CORRELATION BETWEEN NON REASSURING FETAL HEART PATTERN AND CORD BLOOD PH AND ITS PERINATAL OUTCOME

DISSERTATION SUBMITTED IN FULFILLMENT OF THE

REGULATIONS FOR THE AWARD OF

M.D OBSTETRICS AND GYNAECOLOGY

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CERTIFICATE

This to certify that the thesis entitled **CORRELATION BETWEEN NON REASSURING FETAL HEART PATTERN AND CORD BLOOD pH AND ITS PERINATAL OUTCOME** is a bonafide work of **Dr. A.K Chithra** done under my direct guidance and supervision in the Department of Obstetrics and Gynaecology, PSG Institute of Medical Sciences and Research, Coimbatore in fulfillment of the regulations of Tamilnadu Dr. M.G.R Medical University for the award of MD degree in Obstetrics and Gynaecology.

GUIDE HOD

PRINCIPAL

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INTRODUCTION

Electronic intra partum fetal heart rate monitoring was pioneered by Edward Hon in the late 1950's. In the following years the fetal monitor was over whelmingly accepted. Inter observer and Intra observer variations in interpretation of CTG is common.

The clinician responds in various ways with the provided information. One of the most common response is to take advantage of the increased safety of caesarean section thus cutting one's way through the dilemma. Objective analysis of CTG is needed.

Here in this dissertation the correlation between non reassuring CTG using a scoring system and umbilical cord arterial blood pH analysis was done.

REVIEW OF LITERATURE

In the late 1950's CTG, a revolutionary tool of fetal monitoring was first introduced[4].

Electronic fetal monitoring was developed as a non invasive tool to evaluate fetal condition during labour[6]. In 1958, Hon pioneered the use of electronic fetal heart rate monitoring in USA. Also Caldeyrobarcia and Hammacher with Hon reported their observation on various fetal heart rate patterns with fetal distress.

The application of this technology in the antepartum period seemed promising and was implemented wide spread in the 1970's onwards.

Krebs and coworkers devised an antepartum CTG scoring which is one of the most accepted systems. There were also many other scoring systems which are also mentioned here later.

Cardiotocography

CTG is a record of the fetal heart rate either measured from a transducer on the abdomen or a probe on the fetal scalp[7]. In addition to the fetal heart rate another transducer measures the uterine contractions over the

fundus.

The CTG trace generally shows two lines, the upper line is a record of the fetal heart rate in beats per minute (BPM). The lower line is the recording of uterine contractions from the toco[8]. The vertical scale of this trace depends on how the transducer is picking up the contractions. So interpretation needs to be in relation to the rest of the trace.

Pathophysiology

The fetus responds to various stimuli with changes in heart rate. Fetal movements speeds up the heart rate. Many actions decrease the heart rate like rubbing the eyes, scratching the abdomen, urination, grunting, hiccough, grasping the umbilical cord, physiological stimuli[9].

Heart rate changes represent responses to stimuli from the autonomic nervous system. Heart rate is influenced by intrinsic rhymicity. Parasymphathetic system (or Vagus nerve), the sympathetic component of autonomic nervous system. Parasymphathetic system slows the heart rate and represents the dominant influence in the later stages of pregnancy. Sympathetic responses speed up the heart rate, but is a more complex system. Persistent adrenergic stimulation can result in bradycardia.

With persistent stimuli, the fetus generally responds by slowing its

heart rate, maintaining continuous blood flow to the adrenals, head, heart and umbilical circulation[10].

A smooth decrease and recovery of fetal heart rate generally reflects a steady even stimulus. An erratic response consists of unpredictable raises and falls in heart rate, usually produced by mechanical stimuli, such as head or cord compression.

The normal and abnormal FHR pattern[8,11]

The normal fetal heart rate pattern is characterized by a :

- Base line frequency between 120 and 160 BPM
- Presence of periodic acceleration
- A normal heart rate variability with a band width between 5 and 25 BPM
- The absence of decelerations[12]

In this regard the accentuation of the 120 to 160 frequencies is misleading. It originates from the second half of the 19th century. When the normal frequencies by Vonwickel were started to be between 120-160 BPM.

FHR pattern is abnormal when one or more of the following features are observed[13]

1. A base line frequency < 120 or > 160 BPM.
2. Absence of acceleration for > 45 mins.
3. Reduced or absent FHR variability.
4. Existence of repeated variable or late deceleration.

Under normal circumstances the FHR pattern primarily represents the state of the central nervous system, i.e., the 24 hour (circadian) rhythms and the cycling of behavioural status (Ultradian rhythms) when the condition of the fetus is being threatened there is a shift to a more likely representation of disturbances in the regulation of the BP and the cardiac performance, and the ultimately a direct influence of the fetal heart[14,15].

Artificial occlusions of the umbilical cord circulation and the uterine circulation in animal experiments have contributed to a better understanding of the various aspects of the fetal heart rate pattern. Under pathological circumstances, the human situation is far more complex though successive urine contractions vary in duration and intensity. The position of the human fetus may change the amount of amniotic fluid varies from fetus to fetus. Occlusions of the uterine circulation and umbilical vessels and / or placental

circulation can be absent, be partial or even be total. The circulation in the umbilical vein and / or one /both umbilical arteries can be affected. These are just a few of the many combinations on the circulatory level that may occur when the fetal condition is threatened. Next there are the many complex variations regarding the biochemical status of the fetus, in particular the state of acid base balance. It should be remembered that depth, duration and slope of variable decelerations have no direct relation to the acid base balance of the fetus at a particular moment[16].

Variables of CTG:

Baseline rate:

Should be between 120 -160 BPM and is indicated by the fetal heart rate when stable. It should be taken over a period of 5-10mins. The rate may change over a period of time but normally remains fairly constant.

Bradycardia

This is defined as a base line heart rate of < 120 BPM. If between 120 and 100 it is suspicious whereas below 100 it is pathological. A steep sustained decrease in rate is indicative of fetal distress and if the cause cannot be reversed the fetus should be delivered.

Tachycardia

A suspicious tachycardia is defined as being between 160 to 180BPM. Whereas a pathological pattern is above 180[13]. Tachycardias can be indicative of fever or fetal infections and occasionally fetal distress (with other abnormalities). An epidural may also induce tachycardia in the fetus.

Baseline variations

The short term variations in the baseline should be between 10 to 15 BPM (expect during intervals of fetal sleep which should be no longer than 60 mins). Prolonged reduced variability along with other abnormalities may be indicative of fetal distress.

Acceleration

Defined as a transient increase in the heart rate of more than 15 BPM for at least 15 sec. Two accelerations in 20 mins is considered as a reactive trace. Accelerations are a good sign as they show fetal responsiveness and the integrity of mechanical control of the heart.

Deceleration

These may either be normal or pathological.

Early deceleration

Occurs at the same time as uterine contractions and are usually due to fetal head compression and therefore occur in first and second stage labour

and with decent of the head.

They are normally perfectly benign.

Late Deceleration

Persists after the contraction has finished and suggest fetal distress.

Variable deceleration

Vary in timings and shape and respect to each other and may be indicative of hypoxia or cord compression.

A normal CTG is a good sign but a poor CTG does not always suggest fetal distress. A more definitive diagnosis may be made from fetal blood sampling but if this is not possible or there is an acute situations (such as a prolonged l

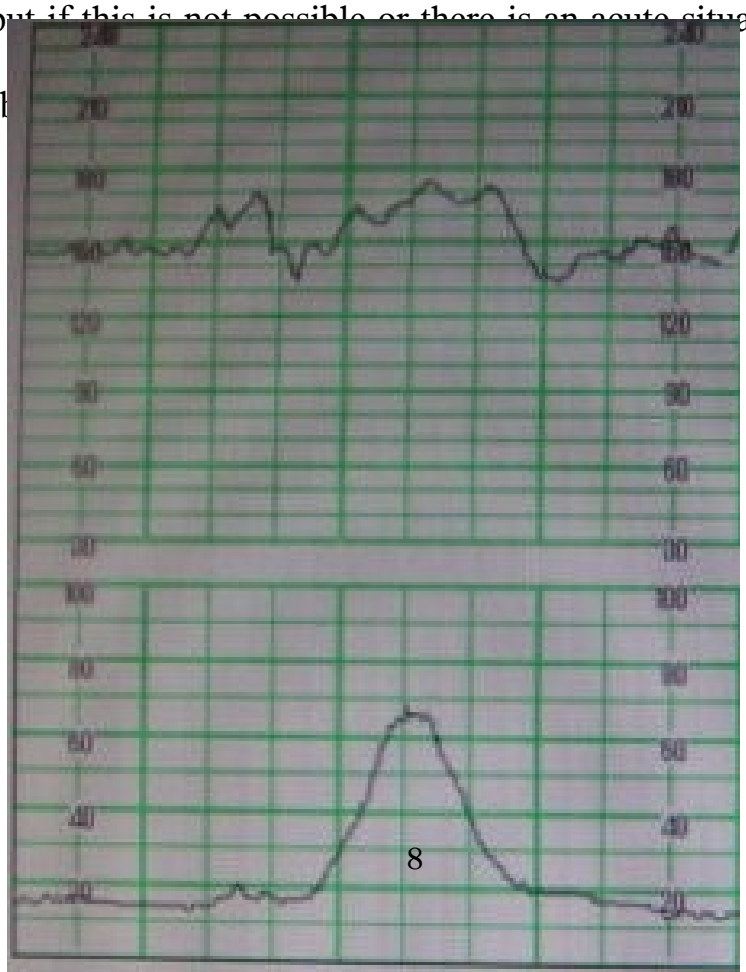


Fig 1: Shows normal fetal heart rate pattern

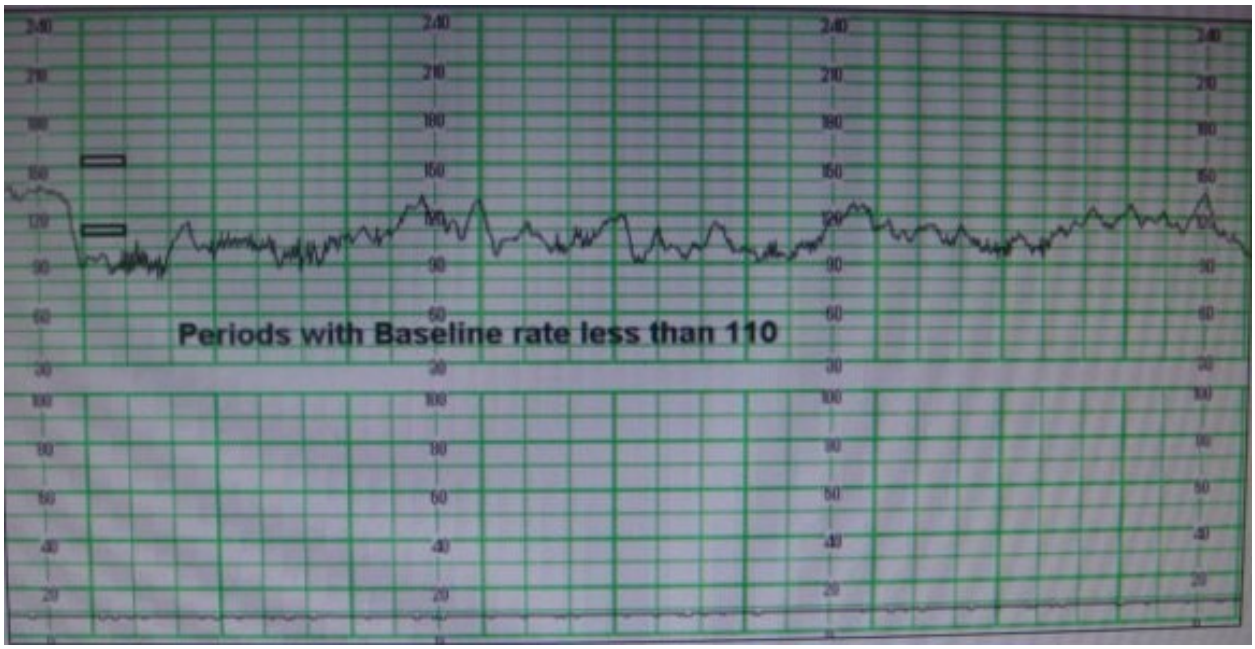


Fig 2: Shows fetal heart tracing with bradycardia

(Baseline rate less than 110)

