"PREDICTION OF NEONATAL ACIDEMIA AT BIRTH WITH INTRAPARTUM TOTAL DECELERATION AREA ON FETAL CARDIOTOCOGRAM – A ONE YEAR CASE CONTROL STUDY"

BY

REG.NO: BJ0121017

Dissertation

Submitted to

KAHER, Belagavi, Karnataka,

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY (M.S.)

in

OBSTETRICS AND GYNECOLOGY

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY

JAWAHARLAL NEHRU MEDICAL COLLEGE, KAHER,

BELAGAVI – 590010, KARNATAKA.

DECEMBER-2024 / JANUARY-2025

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH,

BELAGAVI, KARNATAKA.

Endorsement

This is to certify that the dissertation entitled **"PREDICTION OF NEONATAL ACIDEMIA AT BIRTH WITH INTRAPARTUM TOTAL DECELERATION AREA ON FETAL CARDIOTOCOGRAM – A ONE YEAR CASE CONTROL STUDY"** is a bonafide research work done by **REG No: BJ0121017.**

Dr. Yeshita Pujar MS
Professor & HOD.
Dept of OBG.
J. N Medical College, Belagavi
590010

Date:11-07-2024
Place: Belagavi

KAHER BELAGAVI J.N. MEDICAL COLLEGE

Dr. (Mrs.)N.S.Mahantashetti MS
Principal
J. N Medical College,
Belagavi-590010

PRINCIPAL
J.N. Medical College,
BELAGAVI- 590 010

Date:12-07-2024
Place: Belagavi

UNDERTAKING

"I, Registration number- BJ0121017, hereby declare that the information and data mentioned in my dissertation titled "PREDICTION OF NEONATAL ACIDEMIA AT BIRTH WITH INTRAPARTUM TOTAL DECELERATION AREA ON FETAL CARDIOTOCOGRAM – A ONE YEAR CASE CONTROL STUDY" belongs to me and is original. I am aware of the definition of plagiarism as detailed below:

- An act or instance of using or closely imitating the language and thoughts of another author without authorisation and representation of that author's work as one's own, as by not crediting the original author.

- A piece of writing or other work reflecting such unauthorized use or imitation.

- The deliberate or reckless representation of another's words, thoughts or ideas as one's own without attribution in connection with submission of academic work, whether graded or otherwise.

I hereby declare that the dissertation prepared by me is in original and does not involve plagiarism anywhere. In case at a later stage, it is found that I have indulged in plagiarism, then I am solely responsible for the same and the institution is at liberty to take any disciplinary action against me including cancellation of dissertation or any other penalties imposed by the university."

Date: 12/7/24

Place: Belagavi

Registration number- BJ0121017

PLAGIARISM CLEARANCE

KLE ACADEMY OF HIGHER EDUCATION AND RESEARCH

JAWAHARLAL NEHRU MEDICAL COLLEGE

(A constituent unit of KLE Academy of Higher Education & Research Deemed-to-be-University)

(Recognized by National Medical Commission, New Delhi)

Accredited 'A+' Grade by NAAC (3rd Cycle) **Placed in Category 'A' by MoE (GoI)**

Nehru Nagar, Belagavi- 590 010, Karnataka, INDIA

0831 – 2471350 0831 – 2470759 www.jnmc.edu principal@jnmc.edu

Ref No: MDC/PG/ Date: 28-06-2024

"ACCEPTANCE LETTER"

The softcopy of thesis entitled: **"PREDICTION OF NEONATAL ACIDEMIA AT BIRTH WITH INTRAPARTUM TOTAL DECELERATION AREA ON FETAL CARDIOTOCOGRAM – A ONE YEAR CASE CONTROL STUDY"** has been submitted for anti-plagiarism check through Turnitin software. The scan has been carried out and the scanned output reveals a match percentage of **09%** which is within the acceptable limits of **10%** as per the guidelines given by UGC.

Guide.

KAHER BELAGAVI J. N. MEDICAL COLLEGE

Dr. (Mrs.) N.S. Mahantashetti.
Chairperson-Antiplagiarism Committee &
Principal,
J. N. Medical College, Belagavi.

To,
Reg. No. BJ0121017
Postgraduate Student,
2021-22 Batch,
Department of Obstetrics & Gynaecology
J. N. Medical College, Belagavi.

ETHICAL CLEARANCE

K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to- be- University)
Accredited 'A+' Grade by NAAC in (3rd Cycle) Placed in Category 'A' by MHRD (GoI)

JNMC INSTITUTIONAL ETHICS COMMITTEE
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)

Website: http//www.jnmc.edu
E-Mail : dome@jnmc.edu

Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref No.MDC/JNMCIEC/302 **Date: 11/04/2024**

To,

REG.NO: BJ0121017

PG Student in Obstetrics & Gynaecology
J. N. Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"PREDICTION OF NEONATAL ACIDEMIA AT BIRTH WITH INTRAPARTUM TOTAL DECELERATION AREA ON FETAL CARDIOTOCOGRAM – A ONE YEAR CASE CONTROL STUDY",** is ethical and justifiable. This audit project is approved by ethical JNMC committee with waiver of consent.

(Dr. Smita Sonoli)
Member Secretary
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi.

(Dr. Harsha Hegde)
Chairman,
JNMC Institutional Ethics Committee
J.N.Medical College, Belagavi

LIST OF ABBREVIATIONS USED

CTG	Cardiotocography
TDA	Total Deceleration Area
AUC	Area Under the Curve
OR	Odd's Ratio
CI	Confidence Interval
MC	Chi square test with Monte Carlo simulation
NICU	Neonatal Intensive Care Unit
LSCS	Lower Segment Caeserean Section
EFM	Electronic Fetal monitoring
NICHD	National Institute of Child Health and Human Development
ECG	Electro Cardio Graph
FHR	Fetal Heart Rate
Bpm	Beats per minute
SLE	Systemic Lupus Erythematosis
FGR	Fetal Growth Retardation
MAS	Meconium Aspiration Syndrome
WHO	World Heath Organisation
BD	Base Deficit

DA	Deceleration Area
ACOG	American College of obstetricians and Gynaecologists
MSAF	Meconium-Stained Amniotic Fluid
pH	Potential Hydrogen
PCO2	Partial pressure of carbon dioxide
PO2	Partial pressure of oxygen
HCO3	Bicarbonate
ROC	Receiver Operating Characteristic
BMI	Body Mass Index
PROM	Premature Rupture of Membranes
GDM	Gestational Diabetes Mellitus
HTN	Hypertension
BOH	Bad Obstetric History
DTA	Deep Transverse Arrest
PPV	Positive Predictive Value
NPV	Negative Predictive Value
AU ROC	Area Under Receiver Operating characteristic
LBW	Low Birth Weight
VLBW	Very Low Birth Weight

ABSTRACT

Aims & Objectives: In recent times there is a global rise in the rate of cesarean deliveries, fetal distress being second most common indication. The diagnosis of fetal distress is based on fetal cardiotocography findings during labour.This study aims to analyse the intrapartum total deceleration area on fetal cardiotocogram which will predict neonatal acidaemia at birth.

Materials & Methods: This Case-Control study was conducted for a period of 1 year (Feb 2023- Jan 2024) At KAHERS Dr Prabhakar Kore Hospital, Affiliated to JNMC, Belagavi. The Study participants included women in labor having intrapartum fetal distress who had the 30minutes trace recording of CTG (prior to delivery/decision to delivery) and fetal umbilical cord blood gas analysis at delivery. Neonates having a pH value of ≤7.2 were categorised as Cases and those having a pH of > 7.2 were grouped as Controls. Total deceleration area was calculated and analysed against the cord blood pH obtained at delivery.

Results: A total of 168 participants were analysed, there were, 42 cases and 146 controls were included in the study for further analysis.

The Mean TDA noted in the case and control group was 254.62 missed beats and 165.46 missed beats respectively.

It was observed that an intra partum TDA of 195 missed beats, was associated with neonatal acidemia at birth with an AUC of 0.6576 (0.5305, 0.7847), and a positive predictive value of 83.78%

Conclusion: An intrapartum TDA of 195 missed beats, was significantly associated with neonatal acidemia at birth in this study. The calculation of TDA has simplified the intrapartum fetal monitoring.

Keywords: Total Deceleration Area, TDA, Neonatal acidemia, Cardiotocography.

LIST OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Intrapartum fetal distress and neonatal acidemia are closely linked conditions that lead to neonatal mortality.The neonatal mortality rate at present is 5.5 per 1000 births. Hypoxia is believed to contribute to 90% of deaths occurring during labor and delivery. [1] It can have significant implications for the health of the newborn. Complications of fetal distress also include neonatal morbidity, NICU admissions, birth asphyxia, morbidity like, respiratory distress, neurological damage, hypoxic-ischemic encephalopathy, seizure disorder, developmental delays, and in severe cases, cerebral palsy.

This leads to maternal morbidity in the form of cesarean section as most of the intrapartum fetal distress cases are managed by emergency cesarean sections , for an immediate delivery and a better neonatal outcome.In recent times, there is an exponential rise in the rate of cesarean deliveries across the globe .Fetal distress being the chief indication for primary caesarean sections and second most common indications in overall cesarean sections.[2]A Study conducted in our tertiary care hospital in 2017 showed a cesarean rate of 44.61% . Fetal distress was the second most common indication for cesarean section accounting for 16.24% after previous LSCS [3]

Fetal distress is a vague term. In Clinical practice, it is used to describe a wide range of fetal heart rate abnormalities if not corrected will result in decompensation of physiologic responses and cause permanent central nervous system, damage or death.[4]

Electronic fetal monitoring (EFM) is commonly used to detect fetal distress. In clinical practice, cardiotocography (CTG) based National Institute of Child Health

and Human Development (NICHD) classification is widely used for diagnosis of fetal distress [5] such as Category II and Category III traces which include tachycardia, repetitive variable deceleration, late deceleration , marked variability etc.[5]

It has been observed in previous research that irrespective of type of decelerations, acidotic pH & NICU admissions are seen.[6] Co-relation of acidotic pH is seen with variable/late and even early deceleration.[7]

Hence CTG is criticised for increased rates of caesarean deliveries for fetal distress as false positive rate of diagnosis of fetal distress with CTG is high(60%).[8] Still CTG is preferred due to its medicolegal advantages as it is a documented proof of basis of intervention performed for managing cases of fetal distress. CTG interpretation needs technical training, emphasis is given to avoid fetal acidemia, with respect to late and persistent variable deceleration.

It is observed that there is knowledge gap in identifying a better CTG parameter which can be interpreted easily and aid in the early diagnosis of fetal distress and prevention of neoatalacidemia.

Total Deceleration Area (TDA) a simple formulated calculation is an easy & promising parameter that can used in the detection of fetuses going into the risk of developing acidemia intrapartum irrespective of type of deceleration.Our study aims to evaluate the predictive ability of Total Deceleration Area in detecting fetal acidemia.[20]

AIMS AND OBJECTIVES

Primary Objectives

To study the intrapartum total deceleration area on fetal cardiotocogram which will predict neonatal acidemia at birth.

Secondary Objectives

To compare the predictive ability for neonatal acidemia at birth by following individual components of intrapartum cardiotocography (CTG) described by National Institute of Child Health and Human Development (NICHD) system.

Fetal tachycardia, Fetal bradycardia, variability <5bpm and >25bpm, sinusoidal pattern on cardiotocogram

REVIEW OF LITERATURE

Intrapartum fetal monitoring is the assessment of the fetal well-being during labor and delivery. The purpose is to identify signs of fetal distress or compromise, allowing healthcare providers to intervene promptly, to ensure safety of both the mother and the baby.

History of Intrapartum Fetal monitoring

Fetal heart sounds were initially detected by Marsac in the 1600s, and concept of using fetal heart rate as an indicator of fetal well-being was introduced by Killian during the same period. History of intrapartum fetal monitoring dates back to 18th century when clinicians recognized the need to monitor fetal well-being during labor and delivery. However, it wasn't until 1818 that Mayor and Kergaradec outlined the technique of auscultating fetal heart sounds by placing the ear on the maternal abdomen. Kergaradec also suggested that these sounds could be used to assess fetal viability. [9,10,11,12,13,14,15,16,17]

By 1833, English physician Evory Kennedy had published guidelines for identifying fetal distress, recommending the auscultation of fetal heart rate during labor. In 1893, Von Winkel established a criterion for fetal distress, which remained unchanged until electronic fetal monitoring was introduced, such as passage of meconium, tachycardia, bradycardia, irregular heart rate, and gross alteration of fetal movement.In the early 20th century, prior to the invention of electronic fetal monitoring, clinicians relied on auscultation with a stethoscope to listen to the fetal heart rate, this was the method had limitations in both accuracy and consistency. [9,10,11,12,13,14,15,16,17]

In 1917, David Hillis introduced the concept of the fetal stethoscope, or fetoscope, at the Chicago Lying-In Hospital. However, it was in 1922 that Joseph DeLee, Hillis's superior at the same institution, documented the device, essentially claiming credit for its invention. Over time, it became recognized as the DeLee-Hillis fetoscope and played a prominent role in intrapartum fetal monitoring for the ensuing fifty years. [9,10,11,12,13,14,15,16,17]

In 1906, Cremer introduced the concept of the fetal electrocardiogram (ECG), utilizing both abdominal and intravaginal electrical leads. This innovation spurred further investigation into using electrocardiographic patterns to assess fetal well-being, but researchers ultimately found no consistent patterns indicative of fetal distress. Electronic fetal phonocardiography, initially described by Henly in 1931, involved amplifying the fetal heart rate (FHR) using a microphone. Then, in 1958, Hon, a trailblazer in modern electronic fetal monitoring (EFM), detailed a system for continuous capture of the fetal ECG, marking a significant advancement in fetal monitoring technology. [9,10,11,12,13,14,15,16,17]

During the late 1950s, Doppler ultrasound technology was introduced by an Austrian physicist, Christian Andreas Doppler. This allowed for continuous, non-invasive monitoring of the fetal heart rate, electronically.

Hon introduced the spiral electrode, known as the fetal scalp electrode, in 1972, This method was seen to be invasive and risky. [9,10,11,12,13,14,15,16,17]

As electronic monitoring techniques evolved, more sophisticated methods for distinguishing authentic fetal signals from artifacts emerged, especially when coupled with Doppler technology. These advancements culminated in the development of

modern electronic autocorrelation, offering enhanced accuracy and reliability in fetal monitoring during labor. [9,10,11,12,13,14,15,16,17]

Cardiotocograph (CTG), which combined the electronic monitoring of both fetal heart rate and uterine contractions was developed by Dr. Edward Hon during 1950-1970s.

This allowed for a more comprehensive assessment of fetal well-being during labor. By 21st century, Continuous electronic fetal monitoring during labor became routine in many hospitals, especially for high-risk pregnancies. However, there was increasing recognition of the limitations and potential harms associated with its use, such as false alarms leading to unnecessary interventions and increased rates of cesarean sections.Thus, efforts have been made to refine electronic fetal monitoring techniques and guidelines to improve accuracy and reduce unnecessary interventions. [18]

Intrapartum fetal monitoring is mainly of 2 methods:

Electronic Fetal Monitoring (EFM):

This method uses external monitoring devices to evaluate the fetal heart rate (FHR) and uterine contractions. Two sensors are placed on the mother's abdomen: one to track the fetal heart rate and another to monitor uterine contractions.

The fetal heart rate is usually monitored using Doppler ultrasound, while uterine contractions are monitored using a pressure-sensitive device. [18]

Intermittent Auscultation (IA):

This approach involves periodically checking the fetal heart rate with a handheld Doppler ultrasound device or a fetoscope. It is generally used for low-risk pregnancies.

Normal fetal heart rate patterns, indicate adequate oxygenation and perfusion, while abnormal patterns may suggest fetal distress.

Electronic fetal monitoring (EFM), is an essential tool in obstetrics used to monitor the fetal heart rate (FHR) and uterine contractions during pregnancy and childbirth. It offers critical information about the fetus's well-being, aiding healthcare providers in making informed decisions in managing labor and delivery.

Continuous intrapartum fetal monitoring, is often recommended for high-risk pregnancies, like those involving maternal hypertension, diabetes, post-term pregnancy, or intrauterine growth restriction, as well as in cases of prolonged labor, oxytocin augmentation, or other complications. [18]

Cardiotocograph (CTG)

Cardiotocograph, is a graphic record of the fetal heart rate and uterine contractions, via an ultrasound device placed on the maternal abdomen or through a fetal scalp electrode.

The ‘toco’, registers the uterine contractions through a second transducer placed on the uterine fundus. [18]

CTG was developed by a British obstetrician Dr. Edward Hon during 1950-1970s.The first commercial fetal monitor was released in the year1968.The Fetal Heart Rate, is recorded both on the CTG paper and displayed on the CTG monitor, it

is also heard as an audible signal. This is measured using a doppler ultrasound device. [18]

A Doppler ultrasound device is positioned on the mother's abdomen over the fetus's anterior shoulder, as determined by palpation, and adjusted until a clear signal is obtained. A water-based ultrasonic gel, is applied between the transducer and the woman's abdominal wall to ensure good contact. This gel, having a similar density to the woman's abdomen, allows sound waves to travel through it with minimal interference. The transducer generates and receives sound waves by passing a high-frequency electrical current through a piezoelectric crystal. When the current flows through the crystal, it changes shape, creating a sound wave that travels through the woman's abdomen. The piezoelectric effect is also such that when a piezoelectric crystal is squeezed or released from pressure, it will convert some of this energy into an electric current; this electric current from reflected sound waves is used to determine the FHR. [18]

The 'toco' is positioned on the mother's anterior abdominal wall, over the uterine fundus and secured with a stretchy elastic band, to monitor the frequency and duration of uterine contractions. The amplitude of the tocograph reflects changes in the shape and tone of the anterior abdominal wall rather than the strength of the uterine contractions. During uterine contractions, the uterus beneath the anterior abdominal wall alters its shape and tone, creating a pressure wave that the tocograph records. [18]

All CTG traces should be identified with unique patient identifiers and correct time and date as one would for any other documentation in a patient’s notes. It is very important to check that paper has been loaded in correct orientation. One should be

aware of the 'paper speed', which refers to the speed at which the CTG trace moves. The standard practiced in India is 1cm/min. [18]

Definitions

Baseline fetal heart rate

This is estimated over a 5–10-minute period of CTG, excluding accelerations or decelerations and is recorded in beats per minute (bpm). In normal circumstances the baseline fetal heart rate, is 110–160bpm. [19]

Baseline bradycardia

It is defined as FHR being of a persistently low baseline of below 110bpm. [19]

Baseline tachycardia

It is defined as FHR being of a persistently high baseline of above 160bpm. [19]

Variability

Variability occurs as a result of beat to-beat changes in the heart rate. Normally it is 5–15bpm. Variability < 5bpm is classed as reduced. Variability is measured by analysing a 1-minute stable portion of a CTG. [19]

Accelerations

An acceleration is an increase in the fetal heart rate of 15bpm or more, lasting for at least 15 seconds from the baseline. [19]

Deceleration

A deceleration is a decrease in the fetal heart rate of 15bpm or more, lasting for at least 15 seconds from the baseline.

