STUDY OF ASSESSING THE EFFICACY OF INTRAPARTUM FETAL MONITORING AND FETAL SCALP BLOOD pH IN PREDICTING FETAL JEOPARDY

Dissertation submitted for

M.D. OBSTETRICS AND GYNAECOLOGY

BRANCH II

MADRAS MEDICAL COLLEGE
CHENNAI

FEBRUARY 2006

CERTIFICATE

This is to certify that this dissertation entitled **"STUDY OF ASSESSING THE EFFICACY OF INTRAPARTUM FETAL MONITORING AND FETAL SCALP BLOOD pH IN PREDICTING FETAL JEOPARDY "** has been done by **Dr.M. Vijayalkashmi**, Post Graduate in M.D. (Obstetrics and Gynecology) under my overall supervision and guidance at Govt. Kasturba Gandhi Hospital , Madras Medical College, Chennai in partial fulfillment of regulation of Tamilnadu Dr. M.G.R. Medical University for the award of M.D. Degree in Obstetrics and Gynecology.

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ACKNOWLEDGEMENT

I sincerely thank Prof. Dr. Kalavathy Ponnirivan B.Sc.,M.D**. Dean, Madras Medical college, Chennai for granting me permission to use the facilities of the institution and hospital for this study.**

I express my gratitude and thanks to Prof. Dr. V.Madhini, M.D., D.G.O., MNAMS, **Director , Institute of Obstetrics and Gynecology, Chennai for her guidance.**

I am greatly indebted to Prof. Dr.P.M.Santhamani, M.D.D.G.O., Superintendent **Govt. Kasturba Gandhi Hospital for women and Children, Triplicane, Chennai for providing with the necessary facilities to carryout this study and for her continuous support and guidance.**

I am extremely grateful to Prof. Dr. Rathnakumar, M.D. D.G.O., **Govt. Kasturba Gandhi Hospital for women and children for giving me inspiration, wise directions and encouragement at every stage in bringing out this dissertation.**

I also thank Dr. Muthuvel , M.A., Ph.D**., who helped me in completing statistical work.**

I wish to thank all unit chiefs, Assistants and all my colleagues for their continuous support.

I also thank all the staff members of Labor ward who kindly contributed for the successful competition of this project.

I also thank all the patients for their kind co-operation.

INTRAPARTUM FETAL MONITURING

AND SCALP BLOOD PH

INTRODUCTION

The birth of a healthy baby is a universal aim. About half to one third of perinatal death are antepartum stillbirths. Of these half to two third are due to congenital malformation. Therefore the number of fetuses that can be salvaged by intrapartum surveillance is small.

Each and every fetus has a potential for intrapartum hypoxia or birth injury and an optimal outcome can be concluded only at the end of labor.

However in spite of technological developments in ultra sonogram and electronic fetal monitoring, there has been some disillusion with the result of their clinical application. It is not uncommon to see low risk babies born with asphyxia and normal sized babies delivered operatively for 'fetal distress' in excellent condition. The fetus in labor is not a passive onlooker with no threat posed to its existence **Buckell and wood 1985.**

Risk to fetus has been recently elucidated in terms of biochemical insult and fetal response **kjellmer 1998**.

The original concept of **Little 1996 and Haldane 1992** that cerebral palsy and fetal death are caused by hypoxia at the time of

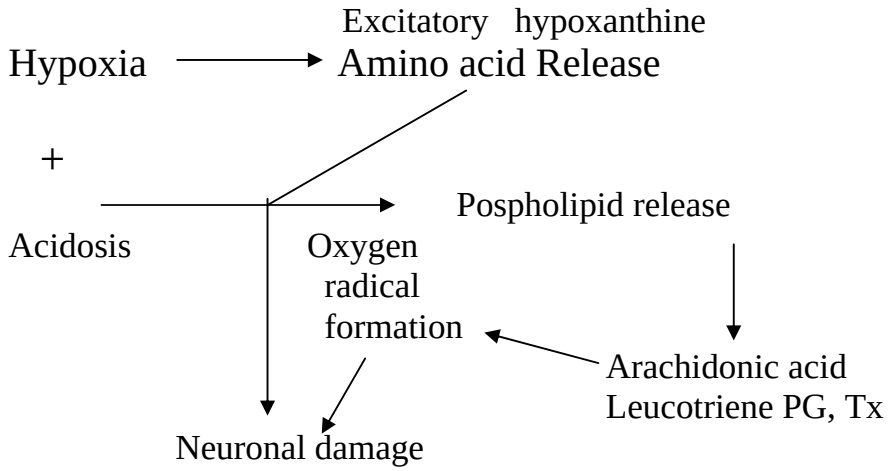
delivery have been challenged by epidemiological data showing that by the time labor begins, the majority of cerebral damage has already taken place **(Nelson and Ellenberg 1996)** and even severe perinatal asphyxia does not result in cerebral palsy.

Despite these contradictions, there is still good evidence that stress of labor in terms of metabolic acidosis can lead to cerebral damage and fetal death (K**jellmer 1998)** and that insults are not limited to high risk pregnancies (ie maternal disease and placental compromise)

Fetal neuronal damage, its occurrence and site are dependant on the degree of asphyxial insult. Total cessation of fetal oxygenation either as a result of total cord occlusion or complete placental abruption result in lesions in the pontine region of brain stem which almost invariably lead to brain death.

Incomplete hypoxia or intermittent reparative cord occlusion leads predominantly to lesions in parietal sub cortical white mater and basal ganglion in a distribution similar to that seen in cerebral palsy. (**kjellmer 1988).**

Causes of Asphyxial Brain damage



Excitatory amino acids and oxygen radicles are profoundly neurotoxic.

Continuos fetal heart rate monitoring was started in the late 1950's.

Edward was the first to recognize the FHR pattern and could predict the neonatal outcome as determined by 5mts. Apgar score.

By the time fetal blood sampling came on the scene pioneered by **SALING IN BERLIN 1960** the FHR was a well-known and established method of identifying fetal compromise

Before the wide spread use of cardiotocogram, the relationship between FHR and development of acidosis in labor was well

described by **Beard et al 1971 Tejani et al 1975[4].** Even in most abnormal pattern, acidosis is present in 50% of babies only.

An improved understanding of patho physiology of fetus and better fetal monitoring has itself contributed to the reduction in perinatal mortality.

FOGSI study 1983 revealed almost two thirds of all perinatal deaths are preventable by regular care, timely admission for delivery, careful monitoring, appropriate management in labor and better neonatal services.

REVIEW OF LITERATURE

Until 17th century no written record of the detection of fetal life in utero was available.

Francois Isaac Mayor in 1818, a surgeon, by applying the ear directly to the pregnant mother's abdomen reported that fetal heart was different from the maternal pulse

Anton Freidrich Hohli in 1834 described the design of fetal stethoscope. In 1876 Pinard designed the fetoscope.

