"EVALUATION OF CARDIOTOCOGRAPHY MONITORING IN INTRAPARTUM FOETAL SURVEILLANCE AND ITS CORRELATION WITH APGAR SCORE AND CORD BLOOD pH"

By

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OBSTETRICS AND GYNAECOLOGY

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MAY 2015

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Dr. ALEKHYA REDDY

LIST OF ABBREVIATIONS

ABG	Arterial Blood Gas
ACOG	American College of Obstetrics Gynecology
APGAR	Appearance Pulse Grimace Activity Respiration.
BP	Blood Pressure
CESDI	Confidential enquires into still births and deaths infancy
CTG	Cardiotocograph
EFM	Electronic fetal monitoring
FHR	Fetal heart rate
FPO	Fetal Pulse oximetry
gm	Gram
IUD	Intra Uterine Death
IUGR	Intra uterine growth retardation
LBW	Low birth weight
LSCS	Lower Segment Cesarean section
ml	Millilitre
mmol	Mili mole
NICE	National institute clinical excellence
NICHHD	National Institute Child Health and Human Development Research Group
NICU	Neonatal Intensive care unit
PROM	Premature Rupture of Membranes
RCOG	Royal College of Obstetrics Gynecology
STAN	ST segment analysis

ABSTRACT

EVALUATION OF CARDIOTOCOGRAPHY MONITORING IN INTRAPARTUM FOETAL SURVEILLANCE AND ITS CORRELATION WITH APGAR SCORE AND CORD BLOOD pH

Objectives:

1. To evaluate the reliability of CTG, for the diagnosis of acute fetal hypoxia.
2. To correlate CTG with Apgar score.
3. To correlate CTG with cord blood pH.

Materials and Methods:

This was a prospective comparative study conducted among 200 women admitted in labour for delivery with singleton term pregnancy in vertex presentation to RLJH hospital, Kolar between 1st January 2013 to 31st July 2014. CTG tracings were taken in active stage, preferably 30 minutes before delivery or even earlier with FHR irregularities. CTG tracing were defined as reactive, suspicious or pathological patterns as per NICHHD guidelines. After delivery Apgar score at 1 and 5 minutes and cord blood gas values were analyzed. NICU admission were analyzed and followed up till discharge.

Results:

Among the 200 patients 100(50%) showed normal FHR patterns, 26(13%) showed suspicious and 74 (37%) showed pathological FHR patterns. Late decelerations were commonest abnormal CTG patterns and seen in 40 cases, next common was variable deceleration in 26, tachycardia in 18 prolonged deceleration in 8, bradycardia in 5 and

early deceleration in 3. Of all cases delivered vaginally, among them 18% were in the pathological FHR group.

Operative interventions were done in 47(23.5%) with cesarean deliveries and 39 (19.5%) with instrumental deliveries. Apgar score < 7 at 1 min (depressed) was 3%, 58% and 73% in normal, suspicious and pathological groups respectively. Apgar score at 5 min were 1%, 3%, and 14% in normal, suspicious and pathological groups respectively. The cord blood pH (<7.2) was seen in 2%, 58% and 70% in normal, suspicious and pathological groups. Moderate umbilical artery acidemia with base deficit of 12 to 16 mmol were seen in 2%, 15% and 26% in normal, suspicious and pathological groups respectively. Severe base deficit > 16 mmol was seen in 0, 8% and 15% of cases of normal, suspicious and pathological groups respectively. Admission to NICU were 1%, 7.6% and 14.8% across the 3 FHR patterns groups.

Conclusion:

1. Normal CTG tracings are reassuring.
2. Late decelerations, variable decelerations and bradycardia are most specific for prediction of fetal asphyxia.
3. The use of EFM is associated with increase in the rate of operative intervention. Additional tests such as fetal ECG, Fetal Pulse Oximetry, Fetal Scalp Blood Sampling for pH or lactate estimation are required before intervention to rule out fetal asphyxia as per the protocol.
4. In presence of pathological CTG patterns cord blood pH and base deficit shows better correlation than APGAR score for prediction of fetal asphyxia.

Key words – CTG, APGAR score, cord blood pH, base deficit

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INTRODUCTION

The practice of modern obstetrics involves the care of the mother and her fetus. The goal of intrapartum assessment of the fetus is to identify fetal asphyxia and intervene to alleviate this.[1] Intrapartum fetal asphyxia is a major risk for neonatal morbidity and mortality. Intrapartum fetal asphyxia with significant metabolic acidosis at delivery was shown to occur in approximately 20-25 infants per 1000 births. Most of these were of mild type with no cerebral dysfunction or brain damage. In 3-4 infants per 1000 births moderate or severe fetal asphyxia was seen with neonatal encephalopathy and other organ system complications. Among these ≥1 infant per 1000 births would have brain damage resulting in early neonatal death or motor or cognitive problems in surviving child. [1-3]

Various methods have been used to assess intrapartum fetal distress. Currently the two standard methods, intermittent auscultation and electronic fetal heart rate monitoring (EFM) by cardiotocograph (CTG) are used. Other newer methods used are fetal scalp blood sampling, scalp stimulation, vibroacoustic stimulation, fetal pulse oximetry, intrapartum doppler velocimetry. After birth fetal asphyxia is subjectively assessed by APGAR score and objectively by Cord Blood pH.[1,4,5]

Intermittent auscultation is simple and as safe as continuous EFM,[4] but does not show accurate information about baseline variability or periodic changes. It requires 1:1 nursing to patient ratio and is difficult to auscultate in obese patients.[5,6] One study has shown that intermittent auscultation was done successfully according to guidelines in only 3% of the cases. [7]

EFM is a non-invasive method and provides better information about baseline variability and is a visual sensitive record. Easy to operate, available under any

hospital situation, possible to use in the absence of the obstetrician and is financially accessible.[5]

One of the study showed 55% reduction of neonatal seizures and reduction of hypoxia related deaths by 1 perinatal death per 1000 births at the expense of an increase in operative vaginal and cesarean delivery for suspected fetal distress by 2-3 fold.[4,6,8] In Confidential Enquires into Still Births and Deaths in infancy (CESDI) report it was shown that over 50% of intrapartum deaths of normally grown fetuses weighing more than 1.5 kg were due to failure to recognize or take appropriate action on CTG abnormalities. Routine clinical practice of EFM use was to reduce perinatal mortality and long term neurological handicaps due to intrapartum hypoxia or acidosis.[4]

In USA, EFM was used among 4% of parturients in 1980, 62% in 1988, 74% in 1992 and 85% in 2002 and with this cesarean sections for fetal distress also significantly increased. From 1974-1991 the incidence of cesarean section for fetal distress increased 15 fold from 0.6%-9.2% to all cesarean section performed in USA. Electronic Fetal monitoring has been associated with unnecessary intervention with increased incidence of cesarean deliveries for fetal distress, operative deliveries and general anaesthesia.[2]

Studies on EFM have showed high false positive rates and poor interobserver and intraobserver reliability.[1,3] EFM has high sensitivity but its specificity is low. Abnormal FHR patterns for which cesarean or instrumental deliveries were undertaken were associated with a 50% of fetal acidosis. Hence RCOG has recommended the use of fetal blood sampling (FBS) in conjunction with EFM to improve specificity. To improve the effectiveness of EFM additional methods like fetal pulse oximetry, scalp blood analysis, ST wave form analysis of fetal ECG and

lactate estimation are advised. All these methods are invasive and cannot be used in presence of intact membranes, infection and low lying placenta.[4,5,8 – 11.]

Hence this study was undertaken to evaluate CTG and fetal blood gas and acid base assessment for prediction and detection of intrapartum fetal asphyxia in a single tertiary care obstetric unit. In addition it was studied whether EFM has been associated with increased incidence of operative deliveries and also the neonatal outcome was noted.

OBJECTIVES

1. To evaluate the reliability of CTG, for the diagnosis of acute fetal hypoxia.
2. To correlate CTG with APGAR score.
3. To correlate CTG with cord blood pH.

REVIEW OF LITERATURE

Methods of Intrapartum fetal surveillance, before 1970, fetoscopes were used to auscultate fetal heart rate and intermittent auscultation was used for intrapartum fetal monitoring during the first half of 20th century.[4] Electronic fetal monitoring (EFM) was pioneered by Hon in 1958 and the first commercially available monitor was produced by United States in 1968.[12] EFM was used for continuous recording of FHR as well as uterine contractions.[13]

Intermittent auscultation was advised by RCOG in all low risk cases. In active stages of labour, intermittent auscultation was advised after a contraction for a minimum of 60 seconds and atleast every 15 minutes in the first stage and every 5 mins in the second stage.[1,4] Continuous EFM was recommended if there is evidence of a baseline < 110 or >160 bpm, any decelerations on auscultation or any intrapartum risk factors. Admission cardiotocography (CTG) in low risk pregnancies was not recommended. In presence of increased risk of perinatal death, cerebral palsy or neonatal encephalopathy continuous EFM was advised. Continuous EFM was also advised with oxytocin use for induction or augmentation.[9]

APGAR score initially developed to identify the newborn who needs resuscitation, is a subjective method and depends on age and maturity of the newborn. Sykes and colleagues in a prospective study of more than 1000 deliveries found only 21% of babies with a 1 minute APGAR score less than seven and 19% of babies with a 5 minute APGAR score less than seven had severe acidosis. In those with severe acidosis, 73% of babies had an APGAR score of seven or more at 1 minute and 86% had such a score at 5 minutes and thus factors unrelated to hypoxia can result in low

APGAR scores. A low APGAR score indicates an abnormal condition but not its cause. Acidosis may be present in a vigorous neonate with a normal APGAR score. APGAR score is not correlated directly with fetal biochemical status except in conditions of extreme acidosis.[14-18] Umbilical artery blood acid base assessment is recognized as the gold standard for fetal condition. Umbilical arterial acidemia has been defined as a pH <7.20, but several studies have shown that umbilical artery pH of 7.10-7.13 as normal and pathological acidemia has been defined at a pH of <7. Instead of pH, base deficit has been proposed for diagnosis of metabolic acidosis of new born. A base deficit of 12-16 mmol /l was considered as mild umbilical artery acidemia and more than 16 mmols/L as severe acidemia.[4,18-28.]

The incidence of pathologic acidemia has been reported between 0.26% and 1.3% in various populations. When the umbilical artery pH is <7 increased incidence of major morbidities, such as intracranial hemorrhages, seizures, respiratory distress and death were observed.[21,29-31]

National Institute of Child Health and Human Development Research Group (NICHHD) suggested other methods for additional evaluation of EFM with fetal scalp blood sampling, fetal stimulation tests, ST segment analysis (STAN) and fetal pulse oximetry (FPO).[13]

Fetal scalp blood sampling (FBS) was first used by Saling in the early 1960's as an adjunct to intermittent auscultation. It is not done regularly because it is invasive, inconvenient to patients, costly and cannot be used in presence of membranes, infection or low lying placenta. A minimum of 35 μl of capillary blood is required. FBS measurements may be inaccurate in presence of scalp edema. Maternal hyperventilation can lead to a rise in pH and obscure true fetal acidosis. National Institute of clinical excellence (NICE) – 2001 has recommended actions on FBS

results. When pH ≥ 7.25 FBS should be repeated if FHR abnormality persists, if it is 7.21 to 7.24 FBS should be repeated within 30 minutes or delivery should be considered or if it is < 7.20 delivery is indicated.[4,9,32,33] Fetal scalp or acoustic stimulation resulting in FHR accelerations of 15 or 10 bpm is used to evaluate an EFM tracing. RCOG has recommended in presence of non reassuring FHR patterns to rule out fetal acidemia. A positive response of fetal heart rate acceleration is associated with a normal scalp pH. It has a 100% negative predictive value, 100% sensitivity and has only 50% positive predictive value.[4,13,34 - 36]

Continuous pH, PO2 and PCO2 probes were developed in 1980s and none of these measurements are used in current clinical practice mainly because of technical problems to record continuously throughout labor. Continuous pH monitoring requires scalp incision of 2 mm and insertion of glass electrode tip to a depth of 3 mm. For continuous PO_2 and PCO_2 monitoring the site requires shaving and attachment with vacuum or glue.[4,21,37]

Fetal arterial oxygen saturation (SPO_2) monitoring or fetal pulse oximetry (FPO) was invented in late 1980s. It measures the ratio of oxyhemoglobin to the sum of oxyhemoglobin and deoxyhemoglobin in blood. The measured ratios are converted to an oxygen saturation percentage (SPO_2). Normal fetal SPO_2 ranges from 30% to 70% and SPO_2 of 30% is the cut off value. The pulse oximeter sensor is placed through the cervix after membrane rupture and is applied along the fetal cheek. ACOG committee has recommended prospective RCT's to evaluate the clinical use of FPO for fetal assessment.[4,8,13,21,38 - 40]

ST segment analysis (STAN) of the fetal ECG was developed in 1980. Studies have shown that hypoxaemia can alter the shape of the fetal ECG wave form by

myocardial hypoxia mainly a shortened PR interval, T wave elevation and ST segment depression. Currently it is not available for general clinical use.[4, 21, 41]

Near infrared spectroscopy is a noninvasive optical method of intrapartum fetal assessment introduced in the late 1980's. Light emitting diodes with wavelengths between 700 and 1000 mm provide measurement of cerebral blood flow, blood volume, oxyhemoglobin, deoxyhaemoglobin and oxidized cytochrome oxidase. It measured cerebral oxygenation as well as perfusion. One study has shown a significant correlation between fetal cerebral oxygen saturation and umbilical cord blood gases at delivery. At present no randomized clinical trial have been conducted and is not commercially available.[4,21]

Fetal scalp blood analysis for lactate - Lactate is a direct marker of anaerobic metabolism and can be measured by a simple technique by using 5 µl of blood on a test strip, in a electrochemical device with in 60 seconds on bedside. Studies have shown that Lactate level of 2.9 to 3.08 mmols / L. (95th to 99th percentiles) are considered suspicious and levels more than 3.08 mmols / L are abnormal and needs intervention. A study has showed a good correlation between lactate levels and pH / base deficit. Not used widely because of non availability of microvolume equipment.[4,21,42-44]

In a prospective study various types of fetal heart rate patterns were analyzed in early labour in low and high risk pregnancies and their correlation with perinatal out come were studied. It was found that 13% of low risk pregnancies had abnormal fetal heart rate patterns and became high risk in labour. No statistically significant difference was seen in base line heart rate and variability patterns in low and high risk pregnancies. All types of decelerations were frequently seen in high risk pregnancies late decelerations were found to have worst fetal prognosis.[45]

