CORRELATING CARDIOTOCOGRAPHY, APGAR SCORE AND CORD BLOOD PH AFTER CAESAREAN SECTION FOR FETAL DISTRESS AT RAHIMA MOOSA MOTHER AND CHILD HOSPITAL

STUDENT

DR TSHIKANDA KHATHUTSHELO ASHLEY

STUDENT NUMBER

A0040359

SUPERVISORS

PROF BUCHMANN E

PROF LOMBAARD H

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UNIVERSITY OF THE WITWATERSRAND, JOHANNESBURG

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Declaration

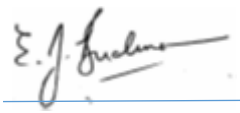
I, Tshikanda Khathutshelo Ashley, hereby declare that this research is my own original work, that I have extensively researched and all the sources used have been recognized and documented. It is being submitted to the Faculty of Health Sciences for the degree of Master of Medicine in Obstetrics and Gynaecology, at the University of the Witwatersrand, Johannesburg. I declare that this research has not been previously submitted in full or partial fulfilment of the requirements for an equivalent or higher qualification at any other institution

Researcher:

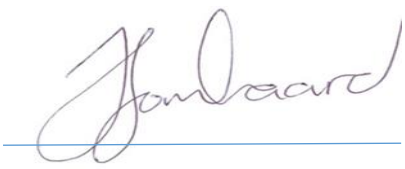
Dr Tshikanda KA

Supervisors:

Prof Buchmann E



Prof Lombaard H



Dedications

I dedicate this research to my parents Mr Tshikanda Rembuluwani Jan and Mrs Tshikanda Matodzi Albertinah. They managed to take me to school and university regardless of struggling to make ends meet. I also would like to dedicate this research to my family, my wife Mrs Tshikanda Nakedi Daphney and our children Rotondwa and Dakalo, for their support throughout.

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Abstract

Background

Fetal distress is one of the most common indications for emergency caesarean section in both developed and developing countries. Intrapartum fetal monitoring was designed with the hope of reducing the prevalence of cerebral palsy and early detection of fetal hypoxia. The most commonly used method of intrapartum fetal surveillance is cardiotocography (CTG). The use of umbilical cord artery blood gases can provide important information about the acid-base status of the newborn. The aim of this study was to correlate intrapartum CTG findings with the outcome of the newborns in terms of umbilical cord pH and Apgar scores, in women who underwent caesarean sections for fetal distress at term and were in labour, and thus to evaluate the accuracy of CTGs.

Methods

This study included 99 women with term singleton pregnancies, who were in labour and underwent caesarean section for fetal distress. CTG tracings were interpreted by a senior obstetrician as normal, non-reassuring or abnormal. The senior obstetrician interpreted these CTGs without knowledge of the acid-base statuses of the newborns. Umbilical artery blood was taken immediately after birth in a heparinized syringe and was analysed in less than 30 minutes. A pH of < 7.1 was taken as acidosis and a pH of ≥7.1 was taken as normal.

Results

The age range was 18-46 years. The number of women with gestational age of 42 weeks or more was 8 (8%). The interval between end of CTG and birth ranged from 21 to 352 minutes. Three women had a hypertensive disorder and none had diabetes mellitus.
The birth weight range was 2125-4240 g. The median Apgar score at 1 minute was 9 (range 1-9), and at 5 minutes was 10 (range 7-10). No infants required admission to the neonatal intensive care unit, none developed hypoxic-ischaemic encephalopathy, and none died. The CTG interpretation found 9 normal, 21 non-reasuring and 69 abnormal tracings.The cord blood pH correlated well with the lactate level ($r=-0.82$; $p<0.001$), and also with the base deficit ($r=-0.78$; $p<0.001$).

The Apgar scores at 1 minute and 5 minutes also correlated well with the pH (both r=0.44 with p<0.001). When the Apgar score at 5 minutes was <9 (n=7), mean pH was 7.04, and when it was ≥9, the mean pH was 7.22 (p<0.001).

Following fetal distress in the latent phase of labour, the mean pH was 7.23, while after the active phase of labour, the mean pH was 7.1 (p=0.017). Normal, non-reassuring and abnormal fetal heart rate (FHR) patterns were followed by cord blood pH values of 7.19, 7.21 and 7.21 respectively p=0.879).

Conclusion:

The predictive value of CTGs for neonatal encephalopathy was zero in this series, although the sample size of was small, given the rarity of neonatal encephalopathy. The neonatal outcome may however have been modified (improved) by the performance of caesarean section. There was a good correlation between Apgar scores and cord blood pH. Within this group of 99 infants delivered for suspected fetal distress, different fetal heart rate patterns could not be related to neonatal outcome. The study confirms the low predictive value of CTG tracings in the diagnosis of neonatal acidaemia and prediction of poor neonatal outcome, but cannot exclude the possibility that a proportion of infants may have benefited from caesarean section following CTG monitoring.