Winkel in 1893 empirically set the limits of fetal heart rate between 120 – 160 bpm. Schwartz 1858, & Vonwinckel (1893) stressed the importance of auscultation throughout labor

Gunn & Wood (1953) reported the amplification and recording of fetal heart rate sounds in the proceedings of the Royal society of medicine.

In 1958, Hon pioneered electronic fetal monitoring in USA

Caldeyro – Barcia in Uruguay and Hammacher in Germany reported their observations on various heart rate patterns associated with fetal distress in 1968. This set the scene for production of the first

commercially available fetal monitor by Hammacher and Hewlett Packard in 1968 soon to be followed By Sonicaid in the U.K.

The Dublin – FHR monitoring study (Mac Donald et al 1985)[15] compared continuous FHR monitoring and intermittent auscultation. The Neonatal seizures in intermittent auscultation Group was slightly higher (8.5 %) than electronically monitored group (2.4%).

EFM is a subject of controversy for the last 2 decades. Several authors criticized the policy of EFM. (Leveno etal 1986, Stil et al 1990) That EFM is criticized as leading to an increase in the number of caesarean section without any evidence of fetal benefits.

Barring acute events, in low risk cases, admission Test (A.T) may be a good predictor of fetal condition for the next few hours (up to 5 hours) of labor in term fetus. (Arulkumaran & Gibb, 1990)[34] In ominous tracing 40 % developed fetal distress and in reactive tracings only 1.4 % developed fetal distress (APGAR < 7)

Hence admission test is useful to pick up fetal distress in low risk cases,. (Arulkumaran et al, Gibb 1992)[34]

Ingemarsson et al 1993[11] reported that even in 5 to 10 % of low risk cases FH changes were found.

Ingemarsson in 1993[11] reported that even though fetal condition was satisfactory on admission, fetal distress that developed several hours later cannot be predicted by admission test.

AT has high predictive value for fetal well being (98.7%) and high specificity but has low positive predictive value of 30% and it also has low sensitivity of 23.5%.

Experience in interpretation of CTG is very important. In occipito posterior or occipito transverse position during second stage of labor bradycardia between 100 – 119 bpm in the absence of other changes is due to head compression. (Youna & Weinstenin 1976).[29]

Saling in Berlin reported the use of fetal blood sampling (FBS) to study fetal pH in 1966.

Kuble and colleagues[45] in --1969 studied the association of FHR patterns with fetal scalp blood pH. In reactive CTG pattern, frequently the pH value is > 7.25 in 94 %. When FHR deceleration was present pH values more than 7.25 is 80 %.

Minor changes in fetal blood gases do not produce changes in FHR (Wood et al 1979)[54]

Clark et al in 1982[5] notes that in non reactive CTG, if scalp stimulation by Allie's clamp (or during FBS) cause acceleration of at least 15 bpm , The pH was almost always 7.22 or greater. Whereas a positive response is reassuring, negative response is not diagnostic of fetal acidosis (Spencer JAD 1991 [20])

If there is acceleration by vibro acoustic fetal stimulation then the pH is usually 7. 22 (Ingemarsson & Arulkumaran (1989)[12] Again negative response is not diagnostic of fetal distress. Whereas in positive response the chance of developing acidosis is only 0.4 %, in negative response the chance of developing acidosis is almost ten times greater (44.4 %)

Computerized evaluation of FHR patterns minimize the inter observer & inraobserver variation. The most extensively used and evaluated system is that which Dawes and associates in Oxford have developed in 1978. The most recent version of this system is oxford Soinicaid monitor.

Introduction of digital system for distant heart rate monitoring and subsequent rapid transmission by telephones to the hospital in less than 30 seconds reduces hospital admission and improves patient's satisfaction (Moore & Still 1990).

Recently Kinetocardiotocography (KCTG) was introduced by Schiesse & Muller. KCTG registers fetal movements by itself and quantifies the fetal movements on CTG. It eliminates human error in perceiving fetal movements.

Walker in 1954[31] found that thick meconium was associated with low umbilical venous pO_2 . If meconium is associated with abnormal FHR pattern, there is a higher risk of acidosis (Miller et al 1975, Steer et al 1989).[16] There is no association between meconium and acidosis if FHR is normal. (Miller et al 1975, Shaw, Clark 1988, Bakes et al 1992).[16]

Addition of E.C.G waveform with CTG reduces caesarean rate to 4.5 %, from 10.05 % when caesarean is dependant upon CTG alone, Whereas the predictive value of abnormal E.C.G in detecting acidosis is 63 % and for CTG alone it is 50 % (Westgate et al 1992).[30]

In predicting neurological damage the assessment of scalp blood lactate is more sensitive than scalp pH since blood lactate is not affected by CO_2 levels (Myser et. et al 1981)[32]. So in predicting neonatal outcome , lactate measurement is more important than scalp pH.

Techniques for the measurement of absorption spectra of both visible range (using pulse oxymeter) and near infra red (NIRG) range using infra red spectroscopy light reflected from fetal scalp are currently being investigated as a means of detecting changes in the degree of O_2 saturation, SaO2 of Hb and cytochrome oxidase in scalp tissues. (Doyle 1994. Johnson et al 1994).[33]

The condition of baby at birth depend on its reserve to tolerate hypoxia as well as on the degree and duration of hypoxia. In determining the neonatal outcome and long term prognosis other factors including.

1. Duration of acidosis,
2. Perfusion of vital organs especially brain,
3. Coexisting PO_2 level,
4. Trauma such as intra cranial hemorrhage,
5. Infection

are equally important. (Chiswik & James 1979, Myer et al 1981).[32]

PATHOPHYSIOLOGICAL BASIS OF

FETAL HYPOXIA

Oxygen supply to the fetus depends principally on

1. Adequacy of uterine perfusion,
2. Placental gas transfer,
3. Fetal circulation.

The fetus has the following physiological advantages to tolerate mild to moderate hypoxia.

1. HbF concentration is higher than in the adults.
2. HbF has higher affinity for O_2
3. HbF delivers more O_2 at tissue level

When the O_2 tension in umbilical vein becomes severe, fetal tissue hypoxia occurs. The fetus switches to anaerobic metabolism leading to accumulation of lactic acid and a consequent fall in tissue and blood pH.

The fetal response to hypoxia depends on the

1. Acuteness of onset and its severity
2. Duration of hypoxia

Acute fall in O_2 tension stimulates fetal chemoreceptor resulting in transient bradycardia. However, if hypoxia is insufficient to produce acidosis, catecholamine released from adrenal gland causes FHR to return back to normal. The increase in sympathetic stimulation overcomes the effect of vagal stimulation and also produces vasoconstriction. So cardiac output is more directed towards the brain rather than peripheral circulation.

The increase in cardiac output is brought about by increase in fetal heart rate, which is followed by decrease in heart rate or loss of baseline variability (BLV) and absence of acceleration due to hypoxia of the brain stem centers. Further hypoxia damages myocardium.