In a study of limitations in the clinical prediction of intrapartum fetal asphyxia, risk factors were present in approximately half the pregnancies at the onset of labor and in 2/3 of pregnancies before delivery. Significant proportion of intrapartum fetal asphyxia occurred in pregnancies with no risk factors. The incidence of intrapartum fetal asphyxia was found to be 2%. Early recognition of intrapartum asphyxia during labour could be screened by EFM and to improve the sensitivity to decrease the false positives, diagnostic methods like fetal ECG or continuous recording of fetal blood gases were recommended.[46]

In another study the fetal heart rate patterns were interpreted as effect of factors like maternal, fetal and labor effects other than fetal hypoxia. Fetal heart rate records were scored for each 20 minutes period for a maximum of 8 hours prior to delivery and different FHR characteristics were assessed. A significant correlation was seen between decreased fetal weight, gestational age percentile and variable deceleration. Significant higher mean baseline fetal heart rate were seen with abnormal labour and decreased or absent baseline FHR variability was seen in patients with abnormal labour. No relationships were demonstrated between abnormal labour and FHR accelerations or FHR decelerations.[47]

In another study of predictive value of electronic fetal monitoring for intrapartum fetal asphyxia with metabolic acidosis. FHR record was scored in 10-minute cycles over the last 4 hours of labour so that the baseline periodic FHR variables could be quantified. It was shown that FHR patterns with absent baseline variability were the most specific for fetal asphyxia and were seen in only 17% of asphyxia group. The estimated positive predictive value ranged from 18.1% to 2.6% and negative predictive value ranged from 98.3% to 99.5%. A narrow 1 hour window of FHR patterns including minimal baseline variability and late or prolonged

deceleration would predict fetal asphyxial exposure before decompensation and new born morbidity. With careful interpretation predictive FHR patterns could be a useful screening test for fetal asphyxia. But supplementary tests were recommended to confirm the diagnosis and identify large number of false positive patterns to avoid unnecessary interventions. Continuous scoring of FHR records were required to identify the predictive FHR patterns.[48]

In another study of correlation of neonatal acid base status with APGAR score and fetal heart rate tracings, intrapartum fetal heart rate patterns and APGAR scores showed a high incidence of false positive results. Acidosis was confirmed in only 44% of abnormal FHR patterns by pH criteria and hence suggested that the combination of FHR monitoring, cord blood pH and APGAR assessment for evaluation of fetal status just before delivery. [16]

In a study of validity of CTG monitoring for diagnosis of acute fetal hypoxia, it was found that out of 100% of abnormal CTG patterns only 36.2% were valid and the remaining 63.8% of infants were born healthy with no signs of hypoxia, unnecessary cesarean sections were done in those cases. The predictive value for different CTG patterns were 47.6% for variable decelerations, 34.5% for late decelerations and 29.3% for early decelerations. The conclusion was to use additional methods like fetal pulse oximetry or scalp blood analysis to reduce unnecessary cesarean sections.[5]

In another study of electronic fetal heart rate monitoring and early neonatal outcomes, FHR tracing was examined for the 1 hour period preceding delivery and classified into normal, fetal stress or as fetal distress. They concluded that the interpretation of fetal heart rate tracings by simple classification system predicts normal outcomes accurately and fetuses in true distress could also be predicted.[49]

In another study on prediction and prevention of intrapartum asphyxia in term pregnancies, FHR patterns were analyzed retrospectively on the findings in six 10-mincycles of FHR recording. They concluded that continuous fetal heart rate monitoring would be useful when supplemented by fetal blood gas and acid-base assessment and this would not prevent all cases of moderate or severe fetal hypoxia. But with intervention and delivery during the first or second stage of labour progression of mild asphyxia to moderate or severe asphyxia could be prevented.[2]

In a study of significance of umbilical artery blood gases and APGAR scores and the effects of perinatal and obstetric factors in new born, 1 and 5 minutes APGAR scores of infants delivered by vaginal or cesarean section were determined and umbilical artery blood gases were analyzed. They found a significant positive correlation between the two. It was concluded to use umbilical artery blood gases and APGAR score together to assess the new born.[50]

In a study of umbilical cord blood analysis of all newborns by vaginal delivery, atleast a 10 minute CTG recording in II stage was done and cord blood analysis was done immediately after delivery and it was considered as the gold standard assessment of uteroplacental function and fetal oxygenation / acid base status at birth. It was found that umbilical cord blood gases value reflect the last moment of fetal oxygenation and acid base balance prior to delivery.[27]

Guidelines for EFM monitoring prior to any form of fetal monitoring the maternal pulse palpation is advised simultaneously with fetal heart rate auscultation in order to differentiate between maternal and fetal heart rate. In case of suspicion of fetal death, despite the presence of apparently recorded fetal heart rate, ultra sound diagnosis of fetal viability is advised.[9,32] Settings of CTG machines is standardized

with a paper speed of 1cm per minute, fetal heart rate range between 50-210bpm are advised. [32]

FETAL DISTRESS

Fetal distress is a syndrome characterized by an alteration in fetal heart rate, either excessive to over 160 beats per minute, or slow, to below 100-110 beats per minute, or irregularities in cardiac rhythm. The passage of meconium by the fetus in the absence of a breech presentation is an additional sign of distress; with it, the risk to the fetus is said to be increased. Under these circumstances, there is a tendency to deliver the fetus by operative measures, by Cesarean section if the cervix is not fully dilated, or by operative vaginal delivery if the second stage of labour has been entered.[51]

The key outcome measures to assess the role of EFM are:

- Absolute outcome measures – Perinatal death
  Cerebral Palsy
  Neurodevelopmental disability
- Intermediate fetal/ Neonatal measures of hypoxia:
  APGAR score at 5 min
  Umbilical artery acid base status
  Neonatal encephalopathy. [32]

CARDIOTOCOGRAPHY

The cardiotocograph (CTG) is a continuous electronic record of the fetal heart rate obtained either via an ultrasound transducer placed on the mother's abdomen, a second transducer is placed on the mother's abdomen over the uterine fundus to record frequency and duration of uterine contractions. Both components are then

traced simultaneously on a paper strip. The International Federation of Gynaecologists and Obstetricians (FIGO) guidelines for interpretation of intrapartum cardiotocogram distinguish 2 levels of abnormalities, suspicious and pathological. The American College of Obstetricians and Gynaecologists (ACOG) published a practice bulletin on intrapartum fetal heart rate monitoring in 2009. [52]

FIG 1: CARDIOTOCOGRAPH

CTG Indications[53]

1. LABOUR

- Induced labour.
- Augmented labour.
- Prolonged labour.
- Prolonged PROM.
- Regional Anesthesia.
- Previous Cesarian Section.
- Abnormal Uterine Activity

2. FETUS

- Multiple Gestation
- Small baby

- Preterm
- Postdated
- Oligohydrominos
- RHO isoimmunised
- Breech

3. FETAL DISTRESS SUSPECTED

- Meconium stained liquor
- Abnormal FHR on auscultation
- Vaginal Bleeding
- Infection

4. HIGH RISK CASES

- Hypertension
- Diabetes
- Renal disorders
- Anemia
- Haemoglobinopathies
- Cardiac
- Hypothyroidism
- Collagen disorders[53]

Factors That Affect Fetal Heart Rate

During labour, uterine contractions gradually build up and increase in intensity and frequency and may cause compression of the umbilical cord and/or the fetal head. These "mechanical compressions" may result in decelerations in fetal heart resulting in early and variable decelerations, respectively. If hypoxic or mechanical insults

persist for a longer period, then the fetus utilizes its adrenal gland to cope with this on going stress, leading to a "stress response" This "stress response" that occurs through the release of catecholamines from the adrenal glands and represents a physiological mechanism for coping with mechanical hypoxic insults of labour may not be fully operational in a preterm baby. This may also be the case when the normal physiological reserves of the fetus may be impaired (intrauterine growth restriction, fetal infection). Inability of a preterm or growth restricted fetus to mount a required stress response may lead to maladaptive responses resulting in permanent hypoxic insult on the fetal brain occurring at a lower threshold than in the term fetus.

Fetal heart rate is regulated by the autonomic nervous system consisting of 2 branches; the parasympathetic and sympathetic branch which exerts opposing influences on the FHR.

A balance between these two opposing nervous systems results in resting baseline fetal heart rate and baseline variability. During fetal development, the sympathetic nervous system that is responsible for survival ("fight or flight" response) develops much earlier than the parasympathetic nervous system ("rest and sleep") that develops during the third trimester. Hence, a preterm fetus may have a higher baseline fetal heart rate with apparent reduction of baseline variability due to unopposed action of sympathetic nervous system.[52]

In 1997, the National Institute of Child Health and Human Development Research Planning Workshop (NICHHD) proposed definitions for Fetal Heart Rate Patterns.[54,55].

In 2005-WHO and ACOG reviewed 1997 document and adopted it in 2005.

Definitions of Fetal Heart Patterns

Baseline - The mean FHR rounded to increments of 5 beats per minute during a 10minute segment, excluding:

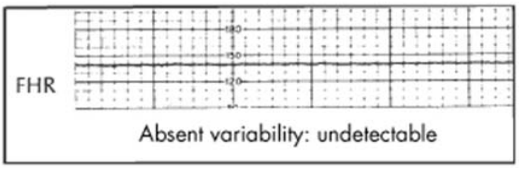
- Periodic or episodic changes
- Periods of marked FHR variability
- Segments of baseline that differ by > 25 bpm
- The baseline must be for a minimum of 2 min in any 10 min segment.

  Baseline Variability – Fluctuations in the FHR of two cycles per min or greater

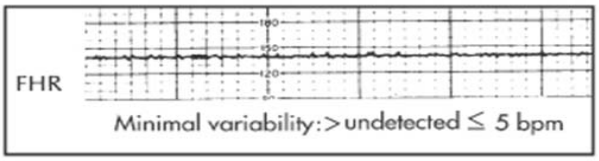
Variability

It is visually quantitated as the amplitude of peak-to troughin bpm

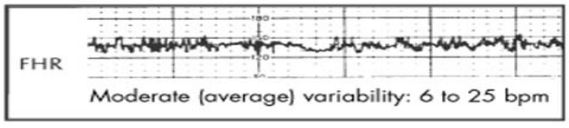
- Absent-amplitude range undetectable



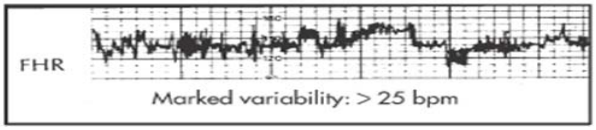
- Minimal-amplitude range detectable but 5bpm or fewer



- Moderate(normal)-amplitude range 6-25bpm

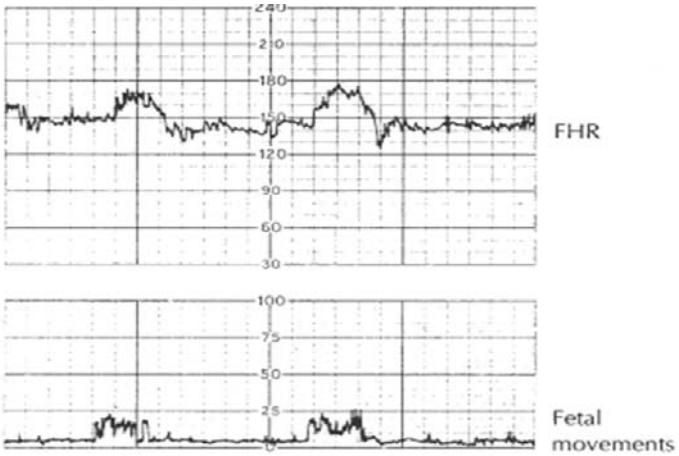


- Marked-amplitude range >25bpm



Acceleration

- A visually apparent increase (onset to peak in < than 30 secs) in the FHR from the most recently calculated baseline.
- The duration of an acceleration is defined as the time from the initial change in FHR from the baseline to the return of the FHR to the baseline.
- At 32 weeks of gestation and beyond, an acceleration has an acme of 15 bpm or more above baseline, with a duration of 15secs or more but < 2min
- Before 32 weeks of gestation, an acceleration has an acme of 10 bpm or more above baseline with a duration of 10secs or more but < 2min
- Prolonged acceleration lasts 2min or more but < 10min
- If an acceleration lasts 10 min or longer, it is a baseline change



Acceleration of fetal heart rate movement.

Bradycardia - Baseline FHR < 110 bpm for ≥10 min

Early Deceleration

- In association with a uterine contraction, a visually apparent, gradual (onset to nadir 30secs or more) decrease in FHR with return to baseline
- Nadir of the deceleration occurs at the same time as the peak of the contraction

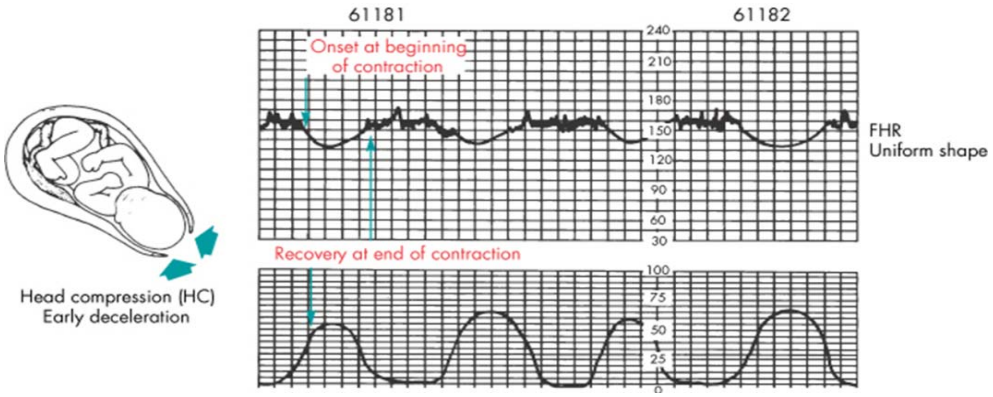
Shape is uniform, symmetrical

Must be repetitive

Typically a mirror image of the corresponding contraction

Rarely drops below 100 bpm

Considered benign with usually no intervention needed (FHR returns to baseline as pressure on fetal head is released)



Early decelerations caused by head compression

Late Deceleration

Late- associated with uteroplacental insufficiency.

