"ONE YEAR COHORT STUDY, ROLE OF UMBILICAL ARTERY DOPPLER AND MODIFIED BIOPHYSICAL PROFILE IN PREDICTING NEONATAL OUTCOME IN INTRAUTERINE GROWTH RESTRICTION, A HOSPITAL BASED STUDY"

By

BJ0111006

Dissertation

Submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

MASTER OF SURGERY (M. S.)
in
OBSTETRICS AND GYNAECOLOGY

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

APRIL – 2014

KLE UNIVERSITY, BELGAUM, KARNATAKA

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ABBREVIATIONS

AC	Abdominal circumference
ACOG	American College Of Obstetrics &Gynaecology.
AEDV	Absent end-diastolic flow.
AFI	Amniotic fluid index.
BPP	Biophysical profile.
CTG	Cardiotocography
CST	Contraction stress test
FHR	Fetal heart rate.
FVW	Flow velocity waveform.
IR	Increase resistance
IUGR	Intrauterine growth restriction.
IVH	Intraventricular Haemorrhage.
LSCS	Lower segment cesarean section
MBPP	Modified biophysical profile.
MCA	Middle cerebral artery
NEC	Necrotizing enterocolitis.
NICHHD	National Institute of Child Health and Human Development
NICU	Neonatal intensive care unit.
NST	Non stress test
PEDV	Positive end-diastolic flow
PI	Pulsatility index.

RI	Resistance index
RDS	Respiratory distress syndrome.
REDF	Reversed end-diastolic flow.
S/D	Systolic diastolic ratio.
SGA	Small for gestational age.
UA	Umbilical artery.
VAST	Vibro-acoustic stimulation test.

ABSTRACT

OBJECTIVES

The present study was undertaken to correlate the role of umbilical artery Doppler and Modified biophysical profile in predicting adverse neonatal outcome in intrauterine growth restriction.

METHODS

This one-year cohort study was conducted on a total of 113 patients diagnosed as IUGR (AC < tenth percentile) on sonography beyond 28 weeks of gestation, singleton pregnancies with known LMP and/or 1st trimester scan underwent umbilical artery Doppler and Modified biophysical profile as a part of antepartum fetal testing. We excluded multiple gestation, fetal anomaly, eclampsia, abruption and placenta praevia. Abnormal fetal testing was defined as umbilical artery Doppler with absent or reversed end diastolic flow and Modified biophysical profile as non-reactive NST and/or AFI<5cms. The adverse neonatal outcomes noted were resuscitation at birth (intubation), intra-uterine death, admission to NICU at birth, respiratory distress syndrome, neonatal seizures, necrotizing enterocolitis and early neonatal death.

RESULTS

In this study the mean maternal age was found to be 25.50 ± 5.38 years in women with good neonatal outcome and 24.01 ± 3.75 years in women with adverse neonatal outcome with more than half (52.21%) of the women that presented as primigravida. 19.46% of the women had abnormal Doppler findings and 25.66% MBPP were abnormal findings. The mean gestational age at delivery was 37.36±1.04

in women with good neonatal outcome and 34.30±2.44 in women with adverse neonatal outcome. In this study, vaginal delivery was noted among 38.93% of the women while 61.06% of the women underwent lower segment caesarean section. All 19 women with AEDF (16.81%) and 2 with REDF underwent LSCS. Similarly, of the 29 women with abnormal MBPP, 8 (7.08%) had vaginal delivery and 21 (18.58%) had lower segment caesarean section.

The mean weight of the babies was 2.14±0.5 Kgs in women with good outcome and 1.42±0.35 kgs in women with adverse outcome. 38 (33.6%) babies required NICU admission at birth, of which 31 admissions were for preterm low birth weight and 6 were for term low birth weight. There were 7 early neonatal deaths, of which 6 were due to necrotizing enterocolitis and 1 due to respiratory distress syndrome and 3 intra-uterine fetal deaths had occurred. Abnormal Doppler was significantly associated with NICU admissions with OR 11.3 (95%CI 3.7-34.3), NEC with OR 12.2 (95%CI 3.2-46.8), RDS with OR 4.8 (95%CI 1.1-21.4) and END with OR 8.2 (CI 1.5-44.9).The sensitivity of Doppler in predicting adverse neonatal outcome was 47.72% with 98.55% specificity, 95.45% PPV and 74.72% NPV. The sensitivity of MBPP in predicting adverse neonatal outcome was 38.64% with specificity 82.61%, 58.62% PPV and 67.86% NPV.

CONCLUSION

Modified biophysical profile can be used as an initial surveillance, although it should be coupled with umbilical artery Doppler as it has high specificity and positive predictive value and is a good predictor of adverse neonatal outcomes. Thus, improving the outcome by appropriately timed intervention in the management of IUGR.

Keywords: AEDF, adverse neonatal outcome, IUGR, Modified biophysical profile, REDF, umbilical artery Doppler.

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INTRODUCTION

Pregnancy is a physiological phenomenon for most women. However, some develop problems during its evolution, affecting the maternal and perinatal outcome.[1] Intrauterine growth restriction (IUGR) is a common complication which can develop in any pregnant mother leading to adverse perinatal outcome. It is a fetus that does not reach its growth potential and is a term used to describe the fetus with an abdominal circumference at or below the 10th percentile for the corresponding gestational age.[2,3]

IUGR at birth may be associated with health risks like respiratory distress, risks associated with prematurity, necrotizing enterocolitis, thrombocytopenia, temperature instability, hypoglycemia, renal failure, meconium aspiration, risks associated with congenital malformations or chromosomal anomalies and neonatal death.[4,5,6,7] Therefore, antepartum fetal surveillance would play an important role in the identifying critical risk factors and directing appropriately timed intervention in the management of IUGR.

Various antenatal fetal tests have evolved since many years like fetal movement counting, contraction stress test (CST), non-stress test (NST), biophysical profile (BPP), uterine artery Doppler and umbilical artery (UA) Doppler. The only antenatal surveillance technique recommended for all pregnant women, with and without risk factors, is fetal movement counting, although the quest to define a quantitative "alarm limit" to define decreased fetal movements has so far been unsuccessful.[8,9] CST was used for its high negative predictive value but has certain limitations like being invasive, time consuming and has a poor positive predictive value with false-positive results in 30% of cases.[10,11] The NST remains an important

component of antepartum care, although current information would suggest that it should not be considered as a "stand-alone" test.[12] Keane et al studied that the NST showed high specificity (81%) but low sensitivity (33%) and false negative rates of 1%.[13] The implications of such results should be supported by other clinical data before obstetric intervention is undertaken. Non-reactive NSTs have about a 55% false positive rate (i.e., a backup test is normal).[14] This has led to its gradual replacement by either complete BPP testing or the modified version of original BPP that was devised, as it was time consuming. A number of investigators have explored the use of uterine artery Doppler for third trimester fetal assessment among women with complicated pregnancies (32-34), but its role in this setting has not been clearly defined. The only testing modality for which there is Level I evidence for its use in the surveillance of IUGR fetuses is UA Doppler due to its high specificity and predictive values.[2,15] The UA Doppler provides early evidence of circulatory abnormalities in the fetus compared to NST evident by the lead time of 5.86 days.[16] But no single testing modality is superior as each has its merits and demerits.[2] Hence, the antepartum fetal testing must be combined to give better results.

In normal pregnancies, UA resistance shows a continuous decline, however, this may not occur in fetuses with utero-placental insufficiency. The most commonly used measure of gestational age specific UA resistance is the systolic-to-diastolic ratio of flow, which changes from a baseline value to an elevated value with worsening of the condition. As the insufficiency progresses, end-diastolic velocity is lost and finally reversed. The clinical significance of this progression has been well documented by Mandruzzato et al, who reported a significant difference in mean birth weight and perinatal mortality for AEDF20% versus REDF68%.[17]

A variety of modifications of the original BPP have been evaluated in an attempt to simplify and reduce the time necessary to complete testing by focusing on the components of the BPP that are most predictive of perinatal outcome. Eden et al have proposed a "modified biophysical profile" by which a reactive NST (with the option of acoustic stimulation), acts as a short-term indicator of fetal acid-base status and along with the AFI as an indicator of long-term placental function.[18] This method has clinical practicalities because the NST data can be obtained in a setting that does not require constant supervision by a technician and the time needed to determine the AFI daily is minimal. Further, the NST component is often done on a more frequent schedule than the AFI determination. A trial by Miller of 56,617 has demonstrated comparable results of the MBPP to the original BPP, namely a false-negative rate (or rate of fetal death within 1 week of a normal MBPP) of 0.8 per 1000.[19]

There must be an identifiable early stage with a sufficiently long latent phase to allow intervention, which will change the outcome. The employed tests should be simple, precise and applicable to the target population.

Doppler and MBPP are the principal antenatal testing modalities. However, the prognostic and diagnostic information gained in each testing modality is in great part independent of each other which makes the diagnosis controversial and question arises that, whether these modalities might be better once combined, or separated. Hence the present study was undertaken to correlate the role of UA Doppler and Modified Biophysical Profile in predicting adverse neonatal outcome in IUGR.

OBJECTIVES

The objective of the present study was;

To correlate the role of umbilical artery Doppler and Modified Biophysical Profile in predicting adverse neonatal outcome in growth restricted fetus.

REVIEW OF LITERATURE

INTRAUTERINE GROWTH RESTRICTION

The term **"intrauterine growth restriction" (IUGR)** is a fetus that fails to reach its growth potential.[2]Intrauterine growth restriction is defined on the basis of abdominal circumference below the 10th percentile for the corresponding gestational age .[2,3]In our study we chose to use the mediscan table over hadlock's table to diagnose IUGR, as a study by Dr. S.Suresh in Chennai in 2010, where he studied 2000 fetuses and derived population based normograms in south Indian population and devised mediscan software. This software avoided over diagnosis of IUGR.[20]

IUGR can complicate 10% to 15% of all pregnancies.[21] India alone accounts for 43.6% of IUGR in the developing world which is more than half of those in Asia.[22] In India, the disparity has ranged from a prevalence of 10% (for the privileged high socio-economic class) to 56% (for the poor urban slum community). IUGR fetuses have 4-8 times increase risk of mortality when compared to non-IUGR fetuses.[23] Fetal and infant morbidity and mortality is sharply increased when the birth weight is less than 5th centile.[6]Perinatal mortality rates for fetuses and neonates weighing less than the 10th percentile, but between 1500 and 2500 g, were 5 to 30 times greater than those of newborns between the 10th and 90th percentiles but for those weighing less than 1500 g, the rates were 70 to 100 times greater.[24]Manning showed that perinatal morbidity and mortality increase if birth weights are below the 10th percentile, and markedly so if below the 6th percentile.[25]Therefore, it becomes

very essential to have antepartum fetal testing as an integral part in the management of IUGR so that timely intervention can be done.

Methods of antepartum fetal surveillance for IUGR patients

The objectives of antepartum surveillance in patients with IUGR are:

- To determine the gestational age and any congenital anomaly in the fetus.
- To detect the abnormalities in fetal growth.
- To detect and determine the severity of acute and chronic fetal asphyxia.
- To confirm the well being of the normal fetus thereby preventing unnecessary intervention.
- To improve the perinatal outcome through the timely diagnosis and treatment of fetal compromise.

The methods available for the antepartum surveillance in IUGR can be broadly classified into two groups namely, non-invasive and invasive

Non invasive methods

<u>Subjective assessment</u>

Daily fetal movement counts (Sadovsky)/ Cardiff count to 10.

<u>Objective assessment</u>

- Biophysical profile i.e.; BPP
- Non-stress test i.e.; NST
- Vibro-acoustic stimulation test i.e.; VAST
- Modified Biophysical profile i.e.; MBPP
- Uterine artery Doppler
- Umbilical artery Doppler

Invasive methods

- Contraction stress test (CST)

Daily fetal kick count

Sadovsky observed that in placental insufficiency, where the fetus dies, fetal movements decreased and stopped 12-48 hrs before the fetal heart stopped.[26] He called it the "movement alarm signal" and suggested that when this signal became manifest, immediate delivery could save the life of the baby. Although several fetal movement counting protocols have been used, neither the optimal number of movements nor the ideal duration for counting them has been defined. Women who report decreased fetal movements (< 6 distinct movements within 2 hours) should have a complete evaluation of maternal and fetal status, including NST and/or BPP.

Contraction stress test

It assesses the fetal heart rate to the stress of stimulated labour and was the primary method of testing in the first half of 1970's, although it has several limitations like length of time required to perform the test is more, need for intravenous access, high incidence of suspicious / equivocal results, high false positive results and is contraindicated in several clinical settings like preterm labour, placenta previa, vasa previa, cervical incompetence, multiple gestation and previous classical caesarean section.

Non-stress test (NST)

Freeman and Lee (1975) observed fetal heart rate accelerations in association with fetal movement, which virtually precluded a subsequent normal CST.[27] NST with false negative rates of 1.9-5/1000 women tested though higher than that of CST,

but when performed twice weekly showed improved performance with fall in false negative rates to 1 % or less.

Freeman and Colleagues (1975) introduced the NST to describe fetal heart rate acceleration in response to fetal movement as a sign of fetal health. By the end of 1970's, the CST was substituted by the NST as the primary method of testing fetal health. The NST was easier to perform and normal results were used to further discriminate false-positive CST. Simplistically, the NST is primarily a test of fetal condition and it differs from the CST, which is a test of utero-placental function. In 1997, the National Institute of Child Health and Human development Research Planning Workshop (NICHHD) in partnership with ACOG proposed definitions for fetal heart rate (FHR) patterns to interpret the readings.[28]

The definition of reactive NST currently recommended by ACOG (2002) is 2 or more accelerations and occurring within 20 minutes of beginning the test. It has also recommended that accelerations with or without fetal movements be accepted.[2]A non-reactive NST lacks sufficient fetal heart rate accelerations over a 40 minute period.[2] Antenatal fetal testing with NST can be started at 28 weeks with 15% incidence of non-reactive NST.[2] NST should be done daily in case of IUGR.[29]

A study done in India,to compare the efficacy of Doppler vascular technic over NST in predicting fetal compromise in utero in cases of severe preeclampsia and IUGR showed that Doppler is useful in recognizing fetal compromise earlier than NST giving a lead time (4-14days) which is important in the management of preterm high risk pregnancies. An abnormal NST following an abnormal Doppler is associated with the worst perinatal outcome. [30]

Amniotic Fluid Index (AFI)

Oligohydramnios i.e. the 5th percentile is present when the amniotic fluid volume is approximately 500ml. Oligohydramnios may be a sign of poor placental function. Because fetal urinary flow is determined in part by the state of fetal hydration, which is in turn determined by placental function. When Cruz and associates compared Doppler studies in patients with oligohydramnios and in healthy pregnant women, those with decreased amniotic fluid had substantially higher flow resistance in both maternal uterine and fetal umbilical arteries suggesting that decreased amniotic fluid is a frequent sign of inadequate placental perfusion.[31] In post-term pregnancies at risk for placental insufficiency, Tongsong and Srisomboon noted that amniotic fluid volume was considerably more accurate in predicting intrapartum fetal distress than was the NST with sensitivity of 73%, specificity of 91 % and positive predictive value of 27%.[32]

AFI described by Phelan and Co-workers (1987) involves adding the vertical depths of the largest pocket in each of four equal uterine quadrants in 330 patients of at risk pregnancies, patients with AFI < 5cm had considerably higher rates of abnormal fetal heart rate testing, meconium passage and caesarian delivery for fetal distress.[33]

AFI IN PREDICTION OF POOR PERINATAL OUTCOME

Perinatal Mortality:

Chamberlain and associates reviewed charts of 7,562 high-risk obstetrical patients referred for Biophysical profile. The corrected perinatal mortality rate for patients with qualitatively normal amniotic fluid was 1.97 in 1000 compared with

4.12 in 1000 when amniotic fluid volume was increased. These investigators also reported a 13-fold increase in perinatal mortality rate (56.5/1000) when amniotic fluid volume was marginal by sonographic assessment and a 47-fold increase (187.5/1000) if severe oligohydramnios was present. [34]

Maximum vertical pocket

This technique involves, selecting the single deepest uninterrupted pocket of amniotic fluid and measuring its depth. Although easy to perform, and reasonably reproducible, the criteria for "Normal" have not been rigorously established. Manning et. al. proposed that oligohydramnios be defined as the absence of any amniotic fluid pocket of at least 1 cm. deep (" 1 cm. rule") and polyhydramnios as any pocket larger than 8 cm.[35]But others investigators have found the "1 cm Rule" poorly predictive, though it was highly predictive of (89% sensitivity) IUGR in the study by Manning and associates.[36]Bottoms and associates noted that the absence of a fluid pocket of at least 1 cm deep was exceedingly rare and may be too restrictive a criterion for oligohydramnios.[37]

Biophysical profile

Manning and colleagues (1980) proposed the combined use of 5 fetal biophysical variables as a more accurate means of assessing fetal health than any single variable alone. They concluded that consideration of 5 variables could significantly reduce both false positive and false negative rates. Required equipment included a real time sonography device with B mode display and Doppler USG to record FHR. Typically these tests require 30-60 minutes of examiner's time. The five components of the biophysical profile are as follows: (1) NST (2) fetal breathing

movements (one or more episodes of rhythmic fetal breathing movements of 30 seconds or more within 30 minutes); (3) fetal movement (three or more discrete body or limb movements within 30 minutes); (4) fetal tone (one or more episodes of extension of a fetal extremity with return to flexion, or opening or closing of a hand; and (5) determination of the amniotic fluid volume (a single vertical pocket of amniotic fluid exceeding 2 cm is considered evidence of adequate amniotic fluid). Each of the components is given a score of 2 (normal or present as defined previously) or 0 (abnormal, absent or insufficient). A composite score of 8 or 10 is normal, a score of 6 is equivocal and a score of 4 or less is abnormal.[38]

Modified Biophysical profile

During the late second or third trimester, amniotic fluid reflects fetal urine production. Placental dysfunction may cause diminished fetal renal perfusion, which can lead to oligohydramnios. Therefore, assessment of amniotic fluid volume can be used to evaluate long-term utero-placental function. This led to the development of the MBPP.[38] The MBPP combines the NST with the AFI, which is the sum of measurements of the deepest cord free amniotic fluid pocket in each of the abdominal quadrants, as an indicator of long-term function of the placenta. An AFI of more than 5 cm is thought to be an adequate volume of amniotic fluid. The MBPP is considered normal if the NST is reactive and the AFI is greater than 5 cm and abnormal if the NST is nonreactive or the AFI is 5 cm or less.[38]

A study was performed to compare diagnostic value of the original BPP to the MBPP. A total of 200 patients were enrolled into the study; 104 pregnancies were managed by the original BPP and 96 pregnancies by the MBPP. There were 30 abnormal (31.3%) in MBPP and 24 (23.1%) abnormal tests in original BPP. There

was significant difference in the incidence of meconium passage between two groups. It was concluded that the original BPP is more costly and time consuming than modified MBPP.[39]

A cross-sectional and prospective analytical study that was performed in Kosar hospital of Qazvin in 2004, 600 high risk pregnant women with gestational age >32 weeks were chosen. For any patient both the test were performed and then sensitivity, specificity, positive and negative predictive value were compared together and then analyzed with statistics. Sensitivity in original BPP was 98.5% and in MBPP was 98.4%. Specificity in original BPP was 82.6% and in MBPP was 81.2%. Positive predictive value in original BPP was 99.7% and in MBPP was 99.7%. MBPP test can be replaced for assessment of fetus health and outcome of pregnancy.[40]

Umbilical artery Doppler changes in IUGR

The use of Doppler ultrasound for the evaluation of the fetal circulation is based on the physical principle of change in frequency of a sound wave when it is reflected by a moving object which was first described in 1842 by the Austrian physicist and mathematician Johann Christian Doppler. Blood velocity and resistance to flow can be evaluated using the Doppler effect-a method that has significantly impacted the evaluation and management of the fetus at risk of hypoxia secondary to placental insufficiency.