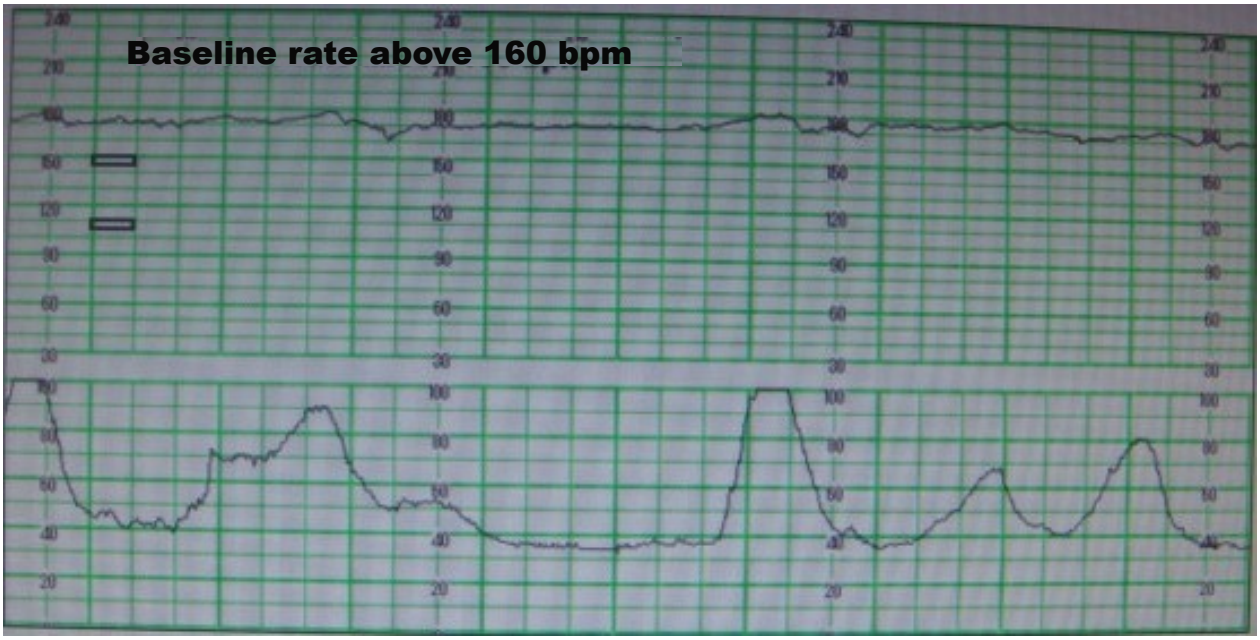


Fig 3: Fetal heart tracing showing tachycardia

(Baseline rate above 160)