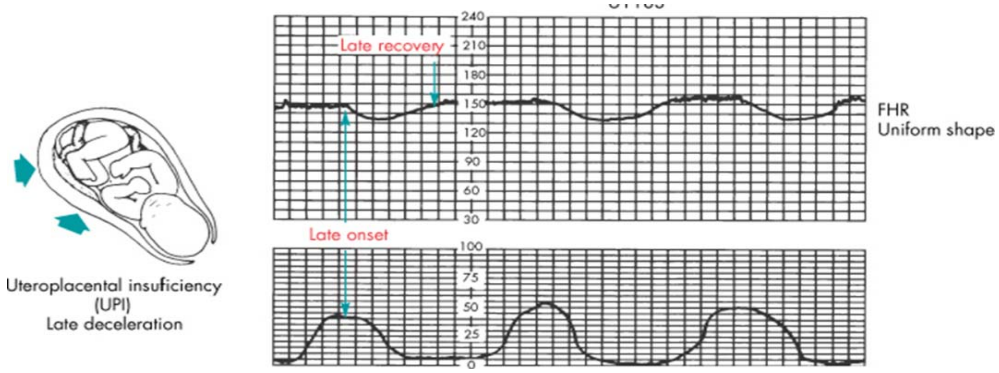
Shape is uniform and symmetrical, a visually apparent, gradual decrease and return of the FHR associated with a uterine contraction

Must be repetitive

Rarely less than 100 bpm; lowest point after peak of uterine contraction.

Clinical significance: Omnious – Indicates decreased O_2 available to fetus; hypoxia

Intervention usually needed.



Late decelerations caused by uteroplacental insufficeincy

Tachycardia – Baseline FHR > 160bpmfor ≥10 min

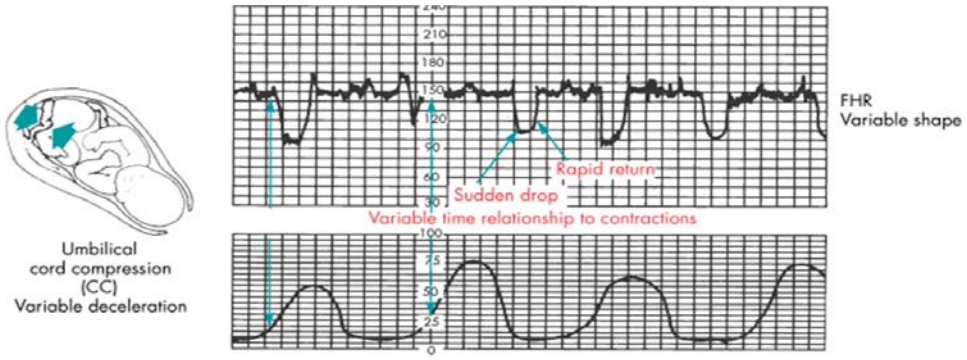
Variable Deceleration

- An abrupt (onset to nadir < 30secs), visually apparent decrease in the FHR below the baseline.
- The decrease in FHR is 15 bpm or more, with a duration of 15 secs or more but <2min, associated with cord compression.

Often drops below 100 bpm

Shape may be U, V, or W

Return to baseline varies; may have rapid or prolonged return to baseline



Variable decelerations caused by cord compression.

Prolonged Deceleration

- Visually apparent decrease in the FHR below the baseline
- Deceleration is 15 bpm or more, lasting 2min or more but <10min from onset to baseline.

Uterine Tone:

The lowest intrauterine pressure between contractions is called resting tone. Normal resting tone is 5-10 mmHg; during labor resting tone may rise to 10-15 mmHg. Pressure during contractions rises to ~25-100 mmHg (varies with stage). A resting pressure above 20 mmHg causes decreased uterine perfusion. Determined by palpation during external monitoring. Montevideo units (MVUs) if internal . Add peak pressures from all contractions in a 10 min period and subtract sum of baseline tones of the contractions – normal 180-250; more common practice today is to just add peak pressures from baseline in a 10 minute period.

RCOG Guidelines to review EFM in a patient without complication is once in every 30 minutes in first stage of labor and every 15 minutes in the second stage. Inpatient with complication is every 15 minutes in the first stage of labor and every 5 minutes during the second stage.[9,32]

The **ACOG committee** recommends the use of the term non-reassuring fetal status instead of fetal distress for features of repetitive late or variable decelerations, tachycardia, loss of variability or bradycardia. In the presence of non-reassuring fetal status fetal scalp blood sampling was advised to continue the labor process.[13]

APGAR SCORE

This scoring system is a useful clinical tool to identify those neonates who require resuscitation as well as to assess the effectiveness of any resuscitative measures (APGAR, 1953). As shown in Table below, each of the five easily identifiable characteristics—heart rate, respiratory effort, muscle tone, reflex irritability, and color is assessed and assigned a value of 0 to 2. The total score, based on the sum of the five components, is determined 1 and 5 minutes after delivery[56].

APGAR SCORE TABLE

SIGN	0 POINT	1POINT	2POINTS
HEART RATE	Absent	<100bpm	≥ 100bpm
RESPIRATORY EFFORT	Absent	Slow irregular	Good, crying
MUSCLE TONE	Flaccid	Some flexion of extremities	Active motion
REFLEX IRRITABILITY	No response	Grimace	Vigorous cry
COLOR	Blue, pale	Body pink extremeties blue	Completely pink

The 1-minute APGAR score reflects the need for immediate resuscitation. The 5-minute score, and particularly the change in score between 1 and 5 minutes, is a useful index of the effectiveness of resuscitative efforts. The 5-minute APGAR score also has prognostic significance for neonatal survival, because survival is related closely to the condition of the neonate in the delivery room. This risk compares with a mortality rate of 1 in 4 for term infants with scores of 3 or less. Low 5-minute scores were comparably predictive of neonatal death in preterm infants. These investigators concluded that the APGAR scoring system is as relevant for the prediction of neonatal survival today as it was almost 50 years ago.

Despite the methodological challenges, erroneous definitions of asphyxia by many groups were established solely based upon low APGAR scores. These prompted the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics to issue a joint statement in 1986 concerning "Use and Abuse of the APGAR Score." The statement was updated in 1996 and has been reaffirmed several times, the most recent in 2006. Important caveats regarding APGAR score interpretation addressed in this statement include the following:

1. Because certain elements of the APGAR score are partially dependent on the physiological maturity of the newborn, a healthy preterm infant may receive a low score only because of immaturity (Amon and associates, 1987; Catlin and co-workers, 1986).
2. Given that APGAR scores may be influenced by a variety of factors including, but not limited to, fetal malformations, maternal medications, and infection, to equate the presence of a low APGAR score solely with asphyxia or hypoxia represents a misuse of the score.

3. Correlation of the APGAR score with adverse future neurological outcome increases when the score remains 3 or less at 10, 15, and 20 minutes, but still does not indicate the cause of future disability (Freeman and Nelson, 1988; Nelson and Ellenberg, 1981).
4. The APGAR score alone cannot establish hypoxia as the cause of cerebral palsy. A neonate who has had an asphyxial insult proximate to delivery that is severe enough to result in acute neurological injury should demonstrate all of the following: profound acidemia with cord artery blood pH < 7 and acid-base deficit 12mmol/L; APGAR score of 0–3 persisting for 10 minutes or longer; neurological manifestations such as seizures, coma, or hypotonia; and multisystem organ dysfunction—cardiovascular, gastrointestinal, hematological, pulmonary, or renal[56].

CORD BLOOD pH

Blood taken from umbilical vessels may be used for acid-base studies to assess the metabolic status of the neonate. Blood collection is performed following delivery by immediately isolating a 10- to 20-cm segment of cord with two clamps near the neonate and two clamps nearer the placenta. The importance of clamping the cord is underscored by the fact that delay of 20 to 30 seconds can alter both the PCO2 and pH (Lievaart and deJong, 1984). The cord is then cut between the two proximal and two distal clamps. Arterial blood is drawn from the isolated segment of cord into a 1- to 2-ml commercially prepared plastic syringe containing lyophilized heparin or a similar syringe that has been flushed with a heparin solution containing 1000 U/mL. The needle is capped and the syringe transported, on ice, to the laboratory. Although efforts should be made for prompt transport, neither the pH nor PCO2 change

significantly in blood kept at room temperature for up to 60 minutes (Duerbeck and associates, 1992). In fact, Chauhan and colleagues (1994) developed mathematical models allowing reasonable prediction of birth acid–base status in properly collected cord blood samples analyzed as late as 60 hours after delivery.[57]

- **Fetal Acid-Base Physiology**

The fetus produces both carbonic and organic acids. Carbonic acid (H_2CO_3) is formed by oxidative metabolism of CO_2. The fetus usually rapidly clears CO2 through the placental circulation, which limits the buildup of carbonic acid. When H_2CO_3 accumulates in fetal blood and there is no concurrent increase in organic acids as occurs in impaired placental exchange the result is termed respiratory acidemia.

Organic acids primarily include lactic and hydroxyl butyric acids. Increased levels of these acids follow persistent placental exchange impairment and result from anaerobic glycolysis. These organic acids are cleared slowly from fetal blood, and when they accumulate without a concurrent increase in H_2CO_3, the result is termed metabolic acidemia. With the development of metabolic acidemia, bicarbonate (HCO_3) decreases because it is used to buffer the organic acid. An increase in H_2CO_3 accompanied by an increase in organic acid reflected by decreased HCO_3 causes mixed respiratory-metabolic acidemia.

In the fetus, respiratory and metabolic acidemia, and ultimately tissue acidosis, are most likely part of a progressively worsening continuum. This is different from the adult pathophysiology, in which distinct conditions result in either respiratory (pulmonary disease) or metabolic (diabetes) acidemia. In the fetus, the placenta serves as both the lungs and to a certain degree, the kidneys. One principal cause of developing fetal acidemia is a decrease in uteroplacental perfusion. This results in the

retention of CO_2 (respiratory acidemia), and if protracted and severe enough, a mixed or metabolic acidemia.

Assuming that maternal pH and blood gases are normal, the actual pH of fetal blood is dependent on the proportion of carbonic and organic acids as well as the amount of bicarbonate, which is the major buffer in blood. This can best be illustrated by the Henderson–Hasselbalch equation:

For clinical purposes, HCO3– represents the metabolic component and is reported in mEq/L. The H2CO3 concentration represents the respiratory component and is reported as the PCO2 in mm Hg.

The result of this equation is a pH value. However, pH is a logarithmic term and does not give a linear measure of acid accumulation. For example, a change in hydrogen ion concentration associated with a fall in pH from 7.0 to 6.9 is almost twice that which is associated with a fall in pH from 7.3 to 7.2. For this reason, the delta base offers a more linear measure of the degree of accumulation of metabolic acid (Armstrong and Stenson, 2007). The change in base, or delta base, is a calculated number used as a measure of the change in buffering capacity of bicarbonate (HCO_3). For example, HCO_3 concentration will be decreased with a metabolic acidemia as it is consumed to maintain a normal pH. A base deficit occurs when HCO_3 concentration decreases to below normal levels, and a base excess occurs when HCO_3 values are above normal. Importantly, a mixed respiratory–metabolic acidemia with a large base deficit and a low HCO_3 less than 12 mmol/L is more often associated with a depressed neonate than is a mixed acidemia with a minimal base deficit and a more nearly normal HCO_3.

- **Clinical Significance of Acidemia**

Fetal oxygenation and pH generally decline during the course of normal labor (Dildy and co-workers, 1994). Using data from more than 19,000 deliveries, the lower limits of normal pH in the newborn have been found to range from 7.04 to 7.10 (Boylan and Parisi, 1994). Thus, these values should be considered to define neonatal acidemia. Most fetuses will tolerate intrapartum acidemia with a pH as low as 7.00 without incurring neurological impairment (Freeman and Nelson, 1988; Gilstrap and associates, 1989). Supportive of this threshold, Goldaber and associates (1991) found that there were significantly more neonatal deaths and infants with neurological dysfunction below a pH of 7.00.[57]

Indications for obtaining Cord blood pH[58]

- Cesarean section for fetal compromise
- Low 5 minute APGAR Score
- Severe growth restriction
- Abnormal FHR
- Maternal thyroid disease
- Intrapartum fever
- Multifetal gestation

FIG 2: PRE HEPARNISED SYRINGES

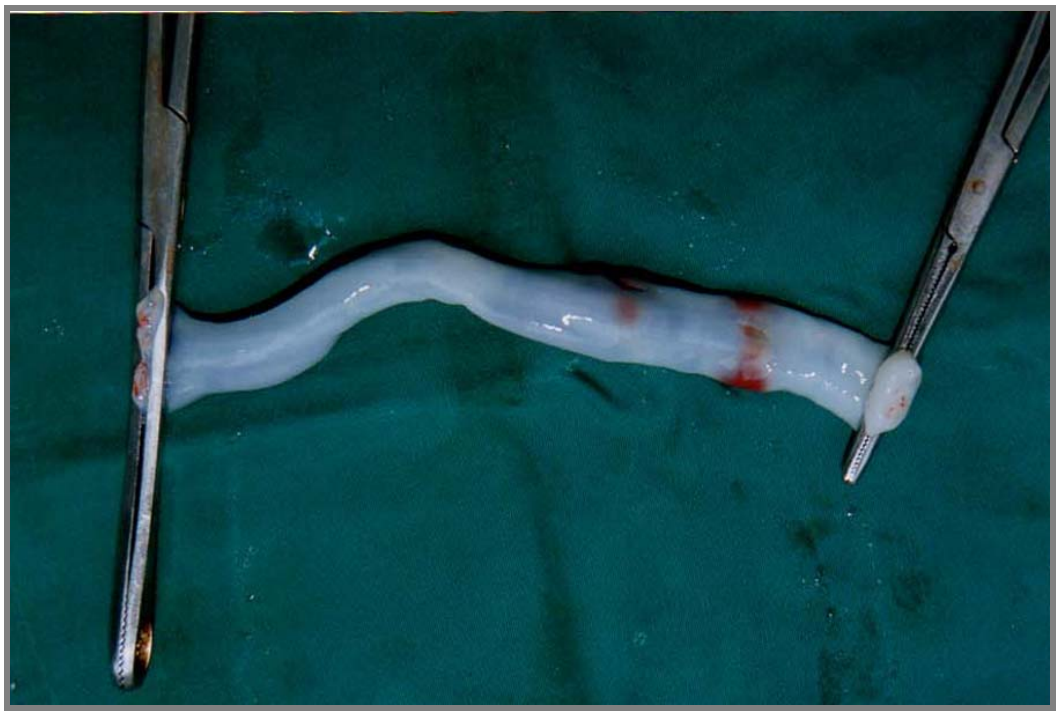


FIG 3 : UMBLICAL CORD

FIG 4 : ABG APPARATUS

MATERIALS AND METHODS

This was a prospective comparative study conducted among 200 women admitted in labour for delivery with singleton term pregnancy in vertex presentation to RLJH hospital, Kolar between 1st January 2013 to 31st July 2014. Ethical committee approval was taken prior to commencement of study. Women fulfilling the following inclusion criteria were recruited to study after obtaining informed consent.

INCLUSION CRITERIA

Singleton pregnancy

Term gestation

Vertex presentation

EXCLUSION CRITERIA

Preterm deliveries

Twin gestations

Antepartum haemorrhage

Women requiring emergency cesarean section on admission.