List of abbreviations:

ACOG- The American Congress of Obstetricians and Gynecologists

BE- Base excess

BPM- Beats per minute

CTG- Cardiotocograph

CS- Caesarean section

DDI- Decision to delivery interval

EFM- Electronic fetal monitoring

FBS- Fetal blood sample

FD- Fetal distress

FHR- Fetal heart rate

HIE- Hypoxic ischaemic encephalopathy

IA- Intermittent auscultation

NICE- National Institute for Health and Care Excellence

NICU- Neonatal intensive care unit

RCT- Randomised controlled trial

STAN- ST waveform analysis

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1. LITERATURE REVIEW

Introduction

The universal aim of maternity care provision is the birth of a healthy baby to a healthy mother. Fetal distress (severe fetal hypoxaemia) is one of the commonest indications for caesarean section (CS), in both developed and developing countries, and is a significant contributor to perinatal morbidity and mortality. CS rates have been rising since 1970 especially in more developed countries.[1-3]

In South Africa the number of CS has increased as shown by a study done by Nathan et al, who found that between 2007 and 2014 the average CS rates were: 55.5% in Steve Biko Academic, 43% in Charlotte Maxeke Academic and 34.5% in Dr George Mukhari Academic Hospitals[1]. The optimum caesarean section rate as per the World Health Organization (WHO) recommendation is between 5 and 15%, as this has been shown to ensure the best outcome for mothers and their babies.[1,2,4,5]

Cardiotocography (CTG) is the method currently used worldwide for intrapartum fetal surveillance and diagnosis of fetal distress. The aetiology of fetal distress comprises multiple factors, such as reduced intervilous perfusion, reduced placental oxygen transfer or reduced umbilical blood flow, often associated with strong uterine contractions in labour. Hypoxaemia activates the fetal autonomic nervous system and affects the fetal heart rate, with abnormalities that can be detected on CTG.[6-8]

In hypoxaemia, there is ongoing production of hydrogen ions (acidosis), which requires a buffering system to maintain acid-base homeostasis. The body has a series of buffering systems to maintain the acid-base homeostasis, and these include bicarbonate in plasma and proteins in erythrocytes. Haemoglobin is one of the prime proteins involved in buffering.[9,10]

In labour the placental circulation is always affected by interruption of blood flow during labour contractions, which results in benign respiratory acidosis due to hypercapnia, which is normally tolerated by a normal fetus. In contrast, acidosis due to hypoxia, with anaerobic respiration, leads to metabolic acidaemia, with resulting disturbances in cellular functions, which are potentially harmful to the fetus.[9,11]

Definition of fetal distress

Fetal distress has been defined as progressive fetal asphyxia that, if not corrected or circumvented, will result in decompensation of the fetal physiological responses (primarily redistribution of blood flow to preserve oxygenation of vital organs) and cause permanent central nervous system and other damage or death.[6,12,13]

Causes of fetal distress

1. Uteroplacental pathology
    - Maternal demographics and exposures (smoking, age)
    - Maternal diseases (hypertension, diabetes, sickle cell)
    - Intrauterine growth restriction
    - Placental separation (marginal bleeds and abruptions)
    - Postmaturity.[14]
2. Fetal pathology:
    - Fetal anaemia (rhesus isoimmunisation, parvovirus infection)
    - Congenital malformations.[15]
3. Labour:
    - Uterine contractions (prolonged or with use of uterotonic drugs)
    - Cord compression
    - Maternal hypotension (Circulatory collapse, aortocaval compression)
    - Obstetric emergencies (uterine rupture, abruption, cord prolapse).[12-16]

Physiology of fetal distress

Oxygen is transferred to the fetus from the environment by maternal and fetal blood, through maternal lungs, heart, vasculature, uterus, placenta and umbilical cord. Disturbance at any of these levels will result in fetal hypoxia. Fetal response to interruptions in the oxygen delivery system consists of the following in a sequence: hypoxaemia, hypoxia, metabolic acidosis and metabolic acidaemia.[14]

Inspired air comprises 21% oxygen, and as the oxygen is being transported to the fetus the PO_2 progressively decreases. By the time oxygen reaches the fetus the PO_2 is about 30

mmHg. After oxygen has been delivered to the fetal tissues, the PO_2 in the deoxygenated blood in the umbilical arteries may be 15-25mmHg.[10,11]

Maternal lungs

During inspiration oxygen is carried from the environment to the alveoli. During transfer to the alveoli the inspired air mixes with the deoxygenated air leaving the lungs, and this causes a drop of the PO_2 from the original 150 mmHg at sea level to 100 mmHg at the alveoli. From the alveoli oxygen diffuses through a thin blood-gas barrier composed of 3 layers to reach the pulmonary capillaries.[10-13]

Factors that can interrupt transfer of oxygen from the environment to the pulmonary capillaries include airway obstruction caused by drugs or maternal convulsions, pneumonia, pulmonary embolus, asthma, atelectasis and acute respiratory distress syndrome.[11-14]

Maternal blood

As soon as oxygen diffuses from alveoli into maternal blood, more than 98% of it is carried by haemoglobin in maternal red blood cells. Factors that can result in interruption of the binding of oxygen to haemoglobin include: anaemia, hemoglobinopathies and methemoglobinemia.[13-15]

Maternal heart

From the lungs oxygenated blood is transported further to the whole body including the utero-placental unit. Transfer of oxygen from the environment to the fetus depends on normal cardiac function. Interruption at the level of the maternal heart results from the factors like arrhythmias, hypovoalemia, compression of the inferior vena cava, impaired heart contractility and structural abnormalities of the heart, heart valves and great vessels.[12-15]

Maternal vasculature

From the uterine arteries oxygenated blood travels through the arcuate, radial and finally the spiral arteries before exiting the maternal circulation and entering the intervillous spaces of the placenta. Interruption of oxygen transfer at the level of the maternal vasculature results from regional anaesthesia, hypovolaemia, impaired venous return, impaired cardiac output, or medication. Conditions that result in chronic vasculopathy include preeclampsia, and

diabetes mellitus. Preeclampsia is associated with poor remodelling of the spiral arteries which results in impedance of blood flow through these vessels.[13-16]