The role of umbilical and middle cerebral artery (MCA) Doppler in the evaluation of fetuses at high risk for poor outcomes has been adequately assessed in randomized control trials and the method has been found to be useful in

complementing NST or BPP to determine more precisely the degree of fetal compromise.

In chronic placental insufficiency, which is the most common cause for IUGR, a substantial increase in the vascular resistance of the feto-placental unit leads to a decrease in end-diastolic flow velocity or its absence in the flow velocity waveform (FVW). Initial studies have demonstrated a relationship between abnormal flow velocity wave forms and decrease in the number of small stem villi , irregular branching of distal villous tree, or reduced vascularization or mal-development of intermediate and tertiary villi.[41,42,43,44] These changes deteriorate the trans-placental oxygen transport and lead to IUGR.

The association of abnormal UA FVW and fetal hypoxemia or acidemia in IUGR fetuses has been documented in studies utilizing cordocentesis.[45] This is further supported by the finding of a significant increase in the nucleated red blood cell counts of neonates in whom abnormal UA FVW with or without signs of redistribution and IUGR were diagnosed prenatally.[46,47,48] The appearance of a reversed end-diastolic flow velocity is the final step in the cascade of events that may lead to intrauterine fetal death. As a consequence of the placental insufficiency, in response to prolonged fetal hypoxic stress, circulatory adaptation occurs, the fetus shifts its blood flow to the vital organs such as brain, heart and adrenal glands. The increased blood flow to the brain is called “Brain Sparing Effect”.[48] Brain sparing effect leads to cerebral vasodilatation leading to increased diastolic flow which is seen as decreased PI in MCA Doppler velocimetry.

There are several methods of analyzing the UA waveforms to provide clinician with a quantitative index of vascular index that is systolic to diastolic ratio

(S/D ratio), pulsatility index (PI), resistance index (RI). In normal pregnancy, these three indices : S/D, PI and RI decrease with advancing gestation in umbilical artery(UA). But in IUGR first there is decreased diastolic flow in UA due to increase in the resistance that occurs in small arteries and arterioles of the tertiary villi. This raises the S/D ratio, Pl and Rl of UA. As the placental insufficiency worsens, the diastolic flow decreases, then become absent, and later reverses. Some fetuses have decreased diastolic velocity that remains constant with advancing gestation and never become absent or reversed which may be due to a milder form of placental insufficiency. In a high–risk population, the use of UA Doppler has been shown to reduce perinatal morbidity and mortality. UA Doppler should be the primary surveillance tool in the IUGR fetus.[3]When UA Doppler flow indices is normal it is reasonable to repeat surveillance every 14 days.[3]

A systematic review of 104 observational studies of accuracy, involving 19, 191 fetuses, found that UA Doppler predicted compromise of fetal/neonatal wellbeing with a pooled LR+ of 3.41 (95% CI 2.68–4.34) and LR– 0.55 (95% CI 0.48–0.62).The technique predicted fetal death (LR+ 4.37, 95% CI 0.88–21.8; LR– 0.25, 95% CI 0.07–0.91) and acidosis (LR+ 2.75, 95% CI 1.48–5.11; LR– 0.58, 0.36–0.94).[49] Bhatt et al reported that abnormal UA flow velocimetry in growth restricted fetuses was a predictor of high risk for necrotizing enterocolitis.[50]

Summary of randomized and quasi-randomized studies indicates that, among high-risk pregnancies with suspected IUGR, the use of UA Doppler assessment significantly decreases the likelihood of labor induction, cesarean delivery, and perinatal deaths (1.2% vs 1.7%; relative risk, 0.71; 95% confidence interval, 0.52–0.98). Antepartum surveillance with UA Doppler should be started when the fetus is

viable and IUGR is suspected. Although Doppler studies of the ductus venous, MCA, and other vessels have some prognostic value for IUGR fetuses, currently there is a lack of randomized trials showing benefit. Thus, Doppler studies of vessels other than the UA, as part of assessment of fetal well-being in pregnancies complicated by IUGR, should be reserved for research protocols.[51]

In a prospective observational study, 100 pregnant women with IUGR confirmed by ultrasound were evaluated by UA Doppler velocimetry after 28 weeks of gestation. Outcome of the pregnancy was recorded for the normal Doppler group (n=54) and abnormal Doppler group (n=46). Abnormal Doppler group consisted of low end diastolic flow group (n=29) and AEDF or REDF group (n=17). Fetuses with abnormal umbilical flow velocimetry had higher incidence of oligohydramnios and abnormal NST compared to the fetuses with normal umbilical flow. The average birth weight and gestational age at delivery were lower in the abnormal Doppler group. Neonates with abnormal UA velocimetry had increased incidence of caesarean delivery, low apgar scores at birth, increased NICU admissions, increased requirement of positive pressure ventilation, and higher perinatal morbidity and mortality.[52]

In a study of outcome of Doppler velocimetry of the UA in three groups of Pregnancies: Those with the positive end diastolic flow (PEDF: n= 214), AEDF: n = 178 and REDF: n = 67, pregnancies complicated by IUGR had high risk of developing absent or reversed end diastolic FVW (odd ratios 3.1). Pregnancies complicated by both IUGR and hypertension had an even higher risk (OR= 7.4). The over all perinatal mortality rate was 28%. Significantly more neonates in the AEDF

flow group needed admittance to the NICU, PEDF group 60% AEDF group 96%, REDF Group 98%.[17]

In a Retrospective study of Doppler velocimetry of 578 singleton pregnancies with diagnosis of IUGR, four subsets were formed: normal UAPI; 334 fetuses; abnormal UAPI; 137 fetuses; AEDF; 70 fetuses; REDF; 37 fetuses. Fetal biometry, amniotic fluid and fetal-maternal Doppler velocimetry were evaluated in all patients, with BPP and routine NST, when indicated. The following outcomes were examined: mean gestational age at delivery, number of preterm deliveries (< 34 weeks), mean neonatal weight, Apgar score at 5 min < 7, prenatal and neonatal deaths (within the first 28 days of life), admission to the NICU and number of days spent after birth in hospital. Neonatal morbidity was analyzed, including respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH, grade 2-3), necrotizing enterocolitis (NEC) and retinopathy of prematurity. They concluded that a strict correlation exists between abnormal umbilical Doppler velocimetry and an increased incidence of perinatal complications in IUGR fetuses.[53]

In a study AEDF or REDF (Doppler II/III) in UA was correlated with poor perinatal outcome, particularly in IUGR fetuses. They also studied the short- and long-term morbidity and mortality among these children. Sixty-nine IUGR fetuses with umbilical Doppler II/III were divided into three groups; Group 1, severe early IUGR, no therapeutic intervention (n = 7); Group 2, fetuses with pathological BPP, immediate delivery (n = 35); Group 3, fetuses for which expectant management had been decided (n = 27). There results were, in Group 1, stillbirth was observed after a mean delay of 6.3 days. Group 2 delivered at an average of 31.6 weeks and two died in the neonatal period (6%). In Group 3 after a mean delay of 8 days, average

gestational age at delivery was 31.7 weeks; two intrauterine and four perinatal deaths were observed (22%). Long-term follow up revealed no sequelae in 25/31 (81%) and 15/18 (83%), and major handicap occurred in 1 (3%) and 2 patients (11%), respectively, for Groups 2 and 3. They concluded that fetal mortality was observed in 22% of this high risk group. After a mean period of follow-up of 5 years, 82% of infants showed no sequelae. According to their management protocols of IUGR associated with UA Doppler II or III does not show any benefit from an expectant management in term of long-term morbidity.[54]

In a study, 70 pregnant women with IUGR confirmed by ultrasound were followed up with UA Doppler. The study group consisted of 35 women, where the Doppler waveform in the UA was compromised (either AEDF or REDF). These were compared with an equal number of controls, where growth- restricted fetuses had normal Doppler waveforms. Outcome measures were evaluated in both groups and analyzed. The periods of gestation at delivery were 27.2 +/- 3.5 weeks in group 1 and 37 +/- 3.3 Weeks in-group II, respectively. Perinatal morbidity and mortality was significantly increased in the group with compromised UA blood group. Birth weight in group I was 742 +/- 126 grams and in group II was 680 +/- 259 grams. This difference was statistically significant (P=0.0001). In comparison to AEDF, REDF fetuses had more morbidity. Perinatal mortality was also significantly increased in this group (P=0.001). They concluded that UA Doppler should be used in the management of IUGR. In those fetuses in normal Doppler, pregnancy can be prolonged. REDF is an indication for termination of pregnancy.[55]

METHODOLOGY

This one year cohort study was conducted in the Department of Obstetrics and Gynecology, KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Study duration and period

The present one year study was conducted during the period from March 2012 to February 2013.

Place

The present study was conducted at Department of Obstetrics and Gynaecology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum a tertiary care hospital attached to Jawaharlal Nehru Medical College, Belgaum.

Source of data

Cases that were diagnosed as IUGR on sonography were enrolled in the study.

Sample size

A total of 113 women diagnosed as IUGR on sonography were studied.

Sampling procedure

The sample size was calculated considering the prevalence based on the formula as below.

n = $4a^2pq/d2$

Where,

a= 1.96

p = Sensitivity of Doppler (64%)

q = 100-P (100-64=36)

d = Error (10%)

Based on this formula the sample size was calculated as 95. However during the study period 113 women fulfilled the selection criteria and hence were enrolled in the study.

Selection criteria

Inclusion

- All IUGR cases with;
  a. Singleton pregnancy
  b. Gestational age beyond 28 weeks
  c. Known LMP or 1st trimester USG

Exclusion

- Multiple gestation
- Fetal anomaly
- Eclampsia
- Abruptio
- Placenta Praevia

Ethical clearance

Prior to the commencement ethical clearance was obtained from the Institutional Ethical committee, Jawaharlal Nehru Medical College, Belgaum.

Informed Consent

Women fulfilling selection criteria were explained about the nature of the study and a written informed consent was obtained (Annexure I) prior to the enrolment.

Method of collection of data

All antenatal women beyond 28 weeks of gestation attending KLES hospital with known LMP and/or 1st trimester scan, diagnosed as IUGR on sonography were enrolled in the study. In this study IUGR was defined as abdominal circumference less than 10th centile for the corresponding gestational age on mediscan software.[2,3,20] After confirming IUGR on ultrasonography they were explained regarding the study and consent was taken for the same. Patients were then admitted in the wards and subjected to the two ante-partum fetal testing modalities i.e. umbilical artery Doppler and Modified biophysical profile (AFI & NST).

These cases were daily subjected to NST and interpreted as reactive or non-reactive. Umbilical artery Doppler and AFI were coupled together while performing ultrasonography. If AFI<5cms, in term gestation, the pregnancy was terminated and the preterm cases were closely monitored on biweekly basis coupled with daily NST and UA Doppler. Doppler was done on weekly basis if the report was normal and pregnancy was terminated at 37 weeks of gestation by induction of labour. Induction

was performed with 0.5mg dinoprostone every 6th hourly followed by 25ug misoprostol every 4th hourly. During intra-partum monitoring if there was fetal distress, non-progress of labour or failed induction patients were taken up for LSCS. If Doppler showed increased resistance then cases were monitored biweekly and pregnancy was prolonged upto 37 weeks and labour was induced. If Doppler changes showed AEDF, then the pregnancy was immediately terminated by induction of labour, provided the bishop score was favourable with low threshold for LSCS. If Doppler changes showed REDF then pregnancy was terminated immediately by LSCS. Thereafter the neonatal outcome was determined. Neonatal outcome was further categorized into good neonatal outcome and adverse neonatal outcome. Good neonatal outcome defined as delivery without any neonatal complications. Neonatal outcomes noted were NICU admissions at birth, neonatal death and intra-uterine death. Other adverse neonatal outcomes noted were resuscitation at birth (intubation), admission to NICU at birth, respiratory distress syndrome, neonatal seizures, necrotizing enterocolitis and early neonatal death. All babies in NICU were followed up until discharge.The findings were recorded on a proforma (Annexure II).

Investigations

The selected women underwent Antepartum fetal surveillance i.e

- Umbilical artery Doppler
- Modified biophysical Profile (NST and AFI)

Umbilical artery Doppler

The Sonography and Doppler was done by 2 sonologist specialised in obstetric ultrasonography to minimize the inter-observer and intra-observer variability. Sonograms and Doppler waveforms were performed by using 3.5MHz Philips HD 11 (Koninklijke Philips Electronics N.V, Bothell, WA, USA).

The umbilical artery waveforms were obtained in one of the many loops floating in the amniotic fluid.[29] The systolic/diastolic ratio, resistance index and pulsatility index recordings were obtained. All these ratios are independent of the angle of isonation.[29] Umbilical artery Doppler was done once a week if normal and twice a week if increased resistance until 37 week.

Modified biophysical Profile

AFI

AFI was obtained by measuring the deepest pocket of all four quadrants on sonography and then adding their values together. [2,3,56] AFI was defined abnormal if less than 5cms. AFI was repeated weekly if normal and biweekly if abnormal.[29]

Non stress test

NST was taken on the Philips Avalon FM 20 machine by making the patient lie in left lateral position. One external transducer was placed on the abdomen for the fetal heart rate with a help of a belt. A 20mins trace was taken out with paper speed at 1cm/min. Reactive NST defined as when there are at least 2 movements over a period of 20mins, each associated with an acceleration of at least 15bpm lasting for at least 15secs.[28] Non-reactive NST defined as presence of less than two fetal heart rate

accelerations within a 20-minute period over a 40-minute testing period.[28] Daily NST was done for all these cases from 28 weeks onwards.[2,29]

Outcome variables

Intrauterine growth restriction

IUGR cases defined as abdominal circumference less than 10 percentile for the corresponding gestational age on mediscan software by Suresh S et al.[2,3,20]

Abnormal fetal testing was defined as case with;

- Umbilical artery Doppler with AEDF or REDF.
- Abnormal MBPP: Non-reactive NST and/or AFI<5cms

Neonatal outcome was further categorized into good perinatal outcome and adverse perinatal outcome.

Adverse perinatal outcome defined as:

a. Resuscitation at birth (intubation),

b. Admission to NICU at birth,

c. Respiratory distress syndrome,

d. Neonatal seizures,

e. Necrotizing enterocolitis,

f. Early neonatal death

g. Intra-uterine death

Good perinatal outcome defined as delivery without any neonatal complications.

Statistical analysis

The data obtained was coded and entered into Microsoft Excel Worksheet (Annexure III). The categorical data was expressed as rates, ratios and proportions and continuous data was expressed as mean ± standard deviation (SD). The accuracy of Doppler and MBPP in predicting adverse neonatal outcome was determined by sensitivity, specificity, positive predictive value and negative predictive value.

RESULTS

This one year prospective cohort study was carried out in the Department of Obstetric and Gynaecology, KLES, Dr. PrabhakarKore Hospital and Medical Research Centre, Belgaum.