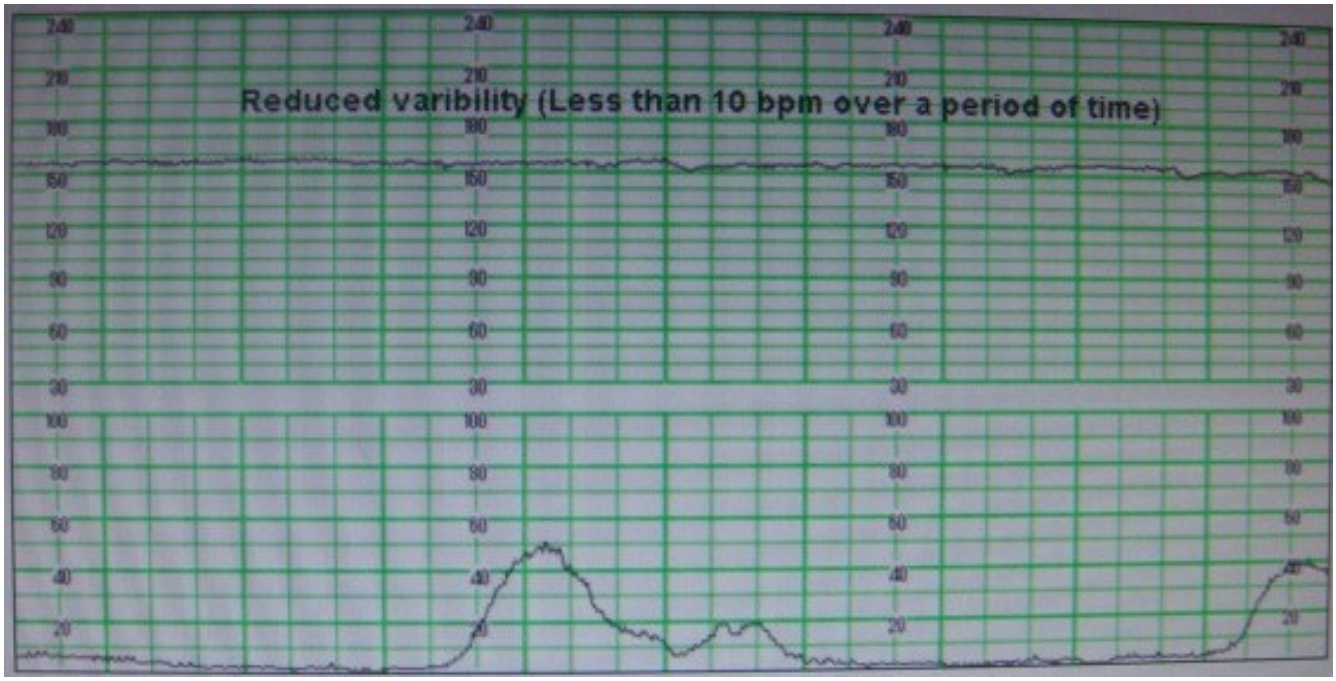


Fig 4: Fetal heart tracing showing reduced variability

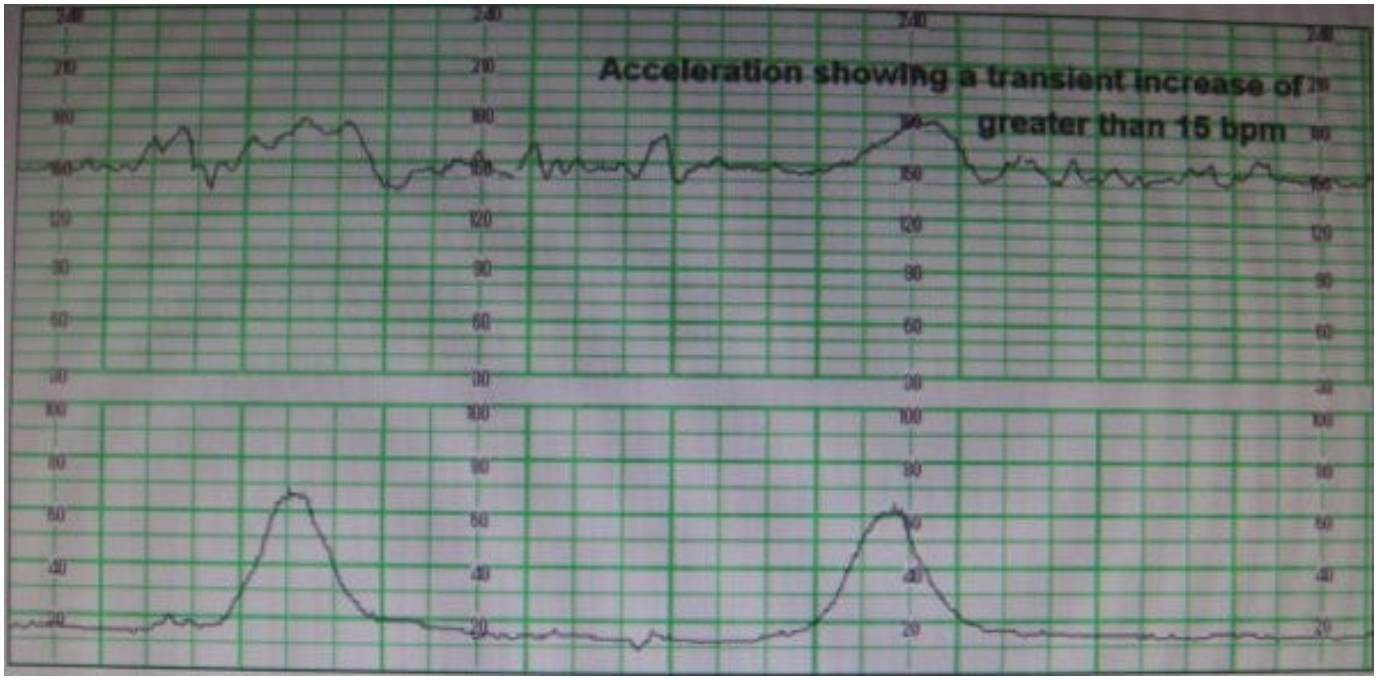


Fig 5: Fetal heart tracing showing acceleration

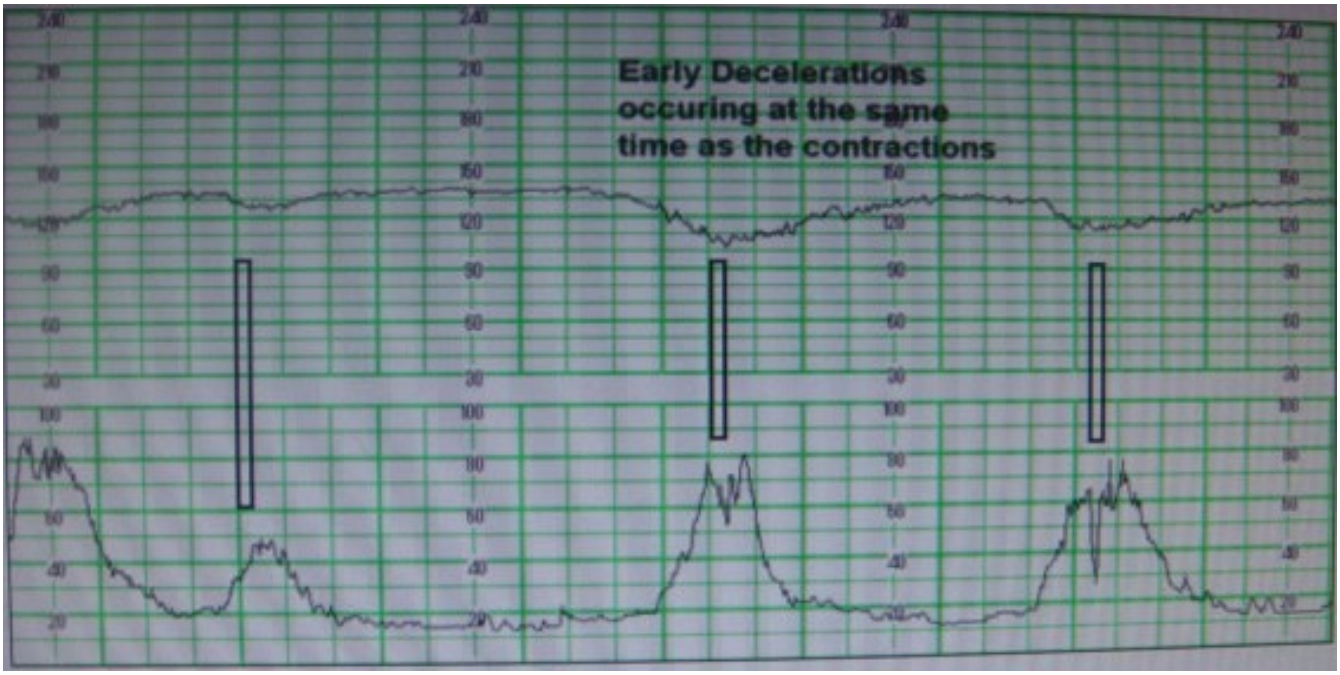


Fig 6: Fetal heart tracing showing early deceleration

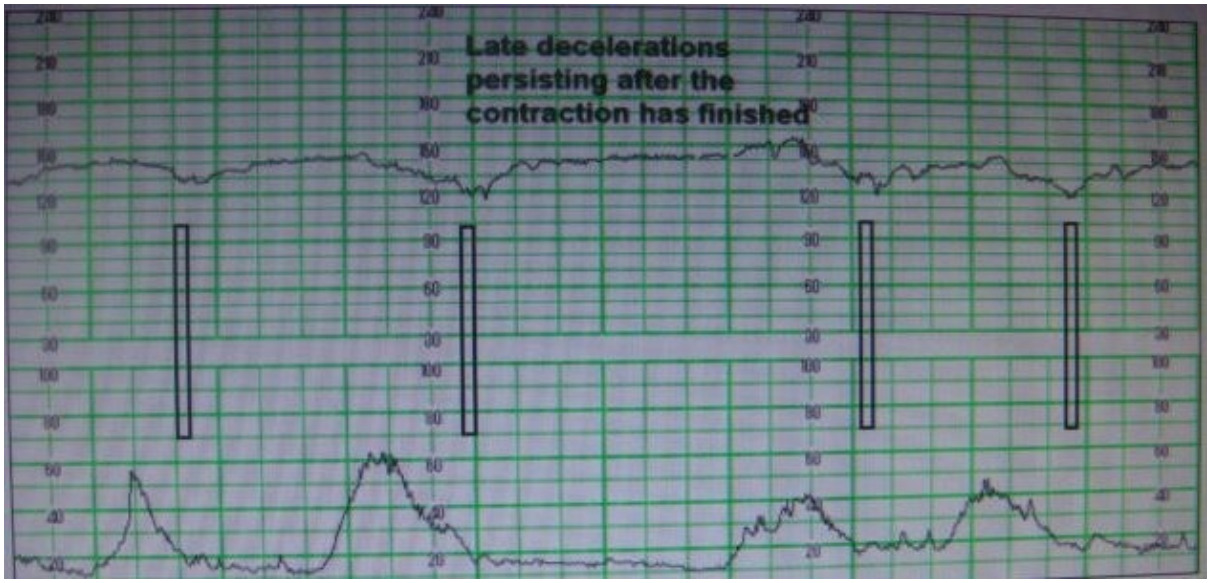


Fig 7: Fetal heart tracing showing late deceleration

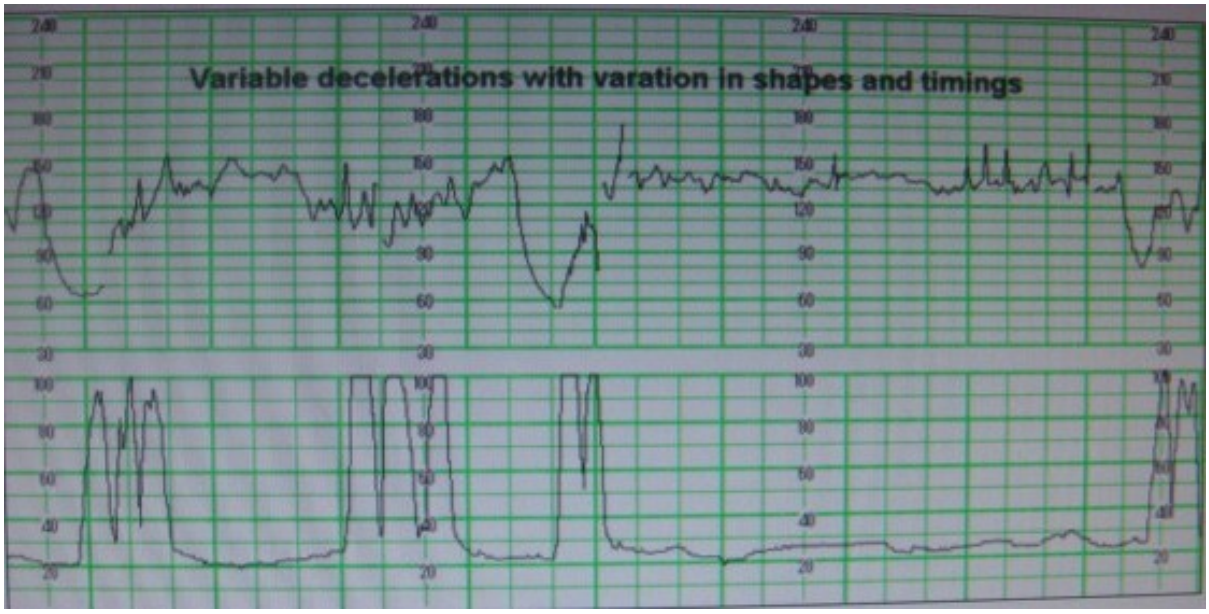


Fig 8: Fetal heart tracing showing variable deceleration

CRITERIA FOR GRADING DECELERATION

by Kubli et al.,

Criteria of grading deceleration	Mild	Moderate	Severe
Variable deceleration level to which FHR drops and duration of deceleration	<30 sec duration irrespective of level >80 BPM irrespective of duration 70-80 BPM <60 secs	<70 BPM lasting for 30-60 secs 70-80BPM lasting >60 secs	<70 BPM for >60 secs
Late deceleration Amplitude of drop in FHR	<15 BPM	15 -45 BPM	>45 BPM

Table 1: shows criteria for grading deceleration by Kubli et al.,

Differences in techniques

For a correct understanding of the fetal heart rate pattern as recorded on paper, it is essential to understand the technical procedures involved with the acquisition and processing of the heart beat signal obtained through ultra sound or direct electrocardiography[18].

The CTG equipment has been improved markedly since the introduction of CTG. Nevertheless, some technical problems have never been fully resolved. Some artificial FHR variability, Halving and doubling of heart frequencies, recording of cardiac arrythmias and recording of the maternal instead of the fetal heart rate frequency[15,16,19].

The major problems, though, occur in clinical practice. There is no uniform opinion regarding the preferred paper speed. Recording speed vary from 1 to 2 to 3 cms per minute depending on the obstetric unit.

A low paper speed facilitate the overview of the recording but has predominant disadvantages:

'Long term' FHR variability is artificially accentuated, 'short term' variability is hardly recognisable and the time relation between uterine contraction and deceleration is more difficult to assess.

The many influences of the fetal heart rate pattern

The healthy near term fetus demonstrates a characteristic FHR pattern with continuous cycling of a reactive and a non reactive FHR pattern representing the cycling of sleep states 2F (REM sleep, active sleep) and 1F (Non REM sleep, quite sleep)[20]. This cycling is interspersed in particular around midnight with periods of fetal activity (Jogging) accompanied by recurrent acceleration, sometimes mimicking fetal tachycardia. The maximum duration of a 1F state is 45 min in a healthy fetus.

When pregnancy advances acceleration increase in number, duration and amplitude. Variability increases during the 2F states, but is nearly constant after 26 wks of gestational age in 1F state[21]. The variability in the 1F state primarily relates to the absence and presence of clusters of regular mouthing movements (associated with oscillation) or breathing movements (associated with short term variability)[22] FHR variability may vary from day to day and week to week in the individual fetus[21].

Interpretation of a CTG tracing is particularly difficult in early preterm. Generally the decision to start CTG is determined by a serious obstetric problem such as presence of IUGR, PROM, vaginal blood loss and PIH. Interpretation then is not only more difficult because of the age of the

fetus and the presenting obstetric problem. But moreover also because of the use of maternally administered drugs. Betamimetic increase the baseline FHR and are associated with reduction in FHR variability. $MgSO_4$ causes decrease in FHR variability. Antihypertensives may cause tachycardia, bradycardia, flattening of accelerations or decrease in variability.

Betamethasone contrary to dexamethasone leads to a reduction in the incidence of fetal body and breathing movements and concomitantly a reduction in the number of accelerations and diminished variability[23].

High intra observer and inter observer variability

Reading, classification and interpretation should be the successive steps in the assessment of CTG tracing. Often too easily a conclusion is reached in (vague) terms of a suspicious, pathological, ominous/ terminal FHR pattern.

The high intra or inter observer variation concerning the classification and interpretation of FHR pattern has sustained in a larger number of publications. Assessment of base line frequency and the presence and absence of acceleration and deceleration is rather uniform, but lacks any uniformity when FHR variability and deceleration types are concerned[24].

Easy, exact guidelines as to how to determine and to interpret FHR

variability are lacking. It appears that in daily obstetric practice variable deceleration far too often are classified as early or late deceleration morely on the basis of time relation between the associated uterine contraction and the deceleration.

The typical characteristics of the variable deceleration is the variation in the shape, duration and depth and on this basis nearly all deceleration occurring during labour should be classified as being of the variable type[25]. The shouldering i.e., the increase in the FHR prior to and follow a variable deceleration can incorrectly be interpreted as an acceleration. Their monotonus and recurrent character is, though, very different from the irregularly spaced acceleration occurring in association with fetal body movements. Recurrent increases in the FHR during uterine contraction should raise suspicious that the umbilical venous circulation is being obstructed.

The low validity, high false positive rate

The low validity of CTG is concluded from a very limited number of earlier performed randomised studies, in which CTG has been compared with intermittent auscultation[24].

These studies suffer from a number of serious draw backs and

inadequacy concerning patient selection, sample size, randomization procedures, inclusion and exclusion criteria, the knowledge of the responsible obstetrician how to classify and interpret CTG tracing and the comparability of applied intermittent auscultation with daily obstetric practice.

A major problem with these and other studies aiming to assess the validity of CTG and related procedures is the lack of solid end points concerning a fetal condition. One of the few hard end points is the status of the acid base balance in arterial and venous cord blood, often applied to assess justification of artificial deliveries for reasons of suspected fetal distress[26]. The primary aim of obstetric intervention in this regard is, though to prevent fetal distress and as such these measures are not very helpful to assess adequacy of obstetric management in individual cases.

CTG increases medicolegal Vulnerability

USG and CTG have been named as the ‘mine fields of obstetrics’ in contrast to antepartum monitoring, intra partum application of CTG which may lead to litigation. The knowledge that their baby was in a good condition at the start of the birthing process is for parents an unbearable though when the baby suffers from handicaps in later life which may possibly relate to the occurrence of fetal distress during the process of labour

and delivery.

The consumers, supported by legal experts, expect from the obstetrician not so much that she / he diagnoses fetal distress, but on the contrary that a serious threat of the fetal condition is being avoided, certainly if it concerns a child with full potentials for later life. He layman wonders why obstetricians do not all apply the same monitoring techniques and cannot understand why FHR pattern cannot be classified and interpreted uniformly.

The medico legal vulnerability associated with the application of the CTG and other monitoring techniques is a very complex area. Many aspects are involved, such as the time path followed, the standard of care at a certain movement the preceding training experienced, at a certain moment the limitations from merely involved information, uncertainty when damage to the infant occur, the confusing nomenclature in use and the lack of exact definition and guidelines.