Uterus

The spiral arteries of the uterus are converted to large low resistance conduits during pregnancy, and this allows blood to flow easily into the intervillous space. During normal uterine contractions there is interruption of blood flow, which a normal fetus can withstand, but excessively long or frequent contractions can lead to fetal hypoxia. Uterine rupture is another important cause of interruption of oxygen delivery at the level of the uterus.[14-16]

Placenta

Oxygenated blood in the intervillous space is in contact with chorionic villi. The placenta facilitates the exchange of nutrients, oxygen and waste products between the maternal blood in the intervillous space and the fetal blood in the villous capillaries.[10]

Fetal villous blood is separated from maternal blood in the intervillous space by a thin barrier similar to the blood-gas barrier in the maternal lungs. The oxygen concentration in the fetal blood is much less than in the intervillous space, therefore resulting in diffusion of oxygen into the fetal capillaries.[14]

Interruptions at this level can be caused by factors like chronic placental insufficiency, pre-eclampsia, intrauterine growth restriction, postmaturity, placental abruption, chorioamnionitis, bleeding placenta praevia or vasa praevia and thrombosis in the intervillous space.[14]

Fetal blood

Fetal haemoglobin has a higher affinity for oxygen than adult haemoglobin and this results in higher oxygen carrying capacity. Factors that could interrupt oxygen delivery to the fetus at this level include alloimmunisation, haemoglobinopathies and enzyme disorders (glucose-6-phosphate dehydrogenase deficiency).[16]

Umbilical cord

Oxygen is carried to the fetus by villous veins, which join to form one umbilical vein in the umbilical cord. Factors that result in interruption at this level include thrombosis, vasospasms, compression, cord prolapse, atherosis, true knot, funisitis, rupture and meconium-associated umbilical vascular myonecrosis.[15]

Fetal response to interrupted oxygen transfer

The fetus responds to interruptions in oxygen supply in many ways depending on the duration, frequency and degree of oxygen interruption. The sequence of events starts with hypoxaemia, which is decreased oxygen content in the blood. Continuous or recurrent hypoxaemia results in hypoxia which is reduced oxygen content in the tissues.[15,16,17]

One of the most important challenges the fetus faces is hypoxia. This can either be acute (often related to labour and contractions) or chronic related to placental insufficiency, preeclampsia or an inflammatory condition during pregnancy such as chorioamnionitis or funisitis. If the fetus has inadequate defence against acute or chronic hyoxia, the fetal brain becomes susceptible to hypoxic brain injury leading to hypoxic-ischaemic brain injury.[15-18]

Normal homeostasis requires oxygen to generate energy through aerobic metabolism. But when oxygen is not sufficient, tissues are forced to change to anaerobic metabolism to generate energy. The anaerobic metabolism generates lactic acid which is then neutralised by buffers like bicarbonate, proteins and haemoglobin. When the hypoxia is sustained, the lactic acid concentration exceeds the buffering capacity then this results in metabolic acidosis.[15-17]

Fetal defences against hypoxia

The fetus is able to cope with short and long term episodes of hypoxia. A number of adaptive mechanisms ensure that the supply of oxygen exceeds its metabolic demands, with contingencies in place to provide an adequate supply of oxygen to vital organs during episodes of hypoxia. [17-19]

The fetus has shunts like the ductus arteriosus and ductus venosus, and these ensure adequate oxygen supply to vital organs. The fetus also possesses mechanisms to hinder oxygen-consuming processes. During episodes of hypoxia a late gestation fetus is able to stop body

movements, breathing movements, and slow down its heart rate and therefore reduce oxygen consumption. The reduction in fetal heart rate prolongs the end-diastolic filling time, increasing cardiac output and perfusion pressure.[15,20]

In addition to cardiac compensatory mechanisms, there is fetal blood redistribution away from peripheral vascular beds, favouring perfusion of the brain, heart and adrenal glands.[20]

Fetal Brain sparing

Neural:

The neural response to hypoxia is mediated by the carotid chemoreflex, where hypoxia triggers these receptors and signals are sent to the fetal brainstem, leading to an increase in both the sympathetic and the vagal outflow. These result in peripheral vasoconstriction and bradycardia respectively.

Endocrine:

Hypoxia has a direct effect on the fetal adrenal medulla, causing the release of catecholamines leading to peripheral vasoconstriction, thereby reducing oxygen consumption.[20,21]

Local:

Hypoxia causes release of vasodilators including nitric oxide and prostanoids, leading to vasodilation in the fetal cerebral vascular circulation and therefore increased blood flow in the brain.[20,21]

Fetal acid-base physiology

The fetus's normal metabolism results in the production of acids which have to be buffered to maintain normal extracellular pH. These acids are either volatile acids (carbonic acid) or non-volatile acids (non-carbonic or organic acids, including lactate). Plasma bicarbonate and haemoglobin are the major buffers that the fetus uses to neutralize these acids. Other buffers include inorganic phosphates and erythrocyte bicarbonate.[9]

Acids:

Carbonic acid: This acid is produced by the fetus during aerobic glycolysis. The fetus handles carbonic acid produced during aerobic metabolism easily because carbonic acid dissociates to water and CO_2, which readily diffuses across the placenta because of a negative CO_2 gradient between the fetal and maternal circulations.[9]