METHODOLOGY

A detailed obstetric history was taken and recorded in the proforma. General and obstetric examination was done to confirm gestational age, lie, presentation, contractions and fetal heart rate. Per vaginal examination to ascertain cervical effacement, dilatation, station of fetal head, pelvic assessment for adequacy done. Labour were monitored by noting uterine contractions and fetal heart rate with intermittent auscultation. Labor was augmented whenever required with oxytocin and instrumental delivery or cesarean section were done if indicated. CTG tracings were taken in active stage of labour, preferably 30minutes before delivery or even earlier with FHR irregularities and correlated with APGAR score and cord blood pH. FOETAL-MONITOR-BPL-FM 9853 and HUNTLIVH BD 4000 excess machines with external transducer with tocodynometer were used for CTG monitoring.

Suspicious and pathological FHR patterns were considered as signs of fetal hypoxia and necessary intervention were:

In case of suspicious CTG, interventions were done

- Change of maternal position (Left lateral position)
- Correction of maternal hypotension
- Increase IV fluid rate or give bolus, especially if maternal hypotension or dehydration present
- Administeration of Oxygen at 8-10 l pm by mask
- Delivery of fetus if pattern not correctable.

A total of 229 cases were included. Among these 29 cases were excluded because of inadequate CTG tracings or delayed cord blood gas analysis.

The fetal heart rate patterns were analysed according to guidelines of National Institute of Child Health and Human Development Research Planning Workshop

(NICHHD) and National Institute of Clinical Excellence (NICE) 2001 and grouped in to normal, suspicious or pathological groups.

Normal: A CTG where all four features fall into the reassuring category.

Suspicious: A CTG whose features fall into one of the non – reassuring categories and the remainder of the reassuring.

Pathological: A CTG whose features fall into two or more non reassuring or one or more abnormal categories.

Reassuring features are those with baseline 110-160 bpm, a variability of ≥ 5 bpm with accelerations and absence of deceleration.

Non reassuring features are baseline between 100-109 or 161 to 180 bpm with avariability of < 5 bpm for > 40 to < 90 minutes, decelerations of early, variable or single prolonged upto 3 minutes with absence of accelerations.

Abnormal features includes a CTG with baseline < 100 bpm ,> 180 bpm or sinusoidal pattern for > 10 minutes, variability < 5 bpm for 90 minutes, a typical variable decelerations, late decelerations or single prolonged decelerations for > 3 minutes.

Following delivery APGAR score was noted at 1 minute and 5 minutes.

Immediately after delivery of the neonate, a segment of umbilical cord (10-15 cm) was double clamped, blood was collected from umbilical artery in a pre-heparinised syringe and blood gas analysis was done immediately (with in 15-20 minutes) in a arterial blood gas analyser.

Umbilical artery blood gas pH of 7.2-7.36 is considered as normal 7.2 to 7.0 as moderate and < 7.0 as severe acidosis. Base deficit was more representative of

metabolic acidosis and the cut off of 12-16 mmol / L for mild and > 16 mmol/L for severe umbilical artery acidemia was used.

All neonatal intensive care unit admissions were noted and followed up till discharge.

STATISTICAL ANALYSIS

Significant figures

+ Suggestive significance (P value: $0.05<P<0.10$)

* Moderately significant (P value:$0.01<P \leq 0.05$)

** Strongly significant (P value : $P \leq 0.01$)

Statistical software: The Statistical software namely SAS 9.2, SPSS 15.0, Stata 10.1, MedCalc 9.0.1 ,Systat 12.0 and R environment ver.2.11.1 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

RESULTS

The study population included 200 women, admitted for delivery with term, singleton pregnancy with vertex presentation. Those with pre term, twin pregnancy, IUD and antepartum haemorrhage were excluded.

Table 1: Age distribution

Age (years)	No. of cases	%
18-20	**55**	**27.5**
21-25	**110**	**55.0**
26-30	**28**	**14.0**
31-35	**6**	**3.0**
36-40	**1**	**0.5**
Total	**200**	**100.0**

Mean ± SD: 22.98±3.37

The age distributions of study population are shown in table 1. Most 110 (55%) of the women were among 21-25 yrs age group, 55 (27.5%) were among 18-20 yrs , 28(14%) were among 26-30, 6(3%) were among 31-35 and 1(0.5%) were above 35 yrs.

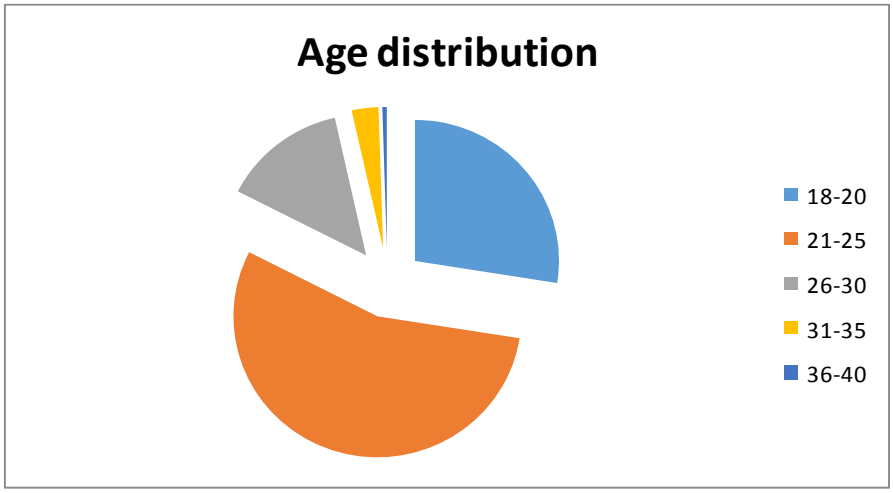


Table 2: Gravidity distribution

Gravidity	No. of cases	%
Primi	**118**	**59%**
G2	**65**	**32.5%**
G3	**11**	**5.5%**
≥G4	**6**	**2%**
Grand Total	**200**	

The Gravidity of study population is shown in table 2.There were mostly primis118(59%) ; 65(32.5%) were gravida 2 and 11(5.5%) gravida 3; 6(3%) were gravida 4 and above.

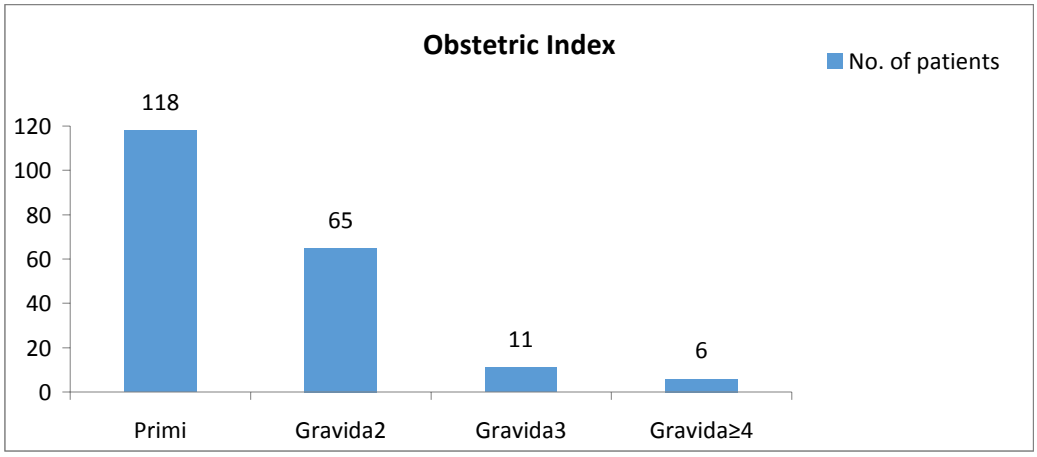


Table 3: Gestational age

Gestational age(weeks)	No. of cases	%
37-40	193	**96.5%**
40-41	7	**3.0%**
Total	**200**	**100.0**

The gestational age of study population are shown in table 3. Most of the women 192 (96%) were in 37-40 weeks ; 7(3.5%) cases in 40-41weeks.

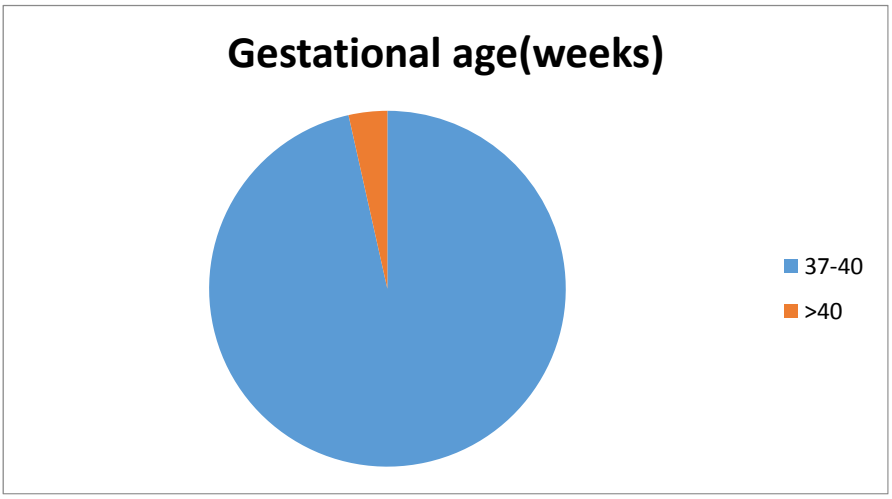


Table 4 Antepartum risk factors

ANTEPARTUM RISK FACTORS	NORMAL CTG (n 100)	SUSPICIOUS CTG (n 26)	PATHOLOGICAL CTG (n 74)
Pre-eclampsia	14	3	10
PROM	6	2	4
Post dated	4	2	1
IUGR			3
Polyhydramnios	2		2
Others(cardiac disease, Rh -ve)	2		3
Total	**28 (28%)**	**7(26.9%)**	**23(31%)**

Antepartum risk factors are shown in table 4.

Among total study population of 200 cases, 58 (29%) were with antepartum risk factors. Pre eclampsia being the most common out of which, 14(14%) in normal ,3(11.5%) in suspicious and 10(13.15%) in pathological CTG pattern. Other antepartum risk factors were PROM, Post Dated pregnancy, IUGR, Polyhydramnios, cardiac diseases, Rh-ve pregnancy. Of all the patients with antepartum risk factors, a total of 28% patients had normal CTG pattern, 26.9% suspicious CTG and 31% pathological CTG.

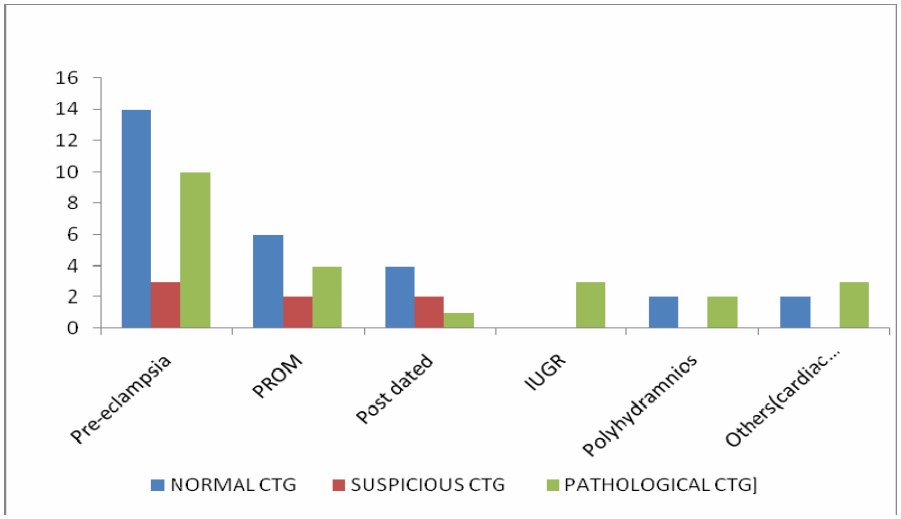


Table 5: Different CTG pattern in the study

CTG Pattern	No. of cases	%
Normal	100	**50.0**
Suspicious	26	**13.0**
Pathological	74	**37.0**
Total	**200**	**100.0**

Among total 200 cases studied 100 (50%) showed normal CTG patterns, 26(13%) suspicious and 74(37%) pathological patterns.

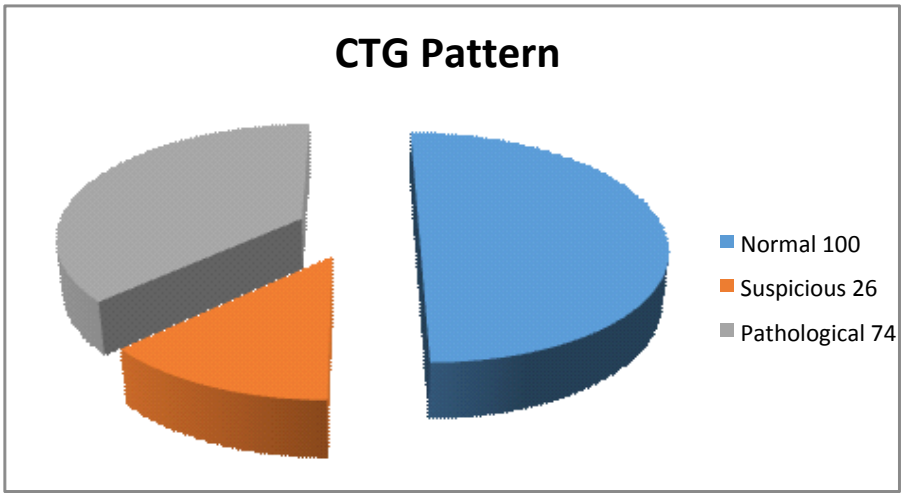


Table 6: Mode of delivery

Mode of delivery	No. of cases	%
Vaginal delivery	114	**57.0**
Instrumental delivery	39	**19.5**
Cesarean delivery	47	**23.5**
Total	**200**	**100.0**

Cesarean deliveries 47(23.5%) were done in 24 cases in active phase, 21 cases in latent phase and 2 cases in second stage. Instrumental delivery were done in 39 (19.5%) 114(57%) cases had vaginal delivery in total study population