Decelerations of the fetal heart rate from the baseline can be classified into the following types:

1. Early
2. Late
3. Variable

Early decelerations

Early decelerations tend to be uniform in shape and occur with each contraction. They often appear, in a mirror image of the contraction. The deceleration begins with the start of the contraction. The heart rate reaches its lowest at the peak of the contraction and returns to baseline by the end of the contraction. [19]

Late decelerations

Late decelerations are usually uniform in shape and depth,it occurs after each contraction. Any deceleration with the lowest point occuring more than 15 seconds after the peak of the contraction is said to be late decelerations. [19]

Variable decelerations

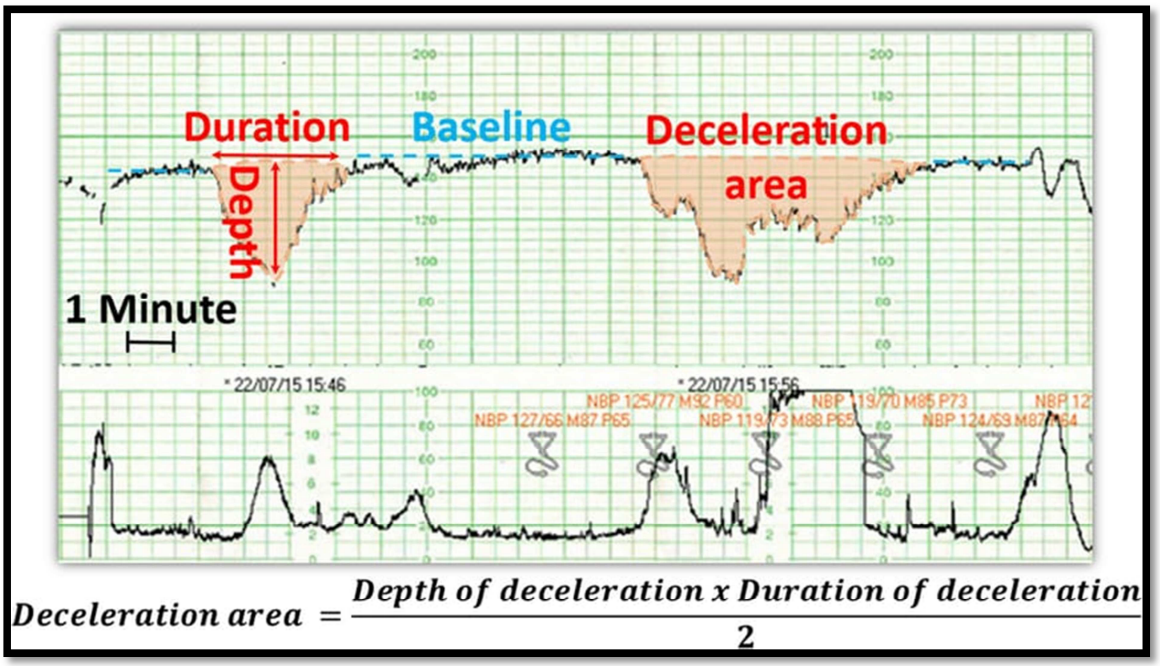
Variable decelerations are inconsistent with shape and frequency and with their relationship to uterine contractions. [19]

Sinusoidal pattern

These patterns are identifiable by the “smooth undulating, sine wave-like baseline”. Variability is absent. The amplitude of the undulations is usually 5–15 beats and the frequency 2–5 per minute.[19]

Deceleration area:

Deceleration area is the width of widest aspect of deceleration (below baseline) measured in minutes, multiplied by maximum depth below the baseline divided by 2.[20]



Neonatal acidaemia at birth:

Umbilical artery cord blood pH of ≤7.2, according to WHO guidelines.[21]

NICHD Guidelines for CTG Interpretation

Normal (category I)

Category I EFM recordings include all following:
- Baseline rate: 110–160 bpm
- Baseline fetal heart rate variability: moderate
- Late or variable decelerations: absent
- Early decelerations: present or absent
- Accelerations: present or absent

Indeterminate (category II)

Category II EFM recordings include all not categorized as category I or III—category II EFM recordings may represent appreciable fraction of those encountered in clinical care

Examples of category II EFM recordings include any of following:
- Bradycardia not accompanied by absent variability
- Tachycardia
- Minimal or marked variability
- Absent variability not accompanied by recurrent decelerations
- Absence of induced accelerations after fetal stimulation
- Recurrent variable decelerations accompanied by minimal or moderate baseline variability
- Prolonged deceleration
- Recurrent late decelerations with moderate baseline variability
- Variable decelerations with other characteristics, such as slow return to baseline, "overshoots," or "shoulders"

Abnormal (category III)

Category III EFM recordings include either:

Absent variability and any of following:
- Recurrent late decelerations
- Recurrent variable decelerations
- Bradycardia

Sinusoidal pattern

EFM, electronic fetal monitoring.

Cahill et al. Deceleration area and acidemia. Am J Obstet Gynecol 2018.

NICE Guidelines for CTG Interpretation

Description	Feature		
	Baseline (beats/ minute)	**Baseline variability (beats/ minute)**	**Decelerations**
Reassuring	110 to 160	5 to 25	None or early Variable decelerations with no concerning characteristics* for less than 90 minutes
Non-reassuring	100 to 109† OR 161 to 180	Less than 5 for 30 to 50 minutes OR More than 25 for 15 to 25 minutes	Variable decelerations with no concerning characteristics* for 90 minutes or more OR Variable decelerations with any concerning characteristics* in up to 50% of contractions for 30 minutes or more OR Variable decelerations with any concerning characteristics* in over 50% of contractions for less than 30 minutes OR Late decelerations in over 50% of contractions for less than 30 minutes, with no maternal or fetal clinical risk factors such as vaginal bleeding or significant meconium
Abnormal	Below 100 OR Above 180	Less than 5 for more than 50 minutes OR More than 25 for more than 25 minutes OR Sinusoidal	Variable decelerations with any concerning characteristics* in over 50% of contractions for 30 minutes (or less if any maternal or fetal clinical risk factors [see above]) OR Late decelerations for 30 minutes (or less if any maternal or fetal clinical risk factors) OR Acute bradycardia, or a single prolonged deceleration lasting 3 minutes or more

Abbreviation: CTG, cardiotocography.

* Regard the following as concerning characteristics of variable decelerations: lasting more than 60 seconds; reduced baseline variability within the deceleration; failure to return to baseline; biphasic (W) shape; no shouldering.

† Although a baseline fetal heart rate between 100 and 109 beats/minute is a non-reassuring feature, continue usual care if there is normal baseline variability and no variable or late decelerations.

Intrapartum care: NICE guideline CG190 (February 2017)

Fetal Distress

Fetal distress, also known as "non-reassuring fetal status", is a condition during pregnancy or labor where the fetus shows signs of inadequate oxygenation.

The condition is detected most often with electronic FHR monitoring through cardiotocography (CTG), allowing clinicians to measure changes in the cardiac response to declining oxygenation.[22]

The fetus relies on the mother, for the exchange of oxygen and carbon dioxide through the placenta. This process depends on the mother having adequate blood gas levels, proper uterine blood flow, efficient placental transfer, and effective fetal gas transport. Any disruption in these areas can lead to fetal hypoxia, and despite the fetus's compensatory mechanisms, it may result in acidosis.The fetus has several compensatory mechanisms, such as increasing heart rate and redistributing blood flow to vital organs, to counteract mild hypoxia.However, these mechanisms have limits.Prolonged or severe hypoxia can lead to anaerobic metabolism (metabolism without sufficient oxygen), producing lactic acid.The accumulation of "lactic acid" causes a decrease in blood pH, leading to acidosis.Acidosis can impair cellular function and may cause long-term damage to fetal tissues and organs. [23]

The causes of fetal hypoxia, and consequently acidosis, can be categorized into maternal, placental, and fetal factors. The severity and duration of acidosis, as well as the pre-existing condition of the fetus, influence the outcomes of acidosis. Additionally, the causes of fetal acidosis can be classified based on their onset: acute (occurring over hours) or chronic (developing over days).Early detection and management of fetal distress are essential to minimize the risk of adverse outcomes for both the mother and the baby. [22]

Acute hypoxia may include maternal causes such as sudden hypotension or hypovolemia like haemorrhage,hypertonic uterine contractions.Placental factors like abruption or fetal causes such as true umbilical knot is also seen to cause acute hypoxia.

Chronic hypoxia may be attributed to maternal pathological conditions such as cardiac diseases, SLE. Placental insuffiecieny resulting in FGR and conditions such as parvovirus infection, anaemia from rhesus disease, alpha-thalassaemia or feto-maternal haemorrhage,are also noted to be associated with chronic hypoxia.[23]

Fetal distress can lead to various complications for both the fetus and the mother. Some potential complications include [23]

1.Fetal Hypoxia and Acidosis: Inadequate oxygenation to the fetus can result in fetal hypoxia (oxygen deprivation), leading to metabolic acidosis (excessive acidity in the blood). Prolonged or severe hypoxia can cause damage to fetal organs, including the brain, heart, and kidneys.

2.Hypoxic ischemic encephalopathy: Prolonged lack of oxygen during labor and delivery can lead to brain damage in the newborn, resulting in conditions like cerebral palsy, developmental delays, or intellectual disabilities.

3. Neonatal Asphyxia: Severe or prolonged fetal distress can result in neonatal asphyxia, a condition characterized by impaired gas exchange and oxygen deprivation at birth. Neonatal asphyxia can lead to long-term neurologic deficits, developmental delays, and other complications, including cerebral palsy.

4. Meconium Aspiration Syndrome (MAS): Fetal distress can result in the passage of meconium into the amniotic fluid. If the baby inhales meconium-stained amniotic

fluid before or during birth, it can lead to meconium aspiration syndrome, a condition characterized by respiratory distress, airway obstruction, and inflammation of the lungs.

5.Birth Trauma: In cases of severe fetal distress, expedited delivery may be necessary to prevent further complications. Instrumental deliveries (e.g., vacuum extraction or forceps) or emergency cesarean sections carry a risk of birth trauma, including injuries to the baby's head, shoulders, or other body parts.

6.Organ damage: Prolonged oxygen deprivation can damage vital organs such as the heart, kidneys, liver, and gastrointestinal tract.

7.Stillbirth: In severe cases of fetal distress, where interventions are not successful in restoring oxygen supply to the fetus, it can lead to intrauterine fetal death, also known as stillbirth.

8.Neonatal intensive care admission: Babies who experience fetal distress may require admission to a neonatal intensive care unit (NICU) for monitoring and supportive care after birth

9.Maternal Complications: Fetal distress necessitates interventions such as cesarean section or instrumental delivery, which can increase maternal complications such as hemorrhage, infection, or surgical complications.

10.Long-Term Neurological Sequelae: Severe or prolonged fetal distress and associated complications such as neonatal asphyxia can result in long-term neurologic sequelae for the baby, including cognitive impairments, developmental delays, learning disabilities, and cerebral palsy.

11.Emotional Impact: Experiencing fetal distress and its associated complications can be emotionally distressing for both parents, leading to anxiety, depression, and post-traumatic stress disorder (PTSD) in some cases. [23]

Prompt recognition and management of fetal distress are crucial to minimizing the risk of complications and optimizing outcomes for both the mother and "baby. Close monitoring during labor, timely interventions, and appropriate medical care can help mitigate the risks associated with fetal distress.[24]

Neonatal acidemia at birth is identified with umbilical artery cord blood pH of < 7.2, according to WHO guidelines. [21]

CTG association with fetal distress

CTG serves as a crucial tool in obstetrics for monitoring fetal well-being and detecting signs of fetal distress. By continuously monitoring the fetal heart rate and patterns, CTG allows healthcare providers to assess the baby's condition throughout pregnancy and labor. Interpreting CTG results necessitates clinical expertise and a thorough assessment of multiple factors to enhance the identification of fetal distress and enhance overall outcomes.

A CTG showing non-reassuring patterns often correlates with different levels of neonatal morbidity at birth.

The research by Paladugu and fellow researchers in Kerala from September 2020 to 2021 revealed a notable link between neonatal acidemia and pathological CTG patterns when compared to suspicious ones. Nevertheless, the study indicated that isolated abnormal fetal heart rate (FHR) characteristics did not demonstrate a significant correlation with acidosis.[25]

Anne-Charlotte Faivre and her team carried out a 4 year study in the Champagne-Ardenne region of France. Their findings demonstrated a significant association between “late decelerations and severe neonatal acidosis” . Furthermore, the study noted that in cases where fetal heart rate (FHR) variability was within normal range, the occurrence of variable decelerations did not result in severe neonatal acidosis in 97% of instances.[26]

This result has was similar to that observed by Cahill e al [12] and Martin Gamboni et al [27]

Using the physiological interpretation of CTG in accordance with the International Consensus Guidelines can aid in identifying the type of fetal hypoxia present. This understanding may allow clinicians to anticipate the rate at which the pH level in the fetal umbilical artery is likely to decline.By predicting the umbilical cord pH at birth, frontline clinicians can make informed decisions and implement timely interventions to optimize fetal oxygenation.This proactive approach could potentially improve outcomes for both the fetus and the mother.[28]

Efforts to investigate the association between intrapartum CTG findings and neonatal outcomes have been ongoing since the early 1970s. In 1971, Trevor Shelley and Richard H. from the Obstetrics “University Department of Obstetrics and Gynecology at The Jessop Hospital for Women in Sheffield” conducted a study where they examined 100 case records. [29]

They focused on analyzing fetal heart rate records during the last hour before delivery. They calculated “the sum of the products of the fall in heart rate and duration for each dip”, which they termed the “dip area”.The dip area served as a

measure of the amplitude, frequency, and duration of bradycardic episodes, disregarding their timing in relation to uterine contractions. [29]

The researchers created scattergrams by plotting the pH of umbilical venous and arterial blood against the dip area.

Their analysis of fetal heart rate records indicated that "the frequency, duration, and amplitude of transient episodes of fetal bradycardia, regardless of their timing in relation to uterine activity, were crucial factors in assessing fetal condition." [29]

They concluded that the dip area, being a quantitative measure of these factors, was easily communicable and avoided the current confusion surrounding the classification and nomenclature of fetal heart rate patterns.[29]

Cahill AG, and colleagues undertook a prospective cohort study involving 8580 women spanning from 2010 through 2015.

The study's main focus was on fetal acidemia, which was defined as an umbilical artery pH of less than 7.10. They chose this threshold to explore whether electronic fetal monitoring (EFM) patterns could help identify term fetuses at risk of abnormal pH levels.

Throughout the 120 minutes leading up to delivery, EFM patterns were analyzed in 10-minute segments, with a particular emphasis on calculating the total deceleration area. This metric was then compared against neonatal outcomes.Their findings indicated that the deceleration area emerged as the most predictive EFM pattern for acidemia. This suggests that monitoring this specific aspect of fetal heart

rate decelerations could be particularly useful in identifying fetuses at risk of acidemia during labor. [20]

Dr. Sabina Marti Gamboa and his research team conducted a study in Spain during 2012-2013, aiming to compare the predictive abilities of individual components of intrapartum cardiotocography (CTG) as described by the "National Institute of Child Health and Human Development (NICHD) system" against the deceleration area.

They analyzed 204 CTG traces obtained 30 minutes prior to delivery. Of these, 102 neonates developed neonatal acidemia, while the other 102 did not. The neonates who developed acidemia were classified as cases, while those who did not were classified as controls. Importantly, the individuals analyzing the traces were blinded to the outcome of the neonates.Their results revealed that Minimal variability (with an area under the curve [AUC] of 0.74), total number of late decelerations (AUC: 0.75), and prolonged decelerations (AUC: 0.77) were the top three "NICHD features" with predictive ability for fetal acidemia in the last thirty minutes of labor. However, total deceleration area demonstrated the highest discrimination power, with an AUC of 0.83, surpassing all other analyzed elements. [27]

They also found that for each cm2 increase in the deceleration area during the last thirty minutes of labor, pH decreased by 0.08 units, base deficit (BD) increased by 0.272 mEq/L, and lactate increased by 0.183 mEq/L. In conclusion, the study found that total deceleration area exhibited the greatest predictive ability for fetal acidemia.

This suggests that measuring the deceleration area could be valuable in estimating intrapartum fetal acid-base status [27]

Angela Agostinelli and their team conducted a study in 2016 at the

"Czech Technical University in Prague and the University Hospital in Brno". Their objective was to examine the relationship between "Deceleration Areas (DAs) in the Second Stage of Labor and Neonatal Acidemia".They analyzed 433 CTG traces, which were then classified into 34 cases and 399 controls.

The median DA values for Cases were 5.32 cm2, while for Controls, they were 1.44 cm2. They observed a significant inverse correlation ($\rho = -0.23$, $P < 10\text{-}6$) between DA in the last 60 minutes before delivery and pH at birth.

Their study concluded that deceleration area represents severe fetal bradycardia and is associated with critical fetal outcomes.

Moreover, they found that the deceleration area increases with rising levels of neonatal acidemia. [30]

Abby Furukawa et al conducted a retrospective cohort study involving singleton cephalic presenting neonates at term gestation.The study was conducted between "February 1, 2015, and January 31, 2017, at Legacy Health, a community-based hospital system in the Portland, OR, and Vancouver, WA metropolitan areas."

Their findings indicated that cumulative deceleration area could effectively replace the need to identify deceleration types.This measure quantifies three crucial aspects of decelerations "frequency, depth, and duration" into a single numerical value.