Organic acids: during ongoing fetal hypoxia, energy production proceeds along an anaerobic pathway. Lactate and ketoacids are produced, but are not readily excreted or metabolized, and are cleared slowly across the placenta, resulting in accumulation in the fetus. Acidemia then results when these organic acids deplete the buffering system.[9]

Diagnosis of fetal distress

There are 3 methods for assessing fetal acid-base status:

Antepartum: percutaneous umbilical cord blood sampling

Intrapartum: fetal scalp capillary blood sampling

Immediately postpartum: umbilical cord blood sampling.[9,22]

The antepartum and intrapartum methods are invasive and require skill and time. Therefore, fetal distress is mainly diagnosed indirectly with detection of an abnormal fetal heart rate pattern on a CTG. Fetal distress can also be diagnosed by intermittent fetal heart rate (FHR) auscultation, but CTG confirmation is preferred. According to the National Institute for Health and Care Excellence (NICE) guidelines, features suggestive of fetal distress on CTG include FHR <100bpm or >180 bpm, sinusoidal pattern > 10 minutes, baseline variability< 5 for 90 minutes or more, single prolonged deceleration for more than 3 minutes and the presence of late decelerations.[23]

Sampling of fetal blood by scalp incision is the ideal for confirmation of acidaemia when CTG abnormalities are found, but, apart from skill and time, this requires ruptured membranes and an adequately dilated cervix, and may transmit infections such as HIV to the fetus. Fetal scalp blood sampling is currently not practiced in South Africa maternity services, at any level of care. Other forms of fetal monitoring include ST waveform analysis (STAN), but it hasn't been shown to be superior to CTG. This was shown in a meta-analysis

by Potti et al, which showed no difference in perinatal outcomes between STAN with CTG and CTG alone.[24]

Procedure of collecting blood from the cord

A 20cm segment of the umbilical cord should be isolated and clamped, immediately after delivery, if this is not done the PH and pCO2 in the cord blood will change as soon as the baby begins to breathe and this will not be reflective of the acid-base status at delivery. Changes in PH, pCO2 and pO2 occur with time as a result of cellular metabolism, but these changes occur slowly. It has been shown that cord blood can be left at room temperature for up to one hour without significantly affecting the results.[24]

Blood does not usually clot while in the cord but the use of heparinised syringes for cord blood sampling is advised. Syringes should be heparinised on site by adding liquid heparin to a 2ml syringe, but care should be taken to avoid heparin to make up more than 10% of the sample, because heparin is acidic, and therefore will alter the acid-base status of the sample. The use of commercially prepared syringes containing lyophilised heparin will prevent errors caused by varying amounts of heparin in each syringe.[24]

Literature suggests that up to 19% of umbilical cord blood samples are invalid. For a sample to be valid the umbilical vein pH should be >0.02 relative to the artery, and the $CO_2 \leq 4$ mmHg. Ideally both the vein and the artery should be sampled.[24]

Difficulty in diagnosing fetal distress

An important problem with the diagnosis of fetal distress via CTG is that it is partially subjective and clinicians can interpret the same tracing differently. This has been shown by Keith et al in 1995, who demonstrated that even experts can have differing opinions of the same CTG tracing. A further difficulty is in the poor specificity of abnormal FHR patterns for cord blood acidaemia. While the sensitivity of CTG for acidaemia is as high as 95% for non-reassuring and abnormal traces, the specificity has been estimated to be as low as 21.8%, indicating a high false positive rate. At times the CTG might look completely normal while the fetus is acidotic, this was shown in a study by Steer et al, who showed that 4 out of 24 (17%) neonates who were acidotic actually had normal CTGs.[25]

Intermittent auscultation

For pregnancies considered to be of low risk, intermittent auscultation (IA) is recommended in labour. This is done using a hand-held Doppler apparatus or a Pinard stethoscope. The maternal heart rate should also be recorded hourly during fetal heart rate monitoring.

In the first stage of labour, IA should be done at least every 15 minutes after a contraction for a minimum of 60 seconds, as per NICE, but according to South African guidelines, it should be half-hourly. In the second stage of labour, IA should be done at least every 5 minutes after a contraction for a minimum of 60 seconds.[26-28]

If abnormalities are detected, auscultation should be prolonged to cover at least three contractions and per vaginal examination should be done to assess progress of labour and exclude umbilical cord prolapse. If abnormalities remain present after prolonged auscultation, continuous electronic fetal monitoring should be considered.[26,27]

Electronic fetal monitoring

Electronic fetal monitoring (EFM) using CTG became commercially available in the 1960s with the emphasis on improving fetal birth outcomes by detecting fetal hypoxia, before it led to perinatal mortality and morbidity. EFM has clearly made a difference in the practice of midwifery and obstetrics. It has taught us a lot about what happens to the fetal cardiovascular system during labour. Its use has however led to a rise in caesarean delivery rates.[26,29]

Labour is a stressful event to the fetus, but healthy fetuses are able to withstand labour. During contractions, there is transient episodic interruption to fetal oxygenation. Normal fetuses also could end up developing fetal distress in circumstances where there are other factors that predispose to fetal distress. These include excessive contractions, cord prolapse and maternal hypertensive disorders. Electronic fetal monitoring can also help to determine antepartum which fetuses are at risk of developing fetal distress.[26]

EFM is a method of assessing and recording FHR patterns and the frequency of uterine contractions and pattern in relation to FHR, using the CTG. The aim of intrapartum EFM is the identification of FHR changes that are associated with inadequate fetal oxygenation so that timely interventions can be made to reduce the possibility of hypoxic injury or death.[26,29]