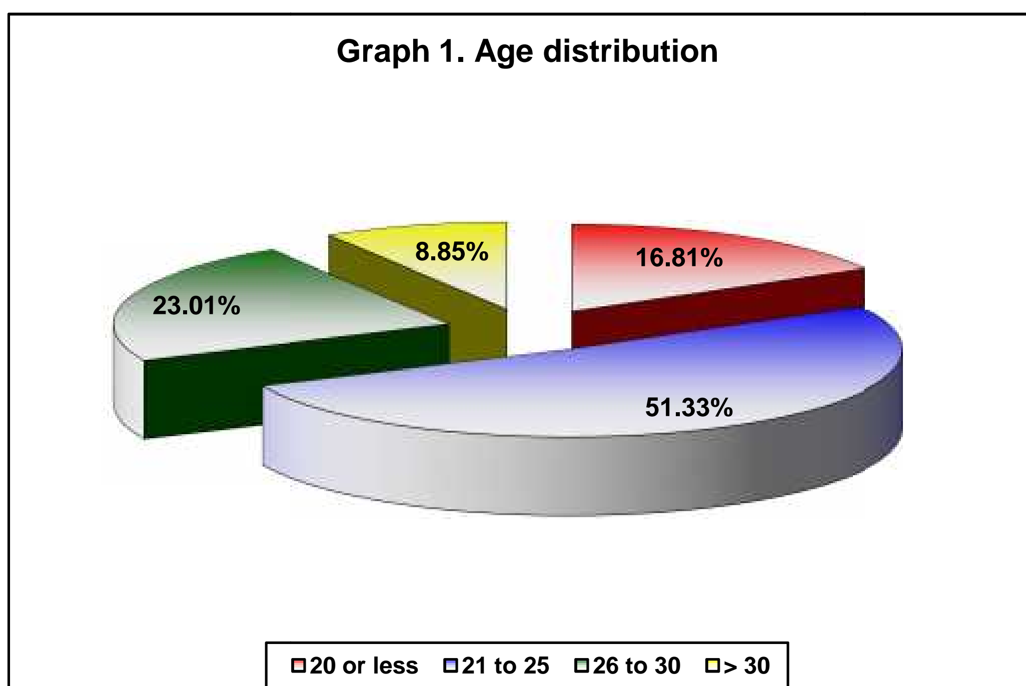
All pregnant women attending antenatal OPD were subjected to ultrasonography. 123 cases of these pregnant women had fetal abdominal circumference below 10th percentile and were labeled as confirmed IUGR. Out of the 123 cases, 10 cases (8.13%) were lost to follow-up as they did not deliver in our hospital.

The data obtained was coded and the master chart was prepared (Annexure III). The data was analyzed and the final results and observations were tabulated as below.

I. MATERNAL CHARACTERISTICS

Table 1. Age distribution

Age group (Years)	Distribution (n=113)	
	Number	**Percent**
20 or less	19	16.81
21 to 25	58	51.33
26 to 30	26	23.01
> 30	10	8.85
Total	**113**	**100.00**

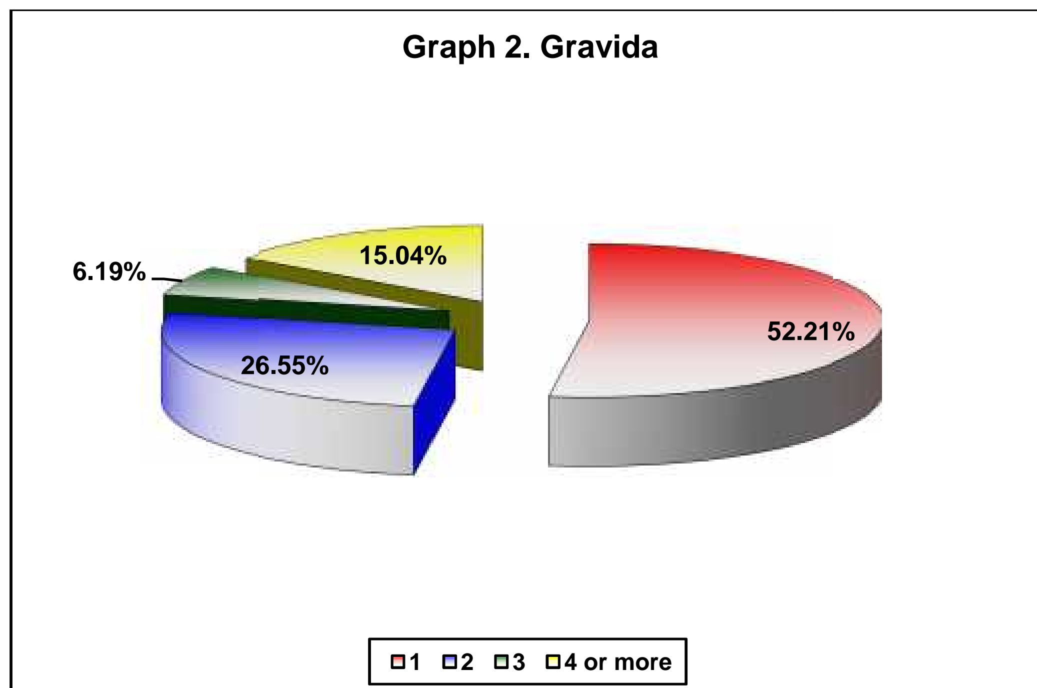


Graph 1. Age distribution

In this study 51.33% of the women were aged between 21 to 25 years. The next commonest group was 26 to 30 years with 23.01% of the women. However, 16.81% and 8.85% of the women had age 20 and 30 years respectively.

Table 2. Gravida

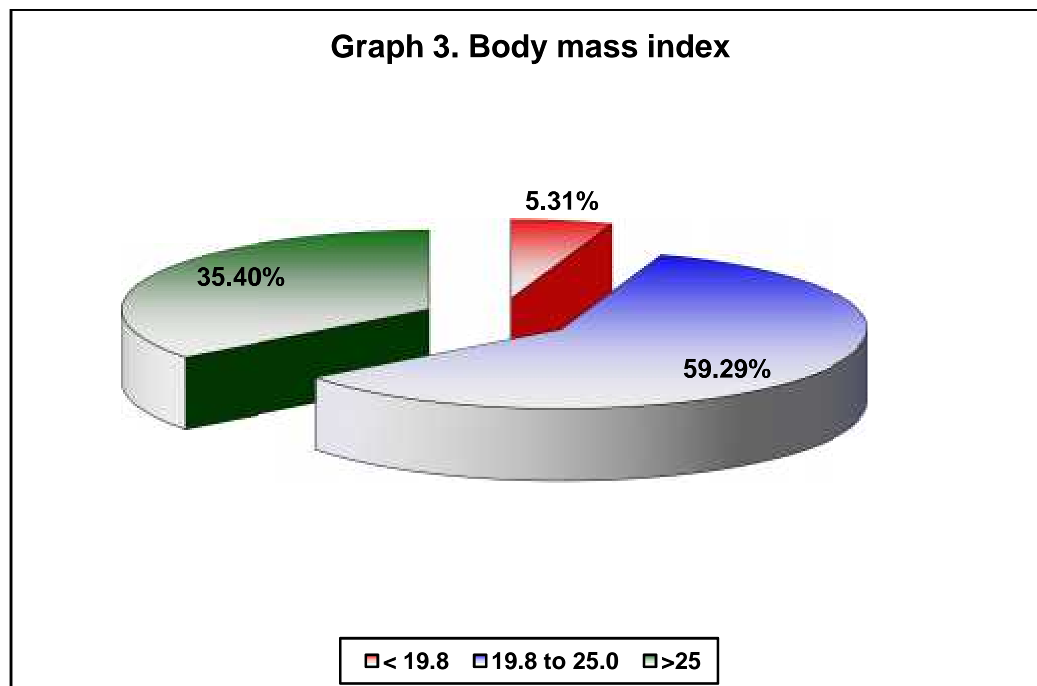
Gravida	Distribution (n=113)	
	Number	Percent
1	59	52.21
2	30	26.55
3	7	6.19
4 or more	17	15.04
Total	**113**	**100.00**



In this study most of the women presented as primigravida (52.21%) and 26.55% with gravida 2. Gravida 3 and 4 or more were noted in 6.19% and 15.04% of the women respectively.

Table 3. Body mass index

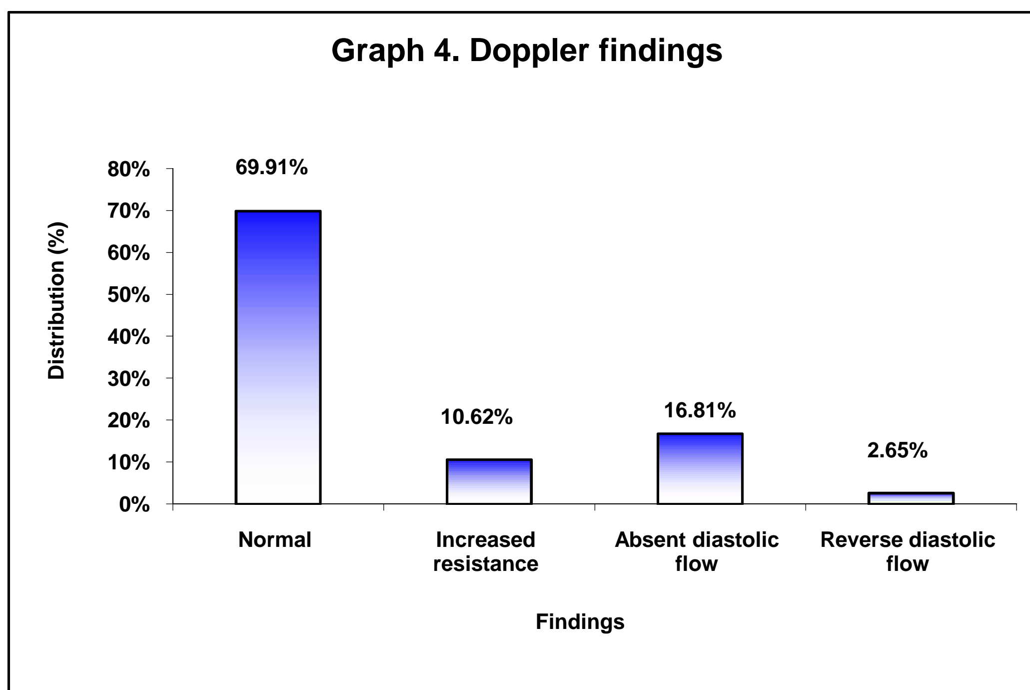
Body mass index (Kg/m^2)	Distribution (n=113)	
	Number	**Percent**
< 19.8	6	5.31
19.8 to 25.0	67	59.29
> 25	40	35.40
Total	**113**	**100.00**



In the present study 59.29% of the women had BMI between 19.8 to 25.0 Kg/m^2 while 35.40% of the women had BMI of > 25 Kg/m^2 and 5.31% had BMI of < 19.8 Kg/m^2.

Table 4. Doppler findings

Findings	Distribution (n=113)	
	Number	Percent
Normal	79	69.91
Increased resistance	12	10.62
AEDF	19	16.81
REDF	3	2.65
Total	**113**	**100.00**

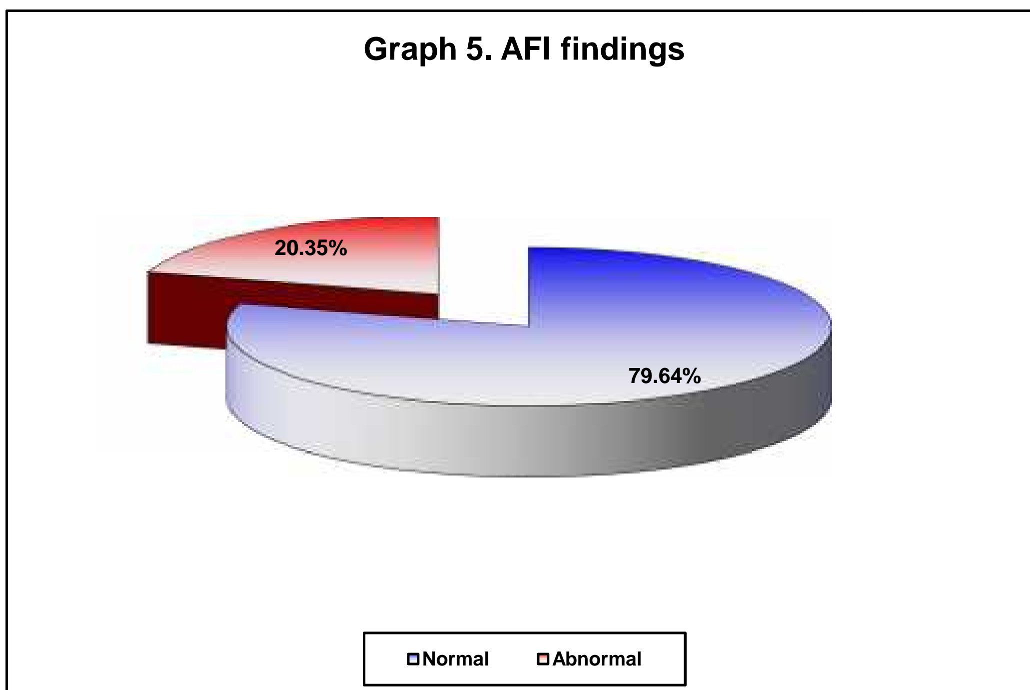


In the present study 19.46% of the women had abnormal Doppler findings while in 80.53% of the women the findings were normal.

Table 5. AFI findings

Findings	Distribution (n=113)	
	Number	Percent
Abnormal	23	20.35
Normal	90	79.64
Total	**113**	**100.00**

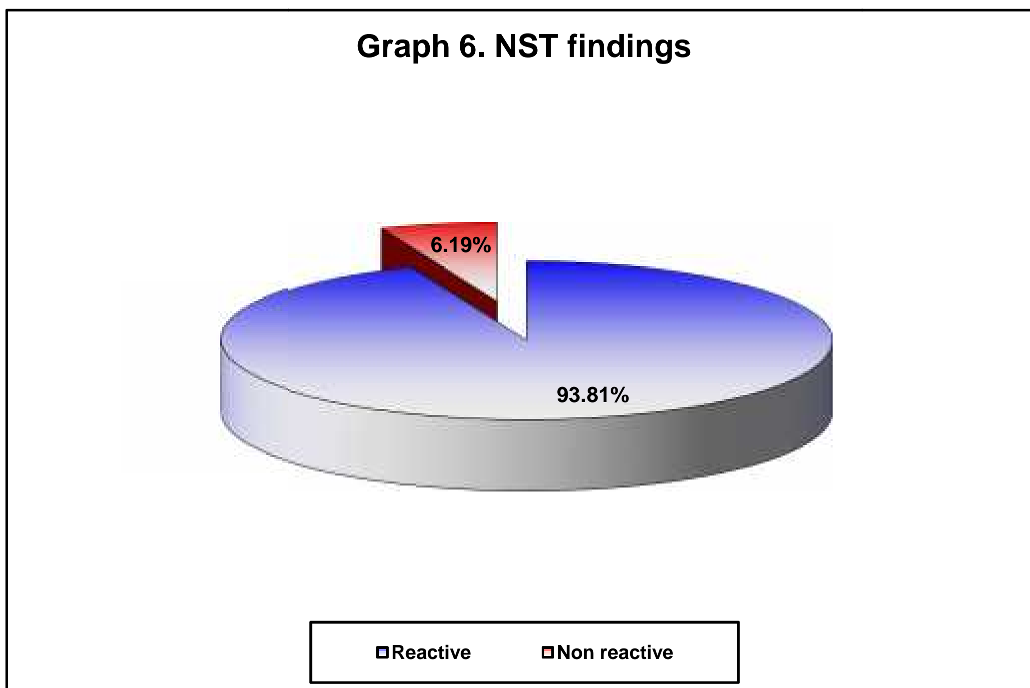
Graph 5. AFI findings



In this study 79.64% of the women had normal AFI findings while 20.35% of the women had abnormal findings.

Table 6. NST findings

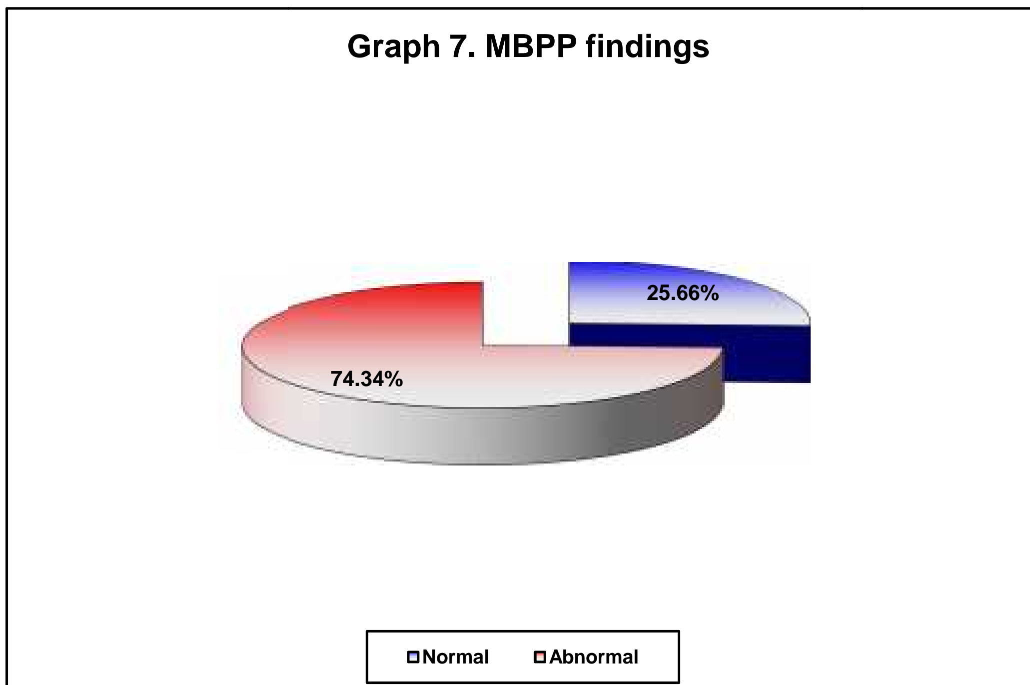
Findings	Distribution (n=113)	
	Number	Percent
Reactive	106	93.81
Non reactive	7	6.19
Total	**113**	**100.00**



In the present study of the 113 cases studied, 7 (6.19%) were non-reactive NST.