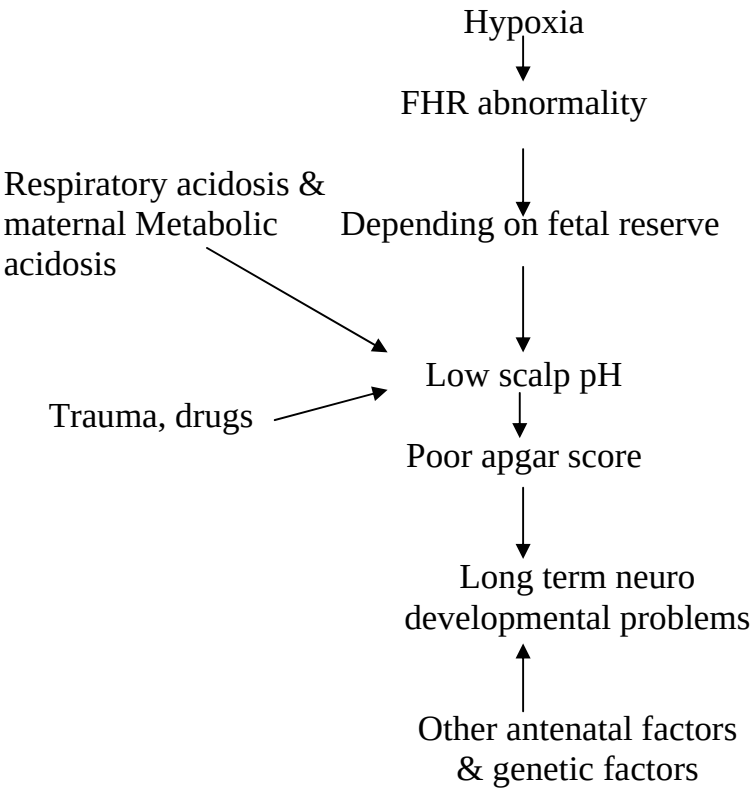
Development of acidosis depends on the reserve capacity of feto placental unit.

With poor placental transfer of gases, CO_2 accumulates in the fetus (respiratory acidosis). The increase in pCO_2 causes increase in fetal hydrogen ion concentration and lowering of pH. This form of acidosis is easily reversed. INGERMARSSON AND ARULKUMARAN 1986.[10] Further prolongation of hypoxia causes anaerobic glycolysis and production of lactate and pyruvate.

Initially the production of metabolic acidosis is buffered and it is the consumption of buffer to restore pH towards normal, that leads to the development of base deficit. A buffer base (bicarbonate and protein) of <34 Mmol/L or a base deficit >11 Meq /L indicates significant metabolic acidosis Lowe 1998. Johnson et al 1990.[50]

Metabolic acidosis cannot be reversed until O_2 delivery to fetal tissue is reestablished. Monitoring of fetal acid base status improves the diagnostic accuracy of FHR monitoring.

RELATIONSHIP BETWEEN HYPOXIA AND NEONATAL OUTCOME



In chronic placental insufficiency, fetal arterial O_2 saturation slowly falls as a result of limited perfusion to the placental bed. The reduction in oxygen delivery to the fetus is slow and does not exceed that of fetal adaptation. Parallel actions of both sympathetic and parasympathetic keep the FHR normal. Other adaptations are, increase in PCV and reduction in metabolism. If reduction in fetal O_2 is severe, it leads to FHR deceleration with uterine contraction. So delivery by caesarean section is imminent.

CONTROL OF FETAL HEART RATE

FHR at any time is under the influence of both sympathetic (increase) and parasympathetic (decrease) systems.

Increase in B.P. stimulates baroreceptor and causes decrease in FHR by increased vagal tone. Decrease in BP increases FHR by inhibition of vagal tone.

Chemoreceptor responds to changes in partial pressure of dissolved oxygen in fetal blood.

Adrenaline and nor adrenaline from fetal adrenal gland have ionotropic and chronotropic effects on the heart muscle.

METHODS OF INTRAPARTUM FETAL MONITORING

1. INTERMITTENT AUSCULTATION

Pinards fetoscope or a Doppler ultrasound device is used for intermittent auscultation.

A.C.O.G 1995 suggests auscultation of FH as follows

Evaluation	Low risk	High risk
1st stage of labor	30mts	15mts
2nd stage	15mts	5mts

It is to be performed after a contraction and for 15 seconds. It is unable to predict the long-term heart rate variability or the relationship of FHR to uterine contraction.

2. PRESENCE OF MECONIUM IN LABOR

If presence of meconium is associated with abnormal FHR pattern, the chance of developing acidosis is higher

3. ELECTRONIC FHR MONITORING

1. Internal method using scalp electrode
2. External method using ultrasound transducer.

In low risk patient, with duration of labor less than 5 hours intermittent auscultation is as effective as EFM. (Dublin study 1985.)[15]

4. FETAL BLOOD SAMPLING (FBS)

To confirm acidosis in abnormal FHR pattern, the pH and base deficit values should be assessed by taking fetal scalp blood.

5. FETAL SCALP STIMULATION TEST

Acceleration of FHR during FBS or on pinching the scalp using Allie's tissue forceps indicates fetus is non acidotic. [Clark and Paul 1985][5]

6. FETAL ACOUSTIC STIMULATION TEST (FAST)

FHR acceleration in response to vibro acoustic stimulation indicates the well being of fetus. Compared to scalp pH, it is less invasive and no chance of introducing any infection.

7.**KINETOCARDIOTOCOGRAPHY ACTOCARDIOGRAPHY)**

It simultaneously measures FHR, uterine activity and fetal movement. Non reactive NST with fetal movement had better perinatal outcome than non-reactive NST with no fetal movement.

8. FETAL PULSE OXIMETRY

This technique measures arterial blood oxygen saturation on a continuous basis

9.FETAL E.C.G.

Hypoxia causes an elevation of ST segment and T wave due to catecholamine surge, beta adreno receptor activation and myocardial glycogenolysis

10. FETAL SCALP BLOOD LACTATE ESTIMATION

5µl of capillary blood are tested in an ACCUSPORT LACTATE METER. If there is any non-reassuring FHR pattern, fetal scalp blood lactate estimation may be done. Cut off level is 4.2 mmol/l

11.FUTURE TRENDS IN INTRA PARTUM FETAL MONITORING

1. Continuous tissue pH monitoring
2. Per cutaneous measurement of pO_2, pCO_2, pH
3. Near Infra Red spectroscopy.

TECHNICAL ASPECTS OF FETAL HEART RATE MONITORING –CTG

Cardiotograph is used to produce continuous recording of FHR and uterine contraction.

CTG can measure the FHR and uterine contraction via external transducer using Doppler ultrasound and Tocodynamometer (strain gauge attached to a belt)

LIMITATION: -

The recording is attenuated if mother is obese or the transducer is poorly applied.

If the transducer moves, the monitor will auto correlate the regular signal derived from larger maternal vessels.

PREDICTIVE VALUE OF CTG IN LABOR

In reactive CTG prior to delivery, 99% of fetuses have 5mts apgar >7, provided the amniotic fluid is not meconium stained.

