. Cognitive outcome of full term small - for gestational age infants at late abdolescent. Obstet. Gynecol. 85 : 452, 1995.

17. Piper et al., 1996. Do growth retarded prematare infants have different rates of perinatal mobidity? Obstet. Gynecol. 87 : 169, 1996.

18. Kleigman, 1997. Intrauterine growth retardation. Neonatal Perinatal Medicine, 6th ed. 1997 - p.203.

19. Campbell et al., 1977. Ultrasound measurement of fetal head to abdomen circumference ratio in assessment of growth retardation. Br. J. Obstet. Gynecol. 84 : 165, 1977.

20. NIcolaides et al., 1991. Cordocentesis in the study of growth retarded fetures. In Divon MY (ed) : Abnormal Fetal growth - 1991.

21. Salafia, 1995. Intra - uterine growth restriction in infants of less than 32 weeks gestation. Am. J. Obstet. Gynecol. 173 : 1049, 1995.

22. Dashe et al., 2000. Impact of asymmetric Vs Symmetric fetal growth. SGI abstract 96 : 321, 2000, Williams 2001.

23. Gardosi J et al., 1999. Controlled trial of fundal height measurement plotted on customized antenatal growth charts. Br. J. Obstet. Gynecol. 106 : 309, 1999.

24. Jensen et al., 1991. Evaluation of symphysis fundus measurements and weighing during pregnancy. Acta Obstet. Gynecol. Scand, 70 : 13, 1991.

25. Walraven et al., 1995. Single pre-delivery symphsis - fundal height measurement as a predictor of birth weight. Br. J. Obstet. Gynaecol. 102 : 525, 1995.

26. Jimenez et al., 1983. Clinical measurements of gestational age in normal pregnancies. Obstet. Gynecol. 61 : 438, 1983.

27. Smith et al., 1997. The relationship between fetal abdominal circumference and birthweight - findings in 3512 pregnancies. Br. J. Obstet. Gynaecol. 104 : 186, 1997.

28. Snijders et al., 1994. Fetal Biometry at 14 to 4- weeks' gestation. Ultrasound Obstet. Gynecol. 4 : 34, 1994.

29. Emmanvel 1992. Integenerational studies of human birth weight from the 1958 birth cohort. Br. J. Obstet. Gynaecol. 99 : 67, 1992.

30. Klebanoff, 1997. Preterm and small for gestational - age birth across generation. Am. J. Obstet. Gynecol. 176 : 521, 1997.

31. Brooks et al., 1995. Birthweight : Nature or Nurture? Early Hum Dev. 2 : 29, 1995.

32. Simpson et al., 1975. Influence on prepregnancy weight and pregnancy weight gain on birth weight. Obstet. Gynecol. 45 : 481, 1975.

33. Abrams et al., 1995. Maternal weight gain pattern and birth weight. Obstet. Gynecol. 86 : 163, 1995.

34. Williams, 2001.

35. Klein et al., 1995. Current concepts of infections of the fetus. Infectious Diseases of the Fetus and Newborn Infant, 1995. p.1.

36. Khoury 1988. Congenital Malformations and Intrauterine growth retardation. P. ediatrics, 82 : 83, 1988.

37. Thelander et al., 1966. Abnormal patterns of growth and development in mongolism. Clin. Pediatr. 5 : 493, 1966.

38. Lindor - 1993. Mosaic trisomy 16 in a thriving infant. Clin. Genet. 44 : 185, 1993.

39. Kalousek, 1993. Uniparental disomy for chromosome 16 in humans. Am. J. Hum. Genet. 52 : 8, 1993.

40. Hanson et al., 1996. Teratology Principles and practice of Medical Genetics, 3rd ed. 1996, p.697.

41. Cliver et al., 1995. The effect of cigarette smoking on Anthropometric measurements of neonate. Obstet. Gynecol. 85 : 625, 1995.

42. Xion et al., 1999. Impact of pregnancy induced hypertension on fetal growth. Am. J. Obstet. Gynecol. 180 : 207, 1999.

43. Stettler and Cunningham - 1992. Natural history of chronic proteinuria complicating pregnancy. Am. J. Obstet. Gynecol. 167 : 1219, 1992.

44. Patton et al., 1990. Cyanotic maternal heart disease in pregnancy. Obstet. Gynecol. Surv. 45 : 594, 1990.

45. Duvekot, et al., 1995. Maternal volume homeoostasis in early pregnancy in relation to growth restriction. Obstet. Gynecol. 85 : 361, 1995.

46. Lunell et al., 1992. Uteroplacental blood flow. Clin. Obstet. Gynecol. 35 : 108, 1992.

47. Howard, 1987. Control of human placental blood flow. Med. Hypothesis 23 : 51, 1987.

48. Hill et al., 1994. Sonographic assessment of twin discordancy. Obstet. Gynecol. 84 : 501, 1994.

49. Lockwood and Rand, 1994. Obstetrical consequences of antiphospholipid antibodies. Obstet. Gynecol. Surv. 49 : 432, 1994.