It was found to be a superior marker of meconium aspiration (MA) compared to "baseline level, baseline variability, and the number of late decelerations". The group with acidemia showed higher deceleration area over the last two hours before

delivery, suggesting that the cumulative area and persistence of repetitive decelerations are clinically important markers.[31]

Marta Chóliz Ezquerro, along with Ricardo Savirón Cornudella and their team, conducted a "retrospective case-control study at Miguel Servet University Hospital in Spain, focusing on 5694 pregnant women between June 2017 and October 2018". They analyzed the 30-minute CTG trace before delivery and found that parameters such as total reperfusion time and total deceleration area have significant predictive ability for neonatal acidemia. Interestingly, these non-ACOG parameters performed better than the ACOG III classification, with no statistical differences between them.

This suggests that these parameters could be valuable indicators of fetal well-being and the risk of neonatal acidemia during labor [32]

Shirel Matmor Loeub and colleagues undertook a study to investigate the relationship between the "cumulative area under the Curve (AUC) of decelerations and accelerations and neonatal acidemia in pregnancies affected by meconium-stained amniotic fluid (MSAF)".

They examined 102 women and found a significant association between the total AUC of decelerations and accelerations and the pH levels in cord blood.This discovery suggests potential implications for the monitoring and care of pregnancies with MSAF to mitigate the risks associated with neonatal acidemia. [33]

Between 2013 and 2019, Geva Y and colleagues investigated 95 instances of low-risk pregnancies complicated by moderate to severe neonatal encephalopathy, a complication arising from Intrapartum fetal distress. Out of these, 33 deliveries (34.7%) were excluded due to insufficient duration of CTG recordings. The remaining

62 cases were compared with 123 controls.Their findings indicated a significant connection between neonatal encephalopathy, and an increased total deceleration area, a decreased total acceleration area, and a reduced acceleration-to-deceleration.[34]

Gal Cohen and colleagues conducted a study in Israel where they analyzed 85 vacuum-assisted deliveries performed due to “non-reassuring fetal heart rate”. They examined 120 minutes of CTG trace preceding delivery and compared it with fetal outcomes in terms of umbilical cord blood pH. They found a negative correlation between deceleration area under the curve (dAUC) in the 60 minutes leading up to delivery and umbilical cord pH.

Specifically, for every 10K units increase in dAUC, the cord pH decreased by 0.02.However, bradycardia/tachycardia and decreased variability in the 30 minutes before delivery did not show a correlation with changes in umbilical cord pH. [35]

This shows that deceleration area is a much more significant tool in identifying neonatal acidemia at birth when compared to other individual components of the NICHD classification.

MATERIALS AND METHODS

Study design: Case control study

Study setting: KAHER'S Dr. Prabhakar Kore Charitable Hospital, Belagavi

Study period: February 2023 – January 2024

Study Population: Pregnant women at or above 37 weeks of gestation, who are admitted to the labour room for delivery in KAHER's Dr. Prabhakar Kore

Hospital and Medical Research Centre, Nehru Nagar, Belagavi during the study period.

Institutional Ethical Clearance obtained, Ref-MDC/JNMC/IEC/302.

CTRI Reg No CTRI/2023/02/049506.

f) Selection Criteria -The study population will be screened as per the inclusion & exclusion criteria. The eligible study participants will be enrolled for the study after taking informed consent.

Inclusion criteria:

- Singleton
- Term pregnancy in labor
- intrapartum fetal distress diagnosis made
- Presence of 30 min CTG trace recording before time of delivery.
- Presence of Umbilical Artery cord blood gas analysis at the time of delivery

Exclusion Criteria:

- Insufficient CTG Trace
- Unavailability of Umbilical Artery cord blood gas analysis at the time of delivery

Methodology – The detailed antenatal history, presenting complaints, obstetric risk factors and complications in present pregnancy, menstrual and obstetric history were noted.

Admission CTG was noted. Intrapartum fetal heart rate monitoring was done with fetal doppler every half an hour during the first stage & every 15 mins in second stage.

CTG trace was repeated anytime during the course of labor if the FHR abnormality is detected.

Based on the CTG findings of non- reassuring fetal heart rate (NICHD criteria) FHR resuscitation was done using intrapartum strategies (left lateral position, started on O2 by face mask, IV Fluids, discontinuation of uterotonics).

The decision for emergent delivery (Vaginal/Instrumental/Caesarean section) was taken by the on-duty obstetric consultant.

Umbilical artery cord blood samples are taken and tested for pH, pCO2, pO2, HCO3.

The study participants were then categorised into 2 groups, case and control, based on the cord blood pH value at birth.

pH less than or equal to 7.2 were considered as cases and pH > 7.2 as controls.[21]

The Intrapartum events, duration of labor, mode of delivery, stage of labor when fetal distress was diagnosed, baby details (Time of delivery, birth weight, sex, resuscitation measures, Apgar score, NICU admission) were noted

The Umbilical cord blood pH value was evaluated against the total deceleration area in 30 mins prior to instrumental delivery/Shifting the patient to Operation Theatre for caesarean delivery.

Sample size

$$n = \frac{(r+1)\left(Z_{\alpha/2} + Z_{\beta}\right)^2}{r\left(\frac{|\mu_1 - \mu_2|}{\sigma}\right)^2}$$

$$d = \frac{|\mu_1 - \mu_2|}{\sigma}$$

Where $\mu 1$ is the mean of the first group, μ_2 is the mean of the second group and σ^2 is the common error variance, for 95% confidence level, $Z_{\alpha/2}$ values are 1.96 and for 80% power Z_{β} value is 0.84 and **r** is the allocation ratio, which is given as $r = n_1/n_2$. N is the total sample size, that is, $N = n_1+n_2$.

Here we assume **d** as moderate, i.e., d = 0.5, with this assumed d, 95% confidence level and 80% power, we take allocation ratio for case to control as 1:3.

Sample sizes are,

***For* case *group*: n_1 = 42**

***For* control *group*: n_2 = 126**

Overall sample size required is 168.

Statistical Analysis:

Data is analysed using statistical software R version 4.3.2 and Microsoft Excel. Categorical variables given in the form of frequency tables. Continuous variables given in Mean ± SD / Median (Min, Max) form. Normality of variable is checked by Shapiro Wilk test and QQ plot. Two sample t test is used to compare the means of variables over groups. Mann Whitney U test is used to compared the distribution of variables over groups. Chi square test is used to check the association of categorical variables with groups. Spearman's rank correlation test is used to check the correlation of different variables with pH. Kruskal Wallis test is used to check the distribution of pH over type of deceleration. Dunn test is used as post hoc analysis. Applicability of TDA to predict neonatal acidemia is checked by Logistic regression and Receiver Operating Characteristic (ROC) curves. Cut off values are obtained by Youden index. Pairwise comparison of ROC curves is done to compare the determinant power of TDA and reperfusion time. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

In this study, a total of 402 participants underwent screening, with 234 individuals excluded, the analysis focused on 168 participants.

Cord blood pH levels were recorded at birth for all foetuses, based on which participants were then categorized into either case or control groups. 42 cases and 146 controls were included in the study for further analysis.

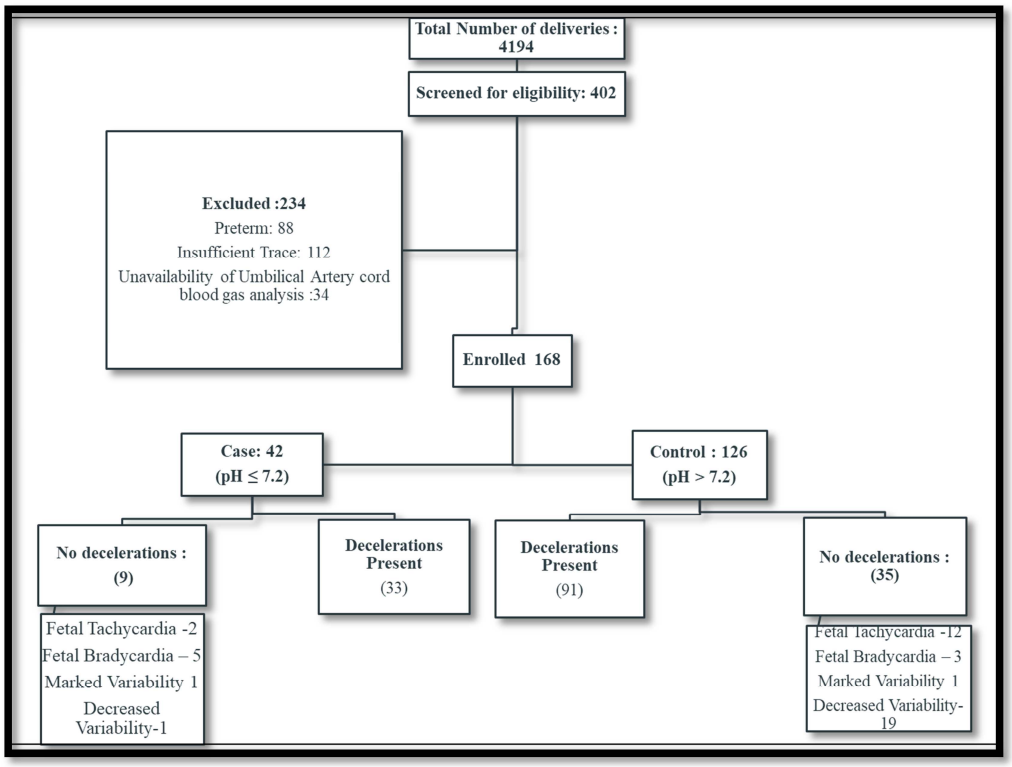
During the study period 4194 deliveries took place, 402 women were screend for eligibility,of which 234 participants were excluded,due to preterm, insufficient trace, unavailability of cord blood pH.

A total of 168 participants were enrolled, Out of which 42 newborns (25%) had neonatal acidemia while 126 (75%) did not.

On further analyzing the Intraparrtum category 2 and category 3 (NICHD) CTG traces, 9 cases were noted to have no decelerations and 35 controls did not have any decelerations. (Figure 1)

The following results are from the data analysis of the 168 participants.

Fig 1 Strobe Diagram



From Table 1 we can observe that the mean age in both case and control group is approximately 24 yrs. On the basis of Socio-economic status, the middle class is more prevalent in both the groups. Primigravidae are seen to be 80% of both the case and control group. The mean BMI in both groups range between 24 to 25 Kg /m^2

From Mann Whitney U test, it is observed that, there is no significant difference in the distribution of age over groups.

From Chi square test, it is observed that there is no significant difference in the distribution of SES and Obstetric score over groups.

From two sample t test, it is observed that there is no significant difference in the mean BMI over groups.

Table 1: Clinical and socio demographic details in Cases & Control group.

Variables	Sub category	Groups		p-value
		Case (n = 42)	Control(n=126)	
Age (years)	Mean ± SD	23.98 ± 3.5	24.52 ± 3.71	0.4685^{MW}
	Median (Min, Max)	24 (19, 35)	24 (18, 38)	
Socio Economic status	Lower Class	12 (28.57%)	52 (41.27%)	0.3678^{MC}
	Middle Class	28 (66.67%)	69 (54.76%)	
	Upper Class	2 (4.76%)	5 (3.97%)	
Obstetric Score	Primipara	35 (83.33%)	103 (81.75%)	0.8161^{C}
	Multipara	7 (16.67%)	23 (18.25%)	
BMI (Kg/m2)	Mean ± SD	25 ± 3.44	24.55 ± 2.89	0.4016^{t}
	Median (Min, Max)	24.62 (19.17, 35.2)	24.6 (18.42, 34)	

Abbreviation: MW – Mann Whitney U test, C – Chi square test, MC – Chi square test with Monte Carlo simulation, t – Two sample t test.

From Table 2 it is seen that nearly 73 to 74% of pregnancies of both the case and control group belong to the high-risk category.

Table 2: Antenatal risk categorisation in case & control group

Sub-Category	Groups	
	Case (n=42)	Control (n=126)
High Risk	31 (73.80%)	94 (74.60%)
Low Risk	11 (26.19%)	32 (25.39%)

Table 3 shows the distribution of high risk factors in both the case and control group ,of the High risk factors, Post datism is the most common accounting to 35.48% in the case group and 34.04% in the control group. Followed by FGR, responsible for 22.58% of high-risk pregnancies in the case group and 18.08% in the control group. Oligohydramnios is seen to the third most common high-risk factor noted in 6.45% of the cases and 7.44% of the controls. PROM is seen in 3.2% of cases and 8.51% of the controls. GDM is responsible for 6.45% of the high-risk pregnancy in the case group and 3.19 % in the control group.

From Chi square test, it is observed that there is no significant difference in the distribution of risk factors over groups.

The following table gives the comparison of Intrapartum risk factors / Complications over groups.

Table 3: Distribution of Antenatal risk factors in case & control group.

Antenatal Risk Factor	Group		P Value
	Case (n=31)	Control (n=94)	
Post datism	11 (35.48%)	32 (34.04%)	0.88
FGR	7 (22.58%)	17 (18.08%)	0.58
Oligohydramnios	2 (6.45%)	7 (7.44%)	0.99
PROM	1 (3.22%)	8 (8.51%)	0.46
GDM	2 (6.45%)	3 (3.19%)	0.59
HTN	2 (6.45%)	4 (4.25%)	0.64
Macrosomia	0	6 (6.38%)	0.35
Hypothyroidism	3 (9.67%)	4 (4.25%)	0.37
Anaemia	1 (3.22%)	5 (5.31%)	0.99
Pre-eclampsia	1 (3.22%)	5 (5.31%)	0.99
Prev LSCS	0	2 (2.12%)	0.99
Prolonged Latent phase	1 (3.22%)	0	0.26
BOH	0	1(1.06%)	0.99

Abbreviation: MC – Chi square test with Monte Carlo simulation, C – Chi square test.

Table 4 shows the risk factor noted intrapartum, a risk factor was identified in 25(59.52%) of the case group and 77 (61.11%) of the control group.

Table 4: Distribution of Intrapartum risk factors / Complications in case & control group

Intrapartum Risk Factor	Group	
	Case (n=42)	**Control (n=126)**
Risk noted	25(59.52%)	77 (61.11%)
No Risk	17 (40.48%)	49 (38.89%)

Table 5 shows the distribution of intrapartum risk factors noted between the case and control group, 59.52 % of the cases and 61.11% of the control had an identifiable intrapartum risk factor which could explain the cause of fetal distress.

MSL contributed to 42.86% in the case group and 37.30% in the control group.

Cord around the fetal was noted in 11.9%of the case and 10.3% of the control group.

Oligohydramnios was seen in 2.38% of the cases and 10.32% of controls.

From Chi square test, it is observed that there is no significant difference in the distribution of Intrapartum risk factor over groups. With a p value of 0.8366^{MC}

Table 5: Distribution of Intra partum Risk Factors over case & control groups

Intrapartum Risk Factor	Group	
	Case (n=25)	Control (n=77)
MSL	18 (42.86%)	47 (37.30%)
Cord around neck	5 (11.9%)	13 (10.32%)
Oligohydramnios	1 (2.38%)	13 (10.32%)
DTA	1 (2.38%)	1 (0.79%)
Intrapartum Hypertension	0	2 (1.58%)
True Knot	0	2 (1.58%)

Abbreviation: MC – Chi square test with Monte Carlo simulation, C – Chi square test.

Table 6 shows the delivery details in both the case and control group.

From Chi square test, it is observed that there no significant difference in the distribution of type of labour, however there is significant difference in the distribution of stage of labour distress detected. Fetal distress detected in the second stage of labour is seen to be significantly associated with academic neonates at birth. (p value 0.0020)

Mode of delivery was also showed significant difference, Instrumental deliveries were significantly noted to be higher in the case group 12 (28.57%).From Mann Whitney U test, it is observed that, there is no significant difference in the distribution of duration of labour of stage 1 over groups. From two sample t test, it is observed that, there is no significant difference in the mean duration of labour of stage 2 over groups.

Table 6: Distribution of delivery details in case and control group in cases and control group

Variables	Sub category	Group		p-value
		Case	Control	
Type of Labor	Induced	24 (57.14%)	73 (57.94%)	0.9281[C]
	Spontaneous	18 (42.86%)	53 (42.06%)	
Stage of Labor when fetal Distress detected	Latent Labor	23 (54.76%)	91 (72.22%)	
	Active Labor	5 (11.9%)	22 (17.46%)	
	2nd Stage	14 (33.33%)	13 (10.32%)	**0.0020[C]***
Mode Of delivery	LSCS	27 (64.29%)	117 (92.86%)	
	Instrumental	12 (28.57%)	7 (5.56%)	**<0.001[MC]***
	FTVD	3 (7.14%)	2 (1.59%)	
Duration of labour Mean ± SD Median (Min, Max)	1st Stage	13.1 ± 9.3 11.5 (1, 50)	12.6 ± 8.43 10 (1, 45)	0.708[MW]
	2nd Stage	0.51 ± 0.29 0.5 (0.1, 1)	0.5 ± 0.18 0.5 (0.16, 0.75)	0.9137[t]

*Abbreviation: MC – Chi square test with Monte Carlo simulation, C – Chi square test, MW – Mann Whitney U test, t – Two sample t test, * indicates statistical significance.*

Figure 2 shows the graphical representation of the distribution of satge of labour when fetal distress was detected between the case and the control group.

Figure 2: Distribution of Stage of labour when fetal distress was detected over groups.