The consumers demand optimal care to each moment inorder to maximise the future potentials for their child. They exact more and more pressure on decision making and easily push for caesarean section. When the condition of their child might be endangered.

CTG Lead to an increase in obstetric interventions

Since EFM has got high false positive rate because of inter and intra observer variability it will lead to unnecessary obstetric interventions like ceasarean section and instrumental deliveries for fetal distress. If EFM is to be used as a screening test for intra partum fetal asphyxia, predictive FHR patterns require supplementary tests to confirm the diagnosis of fetal asphyxia and to identify false positive rates to avoid unnecessary interventions.

Various scoring system

Kubli et al - 1972
Schifferel et al and Caldeyro Barcio - 1973
Pearson Weaver 1978
American scoring system
Fischer scoring system (Krebs)
Mayer mink 1976

A few attempts have been made to associate intra partum FHR tracing patterns with Apgar scores. Hon was the first to recognize that the FHR pattern could predict the neonatal outcome as determined by 5 minutes Apgar score.

Hon's prediction system for Apgar score	
FHR patterns	**Subtract From A Possible Score Of 10**
BASELINE CHANGES	
Tachycardia	
>200 BPM	3
180-199 BPM	2
160-179BPM	1
If any of the above associated with smooth base line additional	2
Bradycardia	
80-100BPM	1
70-79BPM	2
PERIODIC CHANGES	
Variable deceleration	
Frequent (80%of uterine contractions)	1
Increasing	2
< 70 BPM	1 (for each minute of duration)
Late decelerations	
Present	2
Increasing	3
< 100BPM (Each deceleration episode)	1 (For each minute of duration)
< 70 BPM	1

Table 2: Hon's prediction system for Apgar score

Krebs scoring system for CTG

Score	Baseline FHR	Amplitude (Variability BPM)	Variability (OSC. points/min)	Acceleration	Deceleration
0	<100 >180	<5	<3	0	Late or severe variable Deceleration
1	100-119 161-180	5-10	3-6	1-3	Mild or moderate variable Deceleration
2	120-160	>10	>6	>3	Early or No Deceleration

Table 3: Krebs scoring system for CTG

The predictive value of a normal FHR pattern in detecting a neonate with Apgar score ≥ 7 was 99%. The abnormal FHR pattern predicted a depressed neonate with 67% accuracy[30]. The FHR was normal in 98% of neonates with Apgar score ≥ 7 giving the scoring system a high specificity

A normal FHR pattern also could be seen in neonates with Apgar scores <7 but less frequent representing a sensitivity of 77% in various studies. Scores 8-10 were found with normal fetal scalp pH >7.25. Scores 6-7 were seen with pH 7.2-7.25, representing compensated fetal distress. Scores 0-5 were found associated with pH < 7.2, representing significant fetal distress[30,31,33].

Supplementary tests

If EFM is to be used as a screening test for intra partum fetal asphyxia, predictive fetal heart rate patterns require supplementary tests to avoid unnecessary intervention.

A number of supplementary tests are currently being assessed, including fetal vibro acoustic stimulation, the ST segment and PR interval of the fetal electrocardiogram, fetal pulse oximetry, fetal pH electrode, and near infrared spectroscopy. Although potential benefits have been reported, particularly in the identification of false positive fetal heart rate patterns, the

clinical value of these tests remains to be determined.

A fetal blood gas as acid base assessment with evidence of a metabolic acidosis is the gold standard for the diagnosis of fetal asphyxia. The occurrence (or) exclusion of intrapartum fetal asphyxia can be confirmed by a bloo d gas and acid base assessment of umbilical vein and arterial blood at delivery without compromising either the gravida (or) fetus

Umbilical cord blood gas analysis[32,33,34]

Maternal - fetal acid base balance is important to assess the adequacy of fetal oxygenation and fetal well being in utero. Henderson Hesselbach equation is the base to understand the acid base balance of mother and fetus. CO_2 produced by oxidative metabolism is dissociated into H^+ and HCO_3^- ions.

$$CO_2 + H_2O \longleftrightarrow H_2CO_3 \longleftrightarrow H^+ + HCO_3^-$$

The dissociation constant, K, in this equation is

$$K = [H^+]\,[HCO_3^-] / [H_2CO_3]$$

Equating this to get pH

$$pH = -\log [H^+] = p^K + \log \left([HCO3^-]/[H_2CO_3]\right)$$

As the carbonic acid is a weak acid and is in equilibrium with

dissolved CO_2, the equation becomes,

$$pH = p^K + \log \left([HCO3^-] / [CO_2(d)] \right)$$

The concentration of dissolved CO_2 is the function of partial pressure of CO_2 gas and solubility coefficient for CO_2 in plasma.

i.e., $[CO_2 (d)] = S^* PCO_2$

Hence, $pH = 6.1 + \text{Log} \left([HCO3^-] / S^* PCO_2 \right)$ as $p^K = 6.1$

i.e. pH is dependent on ratio of bicarbonate concentration and CO_2 concentration (or) CO_2 pressure on plasma.

With a normal pH of 7.4 the HCO_3^- concentration is 20 times the CO_2 concentration.

In hypoxia, HCO_3^- concentration decrease resulting in reduction in pH causing metabolic acidosis.

In maternal hypoventilation CO_2 concentration increases resulting in reduction in pH causing respiratory acidosis.

In maternal hyperventilation CO_2 concentration decreases resulting in increase in pH causing respiratory alkalosis.

Metabolic alkalosis rarely affects the fetus possible cause is

hyperemesis gravidarum due to loss of HCl in vomitus. HCO_3^- is retained to maintain anionic balance hence pH increases causing metabolic alkalosis.

Causes of acidosis

Placenta is the organ of respiration in fetus. When the placental transfer of oxygen is restricted, fetal metabolism preceeds along anaerobic pathway, producing excess lactic acid, the excess H^+ ions react with HCO_3^- ions in fetal blood causing metabolic acidosis.

However maternal hypoventilation in labour leads to fetal hypoxia (or) hypercapnea, producing metabolic acids. Hence a mixed acidosis always do occur.

Also incomplete oxidation of fatty acids produces ketoacids and some amino acids may also be metabolized to uric acids. These acids traverse the placenta only slowly hence these acids may be accumulated in conditions like diabetic ketoacidosis, starvation ketosis (or) renal failure. This will also decreases HCO_3^- ions resulting metabolic acidosis.

The acid base status

p^H, PCO_2, PO_2, CO_2 & O_2 content of blood can be measured.

Bicarbonate concentration and oxygen saturation can be determined from these measurement. The base excess (or) deficit can be calculated. These values and pH are important to assess the fetal

condition[35,36,37].

pH

Fetal pH is 0.1 less than mothers pH. Also normal pH differs from umbilical artery and vein. In umbilical artery normal pH is >7.15 during labour, >7.2 before labour. In umbilical vein normal pH is 7.2 during labour and >7.26 before labour.

In fetal scalp blood normal pH is >7.25. pH of 7.20 – 7.25 represents border line and pH <7.2 denotes acidemia.

PCO_2

Normal Value is 51 mmHg in umbilical artery and 43mmHg in the vein

PO_2

Normal Value is 18 mmHg in umbilical artery and 28 mmHg in the vein

HCO_3

In the umbilical artery the normal bicarbonate concentration is 24meq/L and 23 meq/L in the vein.

Base Excess / deficit

When fixed acids are produce the pH decreases. Buffering and respiratory compensation leave the pH unchanged. The depletion of the buffer base creates a base deficit.

Base excess = calculated – Normal Buffer Base

Base excess can be calculated from a normogram using pH, PCO_2 and HCO_3.

Normal buffer base = 46 to 49 meq/L, the normal range of the base deficit varies from -2 to +1 meq/L the normal base excess in umbilical artery is -3 meq/L and - 2 meq/L in the vein.

Of various components of the acid base and blood gas measurements PO_2, PCO_2, HCO_3, pH and lactate are measured directly the base deficit is calculated.

The best of these parameters for clinical use is umbilical arterial pH[38] lactate correlate well with this. PCO_2 only does so minimal because it increases only in severe cord compression, with rapid evolution of metabolic acidosis when prolonged severe cord compression occurs. Fetal O_2 changes not accompany sizable reduction in fetal oxygen reserves (As measured by

O_2 saturation)[40]. Fetal PO_2 may not fall measurably until O_2 reserves have been severely depleted. Because of these difficulties, PO_2 value are not reliable in assessing the status of fetus (or) deciding whether to intervene to prevent distress (or) death[13].

Carbonate is one of the most important buffers and can be calculated algebraically (or) from a nomogram.

A metabolic acidosis results in excess acids and decreased buffer base. This is expressed as a base deficit (or) a negative base excess. The concentration of H^+, the other important buffer besides HCO_3 helps determine the level of buffer base and therefore a base deficit (or) base excess. Clinically the base excess or base deficit is more useful than the HCO_3^- concentration.

The pH and the base excess (or) deficit are the most useful values to asses the fetal condition[13].

Cord Blood sampling

Sampling from the umbilical artery is more challenging than the vein because the artery has a smaller lumen, a thicker wall and contains less blood. For this reason it may be easier to sample the artery first because the

distended vein may provide some support to the artery.

Umbilical arterial blood most accurately reflects fetal status because the umbilical arterial blood flow directly from the fetus. In contrast, umbilical venous blood returns from the placenta. Umbilical venous blood gas measurements better reflect maternal acid base status and placental function in addition to fetal acid base status. Therefore, if only one vessel is selected, it should be the umbilical artery rather than the vein. It is not helpful than the vein. It is not helpful to obtain umbilical vein. Blood pH / gas value because it may completely normal when the arterial blood is acidemic.

Analysis the sample

Various blood gas analysers are commercially available. Each instrument will have advantages and disadvantages in a particular environment. The instrument should be caliberated and tested for quality control at specified intervals to ensure that the pH, PCO_2 and PO_2 results correlate with reference standard.

AIMS AND OBJECTIVES

1. Correlation of non reassuring CTG using scoring system with umbilical cord arterial blood pH.
2. To find out the perinatal outcome of non reassuring CTG and abnormal cord blood pH.

MATERIALS AND METHODS

The present study is a prospective study conducted in the department of Obstetrics and Gynaecology in PSG Institute of Medical Science and Research and PSG Hospitals, Coimbatore. 104 patients were included in the study from November 2007 to November 2008.

Inclusion criteria

- Gestational age >34 weeks
- Singleton
- Cephalic presentation

Exclusion criteria

- Elective LSCS
- Breech
- Anomalous babies
- Multifetal Gestation
- Gestational age < 34 weeks

Examination method

All patients were subjected to CTG, in the active phase of labour. The CTG used in the study was Philips Avalon FM30 with the paper speed being 1cm / min with an external transducer.

Uterine contractions were recorded simultaneously

CTG assessed objectively using Krebs scoring system every 30 mins in active labour.

Five parameters used in scoring system were

1. baseline Heart rate
2. baseline variability
3. Amplitude (Frequency)
4. Acceleration
5. Deceleration

Each parameters scored 0-2, giving a total score of 10
Three groups were made from the total score, 0-4, 5-7, 8-10.