In 80% of suspicious FHR and 50% of ominous patterns are not associated with acidosis.

So non reactive FHR pattern has a high negative predictive value of more than 90% and positive predictive value of 30%(Grant 1989)[27]

To reduce the number of false positive and thereby unnecessary intervention, FIGO recommends fetal scalp blood pH estimation in non reactive CTG (FIGO GUIDELINES 1987).[28]

INDICATIONS FOR EFM

1. Medical disorder complicating pregnancy .(LIKE PIH , GDM , RENAL DISEASES , COLLAGEN DISEASES)
2. IUGR
3. Post term pregnancy
4. Oligohydramnios
5. Prolonged labor
6. Prolonged rupture of membrane
7. Regional analgesia
8. Thick meconium stained liquor
9. Abnormal Admission Test

Every fetus has a potential risk of developing intrapartum hypoxia or birth injury and optimal outcome can be concluded only at the end of labor or occasionally much later. Therefore every fetus deserves intra partum monitoring.

CTG TERMINOLOGIES

ADMISSION TEST (A.T)

A short 15-20mts external electronic fetal monitor on admission in labor is Admission Test. (Ingemarsson et al 1986)[11] In a reactive admission test, the chances of fetal hypoxia due to causes other than acute events are unlikely if delivery occurs with in 5 hours.

1. Tachycardia – sustained rise in heart rate >160 bpm

   Mild – FHR between 160-180 beat/mt

   Severe - >180 bpm

2. Bradycardia – a sustained fall in heart rate < 100 beats/mt

   Mild – FHR between 100 – 119 bpm

   Moderate – 80 – 100 bpm

   Severe - <80 bpm

   Prolonged

   bradycarida <80bpm> 10mts

3. FLAT trace – loss of normal long term variability > 35mts.

4. Acceleration – rise in heart rate ≥15bpm ≥ 15 seconds

ABSENCE OF ACCELERATION

There is about a 50% of chance of acidosis if there is no acceleration (clark & colleague 1984)[35] for > 40 mts.

DECREASED BASELINE VARIABLITY (BLV)

BLV is assessed from the bandwidth or amplitude of the fluctuation of FHR measured by the difference between the highest and lowest point of trace in any one minute segment.

Normal variability – 10-25 bpm

Absent variability - <5 bpm

Reduced variability – 5-10 bpm

Increased or saltatory pattern >25 bpm

It is the single most reliable sign of fetal compromise

SINUSOIDAL PATTERN: -normal neural control of heart is lost.

The features are:

1. Regular oscillation above and below a normal baseline rate (110 – 150 bpm)

2. Amplitude of 5 – 15 bpm

3. Frequency of 2 – 5 cycles/mt of long term variability

4. Absent short term variability

5. Absence of acceleration (Young et al 1980)[55]

DECELERATION – fall in FHR ≥15bpm ≥15 seconds

i. Early – deceleration where the lowest point of FHR occurs within 20 seconds of peak of contraction. It is due to head compression and mediated by vagal tone.

ii. Late – deceleration where the lowest point of FHR occurs more than 20 seconds after the peak of contraction. It is due to reduction in retro placental reserve and mediated by chemoreceptors.

iii. Variable – all deceleration not fitting into the definition of early or late deceleration and mediated by baroreceptors.

INTRAPARTUM CTG SCORING SYSTEM

(krebs et al 1979)

Pattern	0	1	2
1.Baseline rate bpm	<100 >180	100-119 161-119	120-160
2.Baseline variability	<3	3-5	6-25
3.frequency/mt	<3	3-6	>6
4.acceleration/30mts	0	1-4 sporadic	>5
5.deceleration	Late, severe	Mild, moderate, variable	None, early

Scoring value	scalp pH
8 – 10	>7.25
6 – 7	7.2 – 7.25
≤5	<7.2

The fetus does not become acidotic as soon as the FHR becomes abnormal. Fleischer el al[36] has shown that a well grown fetus can cope with a hypoxic stress for as long as 90 minutes before pH of fetal blood start to fall (in the absence of any acute event).

FETAL SCALP BLOOD pH ESTIMATION

Fetal scalp blood pH estimation was introduced by Saling in 1962. In reactive CTG, chances of fetal acidosis are extremely rare (Ingemarsson 1986)[10] but non reactive CTG is not always associated with fetal acidosis. Fetal pH is one parameter by which fetal tolerance to hypoxia is assessed.

PREDICTIVE VALUE

For fetal asphyxia, if we take 5-minute apgar < 7 as the cut off point, FBS has a high negative predictive value, but only modest positive predictive value.

False positive low pH <7.2 arises from the following situation

(i) Uterine hypertonus

(ii) Maternal hypotension

(iii) Maternal ketosis (Roversi et al 1975)due to prolonged labor

(iv) Maternal hyperventilation

FBS need not be done in the following situations to avoid wasting precious time.

1. Abnormal FHR pattern with thick meconium stained liquor in early labor
2. Prolonged bradycardia <80bpm >10mts

3. Sinusoidal pattern without acceleration

If pH >7.25 – labor observed

7.2 – 7.25 – FBS to be repeated in 30mts

<7 – immediate termination

In breech presentation, FBS can be taken from the buttocks and interpretation of pH values are same as in cephalic. Since we did not include breech presentation in this study, this was not in this study

Complications (FBS)

1. Scalp infection including abscess formation (< 1% incidence)
2. Scalp hematoma

A single fetal scalp blood pH assessment will give only limited information. Significant hypoxia will produce a progressive acidaemia that will only be recognized by repeated measurement.

AIM OF THE STUDY

This study aims at,

1. Assessing the comparative efficacy of CTG and fetal scalp blood pH in the detection of fetal jeopardy.
2. Assessing the predictability of both these methods for routine screening.

MATERIAL AND METHODS

This is a prospective study conducted in Government Kasthuriba Gandhi Hospital Chennai-5 attached to Madras Medical College during the period October 2004 to August 2005.

Hundred patients in active phase of labour were selected by the inclusion and exclusion criteria for this study.

For all the hundred patients, CTG were taken in the acitive phase of labour. If the CTG was non reactive. I have repeated the CTG after left lateral position, correcting hydration, amnioinfusion etc. if CTG was still non reactive, scalp blood pH was done.