Table 7. MBPP findings

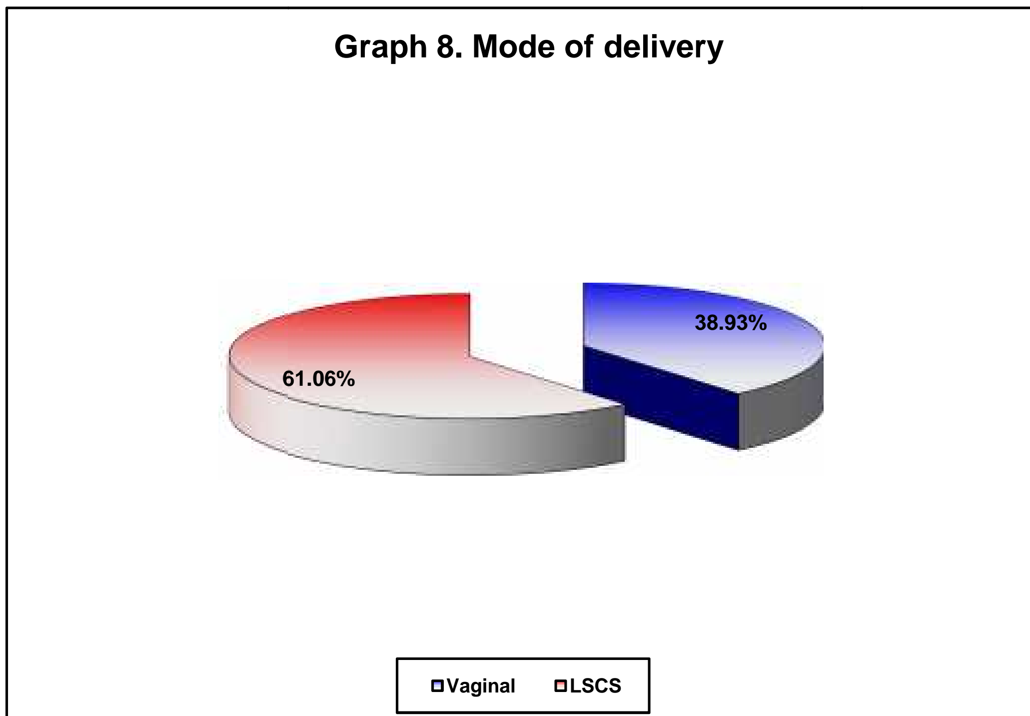
Findings	Distribution (n=113)	
	Number	**Percent**
Abnormal	29	25.66
Normal	84	74.34
Total	**113**	**100.00**



In this study 74.34% of the women had normal MBPP and in 25.66% of the women the findings were abnormal.

Table 8. Mode of delivery

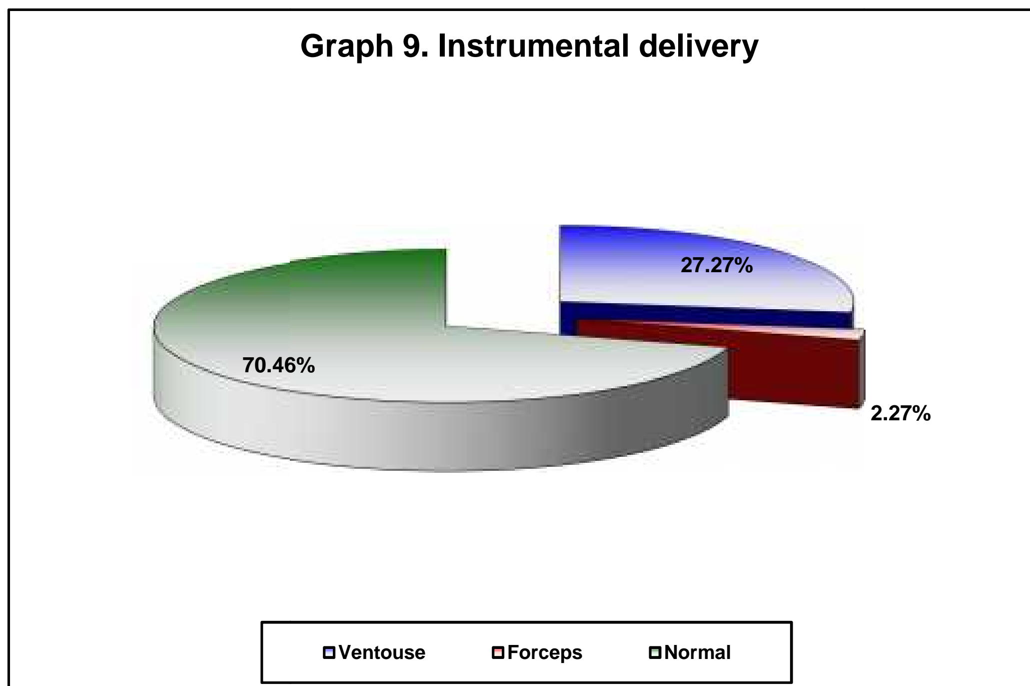
Findings	Distribution (n=113)	
	Number	Percent
Vaginal	44	38.93
LSCS	69	61.06
Total	**113**	**100.00**



In this study 38.93% of the women had vaginal delivery and 61.06% of the women underwent LSCS.

Table 9. Instrumental delivery

Mode	Distribution (n=44)	
	Number	**Percent**
Ventouse	12	27.27
Forceps	1	2.27
Normal	31	70.46
Total	**44**	**100.00**



In this study, of the 44 women who underwent vaginal delivery, 27.27% of the women had ventouse assisted vaginal delivery and 2.27% of the women were delivered by forceps.

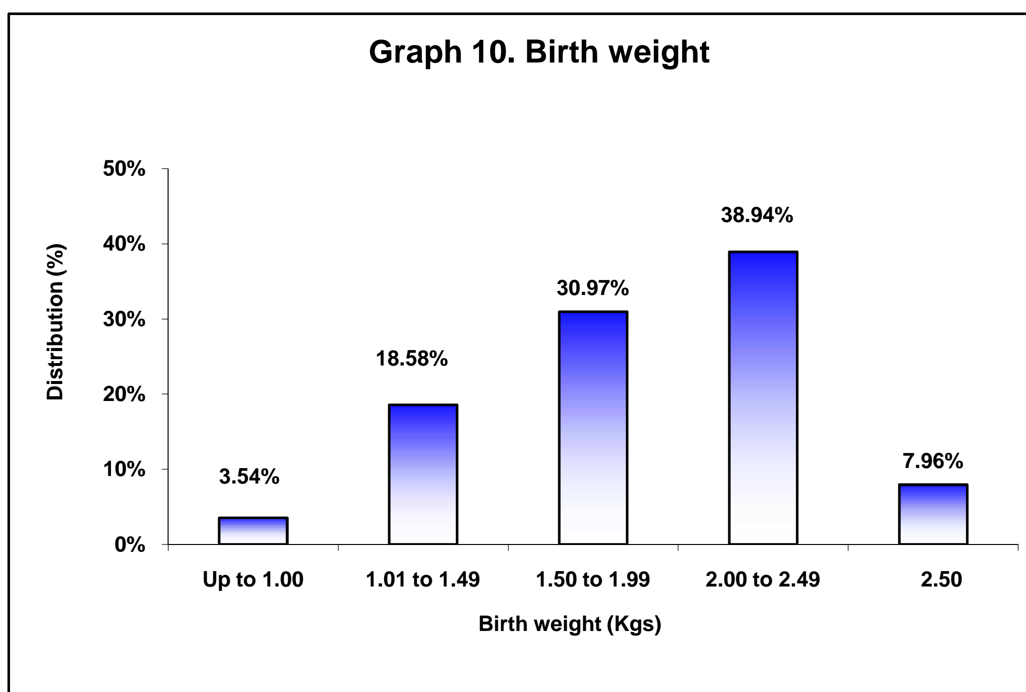
Table 10. Indications for LSCS

Indications	Distribution (n=69)	
	Number	Percent
Previous LSCS	13	18.84
Fetal distress	13	18.84
Failed inductions	9	13.04
AEDF	8	11.59
Uncontrolled PIH	7	10.14
Non progress of labour	4	5.80
Breech	3	4.35
Nil Liquor	2	2.90
Severe oligohydramnios	2	2.90
Transverse lie	2	2.90
REDF	2	2.90
Bad obstetric history	2	2.90
Maternal request	1	1.45
Elderly primigravida	1	1.45
Total	**69**	**100.00**

In this study, of the 69 women who underwent LSCS, the commonest indications were fetal distress and previous LSCS (both 18.84%).

Table 11. Birth weight

Birth weight (Kgs)	Distribution (n=113)	
	Number	Percent
Upto 1.00	4	3.54
1.01 to 1.49	21	18.58
1.50 to 1.99	35	30.97
2.00 to 2.49	44	38.94
2.50	9	7.96
Total	**113**	**100.00**



In the present study 38.94% of the babies weighed 2 to 2.49 kgs while 1.5 to 1.99 birth weight was noted in 30.97% of the babies.

Table 12. Gestational age at delivery and Doppler findings

Doppler findings	Gestational age (Weeks)							
	31.6		32 - 36.6		37 - 39.6		40	
	No.	%	No.	%	No.	%	No.	%
Normal	4	3.54	16	14.16	56	49.56	3	2.65
IR	0	0.00	6	5.31	6	5.31	0	0.00
AEDF	3	2.65	12	10.62	3	2.65	1	0.88
REDF	0	0.00	2	1.77	1	0.88	0	0.00
Total	**7**	**6.19**	**36**	**31.86**	**66**	**58.41**	**4**	**3.54**

In the present study most of the women delivered between 37 to 39.6 weeks of gestation. Among these, 49% had normal Doppler findings, and 5.31%, 2.65% and 0.88% had increased resistance, AEDF and REDF respectively. In women with normal Doppler findings below 37 weeks of gestation had delivered for various causes like uncontrolled PIH, symmetrical IUGR, elderly primigravida and spontaneous labour.

Table 13. Gestational age at delivery and MBPP

Doppler findings	Gestational age (Weeks)							
	31.6		32 - 36.6		37 - 39.6		40	
	No.	%	No.	%	No.	%	No.	%
Abnormal	2	1.77	12	10.62	14	12.39	1	0.88
Normal	5	4.42	24	21.24	52	46.02	3	2.65
Total	**7**	**6.19**	**36**	**31.86**	**66**	**58.41**	**4**	**3.54**

In this study of the 66 women who delivered between 37 to 39.6 weeks of gestation, 14 (12.39%) had abnormal and 46.02% had normal Doppler findings.

Table 14. Mode of delivery and Doppler findings

Mode of delivery	Doppler findings							
	Normal		IR		AEDF		REDF	
	No	%	No	%	No	%	No	%
Vaginal	38	33.63	5	4.42	0	0.00	1	0.88
LSCS	41	36.28	7	6.19	19	16.81	2	1.77
Total	**79**	**69.91**	**12**	**10.62**	**19**	**16.81**	**3**	**2.65**

In the present study of the 19 women with AEDF and 2 REDF underwent LSCS. 1 REDF that underwent vaginal delivery at 32 weeks did not consent for LSCS and NICU expenses.

Table 15. Mode of delivery and MBPP

Mode of delivery	MBPP findings			
	Abnormal		Normal	
	Number	Percent	Number	Percent
Vaginal	8	7.08	36	31.86
LSCS	21	18.58	48	42.48
Total	**29**	**25.66**	**84**	**74.34**

In this study abnormal MBPP findings were noted among 29 women. Among these, 8 (7.08%) had vaginal delivery and 21 (18.58%) had LSCS.

Table 16. Characteristics of study population with good and adverse neonatal outcome

Characteristics	Neonatal outcome		p value
	Good (n=69)	Adverse(n=44)	
	Mean	Mean	
Maternal age (Years)	24.01±3.75	25.50±5.38	0.11
AC (mm)	259.54±2.16	253.88±2.34	0.21
PIH (n)	18	19	0.06
Abnormal MBPP n/(%)	12(17.3%)	17(38.6%)	
Abnormal UA Doppler n/(%)	1(1.44%)	21(47.7%)	
GA at birth (weeks)	37.36±1.04	34.30±2.44	<0.05
Birth weight (Kgs)	2.14±.028	1.42±0.35	<0.05

In this study, the group of patients that experienced an adverse neonatal outcome had a significantly lower gestational age at delivery and the babies who had an adverse outcome had a significantly lower birth weight ($p<0.05$). The groups of patients with and without adverse outcome were similar with respect to maternal age, abdominal circumference and PIH.

Table 17. Neonatal outcome

Outcomes	Number	Percent
NICU admission at birth	38	33.63
IUD	3	2.65
Early neonatal death	7	6.20

In this study, out of the 113 cases, 38 (33.6%) babies required NICU admission at birth, of which 31 admissions were for preterm low birth weight and 6 were for term low birth weight. There were 7 early neonatal deaths, of which 6 were due to necrotizing enterocolitis and 1 due to respiratory distress syndrome. 3 intra-uterine fetal deaths had occurred out of which one was due to intra-partum fetal distress with two tight loop of cord round the neck with thick meconium stained liquor, one was due to severe symmetrical IUGR terminated at early gestation (31weeks) after disregarding fetal prognosis and one at 32 weeks was induced in view of REDF not consenting for LSCS and NICU expenses.

Table 18. Association between abnormal fetal testing and adverse neonatal outcome in IUGR

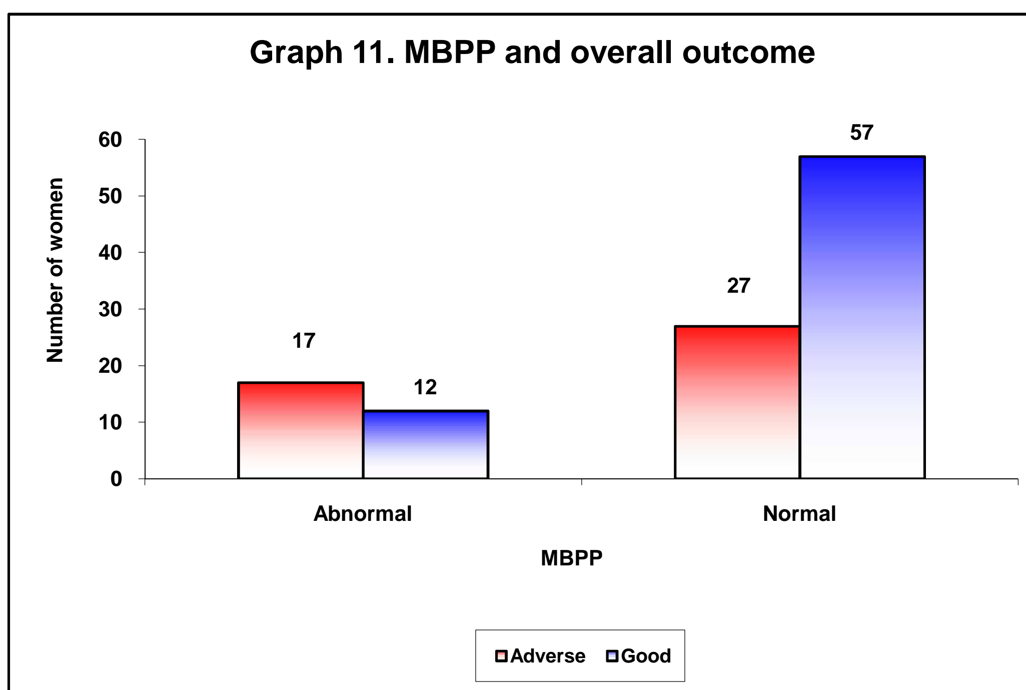
Adverse neonatal outcomes	Antepartum fetal tests					
	Abnormal UA Doppler N=22			Abnormal MBPP N=29		
	Number	OR	CI (95%)	Number	OR	CI (95%)
Resuscitation at birth N=2	1	-	-	2	-	-
NICU admission N=38	17	11.3	3.7-34.3	13	2.7	0.9-6.7
RDS N=8	4	4.8	1.1-21.4	3	1.1	0.2-6.3
Neonatal seizures N=4	3	7.5	0.7-75.3	2	3.0	0.4-22.6
NEC N=12	8	12.2	3.2-46.8	5	3.3	0.9-11.5
END N=7	5	8.2	1.5-44.9	5	8.5	2.0-35.9
IUD N=3	2	4.7	0.4-53.9	2	6.1	0.5-70.4
Apgar score N=8	6	3.2	0.8-12.8	3	1.8	0.4-8.1

In the present study abnormal umbilical artery Doppler was significantly associated with NICU admissions at birth with OR 11.3 (95%CI 3.7-34.3), NEC with OR 12.2 (95%CI 3.2-46.8), RDS with OR 4.8 (95%CI 1.1-21.4) and END with OR 8.2 (95%CI 1.5-44.9), while MBPP was only associated with END with OR 8.5 (95%CI 2.0-35.9).