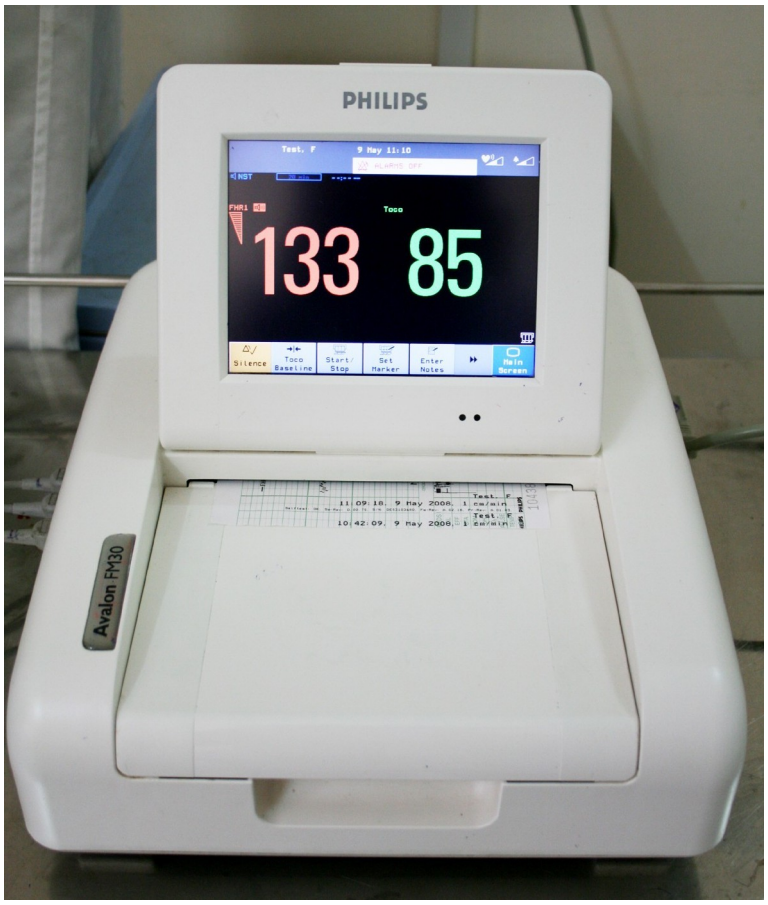


Fig 9: Showing CTG machine with trace

Krebs scoring system

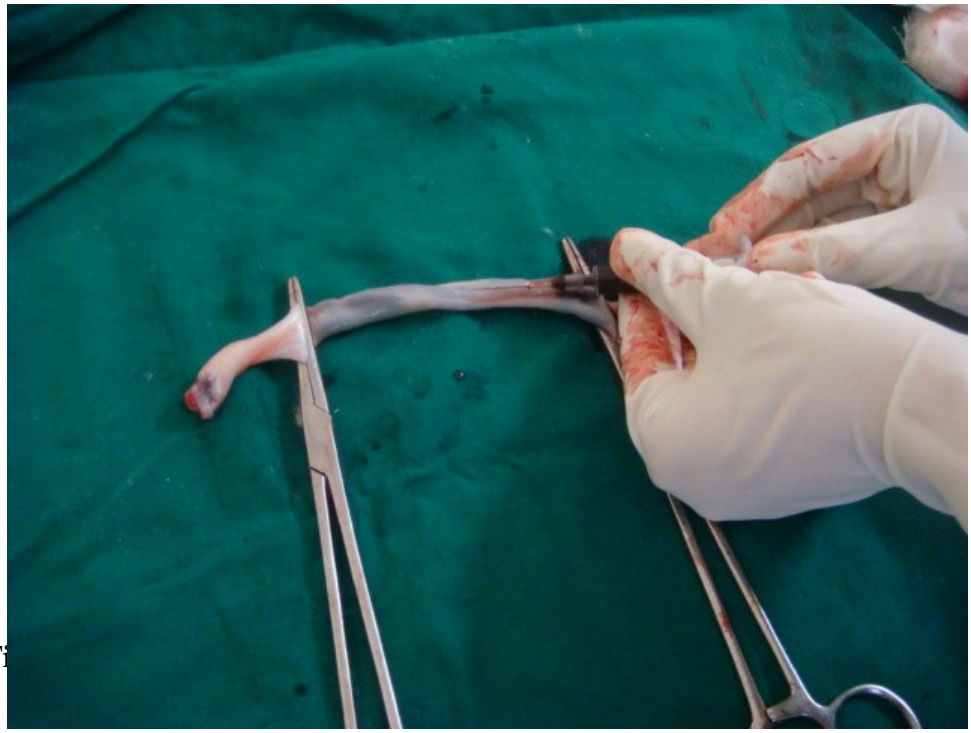
Score	Baseline FHR	Amplitude (Variability BPM)	Variability (OSC. points/min)	Acceleration	Deceleration
0	<100 >180	<5	<3	0	Late or severe variable Deceleration
1	100-119 161-180	5-10	3-6	1-3	Mild or moderate variable Deceleration
2	120-160	>10	>6	>3	Early or No Deceleration

Table 4: Krebs scoring system

Blood collection

Blood collection was performed following delivery, from immediately isolated segment (10 to 20 cm) of cord with two clamps near the neonate two clamps nearer the placenta. The importance of clamping the cord is underscored by the fact that delays of 20-30 secs can alter both the PCO_2 and pH. The cord was then cut between the two proximal and two distal clamps. Arterial blood was drawn from the isolated segment of cord into a 1-2 ml syringe that has been flushed with a heparin solution containing 1000 U/ml. The needle was capped and the syringe transported on ice to the laboratory.

(Although efforts should be made to transport the blood promptly, neither the pH nor PCO_2 change significantly in blood kept at room temperature for up to 60 minutes)**.**



Fi cord

Fig 11: Shows Arterial blood gas analyser

RESULTS AND ANALYSIS

1. Parity

Parity	Frequency (N)	%
Primi	62	59.6
Multi	42	40.4
Total	104	100.0

Table 5: shows the parity

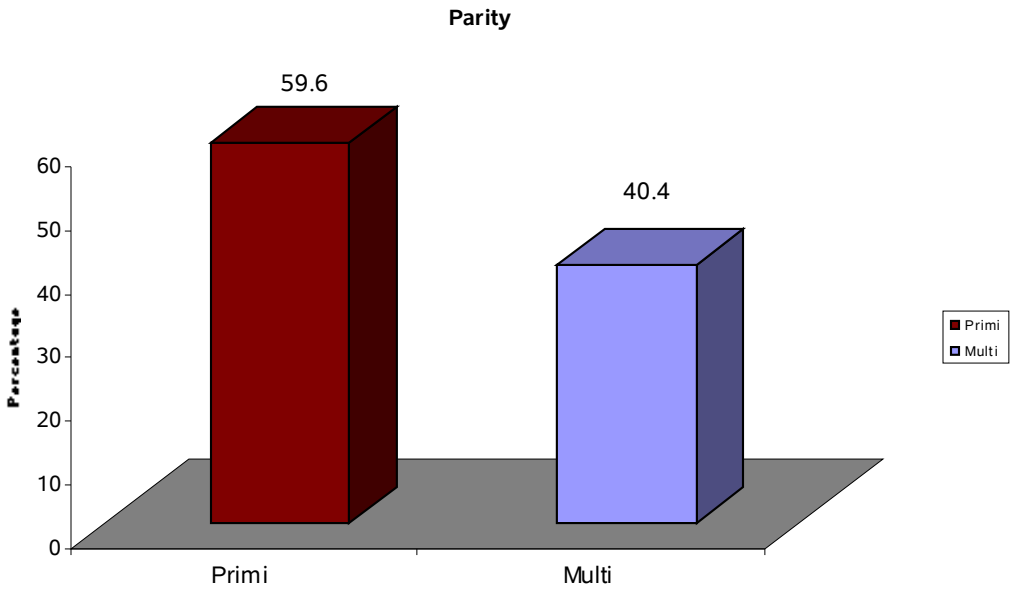


Fig 12: Shows the parity

2. Risk factors in Pregnancy

Parity	Frequency (N)	%
Low risk patients	76	73
High risk patients	28	27
Total	104	100.00

Table 6: Shows the number of low risk and high risk patients

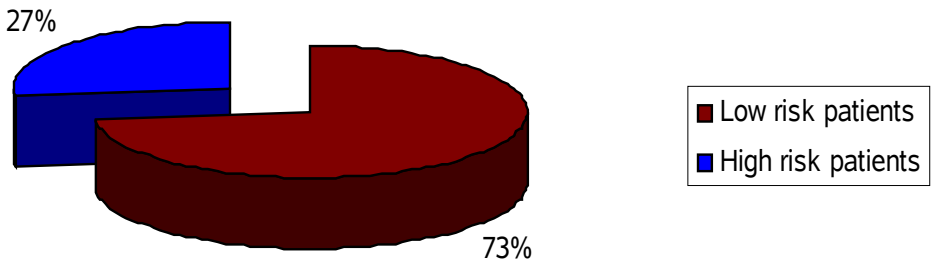


Fig 13: Shows the number of low risk and high risk patients

3. Onset of Labour

Onset of labour	Frequency (N)	%
Spontaneous	74	71.2
Induced	30	28.8
Total	104	100.00

Table 7: Shows the Mode of Onset of Labour

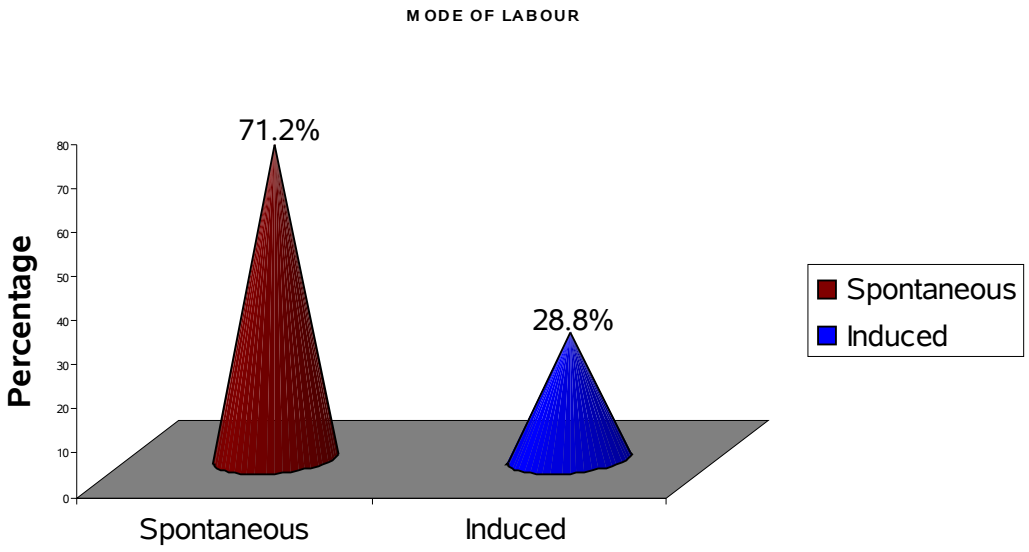


Figure 14: Shows the Mode of Onset of Labour

4. Rupture of membranes

Rupture of membrane	Frequency	%
ARM	79	75.9%
SROM	08	7.6%
PROM	17	16.5%
Total	104	100.00