Low et al showed that the most sensitive FHR pattern variables in terms of predicting acidosis is reduced beat to beat variability and late decelarations.[30] Baseline fetal heart rate has not been shown to be a good predictor of fetal acidosis as shown in a study by Aboulghar et al, were they found 82% had normal baseline fetal heart rate but 34% had acidosis.[31]

As mentioned above, CTGs have poor specificity, with acidaemia confirmed in only a small fraction of the fetuses in which the CTG suggests the presence of fetal distress. There is a need for a more specific tests for fetal hypoxia, but no such tests have yet superseded the CTG.[22] In another study by Williams et al, it was shown that in the presence of minimal/absent variability (amplitude <5) for at least an hour, the incidence of significant acidemia (pH <7.0) was increased.[32]

Continuous EFM is recommended and should be offered in the following circumstances:

- In the presence of antepartum risk factors at the onset of labour
- The development of intrapartum risk factors during labour
- A FHR <110 bpm or >160 bpm, and the presence of decelerations heard after a contraction
- Abnormal fetal growth during pregnancy
- Epidural analgesia
- The presence of meconium stained liquor
- Excessive uterine activity, due to augmentation of labour with oxytocin.[26]

Is it beneficial to use EFM?

There is some evidence that the use of EFM is associated with a reduction in intrapartum death, but there is no proven reduction in long term neurologic impairment. There are no randomized trials to compare continuous fetal monitoring with no intrapartum monitoring. In randomized trials, EFM has not proven to be superior to intermittent auscultation in the reduction of intrapartum deaths and reduction of long-term neurological impairment, for both high and low-risk pregnancies.[33]

A systematic review was done by Devane et al, in 2017 in which continuous EFM was compared to intermittent auscultation.[34] The review included 13 randomized trials with over 37 000 high and low-risk pregnancies. There were no significant differences between the two methods, in terms of acidaemia after delivery, Apgar scores at 5 minutes, neonatal ICU admission, hypoxic-ischaemic encephalopathy, perinatal mortality and neurodevelopmental impairment.[34]

The use of continuous EFM was associated with fewer neonatal seizures but these were not associated with long-term consequences. There is no proven benefit in the reduction of cerebral palsy with the use of continuous EFM as compared to intermittent auscultation. It should be remembered that the randomized trials on EFM involved carefully and optimally performed intermittent auscultation in the control groups, with resource to CTG if evidence of fetal distress was found. Therefore, CTG monitoring was not excluded in the management of control subjects in the trials. CTG monitoring remains the prime adjunct to intermittent auscultation for fetal monitoring in low-risk labours.[35,36]

A weakness of CTG monitoring, related to its subjective interpretation, is the possibility of missing fetal distress. In a study done in Oxford 62 cases of perinatal deaths were reviewed and it was discovered that in 50% there was clear evidence of fetal distress on CTGs, but there was failure to respond by medical staff to these clues from the CTG.[37] In a study by Buchmann et al in South Africa, 123 508 births were surveyed, with 4142 perinatal deaths among infants 1000 g or more and the perinatal mortality rate from intrapartum-related birth asphyxia was 4.8/1000 births. A common avoidable factors was signs of fetal distress interpreted incorrectly in 24.9% of deaths more than the proportion that had no or inadequate monitoring (18.0%).[38]

In a retrospective study by Bogdanovic et al, 68 infants who developed hypoxic ischaemic encephalopathy, 61% of the CTGs were interpreted as pathological which shows a good correlation of CTGs with neonatal outcomes.[39]

Confirmation of fetal distress after delivery

Cord blood pH is considered useful in neonatal evaluation for acidaemia and hypoxaemia. Studies have shown that low cord blood pH is significantly associated with acute and long-

term adverse consequences (low Apgar scores, neonatal encephalopathy and cerebral palsy). A threshold arterial cord blood pH < 7.1 is considered most predictive. However, the vast majority of newborns with acidaemia do not go on to develop encephalopathy.[40,41]

A study done in Oxford's John Radcliffe Hospital in the United Kingdom between 1991 and 2009, where 51 519 neonates had cord blood taken for analysis for pH, found that a cord blood pH of 7.1 and below was strongly associated with adverse neurological outcome. Low Apgar scores were associated with a pH <7.20. Base excess can also be used as a measure of the non-respiratory component of the acid-base status, i.e. the metabolic acid accumulation. The normal ranges of base excess (BE) in neonates is between -5 and +5 mmol/L.[36]

In a study by Modarressnejad, acidosis was defined as a cord blood pH of <7.1.[42] Also in our study we described acidosis as cord blood pH of < 7.1. Kaban et al described acidosis as pH of <7.2.[43]

In South Africa, CTG is the main tool used to diagnose fetal distress, notwithstanding its poor specificity and also the difficulties in CTG interpretation.[38] Currently there are no South African studies to determine the specificity and sensitivity of CTGs.

Interpretation of CTG

In this study interpretation of is done using The National Institute for Health and Care Excellence (NICE) (Appendix A), but there are other guidelines like those of the American Congress of Obstetricians and Gynecologists (ACOG), in which there are three categories: category 1 (normal), category 2 (neither normal nor abnormal and needs further evaluation) and category 3 (abnormal).[44] Also, The International Federation of Obstetrics and Gynecology (FIGO) has recently released new guidelines.