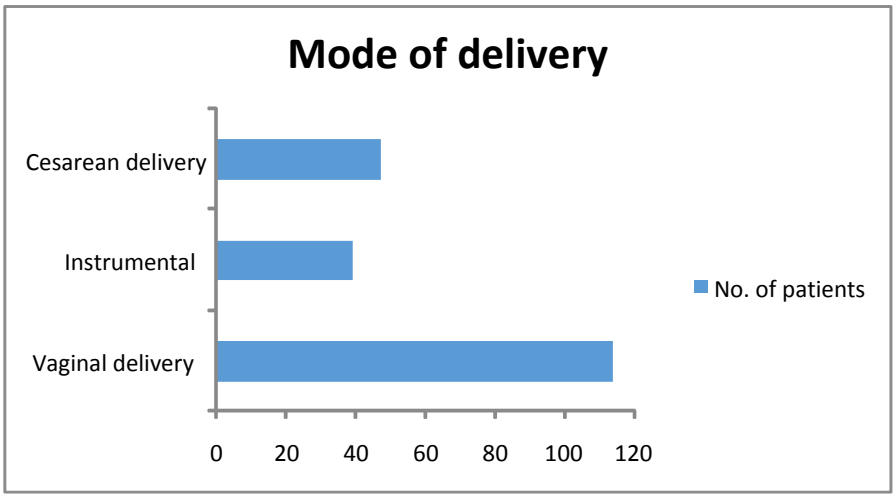


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Table 7: APGAR score

APGAR score	No. of Neonates (n=200)	%	Mean ± SD
1 min			
<7	72	36%	**6.79**
≥7	128	64%	
5 min			
<7	14	7%	**8.50**
≥7	186	93%	

APGAR score at 1 minute <7 was observed in 72 (36%), ≥7 was seen in 128 (64%) with Mean ± SD 6.79. APGAR score at 5 minute <7 was seen in 14 (7%), ≥7 was recodedr in 186 (93%) of neonates with Mean ± SD 8.50

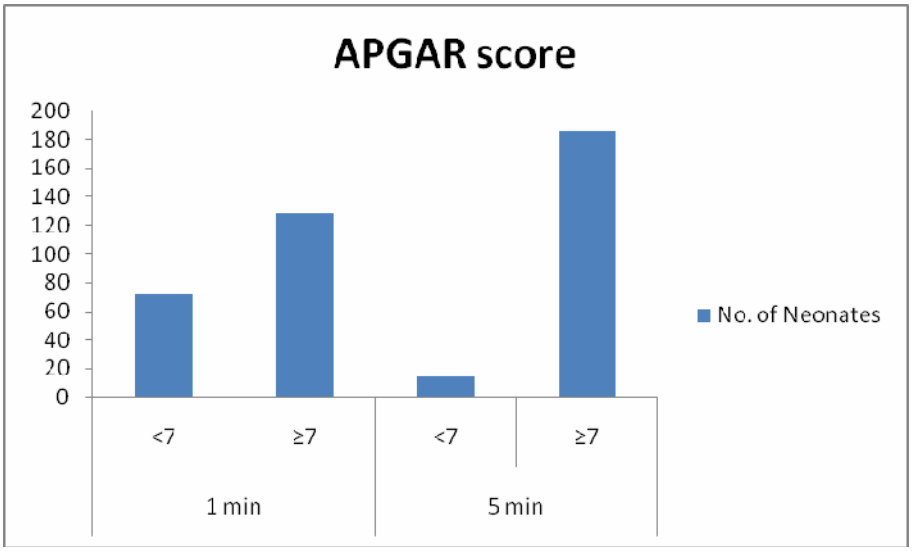

Table 8: Cord blood pH

Cord blood pH	No. of neonates	%
<7.0	13	**6.5**
7.0- 7.19	56	**28%**
>7.2	131	**65.5%**
Total	**200**	**100.0**

Mean ± SD: 7.19±0.09

Moderate and severe acidosis in was noted in 34.5% and severe acidosis in 6.5% of neonates

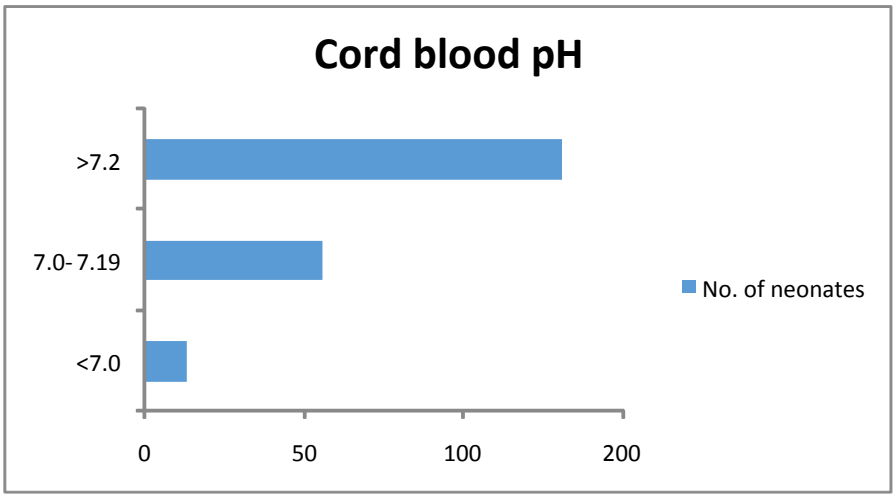

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Table 9: Base deficit

Base deficit mmol/l	No. of neonates	%
<12	162	**81**
12-16	25	**12.5**
≥16	13	**6.5**
Total	**200**	**100.0**

Mean ± SD: 11.51±1.58

In the present study base deficit of 12-16mmol/l were seen in 12.5% and with base deficit of >16mmol/l were seen in 6.5% of the neonates in the pathological groups.

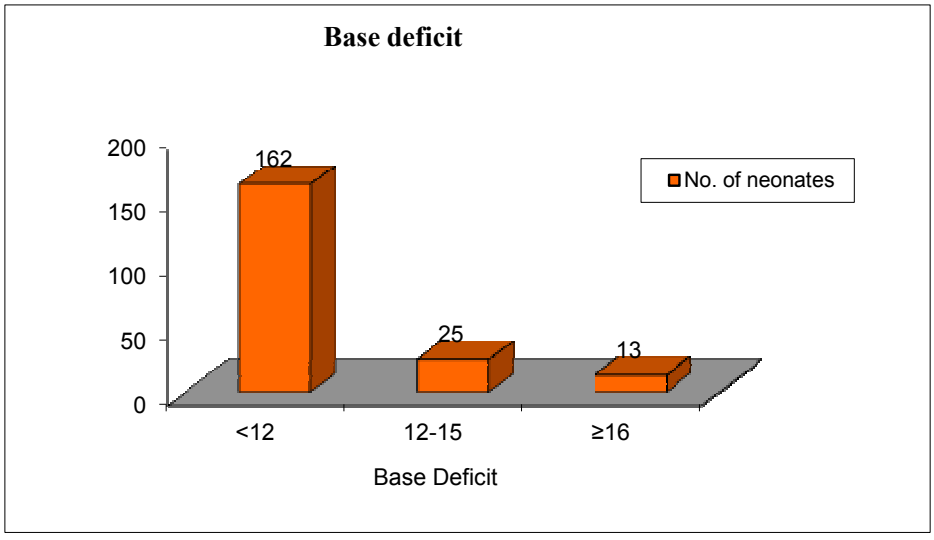


Table 10:Mode of delivery in different CTG patterns

Mode of Delivery	Normal CTG	Suspicious CTG	Pathological CTG	Total
Cesarean Delivery	3 (3%)	8 (31%)	36 (49%)	**47 (24%)**
Instrumental	3 (3%)	10 (38%)	26 (35%)	**39 (20%)**
Vaginal delivery	94 (94%)	8 (31%)	12 (16%)	**114 (57%)**
Total	**100 (100%)**	**26** (100%)	**74** (100%)	**200**

Cesarean sections were done in 24 cases in active phase, 21 cases in latent phase and 2 cases in second stage of labour. Cesarean delivery was done in 3%; 31% and 49% in the normal, suspicious or pathological CTG groups respectively. Instrumental deliveries were done in 3%; 38% and 35% of the cases and vaginal delivery was allowed in 31% of suspicious group and 16% of patients in pathological group. Cesarean section was done in 3 cases for non progress of labor, in 1 case for deep transverse arrest, in 3 cases for abnormal CTG patterns and in all other cases of cesarean section there were different indication. Interventions with instrumental deliveries were done in 39 cases. Out of these 5 cases were prophylactic forceps and in 34 cases with different indications.

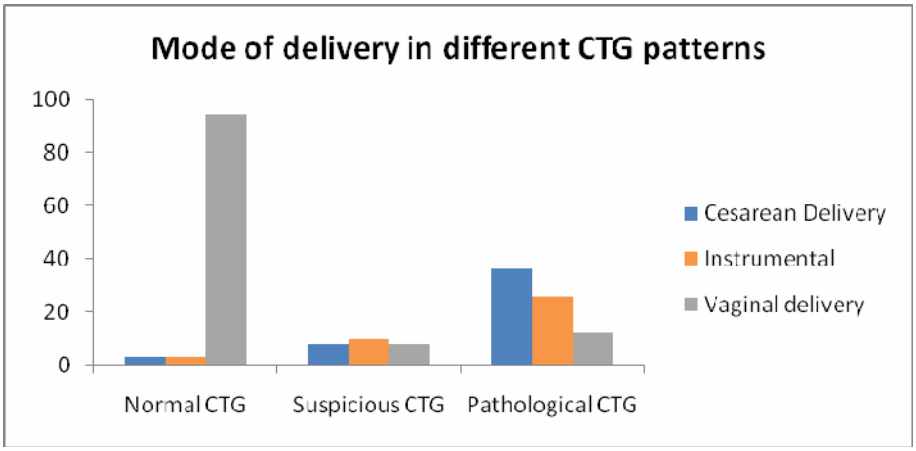


Table No.11 : Correlation of CTG patterns with APGAR score at 1 and 5 minutes

APGAR score	CTG Pattern			Total (n=200)
	Normal (n=100)	Suspicious (n=26)	Pathological (n=74)	
1 min				
<7	3 (3%)	15 (58%)	54 (73%)	**72 (36%)**
≥7	97(97%)	11 (42%)	20 (27%)	**128 (64%)**
5 min				
<7	1 (1%)	3(12%)	10(14%)	**14**
≥7	99 (99%)	23 (98%)	64 (86%)	**186**

APGAR score at 1 minute <7 was seen in 3%, 58% and 73% of the normal, suspicious and pathological groups and showed significant correlation with CTG patterns. APGAR score at 5 minute <7 was seen in 1%, 3% and 14% of the normal, suspicious and pathological groups and showed correlation with CTG patterns.

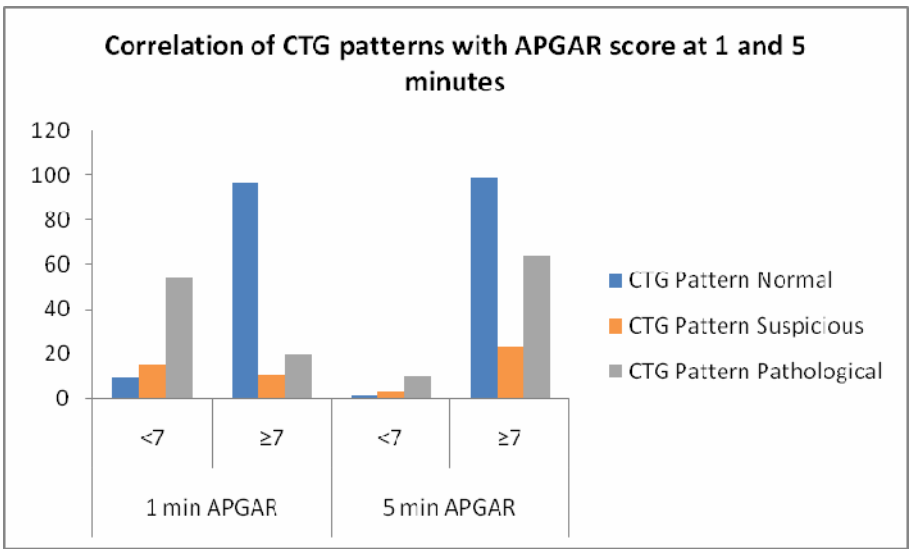


Table 12: Correlation of CTG patterns with cord blood pH

Cord blood pH	CTG Pattern			Total
	Normal	Suspicious	Pathological	
<7.0	0(0%)	1(4%)	12(16%)	**13(6.5%)**
7.0 to 7.19	2 (2%)	14 (54%)	40 (54%)	**56 (28%)**
>7.2	98(98%)	11(42%)	22 (30%)	**131(66%)**
Total	**100(100%)**	**26(100%)**	**74(100%)**	**200(100%)**

Moderate and severe acidosis in pathological group was seen in 70% and severe acidosis in 16% and p value is significant. p=0.001. In suspicious FHR patterns moderate and severe acidosis was seen only in 58% showing evidence of birth asphyxia.

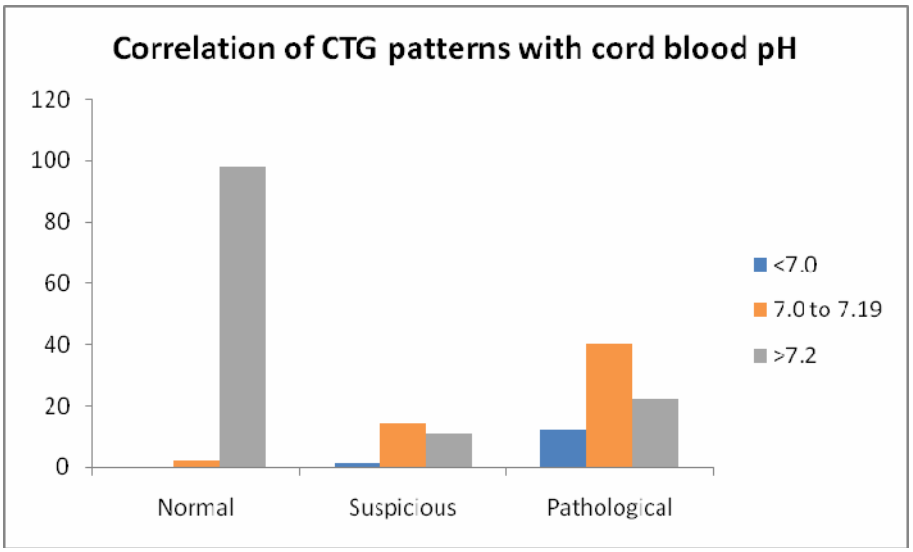


Table 13 : Correlation of CTG patterns with Base deficit

Base deficit mmol/l	CTG Pattern			Total
	Normal	Suspicious	Pathological	
<12	98 (98%)	20(77%)	44 (59%)	**162(81%)**
12-16	2 (2 %)	4 (15%)	19 (26%)	**25(13%)**
>16	0 (0%)	2 (8%)	11 (15%)	**13(7%)**
Total	**100(100%)**	**26(100%)**	**74(100%)**	**200(100%)**

In the present study base deficit of 12-16mmol/l were seen in 26% and base deficit of >16mmol/l were seen in 15% of the cases in the pathological groups.