Table 8: Shows the nature of rupture of membranes

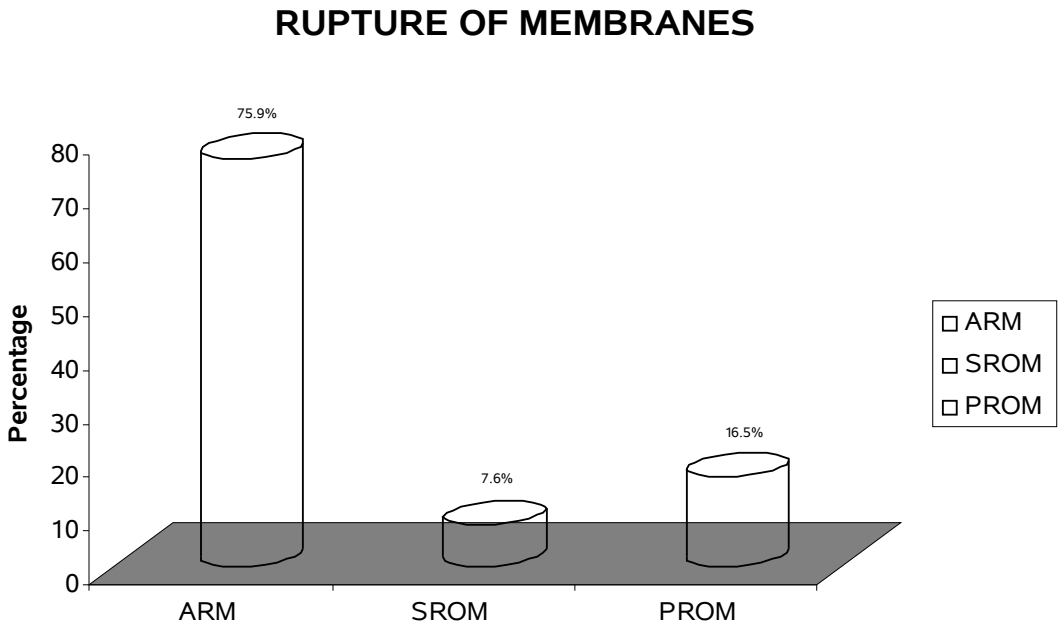


Figure 15: Shows the nature of rupture of membranes

5. Colour of Liquor

Colour of Liquor	Frequency	%
Clear	74	71.1%
Grade I MSL	06	5.9%
Grade II MSL	16	15.3%
Grade III MSL	08	7.7%
Total	104	100.00

Table:9 Shows the Colour of Liquor

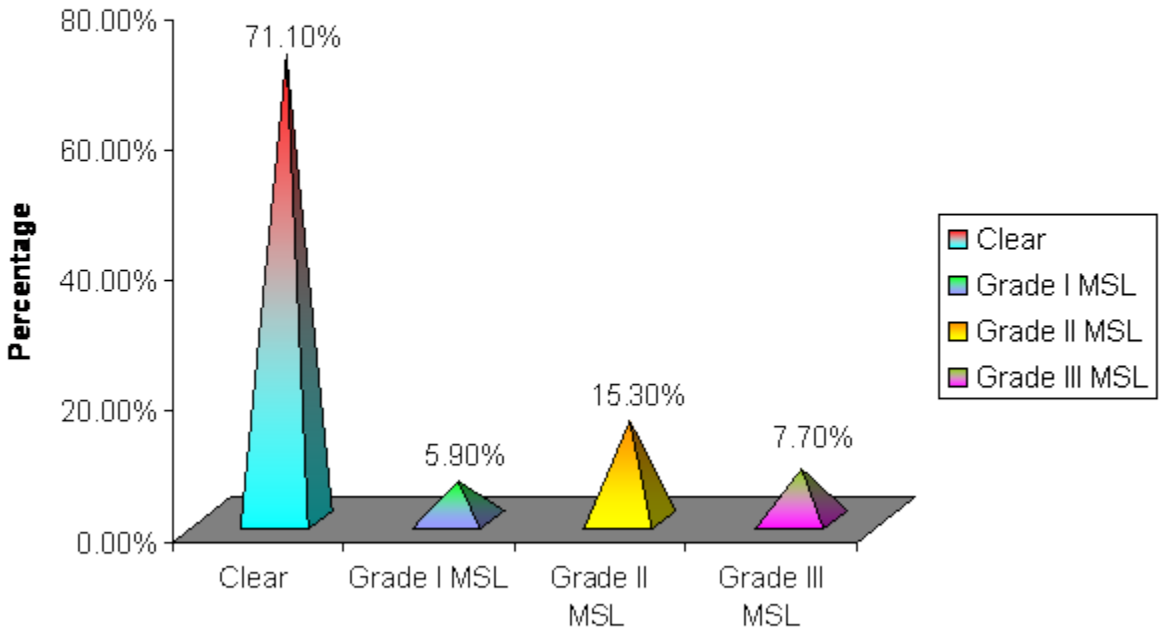


Figure 16: Shows the Colour of Liquor

6. Mode of delivery

Mode of Delivery	Frequency (N)	%
Vag delivery With episiotomy Vacuum Forceps	38 13 08	36.5% 12.5% 7.7%
LSCS	45	43.3%
Total	104	100.00

Table:10 Shows the mode of delivery

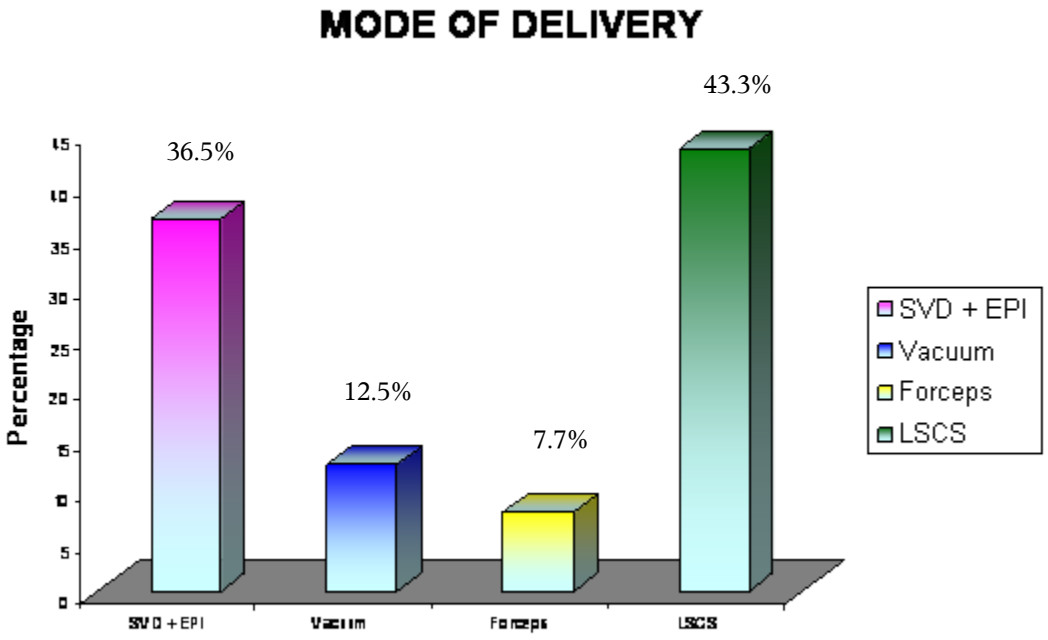


Figure :17 Shows the mode of delivery

Krebs scoring system for CTG

Score	Baseline FHR	Amplitude (Variability BPM)	Variability (OSC. points/min)	Acceleration	Deceleration
0	<100 >180	<5	<3	0	Late or severe variable Deceleration
1	100-119 161-180	5-10	3-6	1-3	Mild or moderate variable Deceleration
2	120-160	>10	>6	>3	Early or No Deceleration

Table 11: showing Kreb scoring system for CTG

CTG Score and correlation with cord blood pH value

Of all the 104 parturients included were on continous CTG monitoring in active phase of labour. The CTG was scored according to Krebs scoring system at an interval of every ½ hour.

Of these 104 patients, 75 patients were in active phase of labour for maximum of 3 hours, 21 patients were in active phase of labour for a maximum of 2 hours. 5 patients were there for a maximum of 1 hour and 3 patients were in the active phase for a maximum of ½ hour.

Based on CTG scores at the beginning of active phase of labour (0-4), (5-7), (8-10). Three groups of parturients were followed throughout the labour, some scores improved some deteriorated and some remained the same. The parturients in these three groups have segregated as group I (initial CTG score 8-10), group II (initial Score 5-7) and group III (initial score 0-4).

Group I (Parturients with a score of 8-10 at the beginning of active phase of labour)

58 parturients started active labour with a score of 8-10, of these 17 patients had persistent score of 8-10 upto delivery with mean pH of 7.30 and 2 neonates had acidosis in this group.

28 of 58 parturients who started active phase of labour with a CTG score of 8-10 had a CTG score of 5-7 before delivery. Had a mean pH of 7.22 and 7 neonates had acidosis in this group.

13 of the 58 parturients who started active phase of labour with a CTG score of 8-10 had shown a progressive fall in score with time. They had a score of 5-7 after 1-2 hours and had a score of 0-4 ½ hr prior to delivery. This group had a mean pH of 7.21 and 3 neonates had acidosis.

Group II (Parturients with a score of 5-7 at the beginning of active phase of labour)

37 parturients had a CTG score of 5-7 at the beginning of active phase of labour. 14 of the 37 parturients had the same CTG score of 5-7 from the beginning of active phase till delivery. This group had a mean pH of 7.25 and 2 neonates had acidosis of this group.

One of the 37 parturients who started active labour with a CTG score of 5-7, had a score of 8-10 after 1 hour of labour and delivered with the same score. The neonate had a cord pH of 7.34.

22 of the 37 parturients who started active labour with a CTG of 5-7 had a score of 0-4 ½ hrs prior to labour. This group had a mean pH of 7.18. 8 noenates in this group had acidosis.

Group III (Parturients with a score of 0-4 at he beginning of active phase of labour)

9 parturients had a score of 0-4 in the active phase on admission.

5 of the parturients had a CTG score of 0-4 for 3 hours prior to delivery. They had a mean pH of 7.07. 2 neonates had acidosis.

2 parturients of the 9 started active phase of labour with a poor CTG score of 0-4 and had delivered with 1 hr with the same score. The mean pH of this group was 7.14 and one neonate had acidosis.

2 of the 9 parturients in this group had been in active phase of labour for only ½ hr. Both the patients delivered by LSCS. The mean pH of this group was 7.25. None of the neonates in this group had acidosis.

Fig 18 represents the graphical representation of the study population comparing the three group viz., Group I with CTG score 8 to 10, Group II with CTG Score 5 to 7, group III with CTG score 0-4 at ½ hr, 1 hr, 2 hrs and 3 hrs. The scores has been plotted against the mean pH.

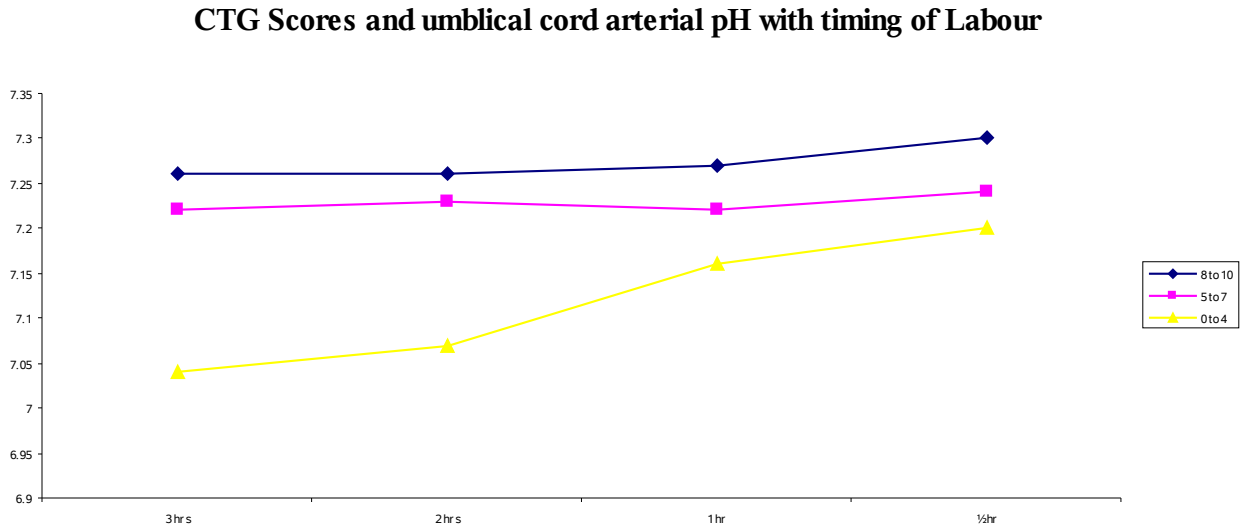


Figure 18: Shows CTG Scores and umblical cord arterial pH with timing of Labour

Correlation between CTG Score ½ hour before delivery and cord arterial blood pH

CTG Score	pH		Mean pH
	≤7.15	>7.15	
0-4	16	29	7.2
5-7	8	34	7.2
8-10	1	16	7.3

Table 12: Correlation between CTG Score ½ hour before delivery and cord arterial blood pH