INCLUSION CRITERIA

1. Suspicious CTG in admission test (AT)
2. Meconium stained liquor
3. Medical disorder complicating pregnancy.(PIH ,GDM, renal disease collagen vascular disease etc.)
4. Oligohydramnios
5. IUGR
6. Prolonged rupture of membrane

7. Where there is no contraindication for vaginal delivery
8. Previous good obstetric history

EXCLUSION CRITRIA

1. Normal CTG in AT
2. Imminent labor
3. Contracted pelvis
4. Previous caesarean section for recurrent indication
5. Acute fetal distress like cord prolapse, abruption, eclampsia. etc.,,

SCALP PH

INCLUSION CRITERIA

In all abnormal CTG patterns

EXCLUSION CRITERIA

1. Thick meconium stained liquor or ominous trace in early labor
2. Reassuring FHR pattern
3. Imminent labor
4. Prolonged and persistent bradycardia
5. Persistent failure to progress

Apparatus used for cardiotocogram in this study was '**WAKCLING AFM 210**' cardiotocogram

CARDIOTOCOGRAM CONTAINS

1. Ultrasound transducer
2. Transducer belt and buckle set
3. Tocodynamometer
4. Chart paper roll
5. Ultrasound coupling gel
6. A.C. line card

FOR FETAL SCALP PH

1. Focusing lamp
2. Speculum
3. Sponge holder
4. 11 blade with BP handle
5. Perineal sheet
6. Heparinised capillary tube
7. Calibrated ph paper/ph meter

FETAL BLOOD SAMPLING (FBS)

Pre requisites: Ruptured membrane,

Cervical dilatation 2-3cm

Station of head –2 or below

METHOD:

After cleaning and draping the perineum, speculum is introduced; fetal scalp skin was wiped with sterile cotton swab and an incision was made by 11 blade through the skin to 2mm depth. The blood was collected into the heparinised capillary tube. pH was measured using calibrated pH paper/ pH meter. Minimal acceptable amount of blood to be collected was 40micro liter. That **is 2 inches in the tube.**

RESULTS

This study was carried out at Government Kasturba Gandhi Hospital, during the period of October 2004 – August 2005. 100 women were included in this study and the outcome analyzed using various parameters. The results are subjected to statistical analysis using the *t* test and chi- square test.

Table 1

n=100

OBSTETRIC CHARECTERISTICS

Sl. No.	Obstetric charecteristics	No of Cases	Percentage
1	PIH	36	36%
2	POST EDD GESTATION	27	27%
3	PROLONGED RUPTURE OF MEMBRANCE	3	3%
4	GDM	7	7%
5	HEART DISEASE	4	4%
6	OLIGOHYDRAMNIOS	6	6%
7	PREVIOUS LSCS	4	4%
8	ANEMIA	10	10%
9	BOH	3	3%

PIH and post EDD gestation constituted 63% of study population

Table 2

AGE DISTRIBUTION

n=100

Age Group	No of cases	Percentage
</=18YEARS	6	6%
19 - 24YEARS	56	56%
25 - 29	30	30%
30-34	5	5%
>35	3	3%

Majorty of the patients(56%)were 19 - 24 Years of age

Table 3
PARITY
n=100

Parity	Number of Patients	Percentage
PRIMI GRAVIDA	59	59%
2nd GRAVIDA	23	23%
3rd GRAVIDA	12	12%
4th GRAVIDA	6	6%

In this study 59% were primi gravida

Table 4
COLOUR OF LIQUOR
n=10

Colour of liqour	Number of cases	Percentage
CLEAR	60	60%
MECONIUM	40	40%

In this study in 60% of cases colour of liquor is clear.

Colour of liquor					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Clear	60	60.0	60.0	60.0
	Meconium	40	40.0	40.0	100.0
	Total	100	100.0	100.0	

Table 5
CARDIOTOCOGRAM FINDINGS
n=100

CTG changes	Number of Patients	Percentage
REACTIVE	28	28%
NON REACTIVE	72	72%

In this stuty 28% of cases show reactive CTG

Table 6
n=72

Nature of abnormality	Number of cases	Percentage
1. PERSISTANT TACHYCARDIA >180	18	25%
2. PERSISTANT BRADYCARDIA <100	8	11.10%
3.VARIABLE DECELERATION		
MODERATE	15	20.83%
SEVERE	12	16.67%
SEVERE WITH REDUCED BLV	8	11.10%
4. LATE DECELERATION	4	5.56%
5.LATE DECELERATION WITH REDUCED BLV	6	8.30%
SINUSOIDAL PATTERN	1	1.39%

In 25%of cases of non reactive CTG, persistant tachycardia is present
(BLV - Base line variability) 48.61% of cases had variable deceleration of varying severity and sinusoidal pattern was seen in 1.39%.

Frequency Table

CTG Abnormalities					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Late deceleration	4	4.0	4.0	4.0
	Late deceleration with reduced BLV	6	6.0	6.0	10.0
	Persistant Bradycardia < 100	8	8.0	8.0	18.0
	Persistant Tachycardia >180	18	18.0	18.0	36.0
	REACTIVE	28	28.0	28.0	64.0
	Sinusoidal	1	1.0	1.0	65.0
	Variable deceleration Moderate	15	15.0	15.0	80.0
	Variable deceleration Severe	12	12.0	12.0	92.0
	Variable deceleration with reduced BLV	8	8.0	8.0	100.0
	Total	100	100.0	100.0	

TABLE NO: 7

n=100 BIRTH WEIGHT

Birth wt	Number of cases	Percentage
2-2.5kg	35	35%
2.5-3kg	39	39%
3-3.5kg	24	24%
>3.5kg	2	2%

39% of cases had birth weight of 2.5-3 kg. There is no correlation between birth weight, CTG abnormalities and neonatal outcome.

Table 8

N=100

MODE OF DELIVERY IN NORMAL AND ABNORMAL CTG PATTERN

Mode of delivery	CTG pattern			
	Normal		Abnormal	
	No of cases	Percentage	No of cases	Percentages
LN	25	89.29%	31	43.06%
VACUUM	1	3.57%	2	2.78%
CAESAREAN	2	7.14%	39	54.17%

In normal CTG No of vaginal delivery -92.86%

Caesarean section -7.14%

Abnormal CTG vaginal delivery - 45.84%

caesarean section - 54.17%

CTG Abnormalities * Mode of Deliviery Crosstabulation
Count

		Mode of Deliviery				Total
		Forceps	LN	LSCS	Vaccum	
CTG Abnormalities	Non Reactive	1	32	38	1	72
	Reactive	1	25	2		28
Total		2	57	40	1	100

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	18.477(a)	3	.000
N of Valid Cases	100		

a 4 cells (50.0%) have expected count less than 5. The minimum expected count is .28.