Table 19. MBPP and neonatal outcome

MBPP	Neonatal outcome		Total
	Adverse	Good	
Abnormal	17	12	29
Normal	27	57	84
Total	44	69	113

Sensitivity	Specificity	PPV	NPV
38.64	82.61	58.62	67.86

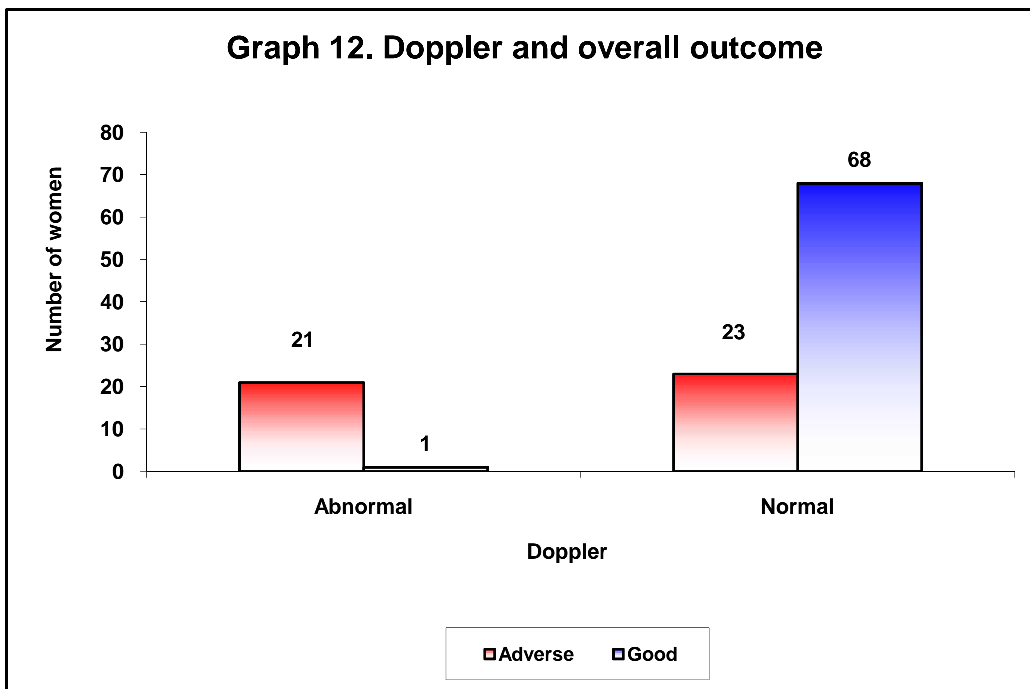


In the present study, of the 44 women with adverse outcome, 17 had abnormal MBPP and 27 had normal MBPP. The sensitivity of MBPP in predicting adverse neonatal outcome was 38.64% and specificity being 82.61%.

Table 20. Doppler and neonatal outcome

Doppler	Neonatal outcome		Total
	Adverse	Good	
Abnormal	21	1	22
Normal	23	68	91
Total	44	69	113

Sensitivity	Specificity	PPV	NPV
47.72	98.55	95.45	74.72



In the present study 44 women had adverse neonatal outcome. Of these, 21 had abnormal Doppler findings and normal Doppler findings were seen in 23 women. The sensitivity of Doppler in predicting adverse neonatal outcome was 47.72% with 98.55% specificity and PPV 95.45%.

DISCUSSION

Antenatal fetal assessment was introduced into the United States in the 1970s. The initial antepartum test, the oxytocin challenge test, later renamed as the contraction stress test, became the gold standard for fetal surveillance. Its labor intensive requirements and contraindications made it inapplicable to some high-risk pregnancies. Other testing schemes were developed subsequently, the NST and its alternative, VAST, the semi-quantitative assessment of amniotic fluid volume, the BPP and its modified version, the MBPP.

The assessment of fetal growth, development and health are considered standard care in most societies. Surveillance has been applied to pregnancies complicated by IUGR to improve fetal outcome hence antenatal fetal surveillance is evolving.[57] In order to prevent complications associated with IUGR, it is important to first detect the condition and once detected, institute appropriate surveillance to asses fetal well being coupled with suitable intervention in case of fetal distress (for example early delivery).[58,59]

Though Doppler and MBPP are the principal antenatal testing modalities, the prognostic and diagnostic information gained in each testing modality is in great part independent of each other, which leads to the controversial diagnosis. However there is lack of data on the integrated or combined Doppler and MBPP in fetal surveillance especially in IUGR complicated pregnancies. This study was aimed to compare the role of UA Doppler and MBPP in predicting adverse neonatal outcome .

This one year prospective cohort study was done on a total of 113 women referred to ANC Clinic, KLES, Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period that is, March 2012 to February 2013 were enrolled.

In this study the most common age group was 21 to 25 years with 51.33% of the women followed by 26 to 30 years with 23.01%. The mean maternal age was found to be 25.50 ± 5.38 years in those women with good outcome and 24.01 ± 3.75 years in women with adverse outcome. A similar study in Jharkhand reported mean maternal age of 24.95±2.52 in those women with good outcome and 23.65±3.57 in women with adverse outcome.[60]

In this study more than half (52.21%) of the women presented as primigravida and gravida 2, 3 and 4 or more were noted in 26.55%, 6.19% and 15.04% of the women respectively. A study done in Karachi reported 60% of the women as multigravidas.[61]

In the present study, 19.46% of the women had abnormal Doppler findings while in 80.53% of the women the findings were normal. On antepartum surveillance with MBPP, 25.66% of the women had abnormal findings. These findings were comparable to a study, which carried out antepartum fetal surveillance with MBPP in 100 cases, of which, 44 cases were with abnormal MBPP and 38 had decreased AFI and 32 cases had non-reactive NST.[62] Another study from Iran reported 30 abnormal tests of the 96 women who were monitored under MBPP including 18 with AFI < 5 and 21 abnormal non-stress tests for MBPP.[39]

In the present study most of the women delivered between 37 to 39.6 weeks of gestation. The mean gestational age at delivery was 36.2 ± 2.30 weeks. A study from Pakistan reported gestational age at delivery as 36.5 weeks.[61] In the present study the mean gestational age at delivery was 37.36±1.04 in women with good outcome and 34.30±2.44 in women with adverse outcome. Similar findings were reported in another study from Jharkhand where the mean gestational age at delivery was 37.12±0.79 in women with good outcome and 36.23±0.99 in women with adverse outcome.[60]

In this study, vaginal delivery was noted among 38.93% of the women while 61.06% of the women underwent lower segment caesarean section. The rate of vaginal delivery in the present study was less compared to a study from Pakistan, which reported 79% of women with vaginal delivery.[61] Also, in another study from Iran reported 27 cesarean sections out of 96 women who monitored under MBPP.[39]

In this study of the 79 women with normal Doppler findings 38 (38.63%) had vaginal delivery and 41 (36.28%) underwent LSCS. In the 12, 19 and 3 women who had increased resistance, AEDF and REDF 6.19%, 16.81% and 1.77% underwent LSCS respectively. Of the 19 women with AEDF & 2 REDF, all (16.81%) underwent LSCS. Similarly, of the 22 women with abnormal Doppler findings, 3 (2.65%) delivered at 31.6 weeks gestation, 14 (12.39%) delivered between 32 to 36.6 weeks, 4 (3.53%) delivered between 37 to 39.6 weeks and 1 (0.88%) had gestation of 40 weeks at delivery. Similarly, of the 29 women with abnormal MBPP, 8 (7.08%) had vaginal delivery and 21 (18.58%) had lower segment caesarean section. These findings could not be compared with the previous literature due to scarcity of the data.

In the present study 38.94% of the babies weighed 2 to 2.49 kgs while 1.5 to 1.99 birth weight was noted in 30.97% of the babies. The mean weight of the babies was 2.14±0.5kgs in women with good outcome and 1.42±0.35 kgs in women with adverse outcome, which was statistically significant, which is comparable to a similar study from Philadelphia which reported mean birth weight as 1.98±0.45kgs in neonates of women with good outcome and 1.02±0.50kgs in neonates of women with adverse outcome.[63]

In the present study, out of the 113 cases, 38 (33.6%) babies required NICU admission at birth, of which 31 admissions were for preterm low birth weight and 6 were for term low birth weight. There were 7 early neonatal deaths, of which 6 were due to necrotizing enterocolitis and 1 due to respiratory distress syndrome and 3 intra-uterine fetal deaths had occurred. Out of the two antenatal testing modalities abnormal Doppler was significantly associated with NICU admissions with OR 11.3 (95%CI 3.7-34.3), NEC with OR 12.2 (95%CI 3.2-46.8), RDS with OR 4.8 (95%CI 1.1-21.4) and END with OR 8.2 (95%CI 1.5-44.9). This was comparable to a study by Bhatt et al that reported high association of UA Doppler to NEC.[50] Study by Juan M et al also showed significant association of abnormal Doppler with RDS.[63] In the present study MBPP was only associated with END with OR 8.5 (95%CI 2.0-35.9).

In the present study 44 women had adverse outcome. Of these, 21 had abnormal Doppler findings and 17 had abnormal MBPP. The sensitivity of Doppler in predicting adverse outcome was 47.72% with 98.55% specificity, 95.45% PPV and 74.72% NPV which was comparable to a study in Toronto in which UA Doppler had sensitivity, specificity, PPV, NPV of 44.6%, 86.6%, 54%, 81.7% respectively.[64] The

sensitivity of MBPP in predicting adverse neonatal outcome was 38.64% and specificity being 82.61% with PPV of 58.62% and NPV of 67.86%.

Gokhan Y et al in 2008, in a retrospective study aimed to evaluate the outcome of IUGR fetuses with AEDF or REDF in the umbilical artery. Their data suggested that pregnancies with AEDF or REDF in the umbilical arteries have high perinatal mortality and morbidity with increased association with respiratory distress syndrome, septicemia, and necrotizing enterocolitis.[65]

A study was done in 1996 by Chan et al in which 71 high-risk fetuses were examined by UA and MCA Doppler US examinations on weekly basis until delivery. In 15.5% (11 of 71) of fetuses, there was perinatal mortality or major morbidity, including major intracranial hemorrhage, periventricular leukomalacia, necrotizing enterocolitis, and major neurologic handicap. By using the last Doppler result for analysis, the UA/MCA RI ratio, compared with the UA S/D ratio, was more sensitive (75% vs 64%) but less specific (60% vs 74%). In conclusion UA Doppler was a better predictor for each of the individual adverse outcomes when separate analyses were performed.[66]

Theodore D et al compared UA Doppler, NST and BPP in IUGR fetuses to predict adverse neonatal outcome. The study concluded that the UA Doppler was the most accurate predictor of adverse neonatal outcome with a sensitivity, specificity, PPV, NPV of 64%, 96%, 88%, 87% respectively.[67]

Juan M et al (2007) in his retrospective cohort study concluded that abnormal Doppler is the best predictor of adverse perinatal outcome in comparison to NST and BPP with a specificity of 88 % and NPV 79% and was significantly associated with

RDS and composite of adverse outcomes while neither an abnormal NST nor BPP were significantly associated with adverse outcomes.[63]

Nageotte MP et al in 1994 concluded that, the MBPP is an excellent means of fetal surveillance and identifies a group of patients at increased risk for adverse perinatal outcome and SGA infants.[19]

Obstetrics and gynecology college of USA (1999) accepted the modified BPP as an acceptable method of fetal antenatal evaluation (Cunningham *et al*., 2005).[68]

Miller (1998) in Los Angles studied on false positive and false negative results of antenatal modified BPP tests concluded that false negative results of modified BPP was lesser than NST and is outstandingly comparative with false negative results of CST and BPP.[69]

Hebek D et al in his prospective study aimed to evaluate variables of the BPP in the assessment of perinatal outcome included 87 pregnant women with singleton pregnancy in the 28th to 42nd week of gestation with clinically and ultrasonically verified fetal growth retardation, where the fetal BPP was assessed antenatally. Through the factor analysis of BPP variables we obtained values indicating the contribution of individual variables to the predictability of perinatal outcome. 70% of the patients were examined in 15 minutes according to the principles of MBPP. The most sensitive variable of the BPP in the prediction of perinatal outcome was the amniotic fluid volume, followed by fetal breathing movements, NST and fetal movements, while the lowest prediction value was assigned to the fetal tone. Authors commented that, the MBPP needs to be perfected on a larger number of pregnant

women, which would advance the predictability of this method in detection of hypoxically endangered fetuses.[70]

Compitak K et al to determine the diagnostic performance of NST, AFI and MBPP for screening fetal acidemia in high-risk pregnancies concluded that, the MBPP had a significantly higher sensitivity than NST or AFI alone in screening for fetal acidemia, so a MBPP should be used to screen for fetal acidemia in high-risk pregnancies.[71]

Very recently a similar study from Jharkhand reported the sensitivity of MBPP as 57.69% with specificity of 70.83%.[60] which was similar to a study done by Bardakci et al (60% and 87.1%).[72] From the available data it clear that, there is a wide variation in the sensitivity and specificity of MBPP as the present study showed sensitivity of MBPP in predicting adverse outcome as 38.64% and specificity as 82.61% which was as high as 87.5% and 96.9% in other studies. One possible explanation for the disparity in these accuracy levels would be the definition of adverse and good neonatal outcome as the present study considered various parameters like, Apgar score at birth, requirement of resuscitation at birth, NICU admission, presence of respiratory distress syndrome, seizures, necrotizing enterocolitis, early neonatal deaths and intra-uterine death which would have resulted in precise selection of cases with good outcome.

MBPP is an effective initial surveillance tool in IUGR cases. Though it has a low sensitivity (38.64%) but it has good specificity, PPV and NPV. By using MBPP as initial surveillance method one saves time, resources and cost of monitoring although it should be coupled with UA Doppler as it has high specificity (98.5%), PPV (95.4%) and NPV (74.7%).

CONCLUSION

1. UA Doppler has a low sensitivity (47.7%) but has a high specificity (98.5%), positive predictive value (95.4%) and negative predictive value (74.7%) in predicting adverse neonatal outcome.

2. Abnormal UA Doppler findings were significantly associated with higher NICU admissions at birth, necrotizing enterocolitis, respiratory distress syndrome and early neonatal death.

3. MBPP has a low sensitivity (38.64%) but has a good specificity (82.61%).

4. MBPP can be used as an initial surveillance method as one saves time, resources and cost of monitoring, although it should be coupled with UA Doppler as it has high specificity and positive predictive value and is significantly associated with adverse neonatal outcomes, so that we can aim to identify IUGR fetuses at risk and improve the outcome by appropriately timed intervention in the management of IUGR.

SUMMARY

The assessment of fetal growth, development and health are considered standard care in most societies. Surveillance has been applied to pregnancies complicated by IUGR to improve fetal outcome. Doppler and MBPP are the principal antenatal testing modalities. The study was done to correlate the role of UA Doppler and MBPP in predicting adverse neonatal outcome.

This one year prospective cohort study was done at ANC Clinic, KLES, Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on a total of 113 women from March 2012 to February 2013.

In this study 51.33% of the women were aged between 21 to 25 years with the mean maternal age found to be 25.50 ± 5.38 years in those women with good neonatal outcome and 24.01 ± 3.75 years in women with adverse neonatal outcome. Most of the women presented as Primigravida (52.21%). 59.29% of the women had BMI between 19.8 to 25.0 Kg/m^2. In the present study 19.46% of the women had abnormal Doppler findings while in 80.53% of the women the findings were normal. On antepartum surveillance with MBPP, 25.66% of the women had abnormal findings of which 20.35% had abnormal AFI and 6.19% had abnormal NST. The mean gestational age at delivery was 37.36±1.04 in women with good outcome and 34.30±2.44 in women with adverse outcome. Vaginal delivery was noted among 38.93% of the women in which 27.27% had ventouse assisted vaginal delivery and 2.27% were delivered by forceps, 61.06% of the women underwent LSCS of which the commonest indications were fetal distress and previous LSCS (both 18.84%). Majority of the abnormal UA Doppler findings (12.39%) were between 32-36.6 weeks of gestation at delivery and 12.39% of abnormal MBPP were between 37-39.6

weeks of gestation at the time of delivery. 19 women with AEDF and 2 women with REDF underwent LSCS. One woman with REDF had a preterm vaginal delivery as she did not consent for LSCS and was not willing for NICU expenses. Among 29 abnormal MBPP, 8 (7.08%) had a vaginal delivery while 21 (18.58%) underwent LSCS. The mean weight of the babies was 2.14±0.5 kgs in women with good outcome and 1.42±0.35 kgs in women with adverse outcome. Out of the 113 cases, 38 (33.6%) babies required NICU admission at birth, of which 31 admissions were for preterm low birth weight and 6 were for term low birth weight. There were 7 early neonatal deaths, of which 6 were due to necrotizing enterocolitis and 1 due to respiratory distress syndrome and 3 intra-uterine fetal death had also occurred.