Chi - Square Analysis was used to correlate pH & CTG scores

½ hour CTG scores Vs pH (Umbilical artery)

CTG Score	% of patients	
0-4	35.5%	64.4%
5-7	19%	80.9%
8-10	5.8%	14.1%

Table 13: Percentage of correlation between CTG Score ½ hour before delivery and cord arterial blood pH

P value = 0.01
<0.05 – significant

This table shows a significant correlation between acidosis and CTG score at ½ hour before delivery

Correlation between CTG Score 1 hour before delivery and cord arterial blood pH

CTG Score	pH		Mean pH
	≤7.15	>7.15	
0-4	7	11	7.16
5-7	14	28	7.22
8-10	4	32	7.17

Table 14: Correlation between CTG Score 1 hour before delivery and cord arterial blood pH

Chi - Square Analysis
1 hour CTG scores Vs pH (Umbilical artery)

CTG Score	% of patients	
0-4	41.1%	64.7%
5-7	33.3%	66.6%
8-10	11.1%	88.8%

Table 15: Correlation between CTG Score 1 hour before delivery and cord arterial blood pH

P value = 0.02

<0.05 – significant

This table shows a significant correlation between acidosis and CTG score at 1 hour before delivery

Correlation between CTG Score 2 hours before delivery and cord arterial blood pH

CTG Score	pH		Mean pH
	≤7.15	>7.15	
0-4	6	3	7.09

5-7	12	29	7.23
8-10	8	37	7.26

Table 16: Correlation between CTG Score 2 hours before delivery and cord arterial blood pH

Chi - Square Analysis was used to correlate pH & CTG scores

2 hours CTG scores Vs pH (Umbilical artery)

CTG Score	% of patients	
0-4	66.6%	33.3%
5-7	29.2%	70.7%
8-10	17.7%	82.2%

Table 17: Percentage of Correlation between CTG Score 2 hours before delivery and cord arterial blood pH

P value <0.01 – significant

This table shows a significant correlation between acidosis and CTG score at 2 hours before delivery

Correlation between CTG Score 3 hours before delivery and cord arterial blood pH

CTG Score	pH		Mean pH
	≤7.15	>7.15	
0-4	3	2	7.07
5-7	7	14	7.22
8-10	9	31	7.26

Table 18: Correlation between CTG Score 3 hours before delivery and cord arterial blood pH

Chi - Square Analysis

3 hours CTG scores Vs pH (Umbilical artery)

CTG Score	% of patients	
0-4	60.0%	40.0%
5-7	33.3%	66.6%
8-10	22.5%	77.5%

Table 19: Correlation between CTG Score 3 hours before delivery and cord arterial blood pH

P value = 0.085
> 0.05 - Insignificant

This table shows no correlation between CTG scores taken 3 hours before delivery and acidosis. This could be due to various intra partum events that change the pH with time in labour.

The outcome of the neonates delivered were also assessed in this study. The outcome was correlated to ½ hour CTG score and the results have been tabulated in table 20.

Of 104 neonates 66 had good outcome,

18 belong to group I

29 belong to group II

19 belongs to group III.

38 needed ICU care,

17 were treated for mild respiratory distress (17= 0+6+11)

13 needed CPAP (13 = 0+5+8),

8 needed ventilator support (8= 0+3+5).

There was no neonatal deaths found

Neonatal outcome

Outcome		CTG Score			Total
		0-4	5-7	8-10	
Good	Count	19	29	18	66
	% of outcome with CTG Score	28.9%	43.9%	27.2%	100.0%
	% of CTG Score at ½ hour	42.2%	69.0%	100.0%	63.4%
CPAP	Count	8	5	0	13
	% of outcome with CTG Score	61.6%	38.4%	0.0%	100.0%
	% of CTG Score at ½ hour	17.7%	11.9%	0.0%	12.5%
Ventilator	Count	5	3	0	8
	% of outcome with CTG Score	62.5%	37.5%	0.0%	100.0%
	% of CTG Score at ½ hour	11.1%	7.14%	0.0%	7.7%
Mild respiratory Distress	Count	11	6	0	17
	% of outcome with CTG Score	64.7%	65.3%	0.0%	100.0%
	% of CTG Score at ½ hour	24.4%	14.2%	0.0%	16.3%
Total	Count	43	43	18	104
	% of outcome with CTG Score	41.3%	41.4%	17.3%	100.0%
	% of CTG Score at ½ hour	100.0%	100.0%	100.0%	100.0%

Table 20: Neontal outcome of various CTG scores

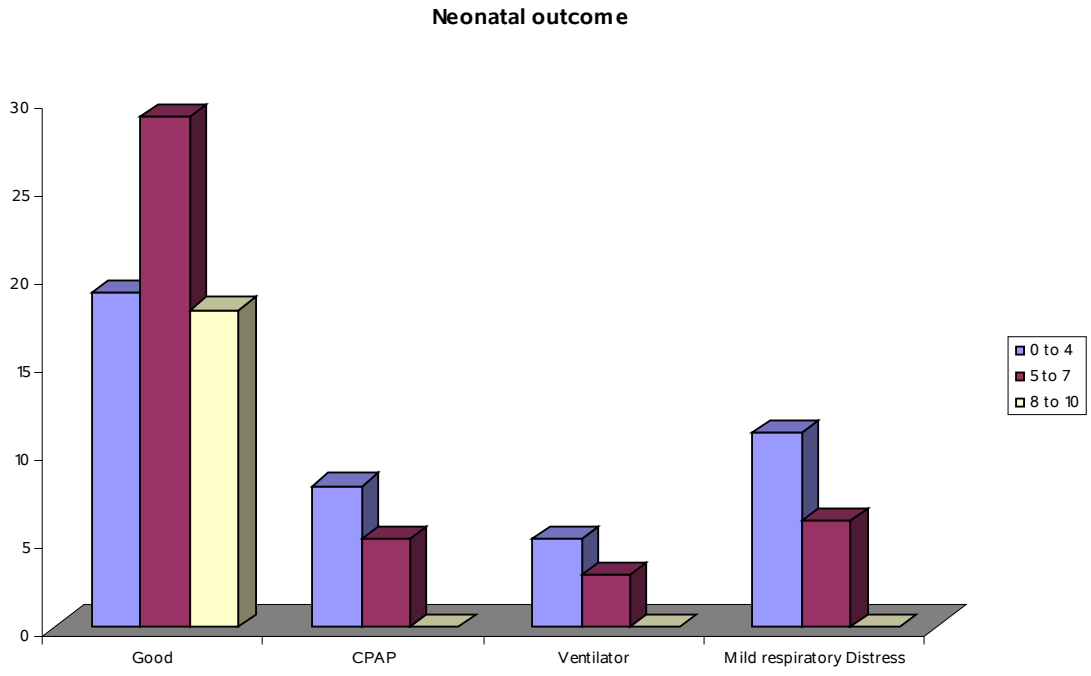


Fig 19: Showing Neonatal outcome of various CTG scores

DISCUSSION

CTG has got high false positive rate and there were also associated increased rate of caesarean section because of inter and intra observer variation. Hence, the objective analysis of CTG is needed. Here a scoring system was used to grade the CTG, based on this scoring system, fetal umbilical cord arterial blood pH was correlated and perinatal outcome of these two parameters were also assessed.

Of 104 patients who were included in this study 62 were primi and 42 were multies, 76 were low risk pregnancies and high risk factors like aneamia, preeclampsia, heart disease, previous neonatal death, bad obstetric history, diabetes mellitus, hypothyroidism were found in 28 patients.

Of 104 patients 74 patients had spontaneous onset of labour pains and the rest 30 patients were induced with prostaglandins/ oxytocics.

Of 104 patients 79 patients had ARM done, 8 patients had spontaneous rupture of membranes and remaining 17 had premature rupture of membranes.

Colour of the liquor was also looked in the study population, because

birth asphyxia is more commonly associated with meconium stained amniotic fluid. Of 104 patients, 74 had a clear liquor, 06 patients had a grade I meconium stained liquor, 16 patients had a grade II meconium stained liquor, 8 had a Grade III meconium stained liquor.

Various modes of delivery were widely distributed in the study population. Of 104 patients 44 patients delivered by LSCS for various causes like fetal distress, protracted descent and dilatation, arrest of descent and dilatation. Of 45 LSCS, 30 were done for fetal distress.

17 parturients had a CTG score of 8-10 from beginning of active phase of labour till delivery. They had a mean umbilical arterial pH of 7.3.

42 parturients (28 from 8-10 group, 20 from 5-7 group) had a CTG score of 5-7 at delivery. They had a mean umbilical arterial pH of 7.22 and 7.25 respectively.

5 parturients had a CTG score of 0-4 from 3 hours prior to and till delivery. They had a mean umbilical arterial pH of 7.07. There was significant correlation (p value <0.01) between low CTG score and acidosis.

Changes of CTG score with time was observed,

CTG score 0-4, if the patients was delivered with in ½ hour/ one hour, the risk of acidosis was 35.5% / 41.1% respectively. If delivery was prolonged to 3 hours, the risk of acidosis was 60.0%.

Correlation between CTG score and pH at time intervals was done,

CTG scoring at ½ hour, 1 hour, 2 hours prior to delivery correlated well with umbilical arterial pH at birth. P value <0.05 for ½ hr score, p value of 0.02 for the 1 hour score, p value of <0.01 for 2 hours score. There was no significant correlation with umbilical arterial pH at birth and CTG score taken 3 hours prior to delivery.

With a CTG score of 8-10,

70.6% had stepped down and only 24.3% of neonates had acidosis with a CTG score of 5-7, 59.4% of patients had stepped down to 0-4 score and of them 36.3% of neonates had acidosis.

Rapid deterioration of CTG score requires immediate intervention to prevent acidosis.

Joel D Larma et al., found sensitivity of 53.8%, specificity of 61.7%, positive predictive value of 50%, and negative predictive value of 88.6% with poor CTG score for outcome as acidosis.

In our study we have found sensitivity of CTG with a poor score of 0-4 for the outcome as acidosis of the neonate is 55.81%, Specificity of the same was 77.04%.

Positive predictive value i.e., with a poor CTG score of 0-4, the probability of acidosis for the neonate is 63.15%

Negative predictive value i.e, with a good CTG score of either 5-7/8-10, the probability of delivering a non acidotic neonate is 71.21%

CONCLUSION

Although FHR monitoring is widely used for fetal surveillance there is still disagreement about the value of CTG and the interpretation of FHR patterns. In our prospective study umbilical cord blood pH values at the time of delivery were related to FHR patterns classified according to Krebs scoring system.

There was a significant correlation ($P<0.01$) between low CTG scores and acidosis. Rapid deterioration of CTG scores were found to require immediate intervention to prevent acidosis.

Sensitivity of CTG with a poor score (0-4) for the outcome as acidosis is 55.81%, specificity of the same was 77.04%.

PPV i.e, with a poor CTG score the probability of the acidosis in the neonate is 63.15%

NPV i.e., with the good CTG score 5-7 / 8-10 the probability of delivery of a non acidotic neonate is 71.21%.

Application of scoring system in interpretation of CTG in labour helps to reduce inter and intra observer variation in interpretation and provides the obstetrician a yard stick to measure fetal well being in labour.