Table 9 n=72

RELATION SHIP BETWEEN CTG ABNORMALITIES SCALP pH AND MODE OF DELIVERY AND APGAR AND NEONATAL COMPLN

CTG Abnormalities	Scalp pH			Mode of Delivery			Apgar		Neonatal				
	Nonacidotic>7.25	preacidotic 7.2-7.25	Acidoic <7.2	LN	Vacuum	Caesaream	No asphyxia>7	Mode rate4-7	severe <4	Complication RDS	MAS	Candn of baby at dischareg	
PERSISTANT TACHYCARDIA>180 N=18	16 (88.9%)	2 (11.1%)	0	13 (72.20%)	0	5 (27.8%)	14	4		2	1	GOOD	NIL
PERSISTANT BRADYCARDIA N = 8	4 (50%)	1 (12.5%)	3 (37.54%)	4 (50%)	1(12.50%)	3 (37.50%)	5	3		3		GOOD	NIL
VARIABLEDECELERATION													
MODERATE N=15	12 80%	0	3 20%	6 40%	0	9 60%	11	4		2	1	GOOD	NIL
SEVERE N=12	7 58.30%	2 16.67%	3 25%	4 33.37%	1 8%	7 58.30%	9	2	1	2	1	GOOD	1 DIED
SEVERE WITH REDUCED BLV N=8	4 50%	2 25%	2 25%	1 12.50%	0	7 87.50%	3	5		3		GOOD	NIL
LATE DECELARATION n =4	2 50%	1 25%	1 25%	2 50%	0	2 50%	3	1		3		OTHERS GOOD	1 DIED
LATE DECELARATION BLV n =6	2 33.30%	2 33.30%	2 33.30%	1 16.60%	0	5 83.30%	3	2	1	3			
SINUSOIDAL n =1			1			1			1	1		GOOD	NIL
TOTAL	**47**	**10**	**15**	**31**	**2**	**39**							
NORMAL CTG				**25**	**1**	**2**	**25**	**3**		**1**		**1GOOD**	**NIL**

TABLE-10
n =28

NUMBER OF FETAL DISTRESS IN NORMAL CTG

APGAR	Number of cases	Percentage
> 7 No Asphyxia	25	89.29%
4-7 Moderate	3	10.71%
< 4 Severe	0	0

Crosstabs

CTG Abnormalities * Apgar Crosstabulation
Count

		Apgar			Total
		Severe Asphyxia	Moderate Asphyxia	No Asphyxia	
CTG Abnormalities	Non Reactive	3	20	49	72
	Reactive		4	24	28
Total		3	24	73	100

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	3.557(a)	2	.169
N of Valid Cases	100		

a 2 cells (33.3%) have expected count less than 5. The minimum expected count is .84.

TABLE – 11

NUMBER OF FETAL DISTRESS IN ABNORMAL CTG PATTERN

n = 72

APGAR	Number of cases	Percentage
> 7 NO ASPHYXIA	48	66.67%
4-7 MODERATE	21	29.17%
4 SEVERE	3	4.17%

In reactive CTG apgar <7 was seen in 10.71% of cases. In non reactive CTG apgar <7 was seen in 33.3% of cases

Relationship between obstetric charecteristics, abnormal CTG, APGAR and neonatal out come

Table 12

Obstetric Characteristics	Normal FHR Pattern	Abnormal FHR Pattern	No Asphyxa >7	Moderate 4-7	Severe <4	Neonatal complication		Baby's condn. at disch	Perinatal death
						RDS	MAS		
1. PIH n=36	14	22	25	9	1	11	1	GOOD	1 DIED
2. POST EDD GEST n=27	4	23	20	6	1	5		GOOD	NIL
3. PROLONGED RUPTURE OF MEMB n=3	1	2	3	0	0	NIL		GOOD	NIL
4. GDM n=7	2	5	6	1	0	1/ Hypoglyc 1		GOOD	NIL
5. HEART DISEASE n=4	1	3	3	1	0	RDS -1		GOOD	NIL
6. OLIGO HYDRAMNIOS n=6	1	5	4	2	0	1	1	GOOD	1 DIED
7. PREVIOUS LSCS n=4	1	3	3	1	0	1	1	GOOD	NIL
8. ANAEMIA n=10	3	7	8	2	0	1	1	GOOD	NIL
9. BOH n=3	1	2	1	2	0	2	0	GOOD	NIL
	28	72	73	24	3				

Though a careful intrapartum monitoring helps in reducing birth asphyxia obstetric characteristic influence in some of the cases (eg) one baby in severe PIH died 5 mts after delivery with thick meconum with MAS. Other one died in case of oligohydramnios with <7.25 scalp pH, immediate caesarian section done. Apgar 6/10 at 5mts. Died after two days with hypoxic ischaemic encephalopathy

Table 13
n=72

SCALP BLOOD pH AND NEONATAL OUTCOME

Ph Values	**No of cases**	**Apgar**			**Neonatal complication**		
		>7	4-7	<4	RDS	MAS	HYPOGLYC
1. <7.2	15	3	9	3	10	2	
2. 7.2 - 7.25	10	0	10	0	7	1	
3. >7.25	47	45	2	0	2	1	1

Table 14
n=47

NUMBER OE FETAL DISTRESS IN NORMAL pH

Apgar	No of cases	percentages
NO ASPHYXIA >7	45	95.74%
MODERATE 4-7	2	4.26%
SEVERE <4	0	0

Scalp pH * Apgar Crosstabulation
Count

		Apgar			Total
		Severe Asphyxia	Moderate Asphyxia	No Asphyxia	
Scalp pH	Acidotic	3	10	6	19
	Pre Acidotic		5	3	8
	Non Acidotic		5	40	45
Total		3	20	49	72

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	28.248(a)	4	.000

a 4 cells (44.4%) have expected count less than 5. The minimum expected count is .33.

Table 15
n=25

NUMBER OF FETAL DISTRESS IN ABNORMAL pH

Apgar	No of cases	percentages
No asphyxia >7	3	12%
moderate 4-7	19	76%
severe <4	3	12%

In normal pH Apgar < 7 = 4.26% of cases

In acidotic pH Apgar < 7 = 88% of cases

Table 16
Relationship between scalp pH and mode of delivery

S No	Scalp pH	Labour natural	Vacuum	Caesarean delievry
1	>7.25 n=47	23	1	23
2	7.2 - 7.25 n=10	3	0	7
3	<7.2 n=15	5	1	9

Crosstabs

Scalp pH * Mode of Deliviery Crosstabulation
Count

		Mode of Deliviery				Total
		Forceps	LN	LSCS	Vaccum	
Scalp pH	Acidotic	1	2	15	1	19
	Pre Acidotic		2	6		8
	Non Acidotic		28	17		45
Total		1	32	38	1	72

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	19.510(a)	6	.003
N of Valid Cases	72		

a 8 cells (66.7%) have expected count less than 5. The minimum expected count is .11.

Table 17
PERCENTAGES

Scalp pH	Vaginal delivery		Caesarean delivery	
	No of cases	**percentage**	**No of cases**	**percentage**
>7.25 n=47	24	51.06%	23	48.94%
<7.25 n=25	9	36%	16	64%

In Normal pH. Vaginal delivery – 51.06%
Caesarean - 48.94 %
Acidotic pH Vaginal - 36 %
Caesarean - 64 %
All Acidotic pH mothers delivered within 30 mts of fetal blood sampling

Table 18
Colour of liquor and neonatal outcome

Colour of liquor	APGAR		
	No Asphyxia >7	Moderate 4-7	Severe
Clear n = 60	48 (80%)	11	1
Meconium N=40	26 (65%)	13	1

Crosstabs

Colour of liquor * Apgar Crosstabulation
Count

		Apgar			Total
		Severe Asphyxia	Moderate Asphyxia	No Asphyxia	
Colour of liquor	Clear	2	11	47	60
	Meconium	1	13	26	40
Total		3	24	73	100

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.647(a)	2	.266
N of Valid Cases	100		

A 2 cells (33.3%) have expected count less than 5. The minimum expected count is 1.20.