Adverse outcomes were noted in 44 women of which, 21 had abnormal Doppler findings and 17 had abnormal MBPP. Out of the two antenatal testing modalities abnormal UA Doppler was significantly associated with higher NICU admissions at birth with OR 11.3 (95%CI 3.7-34.3), NEC with OR 12.2 (95%CI 3.2-46.8), RDS with OR 4.8 (95%CI 1.1-21.4) and END with OR 8.2 (95%CI 1.5-44.9) and MBPP was only associated with END with OR 8.5 (95%CI 2.0-35.9).

The sensitivity of Doppler in predicting adverse neonatal outcome was 47.72% with 98.55% specificity, 95.45% PPV and 74.72% NPV. The sensitivity of MBPP in predicting adverse neonatal outcome was 38.64% and specificity being 82.61% with PPV of 58.62% and NPV of 67.86%.

MBPP can be used as an initial surveillance, although it should be coupled with UA Doppler as it has high specificity and positive predictive value and is significantly associated with adverse neonatal outcomes, so as to improve the outcome by appropriately timed intervention in the management of IUGR.

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ANNEXURE-I

INFORMED CONSENT FORM FOR PARTICIPATION IN THE RESEARCH STUDY

TITLE: One year cohort study, role of Umbilical artery Doppler and Modified Biophysical Profile in predicting neonatal outcome in Intrauterine Growth Restriction, a hospital based study.

Objective/ Purpose of the study: We request you to participate in a study conducted by Dr. _________, , Postgraduate in the Department of Obstetrics and Gynaecology, KLE University's Teaching Hospital, Belgaum, under the direct supervision and guidance of Dr. __________, Department of Obstetrics and Gynaecology, KLE University's Teaching Hospital. The study is an attempt to study the role of Umbilical artery Doppler & Modified Biophysical Profile in predicting neonatal outcome in IUGR fetuses. They are surveillances applied to pregnancies complicated by intrauterine growth restriction (IUGR) to improve fetal outcome, to identify critical risk factors and improve outcome by directing appropriately timed intervention. Patients who fulfil the eligibility criteria will be included in the study. Your participation in the study will help us to derive a conclusion which will be beneficial to the larger population.

Procedures:You will be asked to provide some personal identification information and obstetric history relevant to the study. You will be subjected to three antenatal tests i.e Doppler, AFI & NST and followed up till the delivery.

Risks and benefits: There are no additional risks involved in the procedure. There will be no financial incentives for being a part of the study.

Your participation in the study is purely voluntary. Your decision will not affect your relationship with the institute or in the standard of care provided to you. You are free to withdraw at any time during the study.

Privacy and confidentiality: Every effort will be made to protect the confidentiality of the information provided by you. Results of the study may be published for scientific purposes, but your name will not be used.

If you have any questions about the study, you can contact Dr. _________, Department of Obstetrics and Gynaecology. In case you need any further information regarding your rights as a study participant, you may please.

I, volunteer and consent to participate in the study. I have read the consent or has been read to me. The study has been fully explained to me and I was given an opportunity to ask questions and receive answers.

Signature/thumb impression of participant: ____________________

Signature/thumb impression of witness:______________________

Signature of the investigator:______________________________

Date:

ANNEXURE-II

DATA COLLECTION FORM

TITLE:One year cohort study, role of Umbilical artery Doppler & Modified Biophysical Profile in predicting neonatal outcome in IUGR, a hospital based study .

SLNO: _________ DATE: | | | | | | | | |

GROUP: ________ OPD NO: | | | | | | | | |

IPDNO:________ | | | | | | | UNIT:___

PATIENT'S NAME: ____

AGE: _______________

ADDRESS: ____

____

CONTACT NO (RESIDENCE/MOBILE): _________________________________

CURRENT PREGNANCY

1.) OBSTETRIC INDEX: G _P_ L_ A _D_

2.) L.M.P | | | | | | | | |

3.) E.D.D : | | | | | | | | |

4.) Corrected E.D.D : | | | | | | | | |

5.) Gestational age: | | | WEEKS | | DAYS

6.) Was the Consent Given: YES : | | NO: | |

7.) GENERAL EXAMINATION FINDINGS:

Height (cms) : Weight (kgs) : BMI:

Vitals:-

PR: ______ BP: ________

Pallor:________ Icterus: ________ Oedema: ________

Per abdomen:-

FUNDAL HEIGHT: [ ][ ] weeks

8.) **ULTRASOUND FINDINGS:**

DATE: POG:		
	In weeks	In centiles
BPD		
HC		
AC		
FL		
EFW in grams		
Placental localisation		
Presentation		
Congenital anomalies		
AMNIOTIC FLUID INDEX		
Q1		
Q2		
Q3		
Q4		
TOTAL		

DATE: POG:		
Subsequent scan	In weeks	In centiles
BPD		
HC		
AC		
FL		
EFW in grams		
Placental localisation		
Presentation		
Congenital anomalies		
AMNIOTIC FLUID INDEX		
Q1		
Q2		
Q3		
Q4		
TOTAL		

9.) **UMBILICAL ARTERY DOPPLER FINDINGS:**

	1	2	3
R.I			
P.I			
S/D RATIO			

10.)**NST FINDINGS:**

Reactive : ☐ Non-reactive: ☐

11.)**<u>INTRAPARTUM EVENTS</u>**:

FETAL DISTRESS: ☐

MECONIUM STAINED LIQUOR : ☐

NUCHAL CORD : ☐

12.)**PERINATAL OUTCOME:**

a. Gestational age at birth: ☐☐ weeks
b. Birth weight : ______ Kgs
c. Sex: M ☐ F ☐
d. Live birth: YES ☐ NO ☐
e. FSB: YES ☐ NO ☐
f. MSB: YES ☐ NO ☐
g. Early neonatal death: YES ☐ NO ☐
h. Neonatal seizures: YES ☐ NO ☐
i. Respiratory distress syndrome: YES ☐ NO ☐
j. NICU admission at birth: YES ☐ NO ☐
k. Perinatal mortality: YES ☐ NO ☐
l. Necrotising enterocolitis: YES ☐ NO ☐
m. Normal neonatal outcome: YES ☐ NO ☐
n. Resuscitation at birth YES ☐ NO ☐
o. Apgar score: 1min ☐ / ☐ 5min ☐ / ☐
p. Mode of Delivery:
Vaginal ☐
Ventouse ☐
Forceps ☐
C-section ☐ Elect ☐ Emerg ☐

Indication: ______________________________

ANNEXURE -III
KEY TO MASTERCHART

- AC- Abdominal circumference.
- ASD- Atrial septal defect.
- B- Breech.
- BP- Blood pressure.
- BPD- Biparietal diameter.
- C- Cephalic.
- EDD- Expected date of delivery.
- EFW- Expected fetal weight.
- EL- Elective.
- EM- Emergency.
- F- Female.
- FL- Femur Length.
- FSB- Fresh still birth.
- HC- Head circumference.
- IUGR- Intrauterine growth restriction.
- KMC- Kangaroo mother care.
- LB- Live birth.
- LSCS- Lower segment cesarean section.
- M- Male.
- N-No.
- NPL- Non progress of labor.
- NR- Non reactive.
- PIH- Pregnancy induced hypertension.
- R- Reactive.
- SFH- Symphysio-fundal height.
- T- Transverse lie.
- Y- Yes
- + - Positive.
- x - NIL.