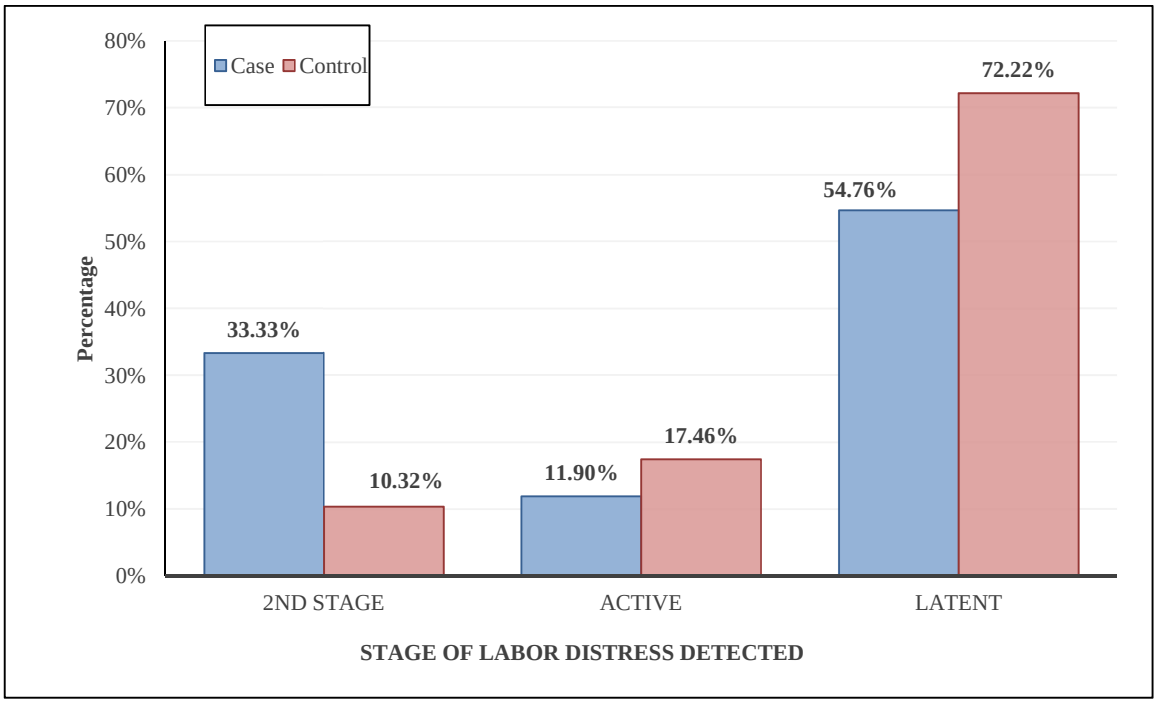


Figure 3 shows the distrubtion between cases and controls with respect to the mode of delivery.

Figure 3: Mode of delivery over groups.

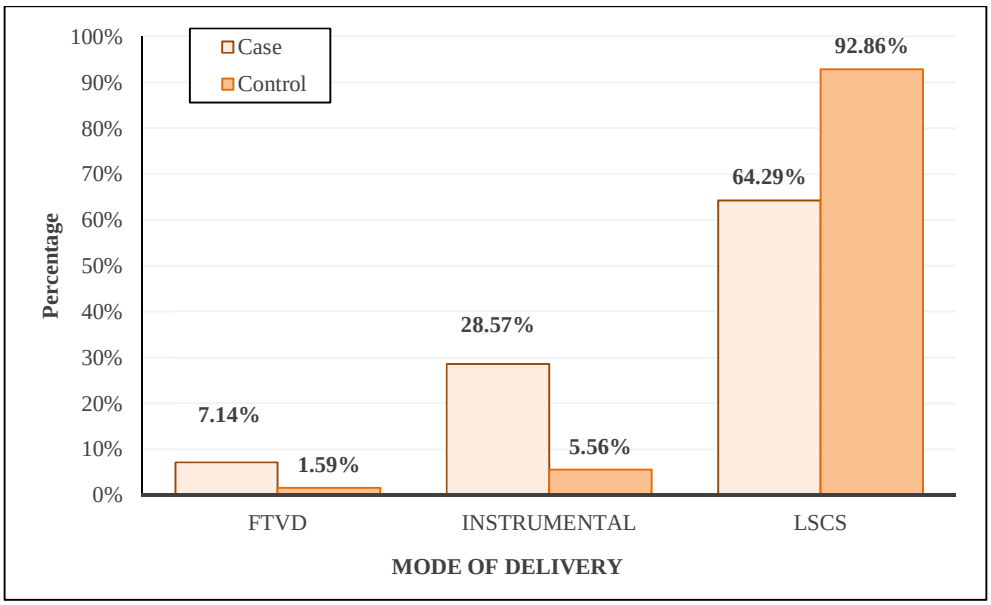


Table 7 show the distribution of NICHD variables between the case and the control group. From Chi square test, it is observed that there is significant difference in the distribution of fetal bradycardia over groups (p value 0.0295). 5 (11.9%) of the academic babies had fetal bradycardia, whereas only 3 (2.38%) of the controls were noted to have fetal bradycaridia.

There is no significant difference noted in the distribution of Fetal Tachycardia, beat to beat variability <5 or >25 and Repeated variable deceleration over groups.

There is a significant difference in the distribution of type of deceleration noted over g roups, Late decelerations are noted to associated significantly with neonatal acidemia, 11 (26.19%) of the case group and 10 (7.94%) of the control group were noted to hav e late decelerations.

Table 7: Distribution of different NICHD variables over case & control groups.

Variables	Sub category	Group		p-value
		Case	Control	
Fetal Tachycardia	Yes	8 (19.05%)	30 (23.81%)	0.5229[C]
	No	34 (80.95%)	96 (76.19%)	
Fetal Bradycardia	Yes	5 (11.9%)	3 (2.38%)	**0.0295[MC]***
	No	37 (88.1%)	123(97.62%)	
Beat to beat variability <5 or >25	Yes	20 (47.62%)	65 (51.59%)	0.6560[C]
	No	22 (52.38%)	61 (48.41%)	
Repeated variable deceleration	Yes	3 (7.14%)	11 (8.73%)	0.7871[MC]
	No	39 (92.86%)	113(89.68%)	
Type of deceleration	Early	2 (4.76%)	15 (11.9%)	
	Late	11 (26.19%)	10 (7.94%)	**0.0035[MC]***
	Variable	20 (47.61%)	82(57.74%)	

*Abbreviation: C – Chi square test, MC – Chi square test with Monte Carlo simulation, * indicates statistical significance.*

Table 8 shows the Comparision of mean pH between different types of decelerations noted.From Kruskal Wallis test, it is observed that, there is significant difference in the distribution of pH level over type of deceleration. Further from post hoc analysis, it is observed that, there is significant difference in pH level between late and early deceleration (p-value = 0.0497), late and variable deceleration (p-value = 0.0134). It can be noted that pH level in late deceleration is significantly less than early and variable deceleration.

Table 8: Comparison of pH value over type of deceleration.

Type of Deceleration	**pH**		**p-value**
	Mean ± SD	**Median (Min, Max)**	
Early	7.28 ± 0.06	7.27 (7.11, 7.36)	**0.0120^{K}***
Late	7.2 ± 0.1	7.21 (6.93, 7.34)	
Variable	7.26 ± 0.08	7.28 (6.96, 7.38)	

*Abbreviation: K – Kruskal Wallis test, * indicates statistical significance.*

Figure 4 shows the mean plot of pH over the type of deceleration, it can be noted that the mean pH is lower in Late decelerations.

Figure 4: Mean plot of pH over type of deceleration.

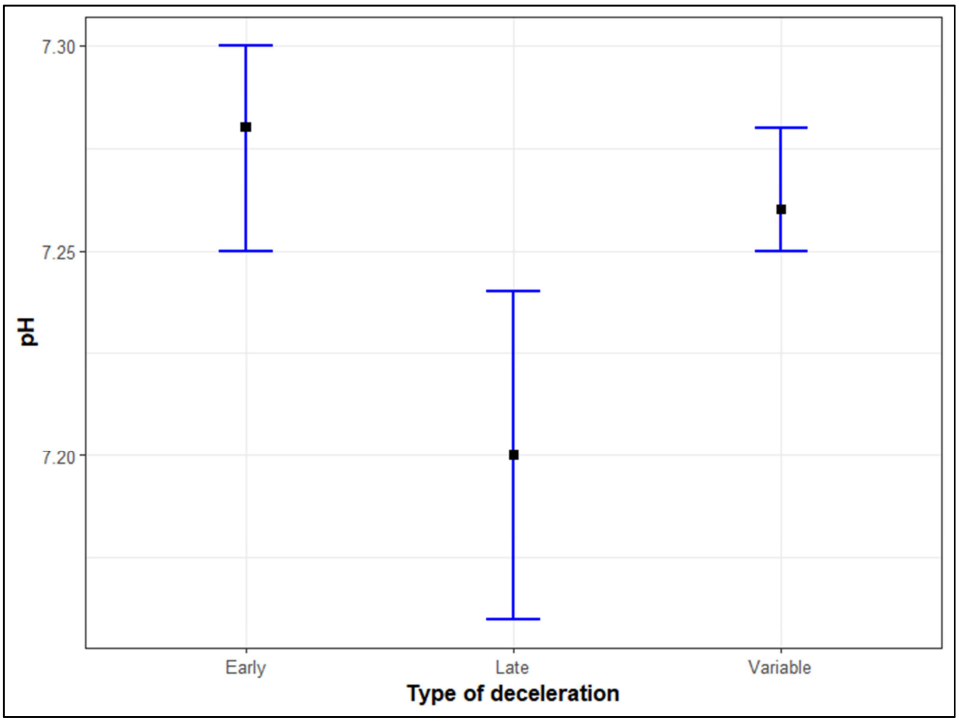


Table 9 shows the various birth characteristics noted between the case and the control groups,from Chi square test, it is observed that, there is significant difference in the distribution of cry at birth and NICU admission over groups. Babies with no spontaneous cry at birth were seen to be significantly associated with neonatal acidemia, 7 (16.67%) of the babies who had neonatal acidemia did not have a spontaneous cry at birth.NICU admissions were also more significantly seen to be associated with the case group when compared to the control group. 9 (21.43%) of the cases had NICU admission.

From Mann Whitney U test, it is observed that, there is no significant difference in the distribution of baby weight over groups.

The mean weight at birth noted was 2.76 kg in the case group and 2.79 kg in the control group.

There is no significant difference in the distribution gender of baby over groups.

Table 9: Birth characteristics of neonates in case & control group

Variables	Sub category	Group		p-value
		Case	Control	
Cry at birth	No	7 (16.67%)	4 (3.17%)	**0.0085^{MC}***
	Yes	35 (83.33%)	122 (96.83%)	
Baby weight	LBW	11 (26.19%)	16 (12.7%)	0.0720^{MC}
	Normal weight	31 (73.81%)	107 (84.92%)	
	VLBW	0	3 (2.38%)	
	Mean ± SD	2.76 ± 0.37	2.79 ± 0.39	0.427^{MW}
	Median (Min, Max)	2.7 (2.2, 3.5)	2.8 (1.8, 3.72)	
Gender of baby	Female	20 (47.62%)	61 (48.41%)	0.9290^{C}
	Male	22 (52.38%)	65 (51.59%)	
NICU admission	No	32 (76.19%)	118 (93.65%)	**0.0030^{MC}***
	Yes	9 (21.43%)	5 (3.97%)	

*Abbreviation: MC – Chi square test with Monte Carlo simulation, C – Chi square test, MW – Mann Whitney U test, * indicates statistical significance.*

Table 10 shows the mean TDA noted in both the case and the control group. from Mann Whitney U test, it is observed that, there is significant (p value 0.0082) difference in the distribution of TDA over groups. Further, it is noted that mean TDA of 254.62 is more among those in case group compared to mean TDA of 165.1 in the control group.

Table 10: Comparison of Total deceleration area over groups.

Variables	**Sub category**	**Group**		**p-value**
		Case	**Control**	
Total Deceleration Area	Mean ± SD Median (Min, Max)	254.62 ± 170.67 228.75 (25, 740)	165.1 ± 94.17 150 (15, 420)	**0.0082MW***

*Abbreviation: MC – Chi square test with Monte Carlo simulation, C – Chi square test, MW – Mann Whitney U test, * indicates statistical significance.*

Figure 5 show sthe mean plot of TDA against the case and the control group, it is noted that the case group has a higher TDA in comparison to the control group.

Figure 5: Mean plot of TDA over groups.

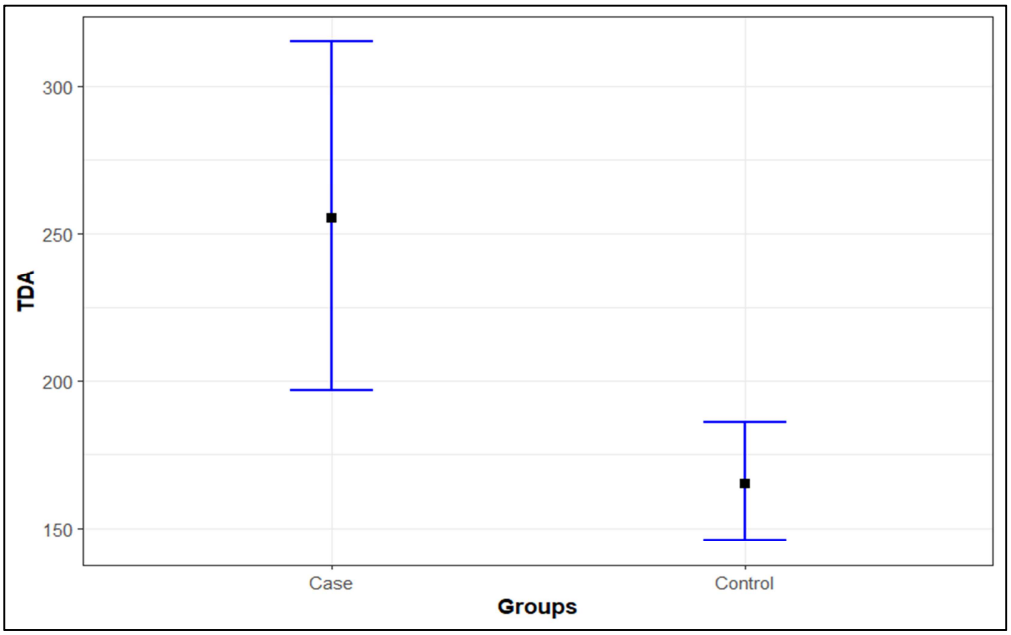


Figure 6 shows the scatter plot diagram of TDA plotted against pH, it is noted that slope is suggestive of negative corelation.

Figure 6: Scatter plot of pH with TDA.

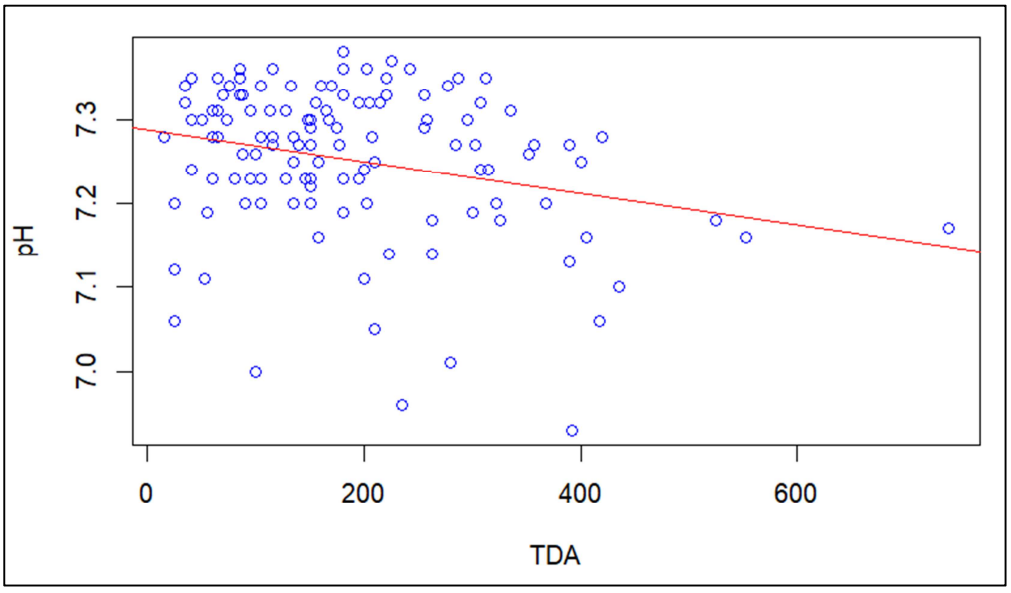


Table 11 shows the data analaysis of Logistic regression of TDA with a cutoff of 195. The AU-ROC for TDA score is 0.6576 at cutoff > 195 with 68.89% sensitivity and 62.50% specificity in predicting neonatal acidemia. From logistic regression, we observe that TDA is significantly predicting neonatal acidemia (p-value **0.0014**).

Table 11: Analysis of TDA for predicting neonatal acidemia.

TDA Cut off for neonatal acidemia	**(>) 195**
Sensitivity (95% CI)	68.89% (58.26%, 78.23%)
Specificity (95% CI)	62.50% (43.69%, 78.90%)
PPV (95% CI)	83.78% (70.64%, 89.35%)
NPV (95% CI)	41.67% (31.05%, 61.58%)
AU-ROC (95% CI)	0.6576 (0.5305, 0.7847)
p-value	**0.0014***

Table 12 shows the multiple logistic regression done over different variables in predicting neonatal acidemia ,from logistic regression, we observe that stage of labour distress detected is significantly predicting neonatal acidemia. Specifically, the odds of having neonatal acidemia is 4.2609 (95%CI: 1.7660 – 10.4473) times higher in the 2nd stage of labour distress compared to the latent stage (p-value = 0.0013). we observe that mode of delivery is significantly predicting neonatal acidemia. Specifically, the odds of having neonatal acidemia is 0.1538 (95%CI: 0.0195 – 0.9708) times lower in LSCS compared to FTVD. we also observe that Fetal bradycardia is significantly predicting neonatal acidemia. Specifically, the odds of having neonatal acidemia is 5.5405 (95%CI: 1.2988 – 28.0501) times more in participants with fetal bradycardia compared to those without fetal bradycardia. we observe that type of deceleration is significantly predicting neonatal acidemia. Specifically, the odds of having neonatal acidemia is 8.25 (95%CI: 1.7461 – 61.2103) times more in participants who had late deceleration compared to those who has early deceleration. we observe that cry at birth is significantly predicting neonatal acidemia. Specifically, the odds of having neonatal acidemia is 0.1639 (95%CI: 0.0409 – 0.5743) times lower in babies who cried at birth compared to those who didn't. we observe that NICU admission is significantly predicting neonatal acidemia. 0.0014* Specifically, the odds of having neonatal acidemia is 6.6375 (95%CI: 2.1419 – 22.9166) times more in babies who had NICU admission compared to those who didn't need NICU admission. we observe that TDA is significantly predicting neonatal acidemia (p-value = 0.0014). With unit increase in TDA, the odds of having neonatal acidemia increases by a factor of 1.0057 (95%CI: 1.0024 – 1.0095).

Table 12: Logistic regression analysis of different variables for predicting neonatal acidemia.