Association of fetal distess with subsequent cerebral palsy

Only 10% of newborns who later develop cerebral palsy have evidence of isolated intrapartum hypoxia as a cause. There are many other causes of cerebral palsy including developmental abnormalities, metabolic abnormalities, autoimmune disease, infections and preterm birth complications. And most events leading to the development of cerebral palsy occur prior to the onset of labour. It has been shown that fetal heart rate patterns are poor

predictors of cerebral palsy.[45] Nelson et al found that fetal heart patterns on CTGs have a false positive rate for cerebral palsy as high as 98.8% even in the presence of late decelerations or reduced beat to beat variability.[46] Nevertheless, according to the American College of Obstetricians and Gynecologists (ACOG), the presence of a severely abnormal fetal heart rate (category 3) on CTG is accepted as evidence of hypoxic-ischaemic injury that may lead to cerebral palsy.[47]

Caesarean section and fetal distress

In South Africa, CS rate has increased from 18.1% in 2000/2001 to 24.4% in 2008/2009.[1] At Rahima Moosa Hospital, the total number of CS was 4469 and normal vaginal deliveries of 6924 resulting in a CS rate of 39.2% in 2016. Udigwe et al showed that in as many as a third of these CS done for fetal distress the diagnosis is not accurate.[48] This rise in CS rate is associated with increased risk of complications. In addition, CS are both exhaustive of resources and labour intensive, compared to vaginal birth.[49,50]

A goal which is continually pursued during labour is to detect early signs of fetal compromise and to intervene immediately to avoid adverse outcomes.[50] But identifying the fetus at risk is not always easy. The methods that are currently used to detect fetal hypoxia have not been proven to be completely reliable. Electronic fetal monitoring and intermittent auscultation have been shown to have less specificity in predicting the degree of fetal hypoxia or in detecting adverse neonatal outcomes.[25] The diagnosis of fetal jeopardy based on CTG alone has led to an increase in CS rate. This was shown by Nelson et al in USA.[45]

A study done in Sri Lanka revealed that 20% of CS were done for fetal distress.[51] The corresponding proportions are 25% in France, and 9.9% in Nigeria.[52] In a study in Pakistan, the most common indication for CS in a teaching tertiary hospital were: previous caesarean section (22.76%), failed progress of labour (18.29%) and fetal distress (15.44%).[51] Fetal distress constitutes a large proportion of indications for CS, albeit a lesser one in poorer countries, possibly related to a lack of resources. There appears to be overwhelming evidence that EFM has led to increased CS rate, as shown in a Cochrane review by Alfirevic et al. Yet, there is lack of direct relationship between CTG patterns and short and long term outcomes of neonates.[53]

Interval between decision for emergency caesarean section for fetal distress and delivery

It seems intuitive that a CS performed for fetal distress should be expedited as quickly as possible to optimise neonatal outcome. Halsey and Douglas determined their decision to delivery time interval (DDI) to be about 43 minutes in 1957, with the shortest being 15 minutes and the longest being 95 minutes.[54,55] More recent guidelines issued by the ACOG and the American Academy of Pediatrics (AAP), suggested that all hospitals offering labour and delivery services should be equipped to perform emergency CS and should be capable of beginning a CS within 30 minutes of the decision to operate.[55]

However, in 1984 a survey of 538 hospitals was done in the USA and reported that all of them had the ability to perform an emergency caesarean delivery within 30 minutes, but this did not lower the peripartum mortality rate, so it is unclear if this "30-minute rule" has any clinical validity.[55] In 2003 Holcroft and others reviewed the "30-minute rule", by assessing DDIs in all caesarean sections performed for non-reassuring fetal status over two years. They found that most neonates delivered urgently for non-reassuring fetal status, even when born after 30 minutes, had normal cord blood acid-base studies.[56]

In some African countries DDIs of 201 minutes to as long as 511 minutes have been described. In a study done in Nigeria by Yakasai et al, the major cause of delay was anaesthesia in 40% of the cases.[57] In a study by Hall et al in a teaching hospital in South Africa, the mean DDI for emergency caesarean sections was 52.9 minutes.[3] In a study by Helmy et al in the United Kingdom, some of the delays were due to the duration of transferring patients to the operating theatre.[58] In a study done in Pakistan, causes of delay included obtaining consent, multiple attempts of spinal anaesthesia and delays in availability of staff.[51]

Onah et al studied the records of emergency caesarean deliveries done in 2012 in a Nigerian tertiary hospital, and found that only 20 out of 352 caesarean deliveries were done in under the recommended 30 minutes. They also concluded that there is a lack of correlation between DDI and perinatal outcomes, and the recommended DDI of 30 minutes may not be

applicable to all emergency CS, especially when faced with infrastructural challenges especially in developing countries.[54]

Categorization of caesarean sections in terms of urgency

A classification for urgency of caesarean section, as classified by Lucas et al, is as follows:

1. Emergency - immediate threat to life of woman or fetus
2. Urgent - maternal or fetal compromise which is not immediately life-threatening
3. Scheduled - needing early delivery but no maternal or fetal compromise
4. Elective - at a time to suit the patient and maternity team.[51]

The conventional classification of caesarean sections, which classifies Caesarean sections as elective or emergency is not useful because some non-elective caesarean sections are more urgent than others. So it is essential to subdivide the no-elective caesarean sections according to their urgency.[59]