Serial Number	Group	Out patient number	In Patient number	Age (Years)	Obstetric history: Gravida	Para	Living	Abortion	Died	Last menopausal date	Expected date of delivery	Corrected EDD	GA: Weeks	Days	Consent	General examination findings: Height (Cms)	Weight (Kgs)	BMI (Kg/m^2)	Vitals: Pulse rate (bpm)	BP: Systolic (mm Hg)	Diastolic (mm Hg)	Pallor	Icterus	Oedema	SFH (Cms)	EFW (gms)	Fundal height (weeks)	Ultrasound findings, First scan: BPD	HC	AC	FL	EFW (gms)	Presentation	Congenital anomalies	I: Total	Umbilical: RI 1	PI 1	Ratio 1		Findings	Intrapartum: Fetal distress	Meconium stained liquor	Nuchal cord	Perinatal outcome: In patient Number	Gestational age (weeks)	days	Mode of delivery	Indication	Pregnancy outcome	Sex	Birth weight (Kgs)	Apgar score: 1 Min	5 Min	Resuscitation at birth	U adm: At birth	Seizures	RDS	NEC	END	Normal outcome
1	Good	1806379	465654	27	4	1	0	2	0	x	x	25.4.12	36	1	Y	150	50	22	80	120	80	x	x	x	28	2.4	32	58	75	4	25	2.1	C	x	12	0.6	0.4	2.3	normal	reactive	x	x	x	478777	37	2	EMLSCS	failed induction with BOH	LB	male	2	7	8	x	x	x	x	x	x	Y
2	Good	1943248	465213	32	1	0	0	0	0	6.7.11	13.4.12	x	36	6	Y	150	52	23	84	120	80	x	x	x	27	2.3	32	50	50	2	18	2.1	C	x	13	0.5	1.2	2.6	normal	reactive	x	x	x	460303	37	5	vaginal	x	LB	male	2	7	8	x	x	x	x	x	x	Y
3	Poor	2021938	467312	24	1	0	0	0	0	1.7.11	8.4.12	x	36	5	Y	150	56	25	80	120	84	x	x	x	23	1.7	26	15	18	5	10	1.7	C	x	9.2	0.6	1	2.7	normal	reactive	x	x	tight loop	411237	37	0	EMLSCS	NPL	LB	female	1.6	7	8	x	Y	x	x	x	x	N
4	Poor	1945210	463710	21	1	0	0	0	0	16.6.11	23.3.12	1.4.12	37	4	Y	152	56	24	84	126	80	x	x	x	27	2.3	34	33	53	5	75	2.3	C	x	4	0.4	0.6	1.7	normal	reactive	x	Y	tight loop	422146	37	4	EMLSCS	fetal distress	FSB	male	2	0	0	Y	x	x	x	x	x	N
5	Good	994013	461438	20	3	1	1	1	0	19.6.11	26.3.12	x	36	3	Y	156	61	25	80	110	70	x	x	x	25	2	32	50	50	9	62	2.2	C	x	11	0.7	1.3	2.4	normal	reactive	x	x	loose loop	460011	37	2	vaginal	x	LB	male	2.5	7	8	x	x	x	x	x	x	Y
6	Good	1433913	461418	22	2	1	0	0	0	10.6.11	17.3.12	X	36	4	Y	157	50	20	86	120	86	x	x	x	28	2.5	32	5	8	2	18	2	C	x	7.4	0.6	0.9	2.39	normal	reactive	x	x	x	460082	38	0	vaginal	x	LB	female	1.9	7	8	x	KMC	x	x	x	x	Y
7	Good	1965925	460982	23	2	1	1	0	0	15.6.11	22.3.12	x	36	3	Y	152	52	22	76	110	70	x	x	x	28	2.4	32	50	33	2	5	2	C	x	8.3	0.7	1.4	2.7	normal	reactive	x	x	x	462007	37	3	vaginal	x	LB	male	2.2	7	8	x	x	x	x	x	x	Y
8	Good	1974402	461111	20	1	0	0	0	0	5.6.11	12.3.12	x	38	1	Y	150	50	22	72	140	90	x	x	+	28	2.4	32	2	19	9	25	2.2	C	x	9.1	0.6	0.9	2.4	normal	reactive	x	x	tight loop	461784	38	5	vaginal	x	LB	female	1.9	6	7	x	KMC	x	x	x	x	Y
9	Good	1646813	444369	19	1	0	0	0	0	13.1.11	20.10.11	13.11.11	39	0	Y	153	50	21	76	110	80	x	x	x	27	2.3	32	25	30	5	15	2	C	x	9.6	0.6	0.9	3	normal	on reacti	Y	Y	x	460029	39	0	EMLSCS	fetal distress	LB	male	1.9	7	8	x	KMC	x	x	x	x	Y
10	Good	1704299	449240	24	1	0	0	0	0	14.3.11	21.12.11	x	38	3	Y	150	44	19	88	140	90	x	x	x	27	2.3	32	25	50	4	38	2.3	C	x	14	0.5	0.8	2.2	normal	reactive	x	x	x	492814	38	4	vaginal	x	LB	male	2.4	7	8	x	x	x	x	x	x	Y
11	Poor	1582503	445075	29	1	0	0	0	0	29.1.11	5.11.11	x	40	5	Y	155	54	22	80	120	80	x	x	x	28	2.4	34	40	52	6	68	2.4	C	x	7.7	x	x	x	diastoli	reactive	Y	Y	tight loop	445091	40	6	EMLSCS	fetal distress	LB	female	2.5	7	8	x	x	Y	x	x	x	N
12	Good	1362286	446670	21	1	0	0	0	0	18.2.11	25.11.11	x	36	3	Y	155	53	22	80	120	80	x	x	x	28	2.4	32	13	58	8	66	2.3	C	x	4	0.4	0.6	1.7	normal	reactive	Y	x	x	446753	37	3	vaginal	x	LB	male	2.2	8	9	x	x	x	x	x	x	Y
13	Good	974528	444866	24	2	1	1	0	0	2.3.11	9.12.11	x	36	0	Y	153	48	21	86	140	90	x	x	+	28	2.4	32	50	44	9	66	2.3	C	x	9.4	0.7	1.1	2.8	normal	reactive	x	x	x	446749	37	3	vaginal	x	LB	male	2.5	7	8	x	x	x	x	x	x	Y
14	Good	1857125	446229	21	1	0	0	0	0	3.3.11	10.12.11	8.12.11	36	6	Y	148	47	21	80	110	80	x	x	x	24	2	32	25	21	4	10	2	C	x	11	0.6	1	2.4	normal	reactive	x	x	x	496354	37	0	vaginal	x	LB	female	1.9	7	8	x	KMC	x	x	x	x	Y
15	Good	1677238	447060	20	1	0	0	0	0	8.3.11	15.12.11	x	36	5	Y	140	47	23	86	110	80	x	x	x	27	2.3	32	2	4	2	16	1.8	C	x	10	0.6	1.4	2.4	normal	reactive	Y	Y	x	460389	37	2	EMLSCS	fetal distress	LB	female	2.1	7	8	x	x	x	x	x	x	Y
16	Good	1630496	447074	28	3	1	1	1	0	18.2.11	25.11.11	x	39	5	Y	161	68	26	80	140	90	x	x	x	25	2.1	28	10	16	8	20	2.6	C	x	5	0.6	1.1	2.64	normal	reactive	x	x	x	447385	40	0	vaginal	x	LB	female	2.3	7	8	x	x	x	x	x	x	Y
17	Poor	1857111	446063	21	2	0	0	1	0	20.3.11	27.12.11	x	34	2	Y	158	50	20	86	140	90	x	x	x	22	1.5	28	5	10	4	2	1.6	C	x	3.4	0.7	1.2	3.3	sed resi	reactive	x	x	x	476323	35	2	vaginal	severe uncontrolled PIH	LB	female	1.5	7	8	x	Y	x	x	Y	x	N
18	Good	1695811	447456	22	1	0	0	0	0	9.3.11	16.12.11	31.12.11	38	3	Y	150	53	22	86	140	90	x	x	+	27	2.3	32	67	71	8	50	2.1	C	x	8	0.6	1.1	2.3	normal	reactive	x	x	x	460398	38	3	vaginal	x	LB	male	2.2	8	9	x	x	x	x	x	x	Y
19	Good	1907851	444506	18	1	0	0	0	0	27.6.11	2.4.12	x	36	1	Y	154	54	23	86	140	90	x	x	+	28	2.4	34	41	50	9	48	2.5	C	x	11	0.7	1.3	2.3	normal	reactive	x	Y	x	445127	37	0	EMLSCS	failed induction	LB	male	2.7	8	9	x	x	x	x	x	x	Y
20	Poor	1907841	466871	20	1	0	0	0	0	27.7.11	2.5.12	x	36	1	Y	154	54	23	80	130	80	x	x	x	24	2	34	33	18	3	50	2	C	x	11	0.7	1.2	3.66	sed resi	reactive	Y	Y	x	466608	36	3	EMLSCS	fetal distress	LB	female	1.9	7	8	x	Y	x	x	x	x	N
21	Poor	2119588	478418	20	1	0	0	0	0	29.9.11	6.7.12	x	36	6	Y	151	48	21	80	150	100	x	x	+	22	1.5	28	4	4	1	3	1.4	C	x	1.5	x	x	x	diastoli	reactive	x	x	loose loop	478456	37	1	EMLSCS	symmmetrical IUGR with ADF with severe oligohydramnios	LB	female	1.4	6	7	x	x	x	x	x	x	N
22	Poor	2124989	479054	30	1	0	0	0	0	29.9.11	5.7.12	x	36	4	Y	154	56	23	80	140	90	x	x	+	22	1.5	34	5	4	<1	1	1.2	C	x	2.8	x	x	x	diastoli	reactive	x	Y	x	479091	37	4	EMLSCS	symmmetrical IUGR with ADF with severe oligohydramnios	LB	male	1.3	7	8	x	Y	x	x	x	x	N
23	Poor	2021477	478037	24	3	1	0	1	0	31.10.11	7.8.12	x	32	1	Y	160	72	26	80	120	70	x	x	x	24	2.1	34	5	16	1	50	1.6	T	x	8	0.6	0.9	2.3	normal	reactive	x	x	x	501211	37	2	EMLSCS	transverse lie	LB	female	1.6	7	8	x	Y	x	x	x	x	N
24	Poor	2122139	478783	25	3	2	1	0	1	20.10.11	26.7.12	1.8.12	33	5	Y	150	53	22	20	168	100	x	x	+	26	1.8	28	20	25	8	38	1.6	C	x	6.6	x	x	x	diastoli	on reacti	Y	x	x	501223	34	3	EMLSCS	NPL	LB	female	1.5	6	7	x	Y	x	x	x	x	N
25	Poor	2129793	479644	42	1	0	0	0	0	5.11.11	11.8.12	x	33	0	Y	153	77	33	88	190	120	x	x	+	28	2.4	28	18	31	5	58	1.6	C	x	15	0.8	1.3	4	sed resi	reactive	x	x	x	472835	33	0	EMLSCS	severe uncontrolled PIH	LB	female	1.4	7	8	x	Y	x	x	x	x	N
26	Good	2129503	476938	20	1	0	0	0	0	28.9.11	4.7.12	x	36	0	Y	154	60	23	86	120	80	x	x	x	25	2.1	34	5	23	7	10	1.9	C	x	7.7	0.6	0.8	2.2	normal	reactive	x	x	x	478312	37	6	vaginal	x	LB	female	2	7	8	x	x	x	x	x	x	Y
27	Good	2129669	476729	20	1	0	0	0	0	5.9.11	11.6.12	x	39	1	Y	156	65	27	80	120	80	x	x	x	27	2.3	36	15	53	3	10	2.2	C	x	4.5	0.6	1	2.5	normal	reactive	Y	Y	x	431513	39	1	EMLSCS	fetal distress	LB	male	2.2	7	8	x	x	x	x	x	x	Y
28	Good	2026501	472899	23	1	0	0	0	0	4.10.11	12.7.12	x	36	2	Y	152	54	19	82	140	90	x	x	x	27	2.3	32	20	61	10	58	2.2	B	x	3.1	0.6	0.8	2.3	normal	reactive	x	x	x	436633	37	4	ELLSCS	severe MS with breech	LB	female	2	7	8	x	KMC	x	x	x	x	Y
29	Poor	2129612	478418	20	1	0	0	0	0	29.9.11	5.7.12	x	36	6	Y	153	53	18	86	150	100	x	x	+	23	1.7	32	2	4	1	1	1.4	C	x	1.5	x	x	x	diastoli	reactive	x	x	x	463299	37	2	EMLSCS	symmmetrical IUGR with ADF with severe oligohydramnios	LB	female	1.4	7	8	x	Y	Y	x	x	x	N
30	Poor	2061361	479566	24	1	0	0	0	0	30.9.11	6.7.12	x	38	0	Y	154	60	23	80	120	88	x	x	+	25	2	32	15	23	2	18	2.1	C	x	10	0.7	1.2	3.5	sed resi	reactive	Y	Y	x	413612	38	0	EMLSCS	fetal distress	LB	female	1.7	7	8	x	Y	x	x	x	x	N
31	Poor	2280911	479644	42	1	0	0	0	0	5.11.12	12.8.12	x	33	0	Y	155	69	30	80	120	86	x	x	+	25	2	28	18	31	5	58	1.6	C	x	15	0.8	1.3	4.08	sed resi	reactive	Y	x	x	416617	34	2	EMLSCS	elderly primi with IUGR	LB	female	1.4	7	8	x	Y	x	x	x	x	N
32	Poor	2289112	479329	39	4	3	2	0	0	3.12.11	10.9.12	x	28	6	Y	158	58	21	86	160	110	x	x	+	22	1.5	26	2	1	1	5	755	C	x	6	x	x	x	diastoli	reactive	x	x	x	424672	29	0	EMLSCS	ADF	LB	female	650	5	6	x	Y	x	Y	Y	Y	N
33	Poor	2211893	480748	23	1	0	0	0	0	24.10.11	30.7.12	8.8.12	34	3	Y	160	68	29	80	120	80	x	x	x	23	1.7	28	25	31	2	38	1.4	C	x	41	0.7	1	2.8	normal	reactive	x	x	x	436789	34	5	vaginal	x	LB	female	1.4	7	8	x	Y	x	x	Y	x	N
34	Good	2210800	481101	24	1	0	0	0	0	10.10.11	17.7.12	x	36	3	Y	156	64	26	82	120	80	x	x	x	25	2	34	58	69	10	67	2.2	C	x	8	0.6	0.8	2.4	normal	reactive	x	x	x	460513	37	1	vaginal	x	LB	male	2.3	7	8	x	x	x	x	x	x	Y
35	Good	2139068	481007	21	1	0	0	0	0	23.10.11	29.7.12	x	36	1	Y	160	55	23	80	140	90	x	x	+	25	2	32	10	21	3	25	1.7	C	x	7.5	0.4	0.6	1.7	normal	reactive	Y	Y	x	489714	37	0	EMLSCS	fetal distress	LB	female	1.9	7	8	x	x	x	x	x	x	Y
36	Poor	2258038	484613	26	3	2	1	0	0	2.1.12	8.10.12	x	29	1	Y	158	56	20	84	120	80	x	x	x	21	1.3	26	10	10	1	2	770	C	x	10	0.7	1.2	3.2	normal	reactive	x	x	x	461247	31	4	vaginal	x	FSB	male	600	0	0	x	x	x	x	x	x	N
37	Good	2258043	484766	25	2	0	0	1	0	20.11.11	26.8.12	x	35	3	Y	157	60	22	86	150	90	x	x	+	26	2.1	32	67	47	9	2	1.8	C	x	6.8	0.8	1.1	3.3	sed resi	reactive	x	x	x	437770	36	0	vaginal	x	LB	male	1.8	8	9	x	KMC	x	x	x	x	Y
38	Poor	2126800	478037	23	3	1	1	1	0	31.10.11	6.8.12	x	34	0	Y	159	65	27	82	110	70	x	x	x	27	2.3	32	5	16	1	53	1.6	C	x	11	0.6	0.8	2.3	normal	reactive	x	x	x	403012	35	2	ELLSCS	placenta praevia with transverse lie	LB	female	1.6	7	8	x	Y	x	x	x	x	N
39	Poor	2147407	482202	22	2	0	0	1	0	10.11.11	16.8.12	x	34	3	Y	160	60	27	76	150	90	x	x	+	25	2	32	50	31	3	25	1.6	C	x	7.4	0.7	1.1	3.2	normal	on reacti	Y	x	x	482443	34	6	EMLSCS	uncontrolled PIH	LB	female	1.6	7	8	x	Y	Y	x	x	x	N
40	Good	2066123	481487	24	1	0	0	0	0	5.10.11	12.7.12	x	39	1	Y	159	64	27	80	130	90	x	x	x	30	2.8	36	50	28	10	50	2.5	C	x	6.6	0.5	0.7	2.1	normal	reactive	x	x	tight loop	481785	39	3	EMLSCS	failed induction	LB	male	2.6	7	8	x	x	x	x	x	x	Y
41	Good	2068194	486300	24	1	0	0	0	0	5.10.11	12.7.12	x	36	6	Y	159	56	20	84	120	80	x	x	x	28	2.4	34	50	28	10	50	2.5	C	x	6.6	0.5	0.7	2.1	normal	reactive	Y	x	x	401016	37	3	EMLSCS	fetal distress	LB	male	2.6	7	9	x	x	x	x	x	x	Y
42	Good	2660534	486028	25	1	0	0	0	0	20.10.11	26.7.12	x	38	2	Y	155	69	30	80	140	90	x	x	+	26	2.1	34	25	50	2	10	2	C	x	10	0.8	1	5.1	sed resi	reactive	x	x	x	501691	39	3	vaginal	x	LB	male	2.2	7	8	x	x	x	x	x	x	Y
43	Good	2605871	488613	20	1	0	0	0	0	25.10.11	31.7.12	x	36	6	Y	154	60	23	80	120	80	x	x	x	25	2	32	50	2	1	50	1.6	C	x	6	0.7	1.2	3.07	normal	reactive	Y	x	x	498561	37	2	EMLSCS	fetal distress	LB	female	1.9	7	8	x	KMC	x	x	x	x	Y
44	Good	1925727	482421	19	1	0	0	0	0	21.10.11	28.07.12	x	36	5	Y	150	44	24	80	120	80	x	x	x	24	2	32	18	25	2	10	2	B	x	7.7	0.5	0.7	2	normal	reactive	x	x	tight loop	482971	37	6	ELLSCS	breech	LB	male	1.9	7	8	x	KMC	x	x	x	x	Y
45	Good	2617261	485547	23	1	0	0	0	0	16.11.11	22.8.12	x	36	6	Y	153	65	28	80	120	80	x	x	x	28	2.4	34	75	58	7	62	2.3	C	x	7.1	0.6	0.9	2.4	normal	reactive	x	x	x	401366	37	6	vaginal	x	LB	female	2.3	7	8	x	x	x	x	x	x	Y
46	Poor	2789112	48669	20	1	0	0	0	0	14.12.11	19.9.12	x	33	5	Y	156	66	28	86	120	80	x	x	x	25	2	30	33	4	1	2	1.2	C	x	4.9	0.4	1.2	1.5	diastoli	reactive	x	x	x	410376	33	6	EMLSCS	NPL	LB	male	1.5	6	7	x	Y	x	Y	x	x	N
47	Poor	2960601	487373	23	1	0	0	0	0	20.12.11	25.9.12	x	33	3	Y	156	68	30	80	120	80	x	x	x	22	1.5	28	18	18	1	5	1.2	C	x	9.1	0.6	0.9	2.5	normal	reactive	x	x	x	411230	34	3	vaginal	x	LB	male	1.3	7	8	x	Y	x	Y	x	x	N
48	Poor	2906611	488049	26	2	1	1	0	0	19.12.11	24.9.12	x	33	0	Y	156	60	23	80	160	110	x	x	+	21	1.4	28	8	43	1	18	1.2	C	x	10	0.7	1.4	3.94	diastoli	reactive	x	x	x	431743	33	0	EMLSCS	severe uncontrolled PIH	LB	male	1.2	6	6	x	Y	Y	x	x	x	N
49	Poor	2990067	487802	27	2	1	1	0	0	18.12.11	23.9.12	x	34	2	Y	150	56	21	80	140	90	x	x	+	21	1.4	30	20	23	1	2	1.3	B	x	3.8	0.8	1.3	4.9	ersal of f	reactive	x	x	x	460035	34	3	EMLSCS	severe uncontrolled PIH	LB	female	1.3	6	7	x	Y	x	x	Y	Y	N
50	Good	2668081	492533	23	3	1	1	1	0	22.11.11	28.8.12	x	36	4	Y	155	58	22	80	120	80	x	x	x	27	2.3	34	58	60	6	60	2.3	C	x	7.6	0.6	1	2.6	normal	reactive	x	x	x	403100	37	5	ELLSCS	prev LSCS	LB	female	2.3	7	9	x	x	x	x	x	x	Y
51	Good	2961261	489132	27	1	0	0	0	0	22.11.11	28.8.12	x	36	6	Y	155	60	23	84	120	80	x	x	x	29	2.6	34	18	50	2	25	2	C	x	9.3	0.4	0.6	1.76	normal	reactive	x	x	x	476537	37	1	EMLSCS	fetal distress	LB	male	2.4	7	8	x	x	x	x	x	x	Y
52	Good	2985062	494779	24	3	2	1	0	1	1.1.12	7.10.12	x	35	1	Y	153	56	21	76	120	80	x	x	x	28	2.4	36	25	31	8	8	1.8	C	x	12	0.6	0.9	2.3	normal	reactive	x	x	x	401312	38	0	vaginal	x	LB	female	2.4	7	8	x	x	x	x	x	x	Y
53	Poor	2960054	490086	29	1	0	0	0	0	23.12.11	28.9.12	x	35	3	Y	154	55	20	86	130	80	x	x	x	26	1.7	32	5	25	5	25	2	C	x	17	0.5	0.8	2.09	normal	reactive	x	x	x	461374	36	4	EMLSCS	ASD with IUGR	LB	female	1.9	7	8	x	KMC	x	x	x	x	Y
54	Good	2900654	495047	24	2	1	0	0	0	30.12.11	5.10.12	x	38	5	Y	155	60	23	80	160	110	x	x	x	27	2.3	34	33	64	10	1	2	C	x	7.1	0.5	0.7	1.9	normal	reactive	x	x	x	461302	38	5	vaginal	x	LB	male	2	7	9	x	x	x	x	x	x	Y
55	Good	2900682	501691	26	2	1	1	0	0	6.12.11	21.9.12	x	36	3	Y	165	68	26	80	120	80	x	x	x	28	2.4	34	58	56	6	58	2.1	C	x	8	0.6	0.9	2.47	normal	reactive	x	x	x	500568	37	2	vaginal	x	LB	male	2.2	7	8	x	x	x	x	x	x	Y
56	Poor	2906058	491309	26	2	1	2	0	0	28.12.11	3.10.12	17.10.12	33	5	Y	154	60	23	86	120	80	x	x	x	21	1.4	30	5	6	2	38	1.5	C	x	8.4	3.1	0.6	1.16	normal	reactive	x	x	x	400360	36	0	EMLSCS	prev LSCS	LB	female	1.2	7	8	x	Y	x	x	x	x	Y
57	Good	2906006	522884	21	1	0	0	0	0	18.11.11	24.8.12	x	36	5	Y	165	70	27	80	120	80	x	x	x	26	2.2	34	5	7	6	8	1.9	C	x	7.6	0.6	0.9	2.57	normal	reactive	x	x	x	566012	37	1	vaginal	x	LB	female	2	7	8	x	x	x	x	x	x	Y