In the suspicious group only 2 cases showed base deficit >16 and 4 cases showed base deficit 12-16 mmol/ l.

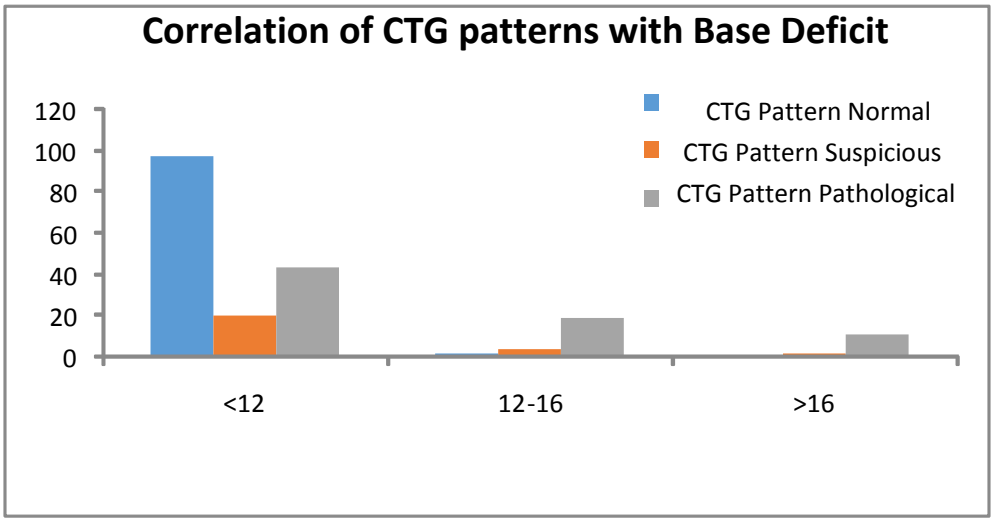


Table 14: Correlation of CTG patterns with other study variables (Mean with SD) of APGAR score at 1min, 5 min, Cord blood pH and Base deficit.

	CTG Pattern			Total	P value
	Normal	Suspicious	Pathological		
APGAR score 1 min	7.58 ±0.71	6.19 ± 1.26	5.93 ± 1.43	**6.79±1.35**	**0.001****
APGAR score 5 min	8.89±0.54	8.11± 1.07	8.12± 0.96	**8.50±0.88**	**0.001****
Cord blood pH	7.23 ±0.02	7.18 ± 0.06	7.13 ± 0.12	**7.19±0.09**	**0.001****
Base deficit	10.80±0.53	11.67±1.75	12.48±2.05	**11.53±1.64**	**<0.001****

In the present study correlation of pathological CTG with APGAR score at 1min, 5 min, cord blood pH and base deficit showed mean with standard deviation of 5.93 ± 1.43, 8.12± 0.96, 7.13 ± 0.12 and 12.48±2.05 respectively showing significant p value.

Similarly correlation of suspicious and normal CTG patterns with APGAR score at 1min, 5 min, cord blood pH and base deficit (mean with standard deviation) showed significant p value

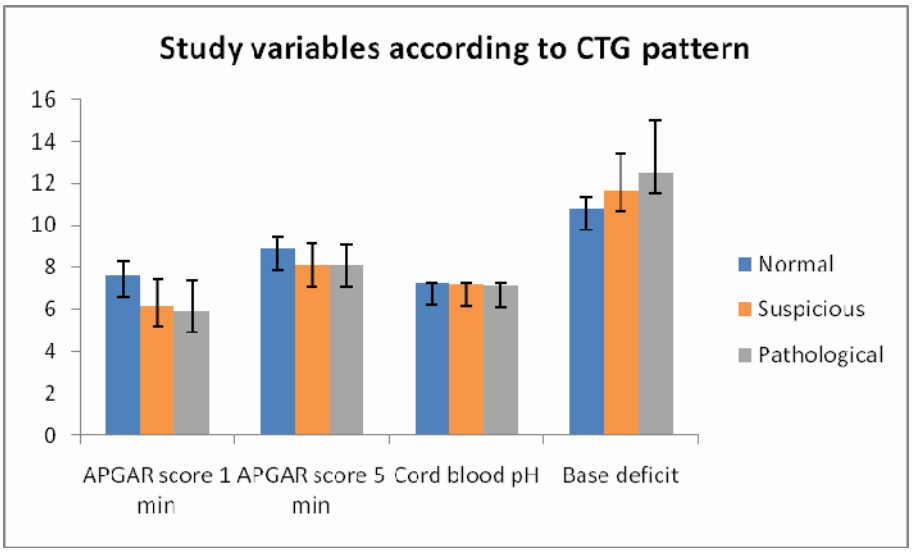


Table 15: Comparison of various FHR patterns with APGAR Score and pH

	n	Mean cord Blood pH	Mean APGAR score at 1 Min	NICU Admissions
Tachycardia	**18**	**7.18**	**6**	**1**
Late decelration	**40**	**7.11**	**5.2**	**4**
Variable decelration	**26**	**7.17**	**6.3**	**4**
Prologned decelration	**8**	**7.02**	**4.9**	**2**
Bradycardia	**5**	**7.00**	**3.75**	**3**
Early decelration	**3**	**7.14**	**8**	
Normal	**100**	**7.28**	**8**	
Total	**200**			**14**

Different FHR patterns are shown in table. Late decelerations were seen in 40 cases, variable decelerations in 26 cases, tachycardia in 18 cases, prolonged decelerations in 8 cases and bradycardia in 5 cases. Among all FHR patterns majority were decelerations and associated with poor neonatal outcome. Among all FHR patterns bradycardia has least mean cord blood pH 7.00, with least mean APGAR score 3.75 at 1min with 3 NICU admissions.

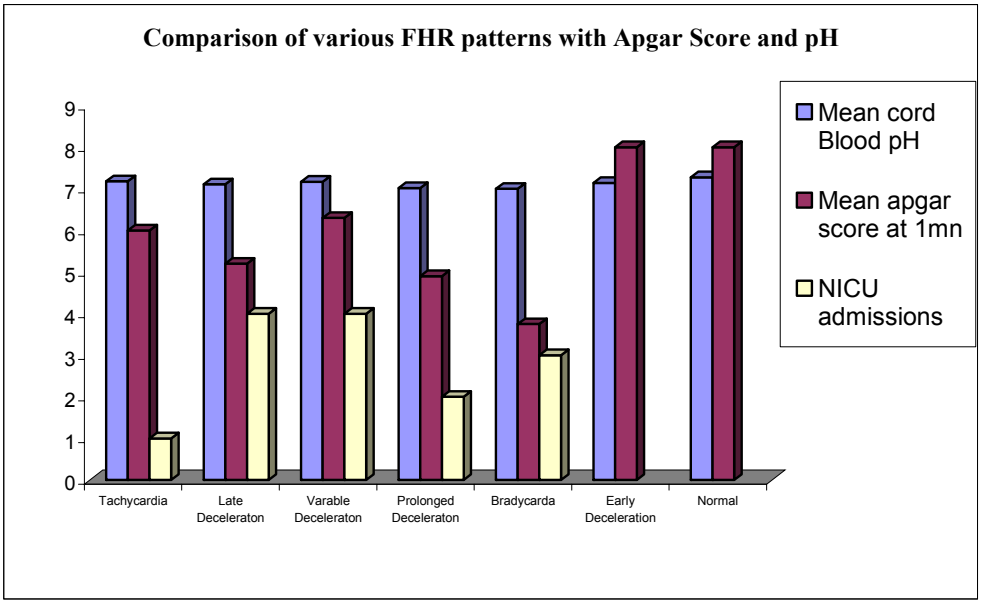


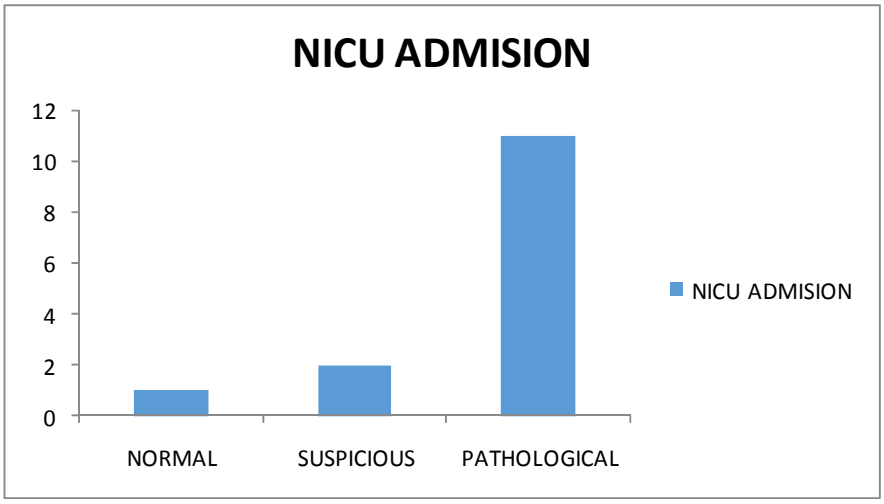
Table 16: NICU ADMISSIONS

CTG Patterns	NICU ADMISION	Percentage
Normal (n 100)	**1**	**1%**
Suspicious (n 26)	**2**	**7.6%**
Pathological (n 74)	**11**	**14.8%**

There were 14 NICU admissions. Out of 14, 1 baby was in normal CTG group and admission was for Low birth weight.

In suspicious CTG group there were 2 NICU admissions, 1 for IUGR and 1 for respiratory distress.

In pathological CTG group babies 11 were admitted to NICU and 4 of them had moderate and 7 severe acidosis. Out of these 7 with severe acidosis group, 4 babies delivered by cesarean section, 2 babies by forceps delivery, 1 baby by Ventouse. In all the CTG groups, babies admitted in NICU survived.



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DISCUSSION

Prediction and prevention of fetal hypoxia by EFM is necessary before intervention to prevent progression into fetal asphyxia. In our study out of the 200 pregnancies, 13% and 37% showed suspicious and pathological FHR patterns respectively and in them interventions were done. 36% of cases showed APGAR score of < 7 at 1 min and 7% showed < 7 at 5 minutes. Severe acidosis was seen in 6.5% and moderate acidosis in 28% of the cases. In suspicious CTG, APGAR at 1 min was 58% which is improved to 12% APGAR at 5 minutes, probably due to effect of neonatal resuscitation.

In a study done by Low JA et al, significant proportion of intrapartum fetal asphyxia occurred in pregnancies with no risk factors. Proportion of pregnancies with risk factors before the onset of labour was 36% and before delivery was 58%, the incidence of intrapartum fetal asphyxia was 2%.[46]

In our study 29% of pregnancies had antepartum risk factors. The incidence of intrapartum fetal asphyxia was 5.5%.

	Risk Factors	**Fetal Asphyxia**
Low JA et al	36%	2%
Our study	**29%**	**5.5%**

In a study done by Frank OP et al acidosis was seen in 44% of abnormal CTG patterns and 12 % of infants with normal APGAR score (< 7at 1 min) showed decreased pH (< 7.20) and 32% of infants with low APGAR scores showed acidosis.[16]

In our study 33.5% of abnormal CTG pattern showed acidosis. 15% of infants with normal APGAR score showed acidosis (<7.20) and 25% of infants with low APGAR score showed acidosis.

In a study done by Neeru Malik et al late decelerations showed worst prognosis with 2 perinatal deaths; variable decelerations were the next common CTG pattern in acidosis group and there were 3 NICU admissions for fetal asphyxia.[45]

In our study of 200 cases late decelerations were seen in 40 cases; mean cord blood pH was 7.11 and mean APGAR score at 1 minute was 5.2; NICU admissions in 4 cases in this group. Variable decelerations were seen in 26 cases; mean cord blood pH was 7.17; mean APGAR score at 1 minute was 6.3, NICU admissions were 4 and no perinatal deaths. Tachycardia was seen in 18 cases; mean cord blood pH was 7.18, mean APGAR score was 6, NICU admission was 1. Prolonged decelerations were seen in 8 cases; mean cord blood pH was 7.02 and mean APGAR score 4.9, 2 NICU admissions in this group. Bradycardia was seen in 5 cases; mean APGAR score is lowest of 3.75 and mean cord blood pH was 7 and 3 NICU admissions

In a study done by Pellantova S et al, out of 100 pathological or suspicious CTG patterns only 36.2% showed moderate or severe acidosis at birth and in the remaining 63.8% of cases there was no acidosis and unnecessary intervention was done. Mean APGAR score at 1 min was 5.4 and pH < 7 was seen in 8.5% of cases. Predictive value for different CTG patterns were 47.6% for variable deceleration, 34.5% for late deceleration and 29.3% for early deceleration.[5]

In our study out of 100 pathological and suspicious CTG patterns operative interventions was done in 80 cases. (44 cesarean sections and 36 operative deliveries). Moderate or severe acidosis was seen in 33.5% of cases, severe acidosis in 6.5% of all

cases. In instrumental deliveries, severe acidosis was seen in 7.4% of cases. In case of cesarean sections moderate and severe acidosis was seen in only 70.3% of cases and unnecessary intervention was done in 29.7% cases. Mean APGAR score at 1 was 5.387, pH < 7 was seen in 6.5% of cases. Predictive value of pathological and suspicious patterns for moderate acidosis was 66%.

	Pathological and suspicious CTG	**Moderate and severe acidosis**	**Mean APGAR at 1 min**	**pH <7**
Pallentova S et al	100 cases	36.2%	5.4	8.5%
Our Study	**100 cases**	**33.5%**	**5.387**	**6.5%**

In a study done by James AL et al, FHR variables associated with fetal asphyxia included absent and minimal base line variability. Late decelerations, prolonged decelerations and absent baseline variability were most specific and identified in 17% of the asphyxia group. In this study FHR record was available for 4 hours before delivery and scored in 10 minute cycles for each FHR variable and selected FHR patterns were examined during the last hour before delivery for their predictive value of fetal asphyxia. [48]

In our study CTG tracings were recorded for the last 30 minutes prior to delivery. Late decelerations, prolonged decelerations, variable decelerations and bradycardia were most specific and seen in 75% of abnormal CTG patterns and severe acidosis was seen in 13.3% of cases.

A study by James AL et al, showed that fetal asphyxial exposures accounted to 15.6% of moderate and severe asphyxia. Interventions and delivery during the 1st or 2nd

stage of labour was done in 78/140 (55.7%) cases of mild asphyxia and intervention and delivery in 20/26 (76.9%) cases of moderate or severe asphyxia.[2]

In our study fetal asphyxial exposures accounted for 36% of moderate or severe asphyxia. Operative deliveries were done in 24 cases during active phase, 21 cases during latent phase and in 2 cases during 2nd stage.