50. Gosling and King, 1975. Ultrasound Angiography - Arterior and Veins, 2nd Edition, p.61 - 98.

51. Sieroszewski P. 2005. Usefulness of uterine artery doppler velocimetry in high risk pregnancy daignosis.

52. Schulman et al., 1986. Umbilical velocity wave ratios in human pregnancy. Am. J. Obstet. Gynecol. 1984, 148 : 985 - 990.

53. Papageorgiou et al., 2002. Have analysed data from 19 studies and have stated the following. J. Maternal Fetal neonatal Med. 2002, Aug : 12(2) : 78 - 88.

54. Chien et al., 2000. How useful is uterine artery Doppler flow velocimetry in the prediction of pre-eclampsia, intrauterine growth retardation and perinatal death? An overview, B. J. Og. 2000; 107 : 196 - 208.

55. Alfirenic and Neilscon - 1995. Doppler ultrasonography in high risk pregnancies. Systematic review with Meta- analysis. Am. J. Obstet. Gynecol. 1995, 172 : 1379 - 1387.

56. Olofsson et al., 1993. A high PI reflects a defective development of placental bed arteries in pregnancies complicated by hypertension. Evr. J. Obstet. Gynecol. Reprod. Biol. 1993, 49 : 161 - 68.

57. Gudmundsson and Marshal, 1991. Umbilical artery blood velocity waveforms in prediction of fetal outcomes - a copmparison. Am. J. Perinatol, 1991C, 8 : 1-6.

58. Wledimiroff et al., 1991. Fetal and umbilical flow velocity waveforms between 10 - 16 weeks gestation. Obstet. Gynecol. 1991 : 78, 812 - 814.

59. Chandran and Colleagues, 1993. Comparison of Doppler and fetal heart testing for predicting hypoxemia at birth.

60. Nicolaides et al., 1990. Relation of rate of urine production to oxygen tension in SGA infants. Am. J. Obstet. Gynecol. 162 : 387, 1990.

61. Al - ghazali et al., 1987. Evidence of redistribution of cardiac output in asymetrical growth retardation. BJ06 : 96 : 697 - 704.

62. Malcus et al., 1991. Umbilical artery velocimetry and non - stress test in monitoring high risk pregnancies. Ultrasound Obstet. Gynecol. 1991b, 1 : 95 - 101.

63. Mari et al., 1992. MCA flow velocity waveforms in normal and SGA fetuses. Am. J. Obstet. Gynocol, 1992, 166 : 1262 - 1270.

64. Arduini et al., 1992. IVC velocities in appropriate and SGA fetuses. Am. J. Obstet. Gynecol. 1992, 166 : 1271 - 1280.

65. Gramellini et al., 1992. Cerebral - umbilical Doppler ratio as a predictor of adverse perinatal outcome. Obstet. Gynecol. 1992, 79 : 416 - 420.

66. Baschat et al., 2000. Relation between arterial and venous doppler and perinatal outcome in FGR. Ultrasound Obstet. Gynecol. October, 2000.

67. Baschat et al., 2001. Arterial and venous doppler in optimising time of intervention in FGR. Ultrasound Obstet. Gynecol. December, 2001.

68. Vyas et al., 1990. MCA waveforms in fetal hypoxaemia. BJOG, 97 : 797 - 803.

69. Newham et al., 1995. Treatment of IUGR. Australian and New Zealand Journal of Obstet. and Gynecol. 35 : 370 - 374.

70. Martin et al., 2002. Final data for 2001. National vital statistics reports. Vol.51, No.1, 2002.

71. Fleischer et al., 1985. Umbilical artery velocity waveforms and intrauterine growth retardation. Am. . Obstet. Gynecol. 1985.

72. Benson et al., Intra uterine growth retardation predictive value of ultrasound criteria for antenatal diagnosis. Radiology, 1986, August. 160(2), 415 - 7.

73. Brodszki et al., 2002. Can the degree of retrograde diastolic flow in abnormal umbilical artery flow velocity waveforms predict pregnancy outcome. Ultrasound in Obstetrics and Gynecology, 19 : 3 : 229 - March 2002.

74. Sterne et al., Abnormal fetal cerebral and umbilical Doppler Measurement in fetuses with IUGR predicts severity of perinatal morbidity. Journal of Clinical Ultrasound Vol.2, Issue 3 page 146 - 151.

75. Trudinger et al., 1988. Low dose aspirin therapy inproves fetal weight in umbilical placental insufficiency. Am. J. Obstet. Gynecol. 1988, 159 : 681 - 685.

76. Gudnunsson et al., 1993. Venous Doppler Velocimetry in fetures with absent end - diastolic blood velocity in the umbilical artery. Journal of Maternal and fetal investigation, 3, 196.

ABBREVIATIONS

IUGR	:	Intrauterine growth restriction
SGA	:	Small for gestational age
CTG	:	Cardio Tocography
NST	:	Non stress test
Umb. Ar	:	Umbilical Artery
MCA	:	Middle cerebral artery
DV	:	Ductus venosus
USG	:	Ultrasonogram
PI	:	Pulsatility index
RI	:	Resistance index
SFH	:	Symphysio - fundal height