59	Poor	2612391	495743	23	1	0	0	0	0	8.3.12	13.12.12	x	29	4	Y	155	59	22	80	120	80	x	x	x	22	1.5	26	58	33	8	50	1.2	C	x	0	0.7	1.2	3.5	normal	reactive	x	x	x	412730	29	4	vaginal	x	LB	female	1.2	5	6	Y	Y	x	x	Y	Y	N
60	Good	2940052	498121	34	3	1	1	0	0	27.1.12	2.11.12	x	36	5	Y	160	68	32	80	120	80	x	x	x	27	2.3	34	58	44	9	50	2.2	C	x	8.8	0.7	1	3.04	normal	reactive	x	x	x	401674	37	4	ELLSCS	prev LSCS	LB	male	2.2	7	8	x	x	x	x	x	x	Y
61	Good	2934029	502074	22	1	0	0	0	0	16.2.12	22.11.12	x	34	4	Y	165	60	25	84	110	70	x	x	x	25	2	34	18	58	9	67	1.9	C	x	13	0.6	0.9	2.5	normal	reactive	x	x	x	501281	37	3	vaginal	x	LB	male	2.4	7	8	x	x	x	x	x	x	Y
62	Poor	2988742	497249	25	5	3	0	1	0	4.3.12	9.12.12	x	33	2	Y	153	65	28	80	150	90	x	x	x	24	1.8	30	42	19	10	25	1.6	B	x	11	0.6	1.1	3.1	normal	reactive	x	x	x	400087	34	0	ELLSCS	breech	LB	male	1.7	7	8	x	Y	x	x	x	x	N
63	Good	2954022	497861	21	1	0	0	0	0	23.1.12	30.10.12	x	36	1	Y	154	56	20	76	130	80	x	x	x	26	2.1	34	58	47	8	50	2.1	C	x	14	0.7	1.1	3.21	normal	reactive	x	x	x	413765	37	0	EMLSCS	failed induction	LB	female	2.4	7	8	x	x	x	x	x	x	Y
64	Good	2073102	500384	27	2	1	0	0	0	7.3.12	12.12.12	24.11.12	37	1	Y	155	50	16	80	120	80	x	x	x	25	2	34	15	31	10	10	1.9	C	x	10	0.6	0.9	2.52	normal	reactive	x	x	x	401671	37	1	vaginal	x	LB	female	2	7	8	x	x	x	x	x	x	Y
65	Poor	2176980	485374	22	2	0	0	1	0	25.11.11	2.9.12	x	35	2	Y	156	56	24	86	140	90	x	x	+	24	1.8	30	33	33	2	62	1.9	C	x	8.1	0.5	0.8	2	normal	reactive	x	Y	x	501224	35	3	EMLSCS	NPL	LB	male	1.9	7	8	x	Y	x	x	x	x	N
66	Good	1936685	487148	24	2	1	1	0	0	11.11.11	18.8.12	x	38	6	Y	144	60	24	88	110	70	x	x	x	28	2.4	36	1	4	4	42	2.2	C	x	5.8	0.5	0.7	2.02	normal	reactive	x	x	x	522011	39	0	ELLSCS	prev LSCS	LB	female	2.3	7	9	x	x	x	x	x	x	Y
67	Good	2081031	487759	22	2	1	1	0	0	4.11.11	11.8.12	x	40	2	Y	154	56	23	80	124	80	x	x	x	30	2.7	34	42	38	9	18	2.6	C	x	12	0.6	1	2.8	normal	reactive	x	x	x	501124	40	2	ELLSCS	prev LSCS	LB	male	2.9	7	8	x	x	x	x	x	x	Y
68	Poor	2160503	486698	20	1	0	0	0	0	14.12.11	26.9.12	x	33	5	Y	156	75	28	80	150	100	x	x	+	26	1.7	32	33	4	1	2	1.2	C	x	4.9	x	x	x	diastoli	reactive	x	x	x	502289	34	0	EMLSCS	failed induction	LB	male	1.5	6	7	x	Y	x	x	x	x	N
69	Good	1844235	446629	21	1	0	0	0	0	24.2.11	4.12.11	x	36	1	Y	164	65	25	68	110	70	x	x	x	26	1.7	34	58	34	7	67	1.9	C	x	5	0.8	1	3.2	sed resi	reactive	x	x	x	446952	37	1	vaginal	x	LB	female	2.2	7	8	x	x	x	x	x	x	Y
70	Good	1844561	500504	22	3	1	1	0	0	5.11.11	12.8.12	x	36	5	Y	158	57	24	62	110	70	x	x	x	27	2.3	32	50	13	8	50	2	C	x	9	0.6	1.2	2.4	normal	reactive	x	x	x	501121	37	0	vaginal	x	LB	female	2.1	7	8	x	x	x	x	x	x	Y
71	Good	2305612	503105	29	2	0	0	1	0	20.2.12	27.11.12	x	39	6	Y	156	62	24	74	120	80	x	x	x	28	2.4	34	58	41	1	2	2	C	x	7.8	0.6	1.1	2.7	normal	reactive	x	x	x	501266	40	0	vaginal	x	LB	male	2.1	7	8	x	x	x	x	x	x	Y
72	Good	2126090	501408	19	2	1	1	0	0	5.3.11	3.12.12	x	36	2	Y	155	56	23	80	120	80	x	x	x	26	1.7	34	50	33	8	30	2.1	C	x	10	0.7	1.1	2.8	normal	reactive	x	x	x	561080	37	o	ELLSCS	prev LSCS	LB	male	1.9	7	8	x	x	x	x	x	x	Y
73	Good	2192011	500504	24	3	1	1	1	0	26.2.12	2.12.12	x	36	2	Y	158	59	24	86	120	80	x	x	+	27	2.3	32	15	33	4	25	2	C	x	8.4	0.6	0.8	2.3	normal	reactive	x	x	x	501299	36	2	vaginal	x	LB	female	2.1	7	8	x	x	x	x	x	x	Y
74	Poor	2210111	591128	25	3	2	1	0	0	5.3.12	10.12.12	x	35	5	Y	160	60	23	80	110	60	x	x	x	26	1.8	32	20	56	2	2	1.7	C	x	7.9	0.5	0.7	2.02	normal	reactive	x	x	x	501121	36	1	ELLSCS	BOH with prev 2 LSCS	LB	male	1.8	7	8	x	x	x	x	x	x	N
75	Good	2210912	501612	25	3	2	2	0	0	28.2.11	6.12.12	x	36	0	Y	154	60	23	80	130	90	x	x	x	27	2.3	34	58	43	6	50	2	C	x	7.8	0.7	1	2.2	normal	reactive	x	x	x	596900	37	0	vaginal	x	LB	female	2.2	7	8	x	x	x	x	x	x	Y
76	Good	2102344	503036	30	3	2	1	0	0	2.3.12	7.12.12	x	38	1	Y	168	69	28	86	110	70	x	x	x	28	2.5	36	42	42	9	75	2.5	C	x	20	0.6	0.8	2.2	normal	reactive	x	x	x	596191	38	1	ELLSCS	prev LSCS	LB	male	2.9	7	8	x	x	x	x	x	x	Y
77	Good	2109600	501123	20	1	0	0	0	0	22.3.12	27.12.12	x	36	0	Y	165	62	25	80	120	80	x	x	x	25	2	32	15	28	1	18	1.6	C	x	12	0.8	1.4	5.6	normal	reactive	x	x	x	501599	37	1	vaginal	x	LB	female	1.8	7	8	x	KMC	x	x	x	x	Y
78	Good	2535035	501966	23	1	0	0	0	0	2.3.12	7.12.12	x	39	3	Y	163	56	21	76	110	70	x	x	x	25	2	32	1	21	1	18	1.7	C	x	0	0.7	0.3	3	normal	reactive	x	Y	x	502305	39	3	EMLSCS	anamnios	LB	male	1.8	7	8	x	x	x	x	x	x	Y
79	Good	2015353	504144	23	1	0	0	0	0	10.3.13	15.12.12	x	38	6	Y	158	61	24	80	120	80	x	x	x	25	2	36	4	23	1	8	1.5	C	x	1.4	x	x	x	rsal of f	reactive	x	x	x	590811	38	6	EMLSCS	ADF	LB	male	1.8	7	8	x	Y	x	x	x	x	N
80	Poor	2099136	505046	26	1	0	0	0	0	25.4.12	13.12.12	x	32	0	Y	158	62	24	80	110	80	x	x	x	25	2	28	2	2	2	10	1.3	C	x	17	x	x	x	diastoli	n reacti	Y	x	x	510817	33	1	EMLSCS	ADF	LB	female	1.2	6	7	x	Y	x	x	Y	x	N
81	Good	2209928	508160	20	1	0	0	0	0	20.4.12	25.1.13	x	36	0	Y	161	65	26	80	140	90	x	x	x	26	2.2	32	33	42	4	18	1.9	C	x	13	0.6	0.9	2.3	normal	reactive	Y	Y	tight loop	511261	37	0	EMLSCS	fetal distress	LB	female	2	7	8	x	x	x	x	x	x	Y
82	Poor	2015089	508178	32	2	1	1	0	0	4.5.12	11.2.12	x	35	5	Y	156	60	23	86	150	90	x	x	+	25	2	32	50	58	5	25	1.5	C	x	10	0.7	1.1	3.5	sed resi	reactive	x	x	x	501281	36	0	ELLSCS	prev LSCS	LB	female	1.7	7	8	x	Y	x	x	x	x	N
83	Good	2016685	508161	22	1	0	0	0	0	17.4.12	22.1.13	x	36	4	Y	170	69	28	84	150	90	x	x	+	25	2	32	5	3	2	25	1.8	C	x	8.3	x	x	x	diastoli	n reacti	x	x	tight loop	521613	36	4	EMLSCS	ADF	LB	male	1.8	7	8	x	KMC	x	x	x	x	Y
84	Poor	2335601	510294	29	1	0	0	0	0	11.6.12	18.3.13	x	28	2	Y	165	74	33	80	120	80	x	x	x	23	1.5	30	10	42	6	50	1.2	C	x	11	x	x	x	diastoli	reactive	x	x	x	521187	30	2	EMLSCS	ADF	LB	female	1.1	6	7	x	Y	x	x	Y	x	N
85	Good	2325802	508526	27	4	3	2	0	0	10.4.12	17.1.13	24.1.13	36	4	Y	163	64	25	72	110	80	x	x	x	26	2.1	32	58	50	2	50	1.9	C	x	6.7	0.6	0.9	2.4	normal	reactive	x	x	x	501231	37	0	vaginal	x	LB	female	2.1	7	8	x	x	x	x	x	x	Y
86	Good	2044480	509112	22	1	0	0	0	0	25.4.12	30.1.13	x	36	0	Y	166	60	22	80	110	70	x	x	x	28	2.4	32	42	64	10	75	2.2	C	x	12	0.6	1	2.96	normal	reactive	x	x	x	511381	37	0	vaginal	x	LB	female	2.1	7	8	x	x	x	x	x	x	Y
87	Poor	2376982	509120	33	1	0	0	0	0	16.4.12	21.113	12.2.13	34	3	Y	174	77	34	80	160	110	x	x	+	25	2	30	10	31	1	18	1.4	C	x	6.7	x	x	x	diastoli	n reacti	x	x	x	522314	34	2	EMLSCS	ADF	LB	male	1	6	7	x	Y	x	x	Y	Y	N
88	Poor	2381702	509664	21	4	3	1	0	2	5.6.12	12.3.13	x	31	0	Y	155	55	27	80	170	110	x	x	+	22	1.5	30	5	5	1	5	1	C	x	3.9	0.6	0.9	2.8	normal	reactive	x	x	x	501233	31	2	vaginal	severe uncontrolled PIH	LB	male	1	6	7	x	Y	x	Y	x	Y	N
89	Good	2382436	509759	22	4	1	0	2	1	15.5.12	19.2.13	11.2.13	35	7	Y	155	56	27	80	110	70	x	x	x	27	2.3	32	25	50	5	38	1.9	C	x	11	0.6	1	2.9	normal	reactive	x	x	x	561321	36	0	ELLSCS	BOH with IUGR	LB	female	2.1	8	9	x	KMC	x	x	x	x	Y
90	Poor	2387426	510617	20	1	0	0	0	0	10.6.12	17.3.12	x	31	2	Y	153	58	22	80	140	90	x	x	+	22	1.5	28	2	2	2	2	1.1	C	x	12	0.7	1.2	3	diastoli	reactive	x	x	x	556126	31	4	EMLSCS	ADF	LB	male	930	5	6	x	Y	x	Y	Y	Y	N
91	Good	2462166	511387	22	2	1	1	0	0	19.5.12	23.2.13	x	34	5	Y	158	68	29	80	110	80	x	x	x	24	2.1	34	42	47	2	18	1.9	C	x	12	0.6	0.9	2.6	normal	reactive	x	x	x	516211	37	0	vaginal	x	LB	male	1.9	7	8	x	KMC	x	x	x	x	Y
92	Poor	2171800	514004	21	1	0	0	0	0	2.6.12	9.3.13	x	35	0	Y	156	56	20	80	120	80	x	x	x	24	1.8	30	10	25	4	30	1.2	C	x	5	x	x	x	diastoli	n reacti	x	x	tight loop	561234	35	0	EMLSCS	nonreassuring NST	LB	female	1.2	6	7	x	x	x	x	Y	Y	N
93	Good	2961001	518750	25	1	0	0	0	0	6.6.12	13.3.13	x	35	0	Y	156	56	20	80	120	80	x	x	x	28	2.4	34	1	2	5	25	2.1	C	x	15	0.6	0.9	2.3	normal	reactive	x	x	x	556236	38	3	vaginal	x	LB	female	2.3	7	9	x	x	x	x	x	x	Y
94	Good	2244478	501611	25	2	1	1	0	0	22.5.12	26.2.13	8.3.13	36	6	Y	160	65	26	80	110	80	x	x	x	26	2.1	34	25	19	8	38	2.2	C	x	12	0.6	1.1	2.9	normal	reactive	x	x	x	596023	37	0	EMLSCS	prev LSCS	LB	male	2.2	7	8	x	x	x	x	x	x	Y
95	Good	2099269	501691	32	2	0	0	1	0	x	x	16.4.13	32	2	Y	158	74	35	80	130	90	x	x	x	25	2	34	25	42	6	48	1.8	C	x	11	0.7	0.9	2.4	normal	reactive	x	x	x	501355	37	0	ELLSCS	maternal request	LB	female	2.4	7	8	x	x	x	x	x	x	Y
96	Poor	2196421	512580	23	2	2	1	0	1	12.6.12	19.3.13	x	33	1	Y	155	66	28	80	150	110	x	x	x	22	1.5	30	2	3	3	2	1.3	C	x	6.5	0.7	1	2.8	normal	reactive	x	x	Y	501135	34	3	EMLSCS	severe uncontrolled PIH	LB	female	1.3	7	8	x	Y	x	x	x	x	N
97	Poor	2196526	515551	26	3	1	1	1	0	13.7.12	20.4.13	x	30	2	Y	157	65	27	80	160	100	x	x	+	24	2	28	42	44	2	50	1.2	C	x	6.4	0.7	1.1	3.2	normal	reactive	x	x	Y	511536	31	0	EMLSCS	severe uncontrolled PIH	LB	female	1.2	5	7	x	Y	x	Y	x	x	N
98	Poor	1785437	517569	26	2	1	1	0	0	6.7.12	13.4.13	28.4.13	31	4	Y	154	80	41	80	120	80	x	x	x	24	1.8	30	1	2	1	1	1	C	x	12	0.6	0.8	2.2	normal	reactive	x	Y	x	536012	35	0	vaginal	x	LB	male	1.1	7	8	x	Y	x	Y	x	x	N
99	Poor	2421650	518334	25	2	1	0	0	0	16.6.12	23.3.13	x	36	5	Y	158	60	23	80	120	80	x	x	x	24	2	34	10	16	5	2	1.8	C	x	7.4	0.8	0.7	4.7	diastoli	reactive	x	x	x	501123	36	5	EMLSCS	ADF	LB	male	1.6	7	8	x	Y	x	x	x	x	N
100	Good	2426089	518998	33	3	1	1	1	0	x	x	26.3.13	36	5	Y	154	51	19	80	120	80	x	x	x	30	2.7	34	2	8	8	5	1.9	C	x	4.8	0.7	1.5	3.8	sed resi	reactive	x	x	Y	523445	37	0	ELLSCS	prev LSCS	LB	female	2.2	7	8	x	x	x	x	x	x	Y
101	Poor	2645678	518379	28	1	0	0	0	0	19.7.12	25.4.13	x	32	0	Y	158	65	27	80	120	80	x	x	x	24	2	28	10	31	2	25	1.4	C	x	7.6	0.7	1.2	4.7	diastoli	reactive	x	x	x	501123	32	5	EMLSCS	ADF	LB	female	1.6	5	6	x	Y	x	Y	Y	x	N
102	Poor	2349890	521594	23	2	1	1	0	0	2.8.12	9.5.13	x	30	5	Y	156	60	23	80	120	80	x	x	x	24	1.8	28	20	42	7	5	1.2	C	x	8.1	0.6	1	2.7	normal	reactive	x	Y	x	523089	33	0	vaginal	second stage labour	LB	male	1.4	5	6	x	Y	x	x	Y	x	N
103	Good	2288682	519473	23	1	0	0	0	0	23.6.12	30.3.13	x	36	3	Y	156	59	22	86	120	80	x	x	x	26	2.1	32	15	18	5	58	2.1	C	x	7.2	0.6	0.9	2.6	normal	reactive	x	x	tight loop	529081	37	0	EMLSCS	failed induction	LB	female	2	7	8	x	x	x	x	x	x	Y
104	Good	2427550	522549	29	2	1	1	0	0	30.6.12	6.4.13	x	36	6	Y	157	61	24	80	120	80	x	x	x	28	2.4	34	42	36	9	10	2.1	C	x	10	0.5	0.8	2.07	normal	reactive	x	x	x	511621	37	3	EMLSCS	failed induction	LB	male	2.5	7	8	x	x	x	x	x	x	Y
105	Poor	2416890	523650	23	1	0	0	0	0	6.7.12	12.4.13	x	35	3	Y	156	65	26	80	120	80	x	x	x	24	1.8	28	10	3	1	10	1.5	C	x	7	x	x	x	diastoli	reactive	x	x	x	516561	36	2	EMLSCS	failed induction	LB	female	1.3	7	8	x	Y	x	x	x	x	N
106	Good	2426089	509261	33	3	1	1	1	0	x	x	26.3.13	36	6	Y	154	69	31	80	150	90	x	x	+	26	2.1	32	2	8	8	5	1.9	C	x	4.8	0.7	1.5	3.8	sed resi	reactive	x	x	x	566126	37	0	ELLSCS	prev LSCS		female	2.2	7	8	x	x	x	x	x	x	Y
107	Good	2436088	520025	25	1	0	0	0	0	2.7.12	13.3.13	x	36	2	Y	156	61	24	80	150	80	x	x	+	26	2.1	32	10	6	2	10	1.8	C	x	1.4	0.5	0.8	2.1	normal	reactive	x	Y	x	566140	36	2	EMLSCS	severe oligohydramnios	LB	female	1.8	7	8	x	KMC	x	x	x	x	Y
108	Good	2451231	530321	28	2	1	1	0	0	5.7.12	11.4.13	x	36	1	Y	156	64	26	80	150	90	x	x	+	24	1.8	32	20	23	2	38	1.9	C	x	8.7	0.6	1	2.7	normal	reactive	x	x	x	571811	36	2	EMLSCS	severe uncontrolled PIH	LB	male	1.9	7	8	x	KMC	x	x	x	x	Y
109	Good	2801311	530332	23	1	0	0	0	0	30.5.12	6.3.13	x	36	1	Y	158	64	26	80	120	80	x	x	x	25	1.9	32	15	42	4	38	2	C	x	11	0.5	0.6	1.8	normal	reactive	x	x	x	591611	37	0	vaginal	x	LB	male	2.1	7	8	x	x	x	x	x	x	Y
110	Poor	2246370	521185	29	2	0	0	1	0	30.7.12	6.513	x	33	0	Y	155	55	20	80	120	80	x	x	x	24	1.8	28	75	52	2	25	1.5	C	x	8.6	x	x	x	diastoli	reactive	x	x	x	561281	34	5	EMLSCS	failed induction	LB	male	1.6	6	7	x	Y	x	x	x	x	N
111	Good	2183976	521561	28	1	0	0	0	0	17.6.12	24.3.13	x	39	1	Y	155	64	26	80	110	70	x	x	x	28	2.4	34	33	36	2	58	2.3	C	x	3.2	0.7	1.2	3.7	sed resi	reactive	x	x	x	522260	39	1	vaginal	x	LB	female	2	7	8	x	x	x	x	x	x	Y
112	Good	2480139	521644	20	2	1	1	0	0	26.4.12	31.1.13	x	38	6	Y	158	66	28	80	120	80	x	x	x	28	2.4	36	2	23	8	82	2.5	C	x	8.2	0.7	1.5	2.6	normal	reactive	x	x	x	512611	39	0	vaginal	x	LB	male	2.3	7	8	x	x	x	x	x	x	Y
113	Poor	2536008	518757	28	4	1	1	2	0	15.7.12	21.4.12	x	33	1	Y	156	64	26	80	110	80	x	x	x	24	1.8	28	1	2	2	1	1.1	C	x	0	x	x	x	rsal of r	reactive	x	x	x	526258	33	4	vaginal	x	FSB	female	1.2	0	0	x	x	x	x	x	x	N