In a study done by Bullent Duran et al, 70% of tracings were normal, 29% suspicious and 1% pathological. Sensitivity for risk of cesarean delivery [stress & distress vs normal] was 35%, NICU admission was 46% and specificities ranged from 66 to 73%.[49]

In our study 50% of tracings were normal, 13% suspicious and 37% pathological. Sensitivity for risk of cesarean delivery was 90%, NICU admission 7% and specificities ranged from 19.3 to 66%.

	Normal CTG	**Suspicious CTG**	**Pathological CTG**	**NICU admission**
Bullent Duran et al	70%	29%	1%	46%
Our study	**50%**	**13%**	**37%**	**7%**

CONCLUSION

- Normal CTG tracings are reassuring.
- Late decelerations, variable decelerations and bradycardia are most specific for prediction of fetal asphyxia.
- The use of EFM is associated with increase in the rate of operative intervention. Additional tests such as fetal ECG, Fetal Pulse Oximetry, Fetal Scalp Blood Sampling for pH or lactate estimation are required before intervention to rule out fetal asphyxia as per the protocol.
- In presence of pathological CTG patterns cord blood pH and base deficit shows better correlation than APGAR score for prediction of fetal asphyxia.

SUMMARY

A prospective comparative study was conducted among 200 women in labor with full term, singleton pregnancies with vertex presentation for evaluation of CTG monitoring for prediction of intrapartum fetal surveillance and its correlation with APGAR score and cord blood pH, done between 1st January 2013 to 31st July 2014 in RLJ Hospital, Kolar. Preterm deliveries, twin gestation, cases of antepartum haemorrhage and women requiring cesarean section on admission were excluded.

- CTG tracings were taken in active stage, preferably 30 minutes before delivery or even earlier in cases with FHR irregularities. CTG tracing were defined as normal, suspicious or pathological patterns as per NICHHD guidelines.
- After delivery APGAR score at 1 and 5 minutes and cord blood gas values were analyzed.
- NICU admission were analyzed and followed up till discharge.
- Among the 200 patients 100(50%) showed reactive/normal FHR patterns, 26(13%) showed suspicious and 74 (37%) showed pathological FHR patterns.
- Late decelerations were commonest abnormal CTG patterns and was seen in 40% of cases, next common was variable deceleration which was seen in 26%, tachycardia in 18%, prolonged deceleration in 8%, bradycardia in 5% and early deceleration in 3%.
- 57% of cases delivered vaginally, among them 16% were in the pathological FHR group.

- Operative interventions were done in 43%, of them 23.5% was with cesarean deliveries and 19.5% with instrumental deliveries.
- APGAR score < 7 at 1 min (depressed) was 3%, 58% and 73% in normal, suspicious and pathological groups respectively with significant p value.
- APGAR score <7 at 5 min were 1%, 12%, and 14% in normal, suspicious and pathological groups respectively with significant p value
- The cord blood pH (<7.2) was seen in 2%, 58% and 70% in normal, suspicious and pathological groups respectively with significant p value.
- Moderate umbilical artery acidemia with base deficit of 12 to 16 mmol/l were seen in 2%, 15% and 26% in normal, suspicious and pathological groups respectively with significant p value.
- Severe base deficit > 16 mmol/l was seen in 0, 8% and 15% of cases of normal, suspicious and pathological groups respectively.
- Admission to NICU occurred in 1%, 7.6% and 14.8% of normal, suspicious and pathological groups respectively.

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ANNEXURES

PROFORMA

Name :

I P. No. :

Age :

Address :

D.O.A. : D.O.D. :

HISTORY : Amenorrhoea -

H/o Present illness -

Obsetetric history : Married life –

Gravida	Para	Living	Abortion
I			
II			
III			

Menstural history :

LMP POG (Dates)

EDD POG (USG)

Past history :

Family history :

General Examination

Pulse Pallor

BP Oedema

Icterus

CVS RS

P/A Ut. Size

Relaxed / Acting

FHS

PV

Diagnosis :

Investigations :

Routine	Hb%
Blood grouping & typing	
HIV	
Hbs Ag	
Urine - Routine	
CTG	
Time of delivery /	Mode of delivery
Vaginal Normal	
Forceps	
Ventouse	
Cesarean section	

Complications (if any)	
Baby’s condition	
Sex	
Weight	
APGAR score	1 Min
	5 Min
Cord blood pH	
NICU Admission	
Stay in NICU days	
Outcome at discharge :	

CONSENT FORM

You are being requested to be a participant in a research study of evaluation of CTG monitoring in intrapartum foetal surveillance and its correlation with APGAR score and cord blood pH. We request you to read this form and ask any question you may have before agreeing to be a participant in this study.

Your participation is voluntary and your decision whether or not to participate will not affect your current and further relation with the hospital. If you decide to participate, you are free to withdraw at any time without affecting the relationship.

This research is being carried out to study the correlation of CTG monitoring with APGAR score and cord blood pH. The procedures involved are non invasive and there are no potential risks or discomforts.

The benefits of taking part in this study are you are being monitored during labour for the foetal hypoxia and after delivery newborns APGAR score is observed and cord blood pH is tested.

The only people who will know about your participation are those in the research team. Privacy and confidentiality will be maintained and your identification will not disclosed.

You are free to ask any questions or doubts without hesitation. If you agree to participate kindly give your approval by signing below.

Date : Signature of Women

Signature of Witness

MASTER CHART

S.NO	Name	Age	I.P. number	Obstetric Index	Gestational age	Antepartum Risk Factors	CTG pattern	mode of delivery	APGAR (1 min)	APGAR (5 min)	Cord blood PH	Base deficit	NICU admission
1	Lakshmi	23	1844	Primi	38	No	Pathological	Em Lscs	4	6	6.9	16	No
2	Aruna	20	2023	G2A1	39	Pre eclampsia	Pathological	Em Lscs	5	8	7.21	15	No
3	Pushpavathi	21	1939	G2P1L1	40	No	Pathological	Em Lscs	4	6	6.8	16	yes
4	Priyanka	22	1029	G2P1L1	38	No	Suspicious	Em Lscs	4	6	7.12	14	No
5	Padma	23	2068	G2P1L1	37	No	Normal	Vaginal delivery	6	7	7.12	11	No
6	Vijaya Bharathi	23	2063	G2P1L1	38	Pre eclampsia	Normal	Vaginal delivery	7	9	7.21	11	No
7	Syed Banu	27	2081	G2P1L1	38	No	Pathological	Em Lscs	6	8	7.22	14	No
8	Preethi D	27	2322	Primi	39	No	Pathological	Em Lscs	5	9	6.89	16	yes
9	Vijayamma	22	2327	Primi	39	Pre eclampsia	Suspicious	Em Lscs	4	6	7.18	10	No
10	Shalini	23	2400	Primi	39	Pre eclampsia	Pathological	Em Lscs	6	8	7.23	11	No
11	Prema l	23	2393	Primi	39	No	Normal	Vaginal Delivery	8	10	7.14	11	No
12	sunitha	22	1013348	Primi	37	No	Pathological	Em Lscs	8	8	7.1	13	No
13	Mala	23	23170	Primi	39	No	Normal	Vaginal delivery	7	9	7.23	10	No
14	Fathima Bee	35	23146	G6P5L5	38	IUGR	Pathological	vaginal delivery	3	6	7.12	11	No
15	Priyanka	22	20343	Primi	39	Pre eclampsia	Normal	Vaginal delivery	7	9	7.22	11	No
16	Saraswati	24	24590	Primi	37	No	Normal	Vaginal delivery	8	9	7.23	11	No
17	Anitha	30	24622	Primi	38	No	Normal	Vaginal delivery	7	9	7.21	11	No
18	Mubeena Taj	22	24633	Primi	38	Pre eclampsia	Normal	Vaginal delivery	8	9	7.22	11	No
19	Veena	23	24634	Primi	38	Pre eclampsia	Normal	Vaginal delivery	8	9	7.22	10	No
20	tulasi	23	1018553	Primi	39	No	Normal	Vaginal delivery	8	9	7.24	10	No
21	Naveena	20	95880	G3P2L1D1	40	No	Normal	Vaginal delivery	8	9	7.24	10	No
22	Heetha	22	25883	G2P1L1	39	No	Normal	Vaginal delivery	7	9	7.23	11	No
23	Manjula	25	25920	G4P2L2A1	40	PROM	Pathological	Vaginal delivery	6	8	7.28	11	No
24	Jyothi	22	875915	Primi	40	No	Normal	Instrumental	7	9	7.24	11	No

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25	Rajini	22	875960	primi	41	Post Dated	Suspicious	Instrumental	6	8	7.18	11	No
26	Soumya	28	907719	Primi	38	No	Suspicious	Instrumental	5	6	7.22	13	yes
27	Amreen Taj	21	25918	G2P1L1	38	No	Normal	Vaginal delivery	8	9	7.22	13	No
28	Renuka	20	25929	Primi	38	No	Normal	Vaginal delivery	8	9	7.24	11	No
29	Manjula	19	25839	Primi	39	No	pathological	Vaginal delivery	4	6	6.88	16	No
30	Gulnaz	23	25895	Primi	39	No	Pathological	Em Lscs	3	6	6.69	16	No
31	Shobha	25	25910	G3P2L2	39	No	Suspicious	Em Lscs	8	10	7.18	10	No
32	Heena Kousar	20	25598	Primi	39	No	Pathological	Vaginal delivery	6	8	7.16	11	No
33	Manjula		909959	Primi	38	No	Suspicious	Instrumental	5	8	6.9	16	No
34	Asharani		913035	G2A1	37	No	Suspicious	Instrumental	5	7	7.18	11	No
35	Anjali	28	6344	G2P1L1	38	Pre eclampsia	Pathological	Em Lscs	6	8	7.22	14	No
36	Sumanjali	24	6352	Primi	38	Pre eclampsia	Pathological	Em Lscs	5	8	7.14	13	yes
37	Jayasudha	19	11850	Primi	38	No	Suspicious	Vaginal delivery	6	8	7.16	11	No
38	Mangala	22	11887	Primi	39	PROM	Pathological	Vaginal delivery	6	9	7.3	13	No
39	Manjula	32	17188	G4P3L1D2	39	No	Suspicious	Vaginal delivery	6	8	7.24	11	No
40	shaheena	20	17644	G2P1D1	37	No	Pathological	Vaginal delivery	6	9	7.19	11	No
41	Geetha	22	7821	G2P1L1	39	No	Pathological	Em Lscs	6	9	7.18	11	No
42	Meenakshi	22	922567	G2A1	40	No	Suspicious	Instrumental	5	8	7.16	16	No
43	Padma	26	17651	G3P2L2	40	No	Suspicious	Em Lscs	6	8	7.26	11	No
44	Saroja	24	17628	Primi	41	Post Dated	Pathological	Em Lscs	5	8	7.22	11	No
45	Kavitha	23	17713	Primi	39	Pre eclampsia	Suspicious	Em Lscs	7	8	7.21	11	No
46	Volliyamma	25	18638	G2P1L1	39	No	Pathological	Em Lscs	6	8	7.16	11	yes
47	Priyanka	25	18642	G3P2L2	38	No	Pathological	Vaginal delivery	5	9	6.89	16	No
48	Veena	22	21422	Primi	37	No	Normal	Vaginal delivery	8	9	7.22	11	No
49	Asha rani	22	21435	Primi	38	Pre eclampsia	Suspicious	Vaginal delivery	8	9	7.21	11	No
50	Sushma	24	22758	Primi	38	No	Normal	Vaginal delivery	7	9	7.23	11	No
51	Supriya	23	22767	G2P1L1	38	No	Normal	Vaginal delivery	7	9	7.23	11	No
52	Uma devi	35	24141	G2A1	39	No	Normal	Vaginal delivery	8	9	7.24	11	No
53	Vidyashri	24	24136	G3P1L1A1	39	No	Normal	Vaginal delivery	8	9	7.23	11	No
54	Shilpa	21	25941	Primi	39	No	Pathological	Vaginal delivery	6	9	7.23	11	No
55	Rupa	26	27908	Primi	39	No	Normal	Vaginal delivery	8	9	7.26	11	No

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56	Zakeera	23	28193	G2P1L1	38	No	Normal	vaginal delivery	7	9	7.26	11	No
57	Thaseema	24	29386	G3P2L1D1	40	No	Pathological	Em Lscs	6	9	7.16	14	No
58	Lakshmi devi	35	29456	G2P1L1	40	Pre eclampsia	Pathological	Em Lscs	3	6	6.88	16	yes
59	Gowramma	20	922940	G3A2	40	No	Pathological	Instrumental	4	9	6.9	16	yes
60	Nagaveni	18	923895	Primi	38	No	Pathological	Instrumental	3	6	6.8	11	yes
61	Pavitra	20	922764	Primi	39	Pre eclampsia	Pathological	Instrumental	4	9	7.16	11	No
62	Parvathi	22	230647	G2P1L1	37	No	Pathological	Em Lscs	5	9	7.22	11	No
63	Sudha	21	30711	Primi	39	No	Suspicious	Em Lscs	5	8	7.17	14	No
64	Thimmamma	22	30712	Primi	38	PROM	Pathological	Em Lscs	5	6	7.32	11	No
65	Naleena	23	30691	G2P1L1	39	Polyhydrominos	Pathological	Vaginal delivery	8	9	7.22	11	No
66	Chitra	22	60637	Primi	39	No	Normal	Vaginal delivery	7	8	7.3	11	No
67	Sushma	26	30687	G2P1L1	38	No	Normal	Vaginal delivery	8	10	7.3	11	No
68	Aisha bhanu	24	33277	Primi	38	Rh -ve	Pathological	Em Lscs	8	8	7.32	14	No
69	Rupa	20	33723	G2A1	38	Polyhydrominos	Normal	Vaginal delivery	8	9	7.27	11	No
70	Gayathri	20	33722	Primi	38	No	Normal	vaginal delivery	7	9	7.26	11	No
71	Harshitha	22	36851	Primi	39	No	Normal	vaginal delivery	7	9	7.26	11	No
72	Bharathi	20	37145	Primi	39	No	Normal	vaginal delivery	7	9	7.26	11	No
73	Nandini	22	37131	Primi	37	No	Normal	Vaginal delivery	9	9	7.23	11	No
74	Naziya	20	37221	Primi	39	No	Normal	Vaginal delivery	8	8	7.23	11	No
75	Bharati	29	874177	Primi	39	IUGR	pathological	Instrumental	6	9	7.23	11	No
76	Renuka	23	37241	Primi	38	No	Normal	Em Lscs	8	9	7.23	11	No
77	Anitha	26	37216	G3P2L2	38	No	Suspicious	Em Lscs	5	7	7.16	14	yes
78	Anitha	24	38362	Primi	38	IUGR	Pathological	Vaginal delivery	6	9	7.18	11	No
79	Deepa	22	976756	G2P1D1	39	No	Normal	Vaginal delivery	8	9	7.23	11	No
80	Kanchana	20	39402	G2P1L1	39	No	Normal	Vaginal delivery	8	9	7.33	11	No
81	Sumaiya sultana	20	39767	Primi	40	No	Normal	Vaginal delivery	8	9	7.22	11	No
82	Swapna	18	39786	Primi	40	No	Normal	vaginal delivery	8	9	7.28	10	No
83	Rihaana	20	39762	Primi	40	No	Normal	Vaginal delivery	7	8	7.24	10	No
84	Jyothi	23	40943	G2P1D1	39	No	Normal	Vaginal delivery	7	9	7.24	10	No
85	Jeevitha	18	40985	Primi	38	Polyhydrominos	Normal	Vaginal delivery	7	9	7.24	11	No
86	Kalpana	20	41001	Primi	39	No	Normal	vaginal delivery	8	9	7.26	10	No