Variable	Sub group	Odds ratio	P value
Stage of labour when fetal distress is detected	2nd stage	4.2609 (1.7660, 10.4473)	**0.0013***
	Active	0.8992 (0.2780, 2.4782)	0.8462
Mode of delivery	Instrumental	1.1429 (0.1266, 8.6506)	0.8968
	LSCS	0.1538 (0.0195, 0.9708)	**0.0459***
Fetal bradycardia	Yes	5.5405 (1.2988, 28.0501)	**0.0232***
Type of deceleration	Late	8.2500 (1.7461, 61.2103)	**0.0153***
	Variable	1.7045 (0.4184, 11.5441)	0.5078
Cry at birth	Yes	0.1639 (0.0409, 0.5743)	**0.0058***
NICU admission	Yes	6.6375 (2.1419, 22.9166)	**0.0014***
TDA	Cut off >195	1.0057 (1.0024, 1.0095)	**0.0014***

*Abbreviation: OR – Odds ratio, CI – Class interval, * indicates statistical significance.*

Referance variable for:

Stage of labor – Latent labor

Mode of delivery – FTVD

Fetal bradycardia – No bradycardia

Type of deceleration – Early

Cry at birth – No cry at birth

NICU admission- No Admission

Table 13 shows the data analysis of logistic regression analysis of different variables in predicting neonatal acidemia.The AU-ROC for Stage of labour distress detected is 0.6199 with 33.33% sensitivity and 89.68% specificity in predicting neonatal acidemia. (Figure 7)

The AU-ROC for mode of delivery (FTVD) is 0.6431 with 35.71% sensitivity and 92.86% specificity in predicting neonatal acidemia. (Figure 8)

The AU-ROC for Fetal bradycardia is 0.5476 with 11.90% sensitivity and 97.62% specificity in predicting neonatal acidemia. (Figure 9)

The AU-ROC for type of deceleration(Late decelerations) is 0.6597 with 39.29% sensitivity and 89.01% specificity in predicting neonatal acidemia.(Figure 10)

The AU-ROC for cry at birth is 0.5675 with 16.67% sensitivity and 96.83% specificity in predicting neonatal acidemia.(Figure 11)

The AU-ROC for NICU admission is 0.5894 with 21.95% sensitivity and 95.93% specificity in predicting neonatal acidemia.(Figure 12)

The AU-ROC for TDA score is 0.6576 at cutoff > 195 with 68.89% sensitivity and 62.50% specificity in predicting neonatal acidemia. (Figure 13)

Table 13: The diagostic values of different variables in predicting neonatal acidemia

Variable	Sensitivity (95% CI)	Specificity (95% CI)	PPV (95% CI)	NPV (95% CI)	AU-ROC (95% CI)
Stage of labour when fetal distress is detected	**33.33%** (19.57%, 49.55%)	**89.68%** (83.00%, 94.39%)	**51.85%** (31.95%, 71.33%)	**80.14%** (72.59%, 86.38%)	**0.6199** (0.5299 - 0.7099)
Mode of delivery	**35.71%** (21.55%, 51.97%)	**92.86%** (86.87%, 96.68%)	**62.50%** (40.59%, 81.20%)	**81.25%** (73.90%, 87.27%)	**0.6431** (0.5662 - 0.7201)
Fetal Bradycardia	**11.91%** (3.98%, 25.63%)	**97.62%** (93.20%, 99.51%)	**62.50%** (24.49%, 91.48%)	**76.88%** (69.56%, 83.17%)	**0.5476** (0.4963 - 0.5990)
Type of deceleration	**39.29%** (21.50%, 59.42%)	**89.01%** (80.72%, 94.60%)	**52.38%** (29.78%, 74.29%)	**82.65%** (73.69%, 89.56%)	**0.6597** (0.5552 - 0.7643)
No Cry at birth	**16.67%** (6.97%, 31.36%)	**96.83%** (92.07%, 99.13%)	**63.64%** (30.79%, 89.07%)	**77.71%** (70.38%, 83.95%)	**0.5675** (0.5084 – 0.6265)
NICU admission	**21.95%** (10.56%, 37.61%)	**95.93%** (90.77%, 98.67%)	**64.29%** (35.14%, 87.24%)	**78.67%** (71.24%, 84.93%)	**0.5894** (0.5229 - 0.6559)
TDA Cut off for neonatal acidemia (>) 195	**68.89%** (58.26%, 78.23%)	**62.50%** (43.69%, 78.90%)	**83.78%** (70.64%, 89.35%)	**41.67%** (31.05%, 61.58%)	**0.6576** (0.5305, 0.7847)

Figure 7: ROC curve of Stage of labour fetal distress detected for predicting neonatal acidemia.

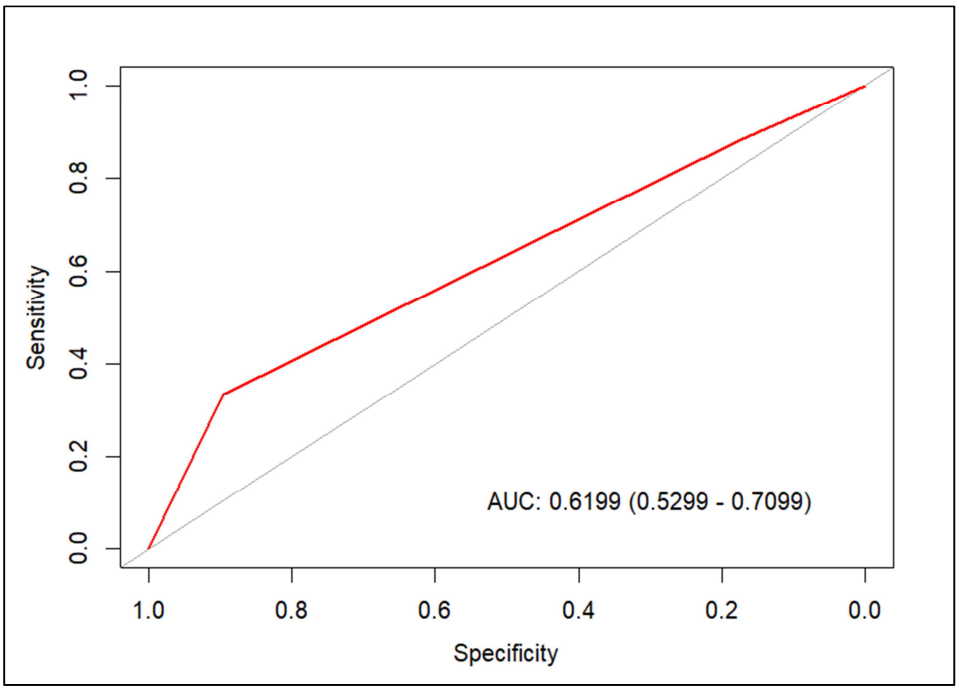


Figure 8: ROC curve of mode of delivery for predicting neonatal acidemia

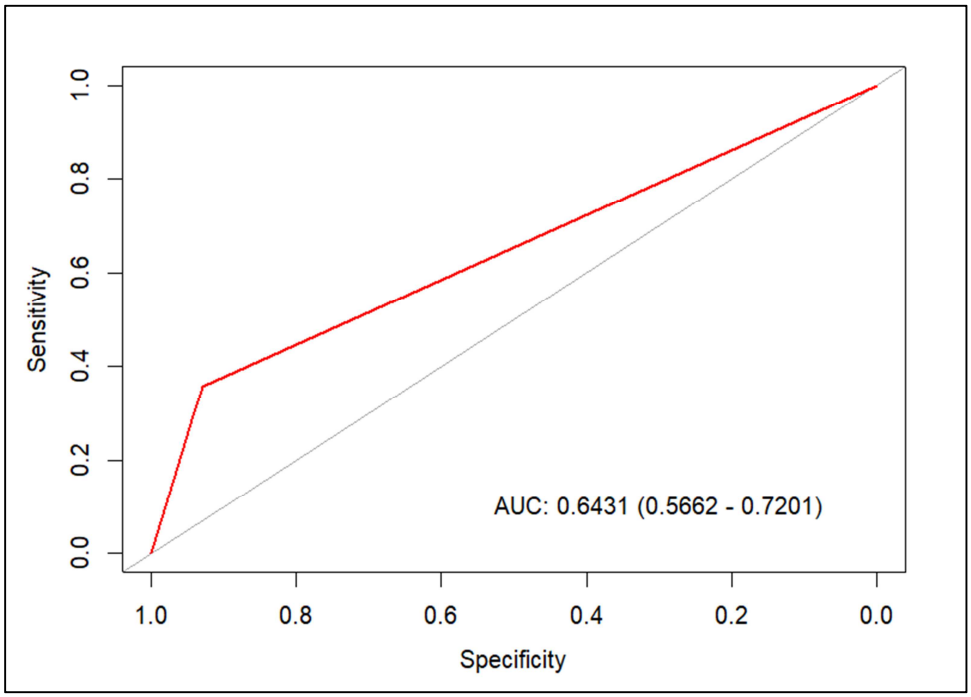


Figure 9: ROC curve of Fetal bradycardia for predicting neonatal acidemia.

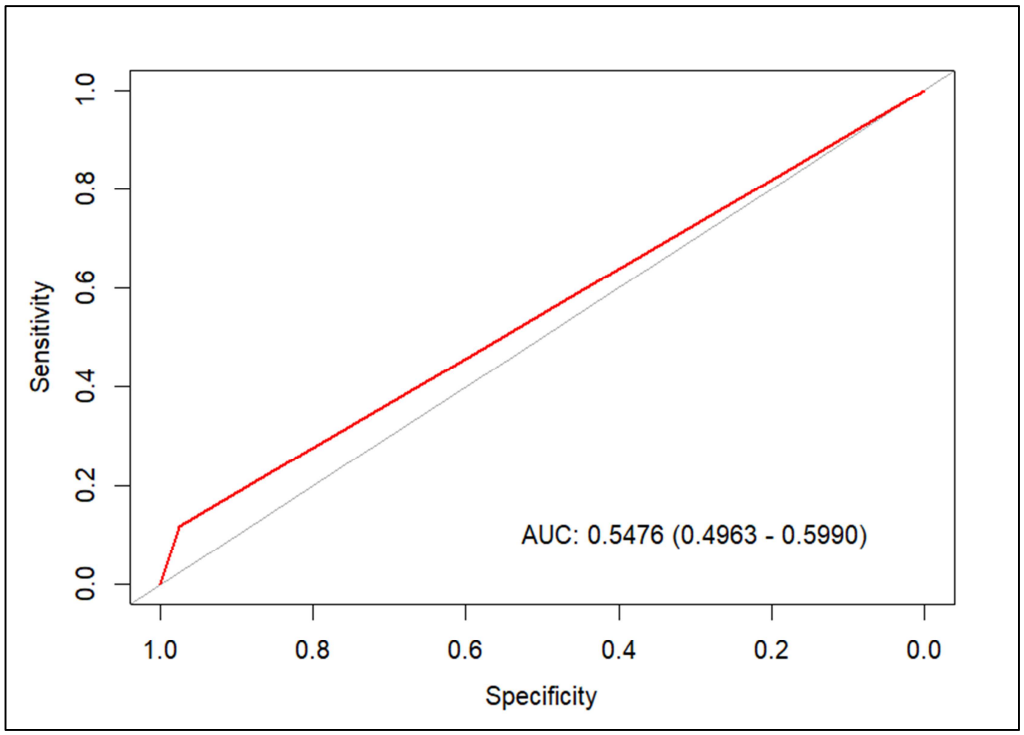


Figure 10: ROC curve of late deceleration for predicting neonatal acidemia.

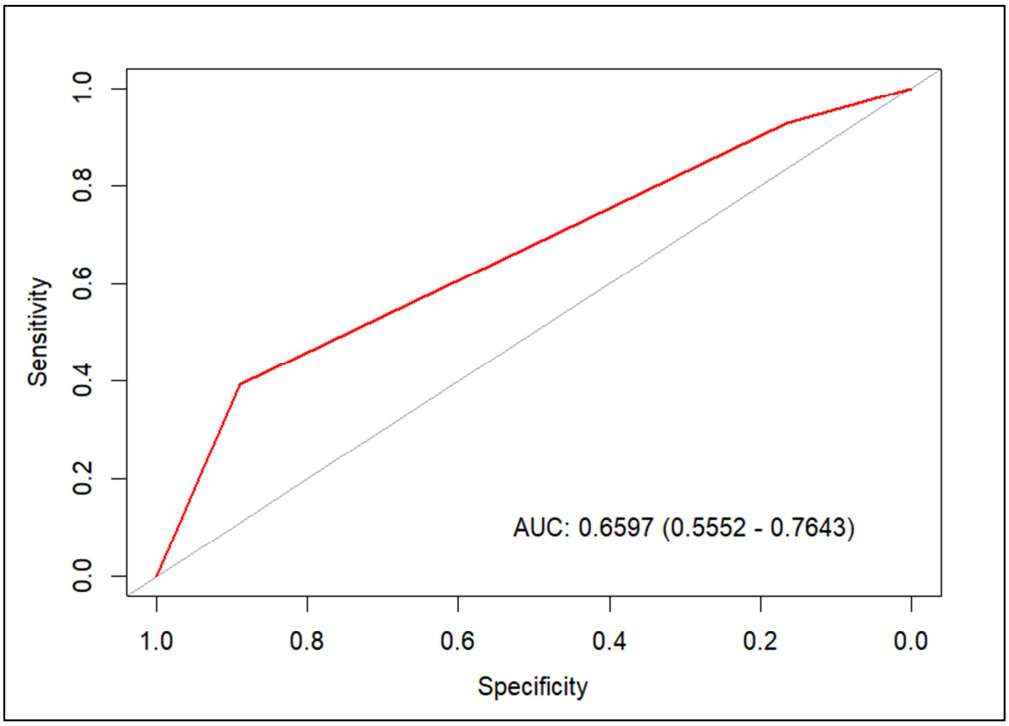


Figure 11: ROC curve of no cry at birth for predicting neonatal acidemia.

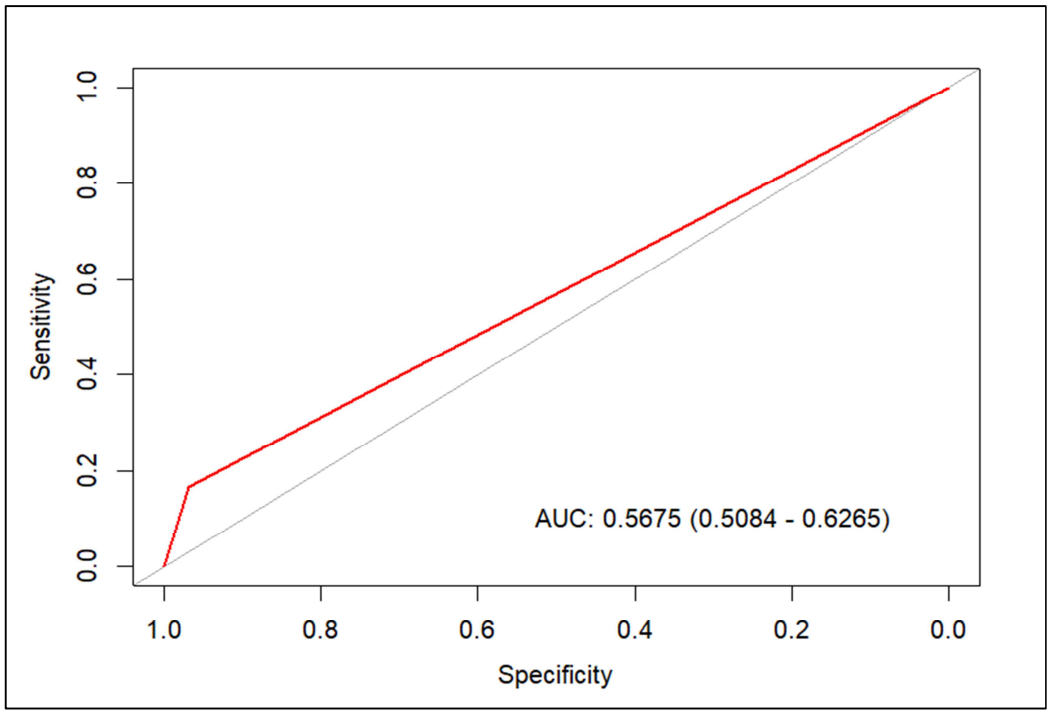


Figure 12: ROC curve of NICU admission for predicting neonatal acidemia.