Reasons for unnecessary caesarean sections done for fetal distress

Fetal distress is one of the leading indications for caesarean section in South Africa. Almost all the diagnosis of fetal distress is based on CTG patterns. The interpretation of CTG traces remains subjective, despite attempts to standardize the interpretation of CTGs. According to a South African opinion paper by de Jong, the following are some of the reasons for unnecessary Caesarean sections done on the basis of the CTG tracing: Fear of poor outcomes and litigation, CTGs not set correctly, e.g. paper speed set at 3 cm/min instead of 1 cm/min, leading to false labelling of CTGs as abnormal, not exploring the reason for an abnormal tracing and not acting upon these reasons, (e.g decelerations following the use of oxytocin in augmentation of labour; instead of stopping the oxytocin, a caesarean section is ordered), detection of maternal heart rate either continuously or intermittently on CTG may be misinterpreted as fetal bradycardia or decelerations.[4]

Ideally, the use of electronic fetal monitoring should be reserved for high risk pregnancies. Intermittent auscultation using a stethoscope or a hand-held Doppler equipment remains the standard for low-risk labour. However, CTG monitoring may be appropriate where there is a

shortage of midwives and regular intermittent fetal heart monitoring would be difficult.53 Many hospitals in South Africa are burdened with a high CS rate which unfortunately increases the rate of morbidity and mortality related to obstetric haemorrhage. There is a need to instil the desire for normal vaginal delivery for patients and to encourage obstetricians and midwives to have a culture of promoting vaginal delivery.[4]

Avoiding the first CS should be a prime goal of midwives and obstetricians. And obstetricians should seek to determine the cause of abnormal CTG tracings first before deciding to book patients for caesarean sections for fetal distress.[60,61] Some causes of abnormal CTG traces can easily be corrected, thus avoiding unnecessary caesarean deliveries for fetal distress. This is likely to reduce the rate of caesarean deliveries and the associated complications. [60,62,63]

1. PROBLEM STATEMENT

Currently there are no studies done in Johannesburg or South Africa, to determine the predictive value of CTG diagnosis of fetal distress, by following up the newborn for acidaemia and or low Apgar scores.

Caesarean section is a useful operation in obstetrics but unfortunately is associated with higher risk to both the mother and fetus, compared to normal vaginal delivery. So it is crucial that CS are performed only when they are absolutely indicated. Fetal distress is one of the common indications for CS and the method used to detect fetal distress is not accurate, possibly leading to a high number of false positives and unnecessary CS, with accompanying maternal morbidity and mortality.

Fear of litigation, among other reasons, may have led to a culture amongst obstetricians of neglecting the basic approach of first establishing the cause of abnormal tracings and attempting to correct the causes before resorting to caesarean section. To what extent this has resulted in unnecessary CS in South African government hospitals, is unknown. Data are needed locally to determine the frequency of poor neonatal indicators and outcomes in caesarean section performed for fetal distress in labour. Such data will provide baseline information to those who plan initiatives that seek to reduce caesarean section rates.

Aims and objectives

Aims:

The aim of this study was to correlate the cardiotocography findings with Apgar scores and cord blood pH after caesarean sections for fetal distress in a tertiary hospital (Rahima Moosa Mother and Child Hospital).

Objectives:

1. To describe CTG findings in cases of caesarean sections for fetal distress using NICE guidelines.

2. To describe Apgar scores and record cord blood acid-base status in infants after caesarean section for fetal distress.

3. To correlate CTG findings with Apgar scores and cord blood acid-base status.

4. To determine how long it takes for emergency caesarean sections for fetal distress to be done once the diagnosis is made in Rahima Moosa Mother and Child hospital.

3. METHODS

Setting:

The study was done in Rahima Moosa Mother and Child Hospital, located in Coronationville, Johannesburg. The hospital is a 332-bed tertiary facility hospital that caters for obstetric and gynaecologic together with paediatrics and paediatric surgery patients. It is a teaching hospital on the circuit of The University of the Witwatersrand. While being a tertiary facility, it does not have referring district hospitals, but has two midwifery clinics, therefore some low-risk women who could give birth in district hospitals, end up giving birth in this hospital. These women are cared for by midwives who refer to doctors in cases of difficulty. CTG monitoring is frequently used for low-risk women, and for all high-risk cases. A dedicated maternity operating theatre is available around the clock, on the floor just above the labour ward. When caesarean sections are performed for fetal distress, it is normal practice for umbilical cord blood to be taken and analysed for blood gas and acid-base status.

Study design and participants:

This was a prospective cross-sectional analytic study. The study population were women who underwent caesarean deliveries for fetal distress. Inclusion criteria were singleton term (>37weeks) pregnancies, with live births and morphologically normal infants. Women whose operations were done for multiple indications were included as long as one of the indications was ‘fetal distress’. For inclusion in the study, there had to be a CTG tracing for at least 30 minutes in the two hours preceding delivery, as well as results of arterial cord blood analysis taken at most one hour after delivery. Because of consent issues, women under the age of 18 years were excluded.

Data collection and analysis

Eligible women were consecutively approached by the researcher during days on which he was available, in the postnatal ward and were briefed about the study and were given the information sheet which also contained the contacts of the ethics office, (attached Appendix C). After they had read and understood the information sheet, they were asked to sign consent, after which data were collected using the data collecting tool. Critical data included demographic information, pregnancy details, obstetric risk profile and neonatal findings such

as recorded Apgars scores, umbilical artery acid-base findings, neonatal intensive care unit (NICU) admission and neonatal encephalopathy (Appendix D). The umbilical cord arterial blood was sampled by a paediatric registrar at each caesarean section for fetal distress and the pCO_2 and pO_2 values were used to ascertain that arterial and not venous blood was sampled. The cord is clamped at two points 20 cm apart and the sampling needle attached to a pre-heparinised syringe is inserted into one of the umbilical arteries and blood is drawn. Photographs were taken of the last 30 minutes of the CTG tracings using a smart phone. These photographs were submitted to a senior obstetrician for CTG interpretation using the NICE guidelines (Appendix A).[23] While the obstetrician was aware that the CTGs had resulted in CS being done for fetal distress, he was not aware of the neonatal findings such as Apgar scores, acid-base status and later clinical condition. .