Table 19

Relationship between Colour of liquor and scalp pH

Colour of liquor	Pre Acidotic 7.2-7.25		Acidotic <7.2	
	NO. of cases	Percentage	NO. of cases	Percentage
Clear n=60	**6**	**10%**	**8**	**14.67%**
Meconium N=40	**4**	**10 %**	**7**	**17.5%**

Table 20

Relationship between Colour of liquor and Birth Aspyxia

Apgar <7

Colour of liquor	Apgar <7		Apgar > 7	
	NO. of cases	Percentage	NO. of cases	Percentage
Clear n=60	**12**	**20%**	**48**	**80%**
Meconium n=40	**14**	**35%**	**26**	**65%**

Table : 21
Relationship between colour of liquor and FHR pattern

Colour of Liquor	Reactive CTG		Non reactive CTG	
Clear n=60	11	18.33%	49	81.6%
Meconium N=40	17	42.5%	23	57.5%

Of these 100 cases 60 cases were clear liquor and 40 cases were thick meconium stained liquor. In clear liquor apgar < 7 was present In 20 % of cases. In Meconium Stained Liquor Apgar < 7 was seen in 35 % of cases. Acidotic pH was present in 14 .67 % of clear liquor cases and 20 % of thick meconium stained liquor. There was not so much difference between these two Groups. Meconium stained liquor is significant if it is associated with abnormal CTG.

Table 22

Predictive value of Abnormal CTG

Test	Preinatal outcome		Total
	Asphyxia Apgar <7	No Asphyxia Apgar >7	
Abnormal CTG	24(a)	48(b)	72(a+b)
Normal CTG	3(c)	25(d)	28(c+d)
Total	27(a+c)	73(b+d)	

1. Sensitivity = a/a+c X 100 = 88.89 %

2. Specificity = d/ b+d x100 = 34.25%

3. Positive predictive Value a/a+b X 100 = 33.33%

4. Negative predict value d/ c+d X 100 = 89.29 %

This study shows that abnormal CTG is associated with predictive value of 89.29% But low positive predictive value (33.33 %). This means that a normal trace indicates that the fetus is not hypoxic. Abnormal CTG is associated with large number of false positive.

Table 23
Predictive value of Scalp pH

	APGAR		
PH Value	**>7**	**<7**	**Total**
> 7.25 n = 47	**45 (a)**	**2 (b)**	**47 (a+b)**
> 7.25 n=25	3 ©	22 (d)	25 (c+d)

1. Sensitivity = a/a+c X 100 = 93.75 %
2. Specificity = d/ b+d x100 = 91.67%
3. Positive predictive Value a/a+b X 100 = 95.74%
4. Negative predictive value d/ c+d X 100 = 88 %

Considering the normal perinatal outcome ie 5 mts Apgar >7 as the end Point
fetal blood sampling has a
Positive predictive Value = 95.74 %
Negative predictive Value = 88 %

DISCUSSION

This prospective study was conducted at Government Kasturba Gandhi Hospital, Chennai-5 attached to Madras Medical College, to assess the efficacy of Cardio Tocogram and scalp blood pH in the detection of fetal jeopardy during the period of October 2004 to August 2005.

In this study all the 100 patients were in the active phase of labor at the time of admission. This study is discussed as follows.

Among 100 patients, CTG is reactive in 28% of cases and non-reactive in 72% of cases.

Among the non reactive CTG, persistent tachycardia was present in 25% of cases.

In reactive CTG, FBS was not done . FBS was done in all non reactive CTG cases.

In non reactive CTG, acidosis was present in 34.72% of cases. As per Beard et al., 1971[4] Tejani et al., 1975 study , acidosis was present in 50% of cases of non reactive CTG.

In reactive CTG of 28 cases apgar <7 was present in 10.71% of cases. In non reactive CTG apgar <7 was present in 34.7% of cases.

Results of AT conducted at KANDANG KERABU hospital in low risk cases . IN REACTIVE CTG Apgar < 7 was present in 1.4% and in non reactive CTG apgar < 7 was present in 50% of cases. As per Arul Kumaran and Gibb 1990 [34] study, in non reactive CTG apgar <7 was present in 40% of cases. In reactive CTG apgar <7 was seen in 1.4% of cases.

As per our study, one baby of mother with PIH and another with Oligohydramnios died . As per kjellmer 1998 development of fetal acidosis and fetal death are not limited to high risk pregnancies . Fetal acidosis and death was also present in low risk cases also.

Among 72 non reactive CTG cases, acidosis was present in 25 cases. Of these 25 cases , 2 babies were died. One baby with PIH mother (case no.21) with scalp pH 7.14 caesarean section was done immediately, baby died after 5 minutes with MAS and another baby with Oligohydramnios mother (case no. 38) with scalp pH 7.23. Even though caesarean section was done immediately baby was died after 2 days with hypoxic ischaemic encepalopathy .

Of the 72 women with non reactive CTG, non acidotic pH of >7.25 was seen in 67.25% of cases. As per Kuble and Colleagues[45] 1969 study , in non reactive CTG pH >7.25 was seen in 80 % of cases.

As per our study Apgar < 7 was present in 4.26% of normal pH cases and in 88% of acidotic pH cases (pH <7.25). As per sykes et al [28] 1982 study Apgar <7 was present in 14.3% of acidotic pH cases and 1.4% of normal pH cases.

In Acidotic pH 64% of cases had caesaren delivery. But in normal pH 48.94% had caesarean delivery. Among non reactive CTG cases caesarean section was done in 54.17% of cases. Addition of FBS has reduced the rate of caesarean section. As per westgate et al., 1992 [30] study, addition of fetal blood sampling reduces caesarean section rate from 10.05% (when CTG alone is used) to 4.5% (when both CTG & FBS were done).

As per our study there is no relationship between colour of liquor and fetal heart rate changes and scalp pH changes. According to Miller et al (1975),[16] Shaw et al(1992) when FHR pattern was normal, there was no difference in scalp pH and cord blood pH or

neonatal outcome between those fetuses who had or had not passed meconium.

Among 72 cases of abnormal CTG, only 2 babies were died. All other babies were discharged in good condition. Because of CTG monitoring perinatal mortality is reduced to 2% . As per Arulkumaran and Gibb 1990 [34] Perinatal death was redueced from 14.5% to 7.9% when CTG was used for fetal monitoring.

In abnormal CTG 3 babies had meconium aspiration syndrome (MAS) and 22 had mild Respiratory Distress Syndrome. Other than 2 , all babies were discharged in good condition.

A study by Grant[27] in 1989 evaluated the value of abnormal intra partum CTG and concluded that though it has a high negative predictive value of 90 %, the positive predictive value is only 30%. In our study also positive predictive value is 33.33%and negative predictive value is 89.29%

This means that a normal trace indicates a fetus that is not hypoxic but an abnormal trace is associated with a large number of false positive.