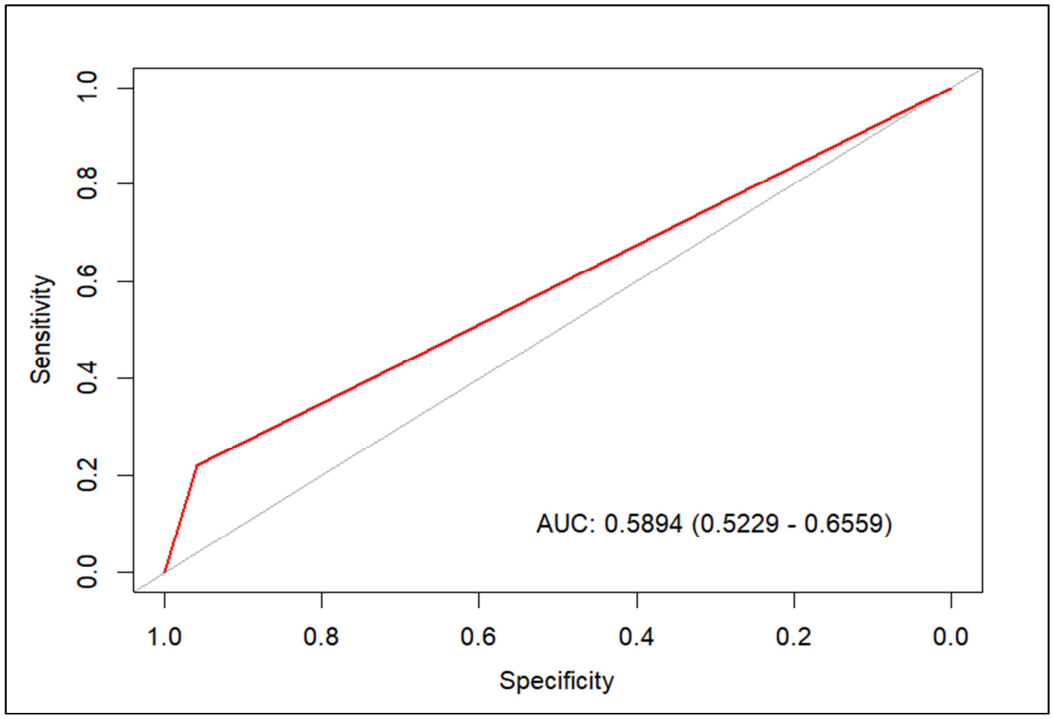
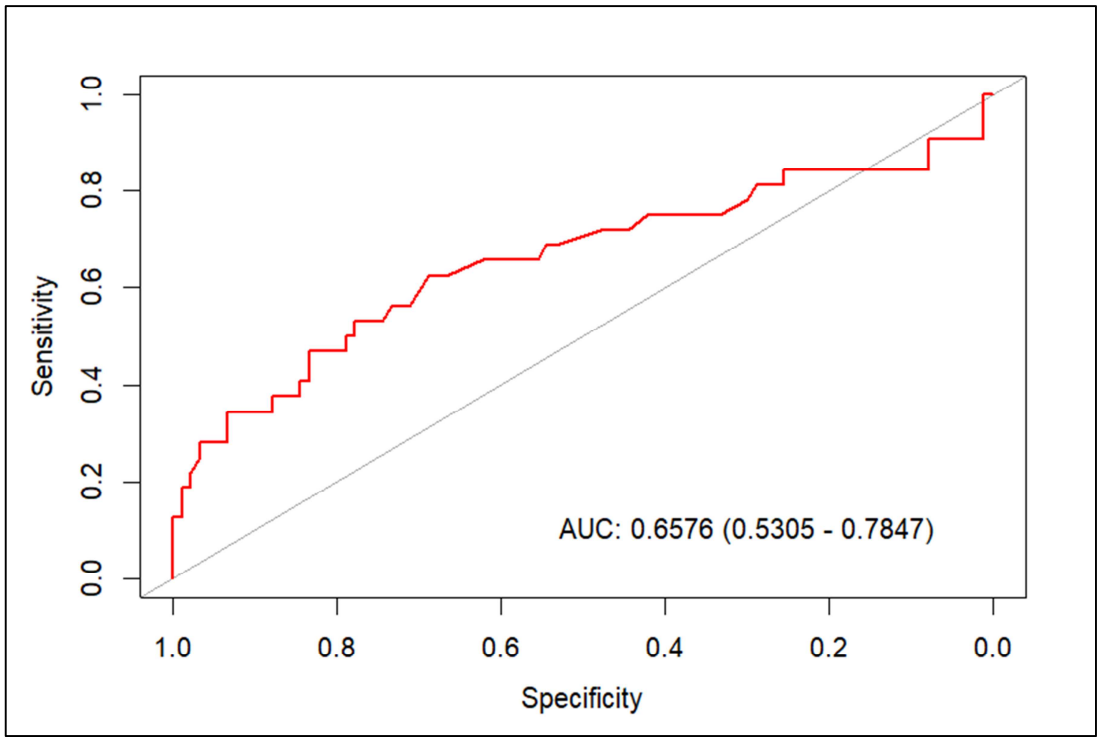


Figure 13: ROC curve of TDA for predicting neonatal acidemia.



DISCUSSION

The results of this case-control study highlighted the predictive ability of TDA observed on fetal cardiotocogram, in the last 30 mins of labor statistically significant (p 0.0014) with neonatal acidemia at birth. It was noted that an intra partum TDA of 195 missed beats, was associated with neonatal acidemia at birth with an AUC of 0.6576 (0.5305, 0.7847), and a positive predictive value of 83.78%

These results are similar to the other studies conducted in various other population settings. In Furukawa et al's research, the TDA was computed based on missed beats, whereas Cahill et al's study utilized seconds, and Sabina et al's study employed square centimetres for calculation. The cutoff obtained in our study is noted to be lesser when compared to other studies, this may be explained by the high risk pregnancy population in our study (75% in both case & control) as compared to the other studies. [20,27,31]

Abby Furukawa et al found that Deceleration Area Of 250 missed beats had the highest Area Under the Curve (AUC) value of 0.702 (95% CI 0.655–0.749), indicating its superior predictive ability for metabolic acidosis.[31] Marti Gamboa et al found that the optimal threshold for predicting neonatal acidemia based on the total deceleration area was 8.37 cm^2 in the last half an hour of labor ,with a sensitivity of 71.6% and a specificity of 71% and an AUC of 0.83; For every square centimetre increase in the area during the final thirty minutes of labour, there was a corresponding decrease of 0.08 units in pH levels.[27]

Cahill et al also observed that TDA is the most predictive electronic fetal monitoring pattern for acidemia, and found the respective Youden cut off point of deceleration area for optimal prediction to be 42,152 for neonatal acidemia with an

AUC of 0.76. Additionally, they found when combined with tachycardia, it poses a significant risk for neonatal morbidity.[20]

Though we found fetal tachycardia in 8 cases(19.05%), and fetal bradycardia in 5 cases (11.9%)in our study, only fetal bradycardia along with TDA ,was significantly associated with neonatal acidemia (p-value 0.029) with an AUC of 0.6597 (0.5552 - 0.7643). [35]

A decreased variability was noted in 11 cases and marked variability in 9 cases. Repetitive variable decelerations were observed in 3 cases (7.14%), both of these variables show no significance in predicting neonatal acidemia at birth.

Different studies have showing varying results with respect to these variables

These findings reported by Marti Gamboa et al and colleagues,noted a significant association between acedemic babies and decreased beat-to-beat variability.[27]

Whereas the study by Cahill et al showed no significance in the predictive ability of decreased or marked variability.[20]

In clinical practice, more than a certain decelerative pattern (early and variable), the depth, duration and frequency of deceleration should be considered important while managing the cases of intrapartum fetal distress.[31]

Similar to previous literature, our study shows a higher incidence of neonates experiencing acidemia in association with late decelerations, noted in 11cases (26.19%), (p value 0.01 and AUC of 0.65). However, it is identified that TDA is more significantly associated with neonatal acidemia than with late decelerations (p value of 0.0014).

Cahill et al in a different study found that combining all significant features (tachycardia prolonged decelerations and, repetitive late and variable decelerations) did not enhance the NICHD model's predictive ability for acidemia, resulting in an AUC of 0.71 [20]

Though early deceleration pattern was not significantly associated with neonatal acidemia, acidic pH with early decelerations was noted in 2 cases (4.67%.) .

Hence, irrespective of the deceleration type observed, neonatal acidemia was consistently noted. It was observed that a few participants with a TDA of less than 195 missed beats also had acidemic babies, this may be due to underlying pathology or risk factor. This could be a knowledge gap which could be explored in future studies.

Among our study population, majority of the participants had antenatal risk factors (73.80% of the Case and 74.60% of the Control groups). Postdatism emerged as the most prevalent risk factor in both groups, (11 participants (35.48%) in case group), followed by fetal growth restriction (7 participants (22.58%) in case group), oligohydramnios and GDM (2 (6.45%) each. Postdatism is seen to be more frequently associated with fetal distress, this may be explained with ageing of post dated placenta resulting in placental insufficiency and intrapartum fetal distress.

Studies show an increased rate of caesarean deliveries are noted in postdatism pregnancies. The most common indication noted was fetal distress.[37]

These findings closely resemble those reported in a study by Schumacher RE et al. they concluded fetal distress was associated with postdatism and dysmaturity. [38]

Our study shows that Induced labor appears to be the predominant type of labor compared to spontaneous labor in both the case and control groups, comprising 57.14% and 57.94%, respectively.

These induction rates align closely with those reported in a study conducted by Cahill et al., where the induction rate was found to be 44.8%, indicating consistency across studies.[20]

This also highlights more likelihood of intrapartum fetal distress with induced labor.

Our study group consisted a greater number of high-risk pregnancies like Postdatism, FGR, oligohydramnios, etc, which get induced for better maternal and neonatal outcome.

In this study, 54.76% of study participants in cases group had fetal distress in latent phase. This could be to attributed increased rate of caesarean sections in women who undergo admission in latent phase. augmentation with oxytocin as caesarean deliveries were also significantly noted more in women admitted in latent labor. [39]

Fetal distress detected during the second stage of labor was seen in 33.3% of the cases, this shows a significant p value of 0.002.

It was observed that 42.8% of cases had intrapartum meconium-stained liquor. It was not found to be significant in determining fetal acidemia. These findings align with results reported in other previous studies. [28,40]

With respect to the mode of delivery, 64.29% of the case group underwent caesarean section (LSCS). Instrumental delivery was performed in 28.5% of cases.

This might be due to most of the fetal distress being diagnosed during latent pahse of labor.

It was noted that 92.86% of the control group underwent caesarean delivery. This shows that an increased number of LSCS is being perfomed for fetal distress, but the neonates are found not to have developed acidemia. This gives power to TDA as a more relevant predictor of fetal acidemia.

In terms of neonatal outcome, of the babies who immediately cried at birth, 83.33% belonged to the case group. Conversely, among babies who did not spontaneously cry at birth, 16.67% belonged to the case group. Notably, babies who did not cry at birth had significantly (p value of 0.0085) worse outcomes compared to those who cried immediately in terms of NICU admissions and need for intensive resuscitation.

In this study, 21.43% of the academic neonates required admission to the Neonatal Intensive Care Unit (NICU) post-delivery. This indicates a significant proportion (p value pf 0.003) of babies with neonatal acidaemia requiring NICU admission compared to those without it.

tudies conducted in other South Asian population show similar results.[7] 73.81% of babies in the case group had a normal birth weight. These findings closely resemble those reported in the study conducted by Cahill et al [20]

It is noted that there's no significant difference in the development of neonatal acidemia concerning gender, as 52.38% of cases were male babies. This suggests that gender may not play a substantial role in the occurrence of neonatal acidemia.

However, others studies have shown results suggesting gender as factor influencing neonatal outcomes. Male babies in primiparas tend to have poorer outcomes during labor, delivery, and the immediate post-birth period compared to female babies.[41]

In our study it is observed that the maternal age group ranged between 21-27 years, with a mean age of 24 years. in both the case and control group which is noted to be similar to the studies conducted by Cahill et al and also to that conducted by Furukawa et al. [20,31]

it is noted that the majority of the study group consists of nulliparous individuals, comprising up to 80% in both the case and control groups. Comparable results were observed in a study conducted by Furukawa and colleagues, where nulliparity was evident in 68.2% of the cases.[31]

Strengths of the Study:

To the best of our knowledge, this is the first study done to identify a TDA cutoff in our Indian population for prediction of neonatal acidemia. This simple calculation can be easily executed by healthcare professionals and paramedical staff with minimal training. It has the potential to become a recommended practice in routine healthcare settings. Large-scale studies specific to the Indian scenario can be conducted to acquire recommendations from recognized national healthcare bodies.

Limitations:

The study was conducted in a small cohort. A few of the acidaemic babies had a TDA of < 195 missed beats. This may be due to underlying pathology. This knowledge can be explored in future studies.

CONCLUSION

An intrapartum TDA of 195 missed beats, was significantly associated with neonatal acidemia at birth in this study. TDA represents a simple calculation in the management of intrapartum fetal distress. This will help clinicians to take a timely decision in management of fetal distress cases in order to prevent fetal acidemia without classifying decelerations on different categories which is subject to interpersonal variations.

BIBLIOGRAPHY

1. Spencer JA. Deaths related to intrapartum asphyxia. BMJ. 1998 Feb 28;316(7132)
2. Arpita Y. Reddy et al 2018. Robson Ten Group Classification System for Analysis of Cesarean Sections in an Indian Hospital. *Research Journal of Obstetrics and Gynecology, 11: 1-8.*
3. Brenda A. Bucklin, in Complications in Anesthesia (Second Edition), 2007
4. "Caesarean section rates continue to rise, amid growing inequalities in access Rising rates suggest increasing numbers of medically unnecessary, potentially harmful procedures". 16 June 2021 Departmental news, WHO.
5. Parer JT et al Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? J Matern Fetal Neonatal Med. 2006 May;19(5):289-94.
6. Ronsmans C, et al. Socioeconomic differentials in caesarean rates in developing countries: A retrospective analysis. Lancet. 2006;368:1516–23.
7. Syed W et al Relationship between immediate postpartum umbilical cord pH, fetal distress and neonatal outcome. Pak J Med Sci. 2020 Nov-Dec;36(7):1529-1532.
8. Ana Pinas, Edwin Chandraharan Continuous cardiotocography during labour: Analysis, classification and management, Best Practice & Research Clinical Obstetrics & Gynaecology, Volume 30,2016,Pages 33-47.
9. Dildy GA. et al. Intrapartum Assessment of the Fetus: Historical and Evidence -Based Practice. Obstet Gynecol Clin N Am 2005; 32:255-271
10. Freeman, RK, Problems with intrapartum fetal Heart rate monitoring interpretation and patient management. Obstet Gynecol 2002; 100: 813-26

11. Garite TJ. Intrapartum fetal evaluation. Gabbe: Obstetrics – Normal an Problem Pregnancies, 4th ed. (2002)
12. Hankins GD, Speer M. Defining the pathogenesis and pathophysiology of neonatal encephalopathy. Obstet Gynecol 2003; 102:628-36
13. National Institute of Child Health and Human Development Research Planning Workshop. Electronic fetal heart rate monitoring: Research guidelines for interpretation. Am J Obste and Gynecol 1997; 177:1385-1390
14. Simpson, K.R. and Creehan, P.A. Perinatal Nursing, 3rd Edition. (2008)
15. Smith JF, Onstad, JH. Assessment of the Fetus: Intermittent Auscultation, Electronic Fetal Heart Rate Tracing, and Fetal Pulse Oximetry. Obstet Gynecol Clin N AM 2005; 32:245-254
16. Steer, PJ, Has electronic fetal heart rate monitoring made a difference? Seminars in Fetal and Neonatal Medicine 2008; 13:2-7
17. Sweha A, Hacker, T. Interpretation of the electronic fetal heart rate during labor. American Family Physician 1999; 59:2487-2500
18. Handbook of CTG Interpretation From Patterns to Physiology Edited by Edwin Chandraharan,2017.
19. CTG made easy 4th edition by Susan Guage
20. Cahill AG, Tuuli MG, Stout MJ, et al. A prospective cohort study of fetal heart rate monitoring: deceleration area is predictive of fetal acidemia. Am J Obstet Gynecol 2018;218:523.e1-12
21. Sendeku, F.W., Azeze, G.G. & Fenta, S.L. Perinatal asphyxia and its associated factors in Ethiopia: a systematic review and meta-analysis. *BMC Pediatr* 20, 135 (2020).
22. American Pregnancy Association. 2014-08-28. Retrieved 2021-09-09.

23. Bobrow CS, Soothill PW Causes and consequences of fetal acidosis *Archives of Disease in Childhood - Fetal and Neonatal Edition* 1999;80:F246-F249.
24. ACOG Practice Bulletin No. 106: Intrapartum Fetal Heart Rate Monitoring: Nomenclature, Interpretation, and General Management Principles. Obstetrics & Gynecology, 114(1), 192-202. (2018).
25. Paladugu V, Sreedhar S, et al.Association of CTG Diagnosis of Intrapartum Fetal Distress and Immediate Postpartum Acidemia in Foetal Umbilical Artery. J Obstet Gynaecol India. 2023 Feb;73(1):28
26. Faivre, A.-C., Tazi, S., et al (2020) Association between Fetal Heart Rate Monitoring during Labor and Neonatal Acidosis in Full-Term Newborns: A Retrospective Multicenter Cohort Study. Open Journal of Obstetrics and Gynecology, 10, 1265-1278)
27. Sabina Marti Gamboa MD, Ph D, Maria Lapresta Moros, Jara Pascual Mancho,et al.(2016): Deceleration area and fetal academia, The Journal of Maternal-Fetal & Neonatal Medicine, DOI: 10.1080/14767058.2016.1256993
28. Gunaratne SA, et al. Prediction of Neonatal Acidosis Based on the Type of Fetal Hypoxia Observed on the Cardiotocograph (CTG). EJMED [Internet]. 2022 Apr. 29 [cited 2024 Apr. 26];4(2):8-18
29. Trevor Shelley & Richard h. Tipton “Dip area. A quantitative measure of fetal heart rate patterns”The Journal of Obstetrics and Gynaecology of the British Commonwealth Aug. 1971. VO~. 78. Pp. 694-701.
30. A. Agostinelli *et al.*, "Relationship between deceleration areas in the second stage of labor and neonatal acidemia," *2016 Computing in Cardiology Conference (CinC)*, 2016, pp. 897-900.

31. Abby Furukawa, Duncan Neilson & Emily Hamilton (2019): Cumulative deceleration area: a simplified predictor of metabolic acidemia, The Journal of Maternal-Fetal & Neonatal Medicine, DOI: 10.1080/14767058.2019.1678130.
32. Marta Chóliz Ezquerro, Ricardo Savirón Cornudella, Luis Mariano Esteban, et al.(2021): Total intrapartum fetal reperfusion time (fetal resilience) and neonatal acidemia, The Journal of Maternal-Fetal & Neonatal Medicine, DOI: 10.1080/14767058.2021.1915977.
33. Matmor Loeub S, Weintraub AY, Rotem R, Geva Y, Yaniv Salem S. Correlation between total deceleration area and fetal cord blood pH in neonates complicated with meconium-stained amniotic fluid at term. *Int J Gynecol Obstet.* 2022; 159: 974-978.
34. Geva Y, Yaniv Salem S.et al. Intrapartum deceleration and acceleration areas are associated with neonatal encephalopathy. Int J Gynaecol Obstet. 2023 Jun;161(3):1061-1068. doi: 10.1002/ijgo.14638. Epub 2023 Jan 9. PMID: 36572017.
35. Cohen, Gal & Gnaiem, Nagam et al (2023). Total deceleration area as a predictor of neonatal acidemia in vacuum extraction deliveries. American Journal of Obstetrics and Gynecology. 228. S476-S477. 10.1016/j.ajog.2022.11.822.
36. Tranquilli AL, Biagini A, et al.The correlation between fetal bradycardia area in the second stage of labor and acidemia at birth. J Matern Fetal Neonatal Med. 2013 Sep;26(14):1425-9.
37. Chaudhari SN et al. Int J Reprod Contracept Obstet Gynecol. 2017 May;6(5):2077-2082.
38. Schumacher RE eta al.The impact of gestational age on dysmaturity/postmaturity. J Perinatol. 1989 Dec;9(4):401-6. PMID: 2593013.