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PROFORMA

Name	:			
Age	:			
Hospital No.	:			
LMP	:			
EDD	:			
Gestational age	:			
Date of admission	:			
Date of delivery	:			
Date of discharge	:			
Obstetric formula	:			
Chief complaints	:	Pain	Bleeding P/V	Leaking P/V
Fetal movements	:	Yes / No		
Menstrual history	:	Cycles Regular / Irregular		
Marital history	:	Married since – Yrs		
Booked /Not	:			

Antenatal complications : PIH/GDM/IUGR/Preterm/Aneamia/oligohydramnios

Past History : Hyper tension/ DM/BA/PTB

Examination

General Examination

Pallor

Edema

Breast

Thyroid

Height

Weight

Pulse rate

Blood pressure

Systemic Examination

CVS

RS

P/A

Height of the uterus

Lie

Presentation

Presenting part – engaged / unengaged

FHR

P/S

Leaking + / -, If + colour

P/V

Cervical,

Consistency

Position

Effacement

Membrane status

Station

Investigation

Hb

RBS

Urine routine

Blood grouping and typing

HIV

Hbs Ag

USG

-Gestational age

- estimated fetal weight

- Amniotic fluid index

- Presentation

Fetal CTG

Time	Baseline	Amplitude BPM	Variability	Acceleration	Deceleration	Score

Labour

Spontaneous

Induced

Risk factors

Low risk

High risk

Duration of stages

I stage

II stage

III stage

Mode of delivery

Spontaneous vaginal delivery with Episiotomy

Vacuum

Forceps

LSCS

Umbilical cord pH

Baby details

Apgar - 1 min

5 mins

Resuscitation needed/not

NICU Admission needed/not

Ventilatory support needed/not

S.No	Age	Obst score	Gest. Age	Risk factors	Rup of mem	Colour of liquor	Initial CTG Score	Baby with Apgar		Umbilical arterial blood pH	Fetal outcome
								1 min	5 min		
1	30	P	T	-	ARM	Clear	8-10	8/10	9/10	7.30	Good
2		M	T	-	ARM	Clear	8-10	8/10	9/10	7.23	Good
3	20	P	T	Aneamia	ARM	Gr II MSL	8-10	8/10	9/10	7.25	Good
4	25	P	36wks	-	PROM	Clear	5-7	8/10	9/10	7.32	Good
5	24	P	T	-	ARM	Gr I MSL	5-7	8/10	9/10	7.22	Good
6	22	M	T	BOH	ARM	Gr III MSL	8-10	6/10	7/10	7.15	CPAP
7	23	P	35 wks	HD	ARM	Clear	8-10	8/10	9/10	7.30	Good
8	25	M	T	-	ARM	Clear	8-10	8/10	9/10	7.15	MRD
9	25	P	PD	-	PROM	Clear	8-10	7/10	8/10	7.10	Good
10	30	M	T	-	SROM	Clear	5-7	8/10	8/10	7.25	Good
11	21	P	T	PE	ARM	GrIII MSL	8-10	7/10	8/10	7.15	Good
12	25	M	T	-	ARM	Clear	8-10	7/10	8/10	7.22	Good
13	20	P	36 wks	PE	ARM	GrII MSL	8-10	8/10	8/10	7.25	MRD
14	27	P	T	-	SROM	Clear	8-10	8/10	8/10	7.15	MRD
15	24	P	T	-	ARM	GrII MSL	5-7	8/10	9/10	7.25	Good
16	32	M	T	GDM	PROM	Clear	5-7	7/10	8/10	7.05	CPAP
17	24	P	T	GDM	ARM	Clear	5-7	7/10	7/10	7.02	*
18	25	P	T	-	ARM	Clear	8-10	8/10	9/10	7.31	Good
19	29	M	T	-	PROM	Clear	8-10	8/10	9/10	7.30	Good
20	23	P	T	-	ARM	Clear	8-10	8/10	9/10	7.31	Good
21	27	M	36 wks	PE	ARM	Gr II MSL	8-10	6/10	7/10	7.05	*
22	29	M	T	GDM	SROM	Gr II MSL	0-4	7/10	7/10	7.15	CPAP
23	25	M	T	-	ARM	Clear	8-10	8/10	9/10	7.12	CPAP
24	23	P	T	-	ARM	Clear	8-10	8/10	9/10	7.25	Good

25	31	M	T	GDM	ARM	Clear	8-10	7/10	7/10	7.14	*
26	27	M	T	-	ARM	Clear	8-10	8/10	9/10	7.27	Good
27	26	P	T	-	PROM	Clear	8-10	7/10	7/10	7.15	*
28	30	M	T	-	ARM	Clear	8-10	8/10	9/10	7.31	Good
29	22	P	T	-	SROM	Gr I MSL	8-10	8/10	9/10	7.25	Good
30	22	M	T	HD	ARM	Clear	8-10	8/10	9/10	7.24	Good
31	19	P	PD	-	ARM	Gr II MSL	5-7	7/10	7/10	7.13	CPAP
32	24	P	T	-	ARM	Clear	8-10	8/10	9/10	7.33	Good
33	17	P	T	-	ARM	GR II MSL	8-10	8/10	9/10	7.25	Good
34	24	M	T	-	ARM	Clear	5-7	8/10	8/10	7.23	MRD
35	18	P	T	PE	PROM	Clear	8-10	8/10	9/10	7.30	Good
36	21	M	T	-	ARM	GRIII MSL	0-4	8/10	8/10	7.32	MRD

37	23	P	T	HD	ARM	Clear	5-7	8/10	9/10	7.20	MRD
38	23	P	PD		ARM	GRII MSL	5-7	8/10	9/10	7.20	MRD
39	27	M	T	GDM	ARM	Clear	8-7	8/10	9/10	7.31	Good
40	22	P	T		ARM	Clear	8-10	8/10	9/10	7.25	Good
41	28	M	PD		PROM	Clear	5-7	7/10	7/10	7.12	*
42	25	P	T		ARM	Clear	8-10	8/10	9/10	7.26	Good
43	24	P	T		ARM	Clear	5-7	7/10	7/10	7.15	CPAP
44	19	P	T	Aneamia	ARM	GRIIIMSL	0-4	7/10	7/10	7.15	CPAP
45	26	M	T		ARM	Clear	5-7	8/10	9/10	7.26	Good
46	25	P	T		SROM	Clear	8-10	8/10	9/10	7.32	Good
47	27	M	T		ARM	Clear	8-10	8/10	9/10	7.18	Good
48	27	M	T		ARM	Clear	8-10	8/10	9/10	7.24	Good
49	20	P	T	PE	ARM	GRI MSL	8-10	6/10	7/10	7.13	CPAP
50	23	P	T		AROM	Clear	8-10	7/10	8/10	7.34	Good
51	25	M	T		ARM	GRII MSL	5-7	7/10	7/10	7.25	CPAP
52	25	P	T		ARM	Clear	8-10	8/10	9/10	7.12	Good
53	29	M	T	GDM	PROM	Clear	5-7	8/10	9/10	7.25	MRD
54	24	P	T		ARM	GRII MSL	8-10	8/10	9/10	7.20	Good

55	23	P	T		ARM	Clear	5-7	7/10	7/10	7.15	MRD
56	22	P	T		ARM	Clear	5-7	8/10	9/10	7.25	Good
57	19	P	T		ARM	Clear	7-10	7/10	7/10	7.23	MRD
58	25	M	T	Prev.NND	ARM	Clear	8-10	8/10	9/10	7.31	Good
59	24	P	T		ARM	Clear	5-7	8/10	8/10	7.27	MRD
60	28	M	T	BOH	SROM	GRI MSL	8-10	8/10	9/10	7.25	Good
61	26	M	T		ARM	Clear	8-10	8/10	9/10	7.23	Good
62	23	P	T	-	ARM	Clear	5-7	8/10	8/10	7.20	MRD
63	28	M	T	Prev.NND	PROM	Clear	0-4	7/10	7/10	7.10	*
64	24	P	T		ARM	Clear	8-10	8/10	9/10	7.32	Good
65	23	P	T		ARM	Clear	5-7	8/10	8/10	7.23	MRD
66	24	P	T	Aneamia	PROM	Clear	8-10	8/10	9/10	7.30	Good
67	27	M	T		ARM	Clear	8-10	8/10	7/10	7.13	CPAP
68	33	M	T		PROM	Clear	5-7	8/10	9/10	7.32	Good
69	24	P	T		ARM	Clear	8-10	8/10	9/10	7.33	Good
70	20	P	35 wks		ARM	GRII MSL	5-7	8/10	8/10	7.02	CPAP
71	35	M	T		PROM	Clear	5-7	8/10	9/10	7.20	Good

72	24	P	T		ARM	GRIII MSL	0-4	8/10	9/10	7.25	Good
73	28	M	T		ARM	Clear	5-7	8/10	9/10	7.25	Good
74	23	P	T		PROM	Clear	5-7	8/10	8/10	7.23	MRD
75	25	P	T		ARM	Clear	5-7	8/10	9/10	7.23	Good
76	26	M	T	Prev.NND	ARM	GRI MSL	5-7	8/10	8/10	7.23	MRD
77	25	P	T		ARM	Clear	0-4	7/10	8/10	7.25	MRD
78	28	M	T		ARM	Clear	8-10	8/10	9/10	7.30	Good
79	30	M	T		PROM	Clear	8-10	8/10	9/10	7.34	Good
80	25	P	T		ARM	Clear	8-10	8/10	9/10	7.25	MRD
81	27	P	T	PE	ARM	GRIIMSL	8-10	8/10	9/10	7.33	Good
82	22	P	T		ARM	Clear	8-10	8/10	9/10	7.30	Good
83	30	M	T		PROM	GRIIMSL	5-7	7/10	7/10	7.13	*
84	22	P	T		ARM	Clear	8-10	8/10	9/10	7.31	Good
85	28	M	T		ARM	Clear	5-7	8/10	9/10	7.31	Good
86	24	P	T	HD	ARM	Clear	5-7	8/10	8/10	7.25	CPAP
87	22	P	T		SROM	Clear	8-10	8/10	9/10	7.25	Good
88	20	P	T		ARM	Clear	0-4	8/10	9/10	7.25	Good
89	19	P	PD	Aneamia	ARM	GRIIIMSL	8-10	8/10	8/10	7.23	CPAP
90	24	M	T		ARM	Clear	8-10	8/10	9/10	7.33	Good
91	24	P	T		ARM	Clear	5-7	8/10	9/10	7.26	Good
92	25	M	T	GDM	ARM	GRIIMSL	8-10	8/10	8/10	7.18	CPAP
93	22	P	T		ARM	Clear	0-4	8/10	9/10	7.25	Good
94	23	P	T		ARM	GR II MSL	8-10	8/10	9/10	7.25	Good
95	26	M	T		PROM	Clear	5-7	8/10	9/10	7.33	Good
96	22	P	T	PE	ARM	GRIMSL	0-4	8/10	9/10	7.33	Good
97	21	P	T		ARM	Clear	8-10	8/10	9/10	7.30	Good
98	19	P	35 wks	PE	ARM	GRIIIMSL	5-7	8/10	7/10	7.10	*

99	20	P	T		ARM	Clear	8-10	8/10	9/10	7.25	Good
100	19	P	T		SROM	Clear	8-10	8/10	9/10	7.25	Good
101	28	M	T		ARM	Clear	5-7	8/10	9/10	7.20	Good
102	27	P	PD		ARM	GRIIIMSL	8-10	7/10	7/10	7.12	CPAP
103	29	M	T		ARM	GRIIMSL	5-7	8/10	9/10	7.33	Good
104	29	M	T		ARM	Clear	8-10	8/10	9/10	7.33	Good