Sykes et al (1982)[28] in a study from Oxford showed that if the end point is taken as apgar of less than 7 , FBS has high negative predictive value and only modest positive predictive value . As per his study only one in 5 babies with 5 minute apgar less than 7 had severe acidosis (pH < 7.2). Similarly only one in 7 babies with severe acidosis had 5 minute apgar <7 confirming that hypoxemia and acidosis are only one of the causes of low Apgar at birth.

According to Beard et al (1971),[4] though the positive predictive value is higher in high risk cases, it is low if it is applied to all cases.

But in our study both positive predictive value (95.74%) and negative predictive value (88%) are higher as we have taken only the high risk cases.

SUMMARY

- This present prospective study " Assessing the Efficacy of Intrapartum fetal monitoring and fetal scalp blood pH in predicting fetal jeopardy " was carried out at Govt. Kasturba Gandhi Hospital Madras Medical College, Chennai during the period October 2004 – August 2005.

- Hundred patients in active phase of labour were selected with inclusion and exclusion criterias.

- In hundred cases PIH and post EDD gestation constituted 63% of study population.

- Majority of the study group (56%) were between 19-24 years of age .

- 59% of this study group were Primi gravidas.

- 60% of the study group had clear liquor.

- CTG is reactive in 28%f cases and non reactive in 72% of cases.

- In 72% of non reactive CTG changes persistant Tachycardia > 180 was present in 25% of cases. variable deceleration of different severity was seen in 48.61% of cases. Sinusoidal pattern was seen in 1.39% of cases.

- 39% of study group had birth weight of 2.5-3 Kg. But there is no correlation between Birth weight, CTG abnormalities and neonatal outcome.

- In reactive CTG, 92.86% were delivered vaginally, 7.14% were delivered by caesarean section . In non reactive CTG 45.84% were delivered vaginally and 54.17% were delivered by caesarean section.

- In normal CTG Fetal distress (Apgar <7) were present in 10.71% of cases. In non reactive CTG Apgar <7 was present in 33.34% of cases. There was no neonatal death in reactive CTG. (p value: 0.169)

- Scalp blood pH estimation was done in 72 cases of non reactive CTG. In reactive CTG , Scalp pH estimation was not done.

- Among the 72cases, normal pH was present in 47 cases . Acidotic pH was seen in 25 cases .

- Among non acidotic pH Apgar >7 was seen in 95.74% cases and Apgar <7 was seen in 4.26% of cases. Among acidotic pH Apgar <7 was seen in 88% of cases.

- In normal pH 51.06% were delivered vaginally and 48.94% were delivered by caesarean section.

- In abnormal pH 36% were delivered vaginally 64% delivered by caesarean section. (p value 0.003)

- There was no neonatal death in non acidotic pH babies.

- In acidotic pH there was 2 neonatal death. One baby of mother with severe PIH wth scalp pH of 7.14 delivered by caesarean section immediately died after 5 minutes due to meconium aspiration syndrome(MAS). Other one with severe oligohydramnios with scalp pH 7.23 delivered by caesarean section immediately with 5 minutes Apgar <6 was died after two days with hypoxic ischaemic encephalopathy.

- There is no correlation between colour of liquor and neonatal outcome.

- Among 72 cases of non reactive CTG caesarean section was done in 54.17% of cases. Addition of FBS and scalp pH estimation reduced the unnecessary caesarean section rate.

- Abnomral CTG is associated with negative predictive value of 89.29% . But low positive predictive value of 33.33 %. This means that a normal trace indicates that the fetus is not hypoxic. Abnormal CTG is associated with large number of false positive.

- For normal perinatal outcome if we take Apgar >7 as an end point. Fetal scalp pH has a positive predictive value of 95. 74 % and negative predictive value of 88%.

- By the use of intrapartum CTG and scalp blood pH, we can reduce the perinatal death due to intrapartum hypoxia.

CONCLUSION

Intrapartum CTG if properly used is a sensitive indicator of fetal well being. But its poor specificity must be improved by appropriately timed scalp blood sampling in carefully selected cases.

The use of continuous electronic fetal monitoring has been associated with great reduction in the incidence of unexpected still birth. Arulkumaran and Gibb 1990[34] has reported that intra partum hypoxia was responsible for 14.5% of perinatal death in 1982 (where only intermittent auscultation was performed). After introduction of continuous EFM during 1986-1992, the perinatal deaths came down to 7.9%. Edington et al 1975 : Parer 1979;[26] Biswas et al 1995;[25] showed similar reduction in perinatal mortality.

Fetal respiratory acidosis secondary to uterine hypertonus or maternal metabolic acidosis secondary to fetal ketosis may produce false positive low pH value (<7.2).

In order to avoid wasting of precious time, scalp blood sampling can be avoided in certain conditions which are obvious clinically.

Fetal pH and acid base deficits are required to confirm metabolic acidosis and thereby prevent unnecessary caesarean sections done for CTG abnormalities.

Electronic fetal monitoring is the method of choice in high risk pregnancies. In low risk cases with reactive admission test, and if labor progresses satisfactorily, regular, conscientious intermittent auscultation is sufficient.

PROFORMA

INTRAPARTUM FETAL MONITORING AND FETAL SCALP BLOOD pH ESTIMATION

Date Time Unit I.P. No

Name Age Gravida Para

Complaints

1. Type of Case Booked Un Booked LMP EDD
2. Associated Complication

   Anemia PIH DM Heart Disease Others
3. Obstetric Complication

   A. Mal Presentation B. Previous LSCS C. PIH D. APH E. IUGR

   F. Others
4. Clinical Examination

   A. Height B. Weight C. Fundal Height D. Abdominal girth
5. FHR / Minute by Pinards Fetoscopy
6. Estimated Fetal Weight Clinical

   USG
7. USG Findings
8. Admission Test A. Reactive B. Equivocal C. Ominous
9. Type of Labour Spontaneous Induced
10. Abnormal FHR Pattern at what time of Labour
11. Membranes Intact Ruptured Normal Colour Meconium stained

12. Bishops Score — Effacement of Cervix
    Dilatation of Cervix
    Station of head
13. CPD
14. Scalp Blood pH — Measured for the First Time
    Repeated measurements if any
15. Maternal PH
16. Duration of Labour Hour / MT
17. Abnormal FHR Pattern- Delivery Interval
18. Scalp PH / Delivery Interval
19. Date & Time of Delivery
20. Type of Delivery
    Vaginal – Normal
    Assisted
    LSCS
21. Out Come of Delivery
    Live Birth
    Intrapartum Death
    Congenital Malformation
22. APGAR
    1 Min 5 Min
23. Birth Weight
24. Baby Sex
25. Neonatal Complication

    Intra cranial Birth Injury | RDS | Hypoglycemia | Others

26. Status at Discharge — Alive / Death
27. If Dead – Cause of Death & Date
    Postmortem findings if any

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