39. Mikolajczyk RT et al. Early versus Late Admission to Labor Affects Labor Progression and Risk of Cesarean Section in Nulliparous Women. Front Med (Lausanne). 2016 Jun 27;3:26.
40. Matmor Loeub S et al Correlation between total deceleration area and fetal cord blood pH in neonates complicated with meconium-stained amniotic fluid at term. *Int J Gynecol Obstet*. 2022; 159: 974-978.
41. Liam Dunn et al.Gender specific intrapartum and neonatal outcomes for term babies, European Journal of Obstetrics & Gynecology and Reproductive Biology, Volume 185,2015,Pages 19-22,ISSN 0301-2115

ANEXURE I – PROFORMA

NAME: **AGE:**

IP NO: **DOA:** **DOD:**

OCCUPATION: **ADDRESS:**

Patient:

Husband:

PHONE NUMBER:

Patient:

Husband:

SOCIO-ECONOMICS : Low / Middle / High

OBSTETRIC SCORE: G P L A

LMP (DD/MM/YY):

EDD:

Period of Gestation(weeks+days):

LOW RISK /HIGH RISK (if high risk factor identified)

Hypertension yes/no

Pre-eclampsia yes /no

If yes mild / severe

Eclampsia yes/no

Antepartum Haemorrhage yes/no

If yes Placenta praevia/ Abruptio placenta

Jaundice in pregnancy yes/no

Rh Negative pregnancy yes/no

GDM yes/no

Macrosomia yes/no

Post-datism yes/no

PROM yes/no

CPD yes/no

Prolonged latent phase yes/no

DTA yes/no

Thyroid abnormality yes/no

FGR yes/no

Heart disease yes/no

Previous LSCS yes/no

Anaemia yes/no

HIV Reactive / Non-Reactive

HbsAg Reactive / Non-Reactive

VDRL Reactive / Non-Reactive

Others:

GENERAL PHYSICAL EXAMINATION:

Built:

General Condition:

Height: **Weight:**

BMI:

Pulse: **Blood pressure:**

Temperature:

Pallor

Icterus

Edema

Breast

Thyroid

SYSTEMIC EXAMINATION:

Respiratory system:

Cardiovascular examination:

Per abdomen examination:

Per Speculum/Per Vaginal examination:

ADMISSION CTG:

CTG findings at time of enrolment

Baseline Heart Rate:

Beat to beat variability:

Acceleration present/absent

Deceleration present/ absent

Deceleration and type:

Total deceleration area:

Reactive / Non-Reactive / Suspicious

If Non-Reactive -abnormality noted:

If suspicious – abnormality noted:

INTRAPARTUM HISTORY:

Induced Labor/Spontaneous labor

Intrapartum status- Uncomplicated / Complicated

If Complicated – Complication noted:

Abruptio Placenta yes/no

Scar Dehiscence/Uterine Rupture yes/no

Cord Prolapse yes/no

Meconium-Stained Liquor yes/no

CPD yes/no

DTA yes/no

Maternal Hypotension yes/no

Maternal Hypertension yes/no

Others:

1st stage of labour duration:

2nd stage of labour duration:

Fetal distress Detected in :

Latent phase of Labour / Active phase of Labour

1st stage of Labour / 2nd stage of labour

Type of delivery:

Full Term Normal Delivery / Full Term Ventose Delivery / Full Term Forceps Delivery / Full Term Emergency LSCS

If Emergency LSCS

Decision to baby out time:

CTG ANALYSIS:(30mins pre delivery)

Total Number of Decelerations:

Total Deceleration area:

$$Deceleration\ area = \frac{Depth\ of\ deceleration\ x\ Duration\ of\ deceleration}{2}$$

Total Fetal Reperfusion time:

Fetal Tachycardia: yes/no

Fetal Bradycardia: yes/no

Beat to beat variability <5bpm: yes/no

Sinusoidal pattern: yes/no

Repetitive variable deceleration: yes/no

Type of Deceleration

DETAILS OF DELIVERY:

Live birth: yes/no

Cried at birth: yes/no

Resuscitation measures taken for baby yes/no

If yes

Bag & mask ventilation yes/no

O2 inhalation yes/no

PPV yes/no

Intubation yes/no

Inotropes yes/no

Others:

APGAR score: 1' 5'

Baby gender: Male / Female

Baby weight at birth:

NICU ADMISSION YES/NO

If Yes Reason:

Duration of NICU admission:

Duration of Hospital stay for baby:

Condition of baby at discharge: Healthy / AMA / Expired

Morbidity Noted:

UMBILICAL ARTERY CORD BLOOD GAS ANALYSIS:

pH: **pCO2:**

pO2: **HCO3:**

SIGNATURE AND NAME OF INVESTIGATOR

ANEXURE II

MASTER CHART

SL	TDA	pH	Age	SES	Obs Score	High Risk Factor	BMI(Kg/m2)	induced/spont labor	Intrapartum Status	Stage of labor Distress detected	Duration of labor	Mode of delivery	Repurfusion time	Type of deceleration	Fetal Tachycardia	Fetal Bradycardia	Beat to beat variability<5 or >25	Repeated variable deceleration	cry at birth	resuscitation measures taken	NICU admission	APGAR 5'	baby weight	sex of baby
1	105	7.2	24	Middle	Primipara	HTN,GDM,Hypothyroid	28	I	U	latent	6	LSCS	23.5	L	N	N	Y	N	Y	N	N	8	2.3	male
2	215	7.32	22	Low	Primipara	post datism	27	S	U	latent	8	LSCS	20	V	N	N	N	N	Y	N	N	8	2.5	Male
3	65	7.31	34	Low	Multipara	Post datism	26	I	MSL	Active	10	LSCS	26.5	E	N	N	N	N	Y	N	N	9	3.2	Male
4	220	7.35	19	Middle	Primipara	Low	24.8	I	U	latent	6	LSCS	18	E	N	N	N	N	Y	N	N	9	2.6	Female
5	315	7.24	24	Middle	Primipara	Low	22.2	S	U	2nd stage	8/0.5	Instrumental	19.5	V	N	N	N	N	Y	N	N	8	2.8	Female
6	55	7.19	26	Middle	Primipara	Post datism, Hbsag +ve	24	I	MSL	latent	12	LSCS	25.5	L	N	N	N	N	Y	N	N	9	3.4	Female
7	390	7.13	20	Middle	Primipara	Post datism	28.5	I	MSL	latent	4	LSCS	15	L	N	N	N	N	Y	N	N	8	3.5	Male
8	140	7.27	18	Low	Primipara	Postdatism	26.2	I	MSL	latent	15.5	LSCS	24	V	Y	N	Y	N	Y	N	N	8	3.2	Female
9	127.5	7.23	22	Middle	Primipara	Post datism	27.5	S	U	latent	8	LSCS	23.5	V	N	N	N	N	Y	N	N	8	2.7	Female
10	115	7.28	19	Middle	Primipara	Oligo,FGR	21.7	I	Cord	latent	17.5	LSCS	23.5	V	N	N	N	N	Y	N	N	9	2.3	male
11	255	7.33	21	Middle	Primipara	PE	30.4	S	U	Active	21.5	LSCS	22	V	N	N	N	N	Y	N	Y, Tachycardia	8	3.3	Female
12	150	7.23	24	Low	Primipara	GDM,Macrosoima	25.47	I	U	latent	18	LSCS	23.5	V	N	N	N	N	Y	N	N	8	3.3	Female
13	357.5	7.27	25	Middle	Primipara	PROM	27.4	I	U	Active	6	LSCS	16	E	N	N	N	N	Y	N	N	8	3	Female
14	157.5	7.16	24	Middle	Primipara	Postdatism	28.9	I	U	Active	5.5/40	Instrumental	6	V	N	N	N	N	Y	N	N	7	3	male
15	390	7.27	22	Middle	Primipara	Low	26.6	S	U	2nd stage	7	LSCS	15	L	N	N	N	N	Y	N	N	7	2.3	Male
16	60	7.23	24	Middle	Primipara	Oligo,Anemia	23.3	I	MSL	latent	8	LSCS	25	V	N	N	N	N	Y	Y,Suctioning	N	8	2.9	male
17	285	7.27	26	Middle	Primipara	Low	25.5	S	MSL	2nd stage	6/0.5	Instrumental	15	V	N	N	N	N	Y	Y,Suctioning	N	9	2.8	Female
18	740	7.17	22	Low	Multipara	Post datism	25.7	S	MSL	latent	10	LSCS	16	L	N	N	N	N	Y	N	N	8	2.9	Female
19	207	7.28	26	Middle	Primipara	HTN	29.2	I	U	latent	8	LSCS	19.5	L	N	N	N	N	Y	N	N	8	2.4	Female
20	155	7.32	25	Low	Primipara	Hypothyroid	23.6	S	U	latent	6	LSCS	25	V	N	N	N	N	Y	N	N	8	3	Male
21	255	7.29	24	Low	Primipara	post datism	24.6	I	U	latent	4	LSCS	21.5	V	N	N	N	N	Y	N	N	9	2.9	Female
22	405	7.16	28	Low	Primipara	Low	25.56	S	U	2nd stage	10-1	LSCS	17	L	N	N	N	N	N	o2,bag & mask	Y, Respiratory distress	6	3	Female
23	295	7.3	28	Low	Primipara	FGR	26	I	U	Latent	9.5	Lscs	16.5	E	Y	N	Y	N	y	y,ppv	Y,LBW	8	2.1	Female
24	-	7.36	23	Middle	Primipara	PROM	34	I	U	Latent	8	LSCS	-	-	Y	N	Y	N	y	n	n	9	3	Male
25	307.5	7.32	31	Low	Primipara	PROM	28	U	U	Active	8	Lscs	21	V	N	N	N	N	Y	N	N	9	2.7	Male
26	135	7.25	21	Middle	Primipara	Anaemia	21.875	I	MSL	2nd Stage	19/0.6	Instrumental	22.5	V	N	N	Y	N	Y	Y,O2	N	8	2.8	Male
27	-	7.35	23	Low	Primipara	disorder	26.02	I	MSL	Latent	16	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	8	3.1	Male
28	235	6.96	20	Low	Primipara	FGR	21.33	S	Cord	2nd stage	4/0.25	Instrumental	9.5	V	N	N	N	Y	Y	Y,PPV	Y,respiratory distress	6	2.3	Female
29	420	7.28	22	Low	Multipara	Low	26.2	S	U	latent	6	LSCS	10.5	V	N	N	N	N	Y	N	N	9	2.6	Female
30	287.5	7.35	28	Middle	Primipara	Low	29.9	S	U	latent	6	LSCS	22.5	V	N	N	N	N	Y	N	N	9	3.2	Female
31	25	7.2	24	Middle	Primipara	Hypothyroid,Polyhydramnios,FGR	31.1	I	U	latent	1	LSCS	-	V	Y	N	N	N	Y	N	N	9	2.2	Male
32	25	7.12	27	Middle	Primipara	post datism	23.8	I	MSL	latent	4	Instrumental	-	V	Y	N	N	N	Y	N	N	8	3	Male
33	105	7.23	32	Low	Primipara	Low	26.8	S	MSL	latent	6	LSCS	25.5	L	N	N	N	N	Y	N	N	8	2.7	Female
34	525	7.18	26	High	Primipara	Low	28.5	S	MSL	latent	10	LSCS	12	V	y	N	N	N	Y	N	N	9	3.2	Female
35	-	7.29	29	Low	Primipara	FGR	28	I	MSL	latent	34	LSCS	-	-	N	N	Y	N	Y	N	N	9	2.5	male
36	220	7.33	21	Middle	Multipara	FGR	25.33	S	U	Latent	8	LSCS	22	V	N	N	Y	N	Y	Y,PPV	N	7	2.5	Male
37	-	7.2	26	Low	Primipara	post datism	28.1	I	U	latent	11	LSCS	-	-	N	N	Y , <5bpm	N	Y	N	N	8	2.8	Male
38	-	7.14	20	Middle	Primipara	FGR,Oligohydramnios	20.13	I	MSL	Latent	20	LSCS	-	-	Y	N	Y	N	Y	Y,PPV	N	8	2.3	Female

39	25	7.06	25	Middle	Primipara	Post datism	24	I	U	2nd stage	14/1	Instrumental	5	L	N	N	N	N	Y	N	N	9	3.4	male
40	177.5	7.27	34	Low	Multipara	Post datism	27.3	I	MSL	Active	25/0.5	LSCS	21.5	E	N	N	N	N	Y	N	N	8	3.3	Female
41	417.5	7.06	25	Low	Primipara	FGR	24.6	I	U	2nd stage	18/0.5	LSCS	18.5	L	N	N	N	N	Y	N	N	8	2.4	Female
42	222.5	7.14	22	Middle	Multipara	GDM	35.2	S	MSL	latent	8	LSCS	22	L	y	N	Y	N	Y	N	Y,Respirator y distress	9	3.4	Male
43	147.5	7.3	24	Low	Primipara	Low	25.2	I	U	Active	18	LSCS	17	V	N	N	N	N	Y	N	N	9	2.7	Female
44	-	7.25	22	Middle	Primipara	Macrosomia	26.4	I	MSL	latent	27	LSCS	-	-	Y	N	N	Y	Y	Y,Suctioning	N	9	2.4	Female
45	135	7.2	26	Middle	Primipara	Postdatism	24.65	I	Cord	Latent	3	LSCS	25.5	L	N	N	N	N	Y	N	N	8	2.6	Male
46	200	7.11	28	Middle	Primipara	GDM	24	S	MSL	Latent	18	LSCS	24.5	E	N	N	N	N	Y	N	N	9	2.3	Male
47	-	7.33	30	Low	Primipara	Doppler Changes,Rh negative	22.9	S	Cord	Active	15.5	LSCS	-	-	N	N	Y	N	Y	N	N	8	3.5	Male
48	277.5	7.34	26	Low	Primipara	FGR,oligohydra mnios	25	I	Cord	Latent	14	LSCS	19	L	N	N	N	N	Y	N	N	8	2.5	Male
49	-	7.32	27	Low	Primipara	Postdatism,Hyp othyroidsm	26.7	I	Cord	Latent	36	LSCS	-	-	N	N	Y	N	Y	stimulation	N	9	2.7	Female
50	335	7.31	22	Middle	Primipara	Low	22.1	S	MSL	2nd stage	10/0.75	LSCS	19	V	N	N	N	N	Y	N	N	8	3.4	Male
51	-	7.3	28	Middle	Primipara	FGR	21.09	I	Anamnios	Latent	14	LSCS	-	-	N	N	Y	N	Y	N	N	8	2.2	Female
52	72.5	7.3	24	Low	Primipara	Polyhydramnios	21.3	I	U	Active	14	LSCS	15	E	Y	N	N	N	Y	N	N	7	3	Male
53	180	7.19	19	Middle	Primipara	Rh negative,Postda tism	24.12	I	MSL	Latent	6	LSCS	22.5	V	N	N	N	N	N	ctioning,Stimul	N	8	2.7	Female
54	552.5	7.16	24	Middle	Primipara	GDM	30	S	Cord	Latent	10	LSCS	13	V	N	N	N	N	Y	N	N	8	2.7	Female
55	95	7.31	22	Low	Primipara	Post datism	24.7	S	U	latent	8	LSCS	23.5	V	Y	N	N	N	Y	N	N	8	3.2	Female
56	-	7.35	24	Low	Primipara	Post datism	31.2	I	MSL	Latent	8	LSCS	-	-	N	N	Y	N	Y	N	N	8	3	Female
57	257.5	7.3	26	Middle	Primipara	Low	26.9	S	MSL	latent	13	LSCS	18.5	L	N	N	N	N	Y	Y,Suctioning	N	8	2.6	male
58	127	7.31	20	Low	Primipara	GDM	30	I	L,True K	Latent	18	LSCS	15.5	V	N	N	N	N	Y	N	N	9	2.3	Male
59	435	7.1	22	Middle	Primipara	Postdatism	25.9	S	U	Active	18	LSCS	15	V	N	N	N	N	Y	N	N	9	3.1	Male
60	-	7.29	19	Middle	Primipara	Post datism	28	I	MSL	Latent	20	LSCS	-	-	Y	N	Y	N	Y	N	N	9	2.5	Male
61	180	7.23	23	Middle	Primipara	GDM	28.7	S	MSL	Active	6/0.16	LSCS	17	V	N	N	N	N	Y	N	N	8	3.3	Male
62	210	7.05	20	Low	Primipara	HTN	32.4	I	U	Active	18	LSCS	9	V	N	N	Y	N	Y	N	N	9	2.7	Male
63	280	7.01	24	Low	Primipara	Hypothyroidism	22.6	S	U	Latent	5	LSCS	21.5	V	Y	N	Y	N	N	Y,Bag&mask	N	7	2.9	Female
64	200	7.24	20	Middle	Multipara	Low	23.7	S	U	2nd stage	15/0.5	Instrumental	22	E	N	N	N	N	Y	N	N	7	2.7	Male
65	105	7.34	22	Low	Multipara	Postdatism	24.45	I	MSL	Latent	5	LSCS	27	V	N	N	Y	N	Y	Y,Suctioning	N	8	2.7	Male
66	-	7.31	21	Middle	Primipara	Postdatism	25.78	I	cohydram	Latent	16	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	8	3.2	Male
67	-	7.24	27	Low	Primipara	Postdatism	22.65	I	U	Active	15	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	9	3	Female
68	50	7.3	28	Low	Primipara	Postdatism	20.4	S	MSL	2nd	22/0.5	Instrumental	22	V	Y	N	N	N	Y	Y,PPV	N	8	3	Male
69	202.5	7.2	23	Middle	Primipara	Postdatism	19.72	S	MSL	Latent	2	LSCS	19	V	N	N	Y	N	Y	,PPV,Suctionir	N	8	2.8	Male
70	225	7.37	24	Middle	Primipara	Low	21.3	S	ROP	Active	6	LSCS	12	V	N	N	N	N	Y	N	N	9	2.5	Female
71	100	7	20	Middle	Primipara	Low	19.17	S	U	2nd stage	13/0.25	Instrumental	0	V	N	N	N	N	N	,bag&mask,PP	Y,3days	7	3.1	Female
72	352.5	7.26	22	High	Multipara	BOH	28.7	I	Cord	Latent	16	LSCS	13.5	E	N	N	N	Y	Y	N	N	9	2.7	Male
73	90	7.2	22	Middle	Multipara	Hypothyroid	24.6	S	Anamnios	latent	16	LSCS	26	E	Y	N	N	N	Y	N	N	9	2.5	Female
74	325	7.18	29	Middle	Multipara	Pre-eclampsisa	21.3	I	MSL	latent	19	LSCS	18	L	N	N	N	Y	Y	ctioning,Stimul	N	8	2.4	Female
75	367.5	7.2	27	High	Multipara	Low	23.5	S	U	Active	10/0.5	FTVD	14	V	N	N	N	Y	Y	Y,PPV	Y	7	2.5	male
76	392.5	6.93	21	Low	Primipara	Postdatism	20.23	S	MSL	Active	15/0.5	Instrumental	9	L	N	N	N	N	N	Y,intubation	Y,Birth Asphyxia,7d ays	5	3.1	male
77	210	7.25	24	Middle	Primipara	Postdatism	24.2	I	MSL	Active	8.5	LSCS	8	E	N	N	N	Y	Y	Y,Suctioning	N	8	2.7	male
78	40	7.35	28	middle	Primipara	Hypothyroid	24	S	MSL	Active	12	lSCS	28	V	N	N	Y	N	Y	N	N	7	3.6	male
79	95	7.23	31	Middle	Primipara	Postdatism	25.9	I	MSL	Latent	14	Lscs	22.5	V	N	N	Y	N	y	y,o2	N	9	2.8	Male
80	-	7.34	23	Middle	primipara	Low	25.5	S	MSL	Latent	6	LSCS	-	-	Y	N	Y	N	Y	Y,O2	N	8	2.8	Female
81	-	7.22	24	Middle	primipara	Anaemia,Oligo hydramnios	24.65	I	ydramnio	Latent	18	LSCS	-	-	Y	N	Y	N	Y	Y,O2	N	9	2.7	Female