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87	Mamoha	21	40797	Primi	39	No	Normal	Vaginal delivery	9	10	7.26	10	No
88	Kavitha	22	41011	Primi	38	No	Suspicious	Em Lscs	5	8	7.18	11	No
89	Lakshmi devi		926206	Primi	38	No	Pathological	Instrumental	8	8	7.21	14	No
90	Meenakshi		922847	Primi	38	Pre eclampsia	Pathological	Instrumental	7	9	7.22	13	No
91	Amreen Taj		929775	primi	38	No	Pathological	Instrumental	8	9	7.22	13	No
92	Lakshmi	21	41458	Primi	38	No	Pathological	Em Lscs	4	6	6.96	16	yes
93	Shobha	36	41545	G2P1L1	39	No	Normal	Vaginal delivery	7	8	7.22	11	No
94	Anitha	22	41484	Primi	37	PROM	Suspicious	Vaginal delivery	6	8	7.18	11	No
95	Sunanda	24	41471	Primi	37	No	Normal	Vaginal delivery	8	9	7.22	11	No
96	Sharadamma	20	42463	Primi	37	Rh -ve	Pathological	Vaginal delivery	5	8	7.14	11	No
97	Lakshmi devi	22	41478	G2P1L1	39	No	Pathological	Vaginal delivery	5	8	7.12	11	yes
98	Savitha	24	45096	G2P1L1	37	No	Normal	Vaginal delivery	8	9	7.23	11	No
99	Radha	24	45138	G2P1L1	38	Rh -ve	Normal	Vaginal delivery	8	9	7.23	11	No
100	Sujatha	20	943424	Primi	37	No	Pathological	Instrumental	5	8	7.15	11	No
101	Khamar Taj	29	10831	G4P3L1D2	39	No	Pathological	Em Lscs	6	8	7.16	11	No
102	Bhagya	20	16845	Primi	40	Pre eclampsia	Pathological	Em Lscs	6	8	7.24	11	No
103	Lakshmi	21	983207	Primi	40	Pre eclampsia	Pathological	Instrumental	4	6	7.3	10	No
104	Mubeena Taj	20	16844	G2A1	39	No	Normal	Vaginal delivery	8	9	7.26	10	No
105	Pavitra	21	26359	G2P1L1	39	No	Normal	Vaginal delivery	8	8	7.26	10	No
106	Archana	19	26458	Primi	38	No	Suspicious	vaginal dgelivery	6	8	7.16	11	No
107	Lakshmi	29	26466	Primi	39	No	Suspicious	Vaginal delivery	7	8	7.16	11	No
108	Roopa	22	26481	Primi	41	Post Dated	Normal	Vaginal delivery	7	8	7.22	11	No
109	Leelavati	23	17743	Primi	40	No	Normal	Em Lscs	7	8	7.22	11	yes
110	Suma	20	26487	G3P1L1A1	39	No	Pathological	Em Lscs	8	9	7.16	11	No
111	Lakshmi	20	983922	Primi	39	PROM	Pathological	Instrumental	8	9	7.18	11	No
112	Shamshad	22	26989	G2P1L1	39	No	Normal	Em Lscs	8	9	7.23	11	No
113	Sudha	32	1018416	G6P5L5	39	No	Normal	Vaginal delivery	8	9	7.21	11	No
114	Sujatha	20	27755	Primi	39	PROM	Normal	Vaginal delivery	8	9	7.24	11	No
115	Anitha	28	27767	G4P3L3	39	No	Normal	Vaginal delivery	8	9	7.24	11	No
116	Lakshmidevamma	22	27793	Primi	39	No	Suspicious	Vaginal delivery	8	9	7.21	11	No
117	Prameela	23	27949	G2P1L1	38	No	Suspicious	Vaginal delivery	7	9	7.16	11	No

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118	Ayesha	20	27784	Primi	38	No	Normal	Vaginal delivery	7	9	7.21	11	No
119	Nagarathna	24	27797	Primi	41	Post Dated	Normal	Vaginal delivery	8	9	7.22	11	No
120	Varalakshmi	26	24726	G2A1	41	Post Dated	Normal	Vaginal delivery	9	9	7.22	11	No
121	Roohila Taj	20	27772	Primi	37	Pre eclampsia	Normal	Vaginal delivery	8	9	7.26	10	No
122	Chitra	18	24702	G2P1L1	37	No	Normal	Vaginal delivery	8	9	7.24	10	No
123	Kalyani	22	29075	G2P1L1	39	Pre eclampsia	Normal	Vaginal delivery	8	9	7.26	10	No
124	Hemavathy	20	29070	Primi	39	No	Normal	Vaginal delivery	8	9	7.25	10	No
125	Radhamma	25	29061	G2P1L1	39	No	Normal	Vaginal delivery	7	9	7.26	10	No
126	Noor Husna	19	985719	G2P1L1	39	No	Pathological	Instrumental	6	8	7.23	14	No
127	Pallavi	24	30017	Primi	38	No	Pathological	Em lscs	8	8	7.33	13	No
128	Manjula	22	26338	G2P1L1	38	Pre eclampsia	Pathological	Em lscs	5	9	6.94	16	No
129	Ayesha	24	30226	Primi	39	No	Pathological	Em lscs	6	9	7.14	11	yes
130	Prema l	19	30254	Primi	38	No	Pathological	Em lscs	7	8	7.14	11	No
131	Nirmala	20	991583	Primi	39	No	Pathological	Instrumental	8	8	7.19	11	No
132	Krishna veni	20	30304	Primi	38	No	Pathological	Em Lscs	8	9	7.1	11	No
133	Ashwini	25	29085	G2A1	37	No	Normal	Vaginal delivery	7	9	7.22	11	No
134	Jayanthi	24	29087	G2P1L1	39	Pre eclampsia	Normal	vaginal delivery	8	9	7.23	11	No
135	Shabreen taj	27	29087	Primi	38	Pre eclampsia	Normal	Vaginal delivery	7	9	7.22	11	No
136	Mariyam	23	31193	G2P1L1	38	No	Normal	Vaginal delivery	8	9	7.23	11	No
137	Asma taj	19	31181	Primi	39	No	Normal	Vaginal delivery	8	9	7.23	11	No
138	Anitha	23	31655	G1A1	38	Cardiac Disease	Normal	vaginal delivery	7	8	7.21	11	No
139	Shylaja	19	989355	Primi	39	No	Pathological	Instrumental	6	8	7.12	15	No
140	Mumtaj	26	11046	G3P1L1A1	38	No	Pathological	Em Lscs	8	8	7.1	15	No
141	Nandini	28	31503	G2P1L1	37	No	Pathological	Em Lscs	8	9	7.28	14	No
142	Parvathi	24	32874	G2P1L1	37	PROM	Normal	Vaginal delivery	8	9	7.24	10	No
143	Sunanda	20	32876	Primi	37	PROM	Normal	Vaginal delivery	8	9	7.24	10	No
144	Bharati	22	32870	G2A1	38	No	Normal	Vaginal delivery	8	8	7.22	11	No
145	Ambika	24	32842	G2A1	38	No	Normal	Vaginal delivery	7	8	7.21	11	No
146	Sameena taj	23	34183	Primi	38	Pre eclampsia	Normal	Vaginal delivery	7	9	7.21	11	No
147	Varalakshmi	24	32824	Primi	38	No	Normal	Vaginal delivery	8	9	7.23	11	No
148	Taj Unissa	25	34231	G2P1D1	39	No	Normal	Vaginal delivery	8	9	7.22	11	No

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149	Shireen taj	23	34233	Primi	39	No	Normal	Vaginal delivery	7	9	7.23	11	No
150	Maithra	28	16748	Primi	39	Pre eclampsia	Normal	Vaginal delivery	8	9	7.23	11	No
151	Melna	25	35362	Primi	39	No	Normal	vaginal delivery	7	9	7.21	11	No
152	Manjula	22	999075	Primi	39	No	Pathological	Instrumental	6	8	7.18	11	No
153	Anitha	26	992358	Primi	39	No	Pathological	Instrumental	6	8	7.26	10	No
154	Suguna	18	33795	Primi	37	No	Pathological	Em Lscs	5	9	7.18	11	No
155	Vindhya	18	1002216	Primi	40	PROM	Suspicious	Instrumental	8	10	7.21	11	No
156	Sheela	33	19509	G2P1L1	40	No	Pathological	Em Lscs	8	9	7.12	11	No
157	Yellamma	20	1006391	G2P1L1	39	No	Pathological	Instrumental	8	9	7.14	11	No
158	Nagaveni	26	33988	G2P1L1	39	No	Pathological	Em Lscs	6	8	7.14	14	No
159	Arathi	20	35447	Primi	41	Post Dated	Normal	Vaginal delivery	7	9	7.22	13	No
160	Roshan taj	20	35453	Primi	40	Pre eclampsia	Normal	Vaginal delivery	7	9	7.22	11	No
161	Reehana Taj	25	35460	G2P1L1	39	No	Normal	Vaginal delivery	8	9	7.23	11	No
162	Asha rani	22	35455	Primi	39	No	Normal	Vaginal delivery	8	9	7.21	11	No
163	Roopashree	28	35459	G2P1L1	39	No	Normal	Vaginal delivery	8	9	7.24	10	No
164	Nirmala	22	35449	G2P1L1	38	PROM	Normal	Vaginal delivery	7	9	7.24	10	No
165	Renuka	22	35389	Primi	38	No	Normal	Vaginal delivery	8	10	7.22	11	No
166	Kulsar Bee	18	1007686	Primi	38	Cardiac Disease	Pathological	Instrumental	5	9	7.18	14	No
167	Bhagyamma	27	36473	Primi	37	No	Normal	Vaginal delivery	8	10	7.21	11	No
168	Shashikala	20	36790	Primi	37	No	Normal	Vaginal delivery	8	10	7.22	11	No
169	Geetha	20	38077	Primi	39	No	Normal	Vaginal delivery	8	9	7.22	11	No
170	Vani	22	1018756	Primi	39	PROM	Normal	vaginal delivery	7	9	7.21	11	No
171	Mamatha	26	38087	Primi	39	No	Normal	vaginal delivery	8	9	7.22	11	No
172	Akhila Khanym	28	1018757	G2P1L1	38	No	Pathological	Em Lscs	6	9	6.91	16	yes
173	Madhav	26	31179	G3P2L2	38	Polyhydrominos	Pathological	Em Lscs	7	8	7.18	11	No
174	Nandini	21	826	G2P1L1	39	No	Pathological	Em Lscs	8	9	7.18	11	No
175	Anuradha	30	1013803	Primi	40	No	Suspicious	Instrumental	7	9	7.26	10	No
176	kokila	20	1013809	Primi	41	Post Dated	Suspicious	Instrumental	7	9	7.26	10	No
177	Jyothi	21	1014712	Primi	39	No	Pathological	Instrumental	6	9	7.18	11	No
178	Baseera Begum	20	37672	Primi	39	No	Normal	Vaginal delivery	4	6	7.22	11	No
179	Lakshmi	20	3767	Primi	39	Pre eclampsia	Normal	Vaginal delivery	8	9	7.22	11	No

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180	Bharati	23	37649	G2P1L1	38	No	Normal	Vaginal delivery	8	9	7.21	11	No
181	Deepa	22	3847	G2P1D1	38	Pre eclampsia	Normal	Vaginal delivery	7	9	7.24	11	No
182	Leelavati	24	38749	Primi	37	Pre eclampsia	Normal	Vaginal delivery	8	9	7.21	11	No
183	Bhavabi	21	20289	Primi	38	No	Normal	Instrumental	7	9	7.21	11	No
184	Kavita	22	20745	Primi	38	No	Pathological	Instrumental	6	8	7.17	11	No
185	Sultana Begum	20	20987	Primi	39	No	Suspicious	Instrumental	8	9	7.22	11	No
186	Sudha	23	21443	Primi	38	No	Suspicious	Instrumental	7	9	7.22	11	No
187	Sukanya	22	21363	Primi	38	No	Pathological	Instrumental	6	8	7.18	11	No
188	Lakshmi	30	23623	Primi	39	No	Pathological	Instrumental	8	8	7.19	11	No
189	Raziya Sultana	20	23096	G2A1	39	No	Pathological	Instrumental	5	8	7.18	13	No
190	Parvathamma	24	37999	G2P1L1	37	No	Pathological	Em Lscs	5	8	7.18	11	No
191	Uma	20	9131	Primi	37	No	Normal	Instrumental	7	8	7.24	10	No
192	Shilpa	20	9428	Primi	38	No	Pathological	Instrumental	5	9	7.16	11	No
193	Sarawathamma	23	40215	G2P1L1	37	No	Normal	Vaginal delivery	7	9	7.26	10	No
194	Bhagyalakshmi	24	40194	Primi	38	No	Normal	vaginal delivery	8	9	7.26	10	No
195	Shylaja	25	41376	G2P1L1	39	PROM	Normal	Vaginal delivery	5	9	7.23	11	No
196	Yashodamma	27	40450	G2P1D1	38	No	Normal	Vaginal delivery	8	8	7.23	11	No
197	Veena	22	41175	Primi	38	No	Normal	Vaginal delivery	8	9	7.24	10	No
198	Bharti	22	10125	G2P1L1	40	No	Pathological	Instrumental	8	8	7.04	11	No
199	Ashwini	20	10757	Primi	40	No	Pathological	Instrumental	6	8	7.08	11	No
200	Gomathy	22	10859	G2P1L1	39	No	Pathological	Instrumental	6	8	7.14	11	No