82	65	7.35	25	MIddle	Primipara	Postdatism,Rh neg,Oligo		S	canty liq	Latent	4	lSCS	25	V	N	N	N	N	Y	n	n	8	2.7	Female
83	-	7.16	20	Middle	Primipara	Postdatism	24.8	I	U	2nd stage	16/0.18	Instrumental	-	-	N	Y	Y	N	y	n	n	9	3.4	Male
84	85	7.35	24	Middle	Primipara	HTN	23.05	I	MSL	Latent	38	lSCS	24	V	N	N	Y	N	y	y,suctioning		8	2.5	Male
85	180	7.38	21	Middle	Primipara	Postdatism	30	I	MSL	Latent	18	lSCS	0	V	N	N	N	N	y	y,suctioning		9	3	Male
86	-	7.34	21	Middle	Primipara	Postdatism	24.3	I	MSL	Latent	4	LSCS	-	-	Y	N	N	N	y	y,O2	n	8	2.7	Female
87	80	7.23	20	Low	Primipara	Low	25.47	S	MSL	Latent	7	LSCS	28	V	Y	N	Y	N	Y	Y,Ppv	N	8	2.7	Female
88	-	7.24	26	Low	Primipara	Postdatism	21.36	I	U	Latent	17	LSCS	-	-	N	N	Y	N	y	n	n	8	2.7	Female
89	262.5	7.14	35	Middle	primipara	Thyroid disorders	24.8	S	MSL	Latent	8	Lscs	3.5	V	N	N	Y	N	y	y,ppv	n	8	2.7	Female
90	-	7.28	25	Middle	Primipara	Postdatism	24.38	I	U	Latent	12	Lscs	-	-	N	N	Y	N	y	n	n	10	3	Female
91	160	7.34	28	Middle	Multipara	FGR,Postdatism	18.42	I	MSL	Latent	12	Lscs	22	V	N	N	Y	N	y	y,O2	n	9	2.4	Female
92	-	7.35	21	Middle	Primipara	Oligohydramnios	25.7	I	gohydram	Latent	20	LSCS	-	-	Y	N	N	N	Y	Y,O2	N	9	3.1	Male
93	195	7.32	27	Low	Multipara	FGR	21.56	I	U	Latent	5	Lscs	17	V	N	N	N	Y	y	n	n	8	2	Male
94	87.5	7.33	21	Middle	Primipara	HTN,Postdatism,FGR	24	I	MSL	Latent	26	LSCS	26.5	V	Y	N	Y	N	Y	Y,O2	N	8	2.8	Female
95	105	7.28	26	Middle	Primipara	Low	21.3	I	U	Latent	11	Lscs	26	V	N	N	Y	N	y	y,o2	n	9	2.8	Female
96	-	7.35	23	Middle	Primipara	Rh Negative,Anaemia	19.5	I	Cord	Latent	8	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	9	2.8	Male
97	60	7.28	22	Middle	Multipara	FGR	22	I	Cord	latent	16	Lscs	28	V	N	N	Y	N	Y	N	N	8	2.7	female
98	40	7.3	23	Middle	primipara	Postdatism,Anaemia	20.8	I	gohydram	Latent	22	LSCS	20	V	N	N	Y	N	Y	N	N	9	3	Female
99	-	7.19	30	Middle	Primipara	FGR,Thyroid disorder	20.54	I	MSL	Latent	18	LSCS	-	-	Y	N	Y	N	Y	Y,PPV	N	9	2.6	Female
100	-	7.19	24	Middle	Primipara	FGR	19.3	I	U	Latent	24.5	Lscs	-	-	N	N	Y	N	y	y.o2	n	9	2.4	Male
101	195	7.23	24	middle	multipara	RH negative	26	S	MSL	Latent	8	Lscs	21	V	N	N	Y	N	y	n	n	9	2.9	Female
102	-	7.33	29	Low	Primipara	Low	23.43	S	MSL	Active	8	LSCS	-	-	Y	N	N	Y	N	Y,PPV	N	9	2.8	Male
103	-	7.29	23	Middle	Primipara	Rh Neg,Postdatism,PROM	21.63	I	MSL	Latent	38.5	Lscs	-	-	N	N	Y	N	Y	Y,o2	N	8	3.1	Male
104	-	7.25	26	High	Primipara	GDM	22.65	S	MSL	Active	13	Lscs	-	-	Y	N	N	N	y	y,O2	n	8	3.5	Male
105	15	7.28	25	High	Multipara	GDM,Macrosomia	24.65	S	Cord	Latent	2	LSCS	27	V	Y	N	N		Y	Y,O2	N	8	2.9	Female
106	132.5	7.34	22	Low	Multipara	Low	24	I	U	Latent	6	LSCS	23	V	N	N	Y	Y	Y	N	N	9	2.8	Female
107	312.5	7.35	33	High	Primipara	Postdatism	25	S	U	Active	5.5	Lscs	18	V	N	N	N	N	y	n	n	8	3.4	Female
108	35	7.34	29	Middle	Primipara	Postdatism	23.73	I	U	Latent	30	LSCS	28	V	N	N	Y	N	Y	Y	N	8	3.3	Female
109	165	7.31	26	Low	Primipara	FGR,oligo	26.6	S	U	Latent	2.5	Lscs	22	V	N	N	Y	N	y	y	n	9	2	Male
110	75	7.34	28	Middle	Primipara	FGR	27.5	I	U	Active	20	Lscs	25.5	L	N	N	N	N	y	n	n	9	2.6	Male
111	85	7.36	22	Low	Primipara	HTN,Postdatism	26.6	I	Mat HTN	Latent	10	Lscs	25	E	Y	N	N	N	Y	n	n	9	3.1	Female
112	-	7.25	19	Middle	Primipara	Macrosomia,Thyroid disorder,Anaemia	25.78	I	U	Latent	34	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	8	3.2	Male
113	262.5	7.18	28	Middle	Primipara	Low	25.5	I	U	Latent	14	Lscs	19.5	V	N	N	Y	N	y	n	n	9	2.4	Female
114	150	7.3	19	Middle	Primipara	Low	20.5	S	MSL	Latent	14	Lscs	24	V	N	N	N	Y	y	y,suctioning	n	8	2.6	Male
115	205	7.32	30	Middle	Primipara	Oligohydramnios	28.12	I	U	Latent	10	Lscs	19	V	Y	N	N	N	y	n	n	9	3	Female
116	-	7.21	19	Low	Primipara	Low	27	I	MSL	Latent	1	Lscs	-	-	N	N	Y	N	y	n	n	9	3.1	Male
117	170	7.34	26	Middle	Multipara	Low	23	S	U	Latent	4	Lscs	24	V	Y	N	Y	N	y	n	n	10	3.4	Male
118	-	7.32	24	High	Primipara	Postdatism	29.6	S	MSL	Latent	10	Lscs	-	-	N	N	Y	N	y	n	n	10	3.72	Male

119	157.5	7.25	23	Middle	Multipara	Postdatism, FGR	24.2	S	MSL	Latent	10	Lscs	24.5	V	N	N	N	N	y	y,BM	Y,LBW	9	1.8	Female
120	80	7.23	20	Low	Primipara	PE,FGR,HELLP	26.4	I	ertension,	Latent	18	LSCS	25	V	N	N	Y	Y	N	Y,PPV	Y	8	1.9	Male
121	35	7.32	24	Low	Primipara	Anaemia	20.7	S	U	Latent	10	LSCS	24	V	N	N	Y	N	Y	Y,PPV		9	2.8	Female
122	53	7.11	21	Low	Multipara	Low	25.47	S	Cord	2nd	8/0.21	Instrumental	-	V	N	Y	Y	N	n	y,BM	n	8	2.7	Male
123	-	7.16	20	Middle	Primipara	FGR	22.22	S	Cord	2nd	12.5/0.1	Instrumental	-	-	N	N	Y	N	y	y,suctioning		10	2.25	Female
124	180	7.33	19	Low	Primipara	Thyroid abnormality	25.78	I	MSL	2nd Stage	18/0.5	Instrumental	21	V	N	N	Y	N	Y	Y,O2	N	9	2.9	Male
125	150	7.2	26	Low	Primipara	PROM,FGR,Thyroid abnormality	25.9	I	MSL	2nd Stage	18/0.5	Instrumental	0	V	N	N	Y	N	Y	Y,O2	N	9	2.49	Female
126	-	7.25	30	Low	Multipara	prev LSCS	24.2	S	U	Latent	1	lscs	-	-	Y	N	N	-	y	n	n	10	2.5	Female
127	167.5	7.3	21	Low	Primipara	HTN,Post datism	21.8	I	ROP	Latent	12	Lscs	19.5	L	N	N	N	Y	y	n	n	8	3.1	Female
128	-	7.19	26	Middle	Primipara	Prolonged labor	22.8	I	U	2nd Stage	50/0.33	FTVD	-	-	N	Y	Y	N	Y	Y	N	7	2.5	Male
129	-	7.06	23	Low	Primipara	Postdatism,FGR,Thyroid disorder	24.03	I	MSL	2nd Stage	18/0.5	Instrumental	-	-	N	Y	Y	N	N	Y,PPV	N	8	2.7	Female
130	-	7.27	24	Low	Multipara	Low	20.7	S	Oligo	Active	7	LSCS	-	-	Y	N	Y	N	Y	Y,O2	N	9	3	Male
131	-	7.3	23	Middle	Primipara	FGR	19.7	S	U	2nd Stage	4.5/0.75	Instrumental	-	-	N	N	Y	N	Y	Y,O2	N	8	2	Female
132	150	7.27	24	Middle	Primipara	Oligohydramnios	21	I	ohydram	Latent	9	LSCS	25	V	N	N	N	N	Y	N	N	8	2.8	Male
133	60	7.31	23	Low	Multipara	Low	23	S	MSL	Active	45	LSCS	27	V	N	N	Y	N	Y	Y,O2	N	9	3	Female
134	-	7.27	22	Low	Primipara	Macrosomia	21.3	I	U	Latent	8	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	8	3.1	Male
135	-	7.16	22	Middle	Primipara	Low	21.8	S	U	2nd	5.5/1	Instrumental	-	-	N	N	Y	N	Y	Y,O2	Y,respiratory distress	8	3.1	Male
136	-	7.25	24	MIddle	Primipara	Postdatism	25.5	I	U	2nd	14/0.38	FTVD	-	-	N	Y	Y	N	Y	Y,O2	N	8	2.8	Male
137	115	7.36	28	Low	Primipara	Postdatism,HIV +ve	27.12	I	MSL	Latent	10	LSCS	25	E	Y	N	N	N	Y	Y,o2	N	8	2.9	Female
138	-	7.29	38	Middle	Multipara	Postdatism	20.23	I	U	2nd Stage	11/0.25	FTVD	-	-	N	Y	Y	N	Y	N	n	8	2.6	Female
139	127.5	7.23	23	Middle	Primipara	FGR	18.9	S	ohydram	Latent	7	LSCS	24	V	N	N	Y	N	Y	N	N	9	2.9	Male
140	95	7.23	31	Low	Multipara	PROM,FGR,Prev LSCS	22.8	S	U	Latent	2	LSCS	11	L	N	N	Y	N	Y	N	N	8	2.1	Male
141	302.5	7.27	27	Middle	Primipara	Postdatism,Thyroid Disorder	23.4	S	MSL	Latent	5	LSCS	19.5	L	N	N	N	N	Y	Y,O2	N	8	3.1	Female
142	115	7.27	19	Middle	Primipara	Low	25.5	S	Cord	Latent	3	LSCS	23	E	N	N	N	N	Y	N	N	9	2.6	Male
143	202.5	7.36	26	Middle	Primipara	PROM	29.2	S	MSL	Latent	4	LSCS	21	V	Y	N	N	N	Y	N	N	8	3.1	Male
144	-	7.3	23	Low	Primipara	Thyroid abnormality	20.77	S	MSL	Active	12	LSCS	-	-	Y	N	Y	N	Y	Y,O2	N	9	3.3	Female
145	-	7.23	25	Low	Primipara	Oligohydramnios,FGR	25.33	I	ohydram	2nd Stage	30/0.3	LSCS	-	-	Y	N	Y	N	Y	N	N	9	1.8	Female
146	-	7.3	26	Low	Primipara	Pre eclmapsia	25.5	I	MSL	Latent	8	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	9	2.5	Male
147	180	7.36	27	Middle	Multipara	FGR,Prev LSCS	24.8	S	Cord	Latent	1	LSCS	23	V	N	N	Y	N	Y	N	Y,respiratory distress	3	2.36	Male
148	85	7.33	26	Middle	Primipara	Oligohydramnios	24.6	S	MSL	Latent	10	LSCS	26.5	V	N	N	N	N	Y	Y,PPV	N	8	2.7	Female
149	150	7.22	21	Middle	Primipara	FGR	31.22	I	U	Latent	11	LSCS	22	V	N	N	Y	N	Y	N	N	9	2.3	Male
150	65	7.28	21	Middle	Primipara	FGR,Thyroid disorder	25.96	I	U	Latent	10	LSCS	24	V	N	N	Y	N	N	Y,o2	N	8	2.2	Female
151	-	7.34	32	Middle	Multipara	Prev LSCS	24.6	S	Cord ,Olig	Latent	3	LSCS	-	-	N	Y	Y	N	Y	Y,O2	N	8	2.7	Female
152	175	7.29	26	Low	primipara	Postdatism,PROM	21.3	I	ohydram	Latent	12	LSCS	26.5	V	N	N	Y	N	y	n	n	9	2.9	Male

153	-	7.07	23	Middle	Primipara	Low	25.9	S	MSL	2nd Stage	38/0.6	FTVD	-	-	N	Y	Y	N	Y	Y,PPV	Y,respiratory distress	6	2.5	Male
154	242.5	7.36	22	Low	Primipara	Low	23.45	S	U	Active	7	LSCS	18	E	N	N	Y	N	Y	N	N	9	2.6	Female
155	307.5	7.24	25	Low	Multipara	Low	23.1	I	U	Latent	12	LSCS	16	V	N	N	N	Y	Y	N	N	8	2.8	Female
156	100	7.26	20	Low	Primipara	Postdatism,FGR	22.1	I	U	Latent	9	LSCS	3	V	N	N	Y	N	Y	N	N	9	2.8	Male
157	112.5	7.31	23	Middle	primipara	PROM,FGR	22.6	I	oligohydra	Latent	18	LSCS	26	V	Y	N	Y	N	Y	Y,PPV	N	8	2	Female
158	135	7.28	20	Low	Primipara	Low	21.6	S	U	Latent	8	LSCS	24	L	Y	N	N	N	Y	N	N	10	2.9	Male
159	145	7.23	30	Low	Primipara	Macrosomia	24	I	DTA	2nd	16/0.75	LSCS	20.5	V	N	N	N	Y	Y	Y,O2	N	8	3.5	Male
160	322.5	7.2	21	Middle	Primipara	PROM,Oligohydramnios	24.23	I	MSL	Latent	8	LSCS	14	V	N	N	N	N	Y	Y,PPV	N	8	2.8	Male
161	70	7.33	24	Middle	Primipara	Postdatism	21.33	I	MSL	Latent	25	LSCS	26	V	N	N	N	N	Y	Y,O2	N	10	3.5	Male
162	300	7.19	28	Middle	Multipara	Anaemia	25.9	I	DTA	2nd Stage	22	LSCS	0	V	N	N	Y	N	Y	Y,PPV	Y,respiratory distress	8	2.7	Female
163	-	7.36	21	Middle	Primipara	Rh Negative	23.45	S	MSL	Latent	8	LSCS	-	-	N	N	Y	N	Y	Y,PPV	N	9	2.5	Male
164	87.5	7.26	23	Middle	Primipara	Hypertension	24.6	S	U	Active	8	LSCS	27.5	V	Y	N	Y	N	N	Y,PPV	N	8	2.8	Male
165	150	7.29	22	Middle	Primipara	PROM	24.8	I	ohydram	Latent	22	LSCS	24.5	V	N	N	N	N	Y	Y,O2	N	8	2.6	Female
166	40	7.24	31	Middle	Primipara	Postdatism	24.8	I	MSL	Latent	20	LSCS	27	E	Y	N	Y	N	Y	Y,PPV	N	8	2.5	Male
167	-	7.31	26	Low	Primipara	PROM	21.8	S	MSL,Cord	Latent	6	LSCS	-	-	N	N	Y	N	Y	Y,O2	N	9	2.8	Male
168	400	7.25	27	Low	Primipara	Postdatism	22.5	I	U	latent	18	Lscs	12	E	N	N	Y	N	Y	N	N	7	2.9	Female