Data analysis:

The data were entered in a Microsoft Excel spread sheet and imported into Stata 13 statistical software (Statacorp, College Station, Texas, USA). Categorical variables were summarised using frequencies of occurrence with proportions expressed as percentages. Continuous variables were summarised using means ± standard deviations, and medians with interquartile ranges and ranges. Fisher's exact test was used for comparison of proportions. Student's t-test was used for comparison of two means, and the ANOVA test was used for comparison of three means. Statistical significance was accepted at a p-value <0.05. Odds ratios were reported with 95% confidence intervals.

Ethics Approval:

Written informed consent was obtained from all participant women, after delivery. Permission to conduct this research was granted by the Human Research and Ethics Committee (HREC) on 08/02/2017. The ethics approval number is M161020 (Appendix C). Permission was also granted from the clinical manager of Rahima Moosa Mother and Child Hospital (Appendix D).

Study period:

February 2017 to April 2017

Sample size

99 participants were involved in this study.

Limitations

The small sample size may not reflect the true picture of neonatal outcomes following caesarean sections for fetal distress. Some CTGs do get lost and the last CTG before caesarean section might not be found.

4. RESULTS

In this study there were 100 consenting participants but one was excluded due to very short CTG, leaving 99 for analysis. The mean age of participants was 27.4 years and 44% of women were nulliparous (Table 1). The mean gestational age at birth was 38.9 weeks. The age range of the women was 18-46 years. The number of women with gestational age of 42 weeks or more was 8 (8%). The decision to delivery interval ranged from 21 to 352 minutes. The mean interval between CTG and birth was 87 minutes. There were three women with a hypertensive disorder and none with diabetes mellitus. One woman had fever and one had an antepartum haemorrhage. There were no cases of cord prolapse.

Table 1. Maternal obstetric findings (n=99); mean ± standard deviation, median (interquartile range) and n (%).

Age (years)	27.4±5.9
Parity:	
0	44 (44%)
1	30 (30%)
2	16 (16%)
≥3	9 (9%)
Gestational age (weeks)	38.9±1.5
Cervical dilatation (cm)	3 (1-6)
Active phase of labour (≥ 4 cm dilated)	48 (48%)
Interval between end of CTG and birth (minutes)	87 (64-133)
HIV seropositive	21 (21%)
Induction of labour	4 (4%)
Augmentation of labour with oxytocin	9 (9%)

Table 2 shows how the CTGs were interpreted by the senior obstetrician. It shows the different CTG characteristics that the senior obstetrician used to interpret the CTG traces. Uterine contractions were present in all 99 cases.

Table 2. Cardiotocograph (CTG) findings at the time of diagnosis of 'fetal distress' (n=99).

	Normal	Non-reassuring	Abnormal
Uterine contractions	79		20
Baseline fetal heart rate	74	17	8
Baseline fetal heart rate variability	75	21	3
Fetal heart rate decelerations	13	31	55
Fetal heart rate accelerations	Present: 28	Absent: 71	
Overall CTG interpretation	9	21	69

The birth weight range was 2125-4240 g. The Apgar scores at 1 minute ranged from 1 to 9, and at 5 minutes ranged from 7 to 10. Bag-mask ventilation was administered in 84% of newborns (Table 3). None required admission to the NICU and none developed hypoxic-ischaemic encephalopathy. There were no neonatal deaths. Eighteen percent of infants were admitted to the neonatal care unit. Meconium staining of the amniotic fluid was noted in 45% of births.

Table 3. Infant details and condition (n=99); mean ± standard deviation, median (interquartile range) and n (%).

Birth weight (g)	3100±399
Apgar score at 1 minute	9 (1-9)
Apgar score at 5 minutes	10 (7-10)
Meconium staining of the liquor at birth: None thin thick	 55 (55%) 12 (12%) 32 (32%)
Resuscitation required: None Bag-mask ventilation Cardiopulmonary resuscitation	 13 (13%) 84 (84%) 2 (2%)
Admission to the neonatal unit	18 (18%)

The mean cord blood pH was 7.20, and 17% of pH measurements were less than 7.1 (Table 4). The median interval from birth to cord blood sampling was 14 minutes (interquartile

range 8-18 minutes). Six specimens were taken after 30 minutes, with the longest duration being 39 minutes. The cord blood pH correlated well with the lactate level (r=-0.82; p<0.001), and also with the base deficit (r=-0.78; p<0.001). The Apgar scores at 1 minute and 5 minutes correlated with the pH (both r=0.44 with p<0.001). When the Apgar score at 1 minute was <7 (n=9) the mean pH was 7.02, and when it was ≥7, the mean pH was 7.22 (p<0.001). When the Apgar score at 5 minutes was <9 (n=7) the mean pH was 7.04, and when it was ≥9, the mean pH was 7.22 (p<0.001).

Table 4. Arterial cord blood gas and acid-base results (n=99); mean ± standard deviation and n (%).

PaO_2 (mmHg)	17.9±6.5
$PaCO_2$ (mmHg)	54.9±14.2
Bicarbonate (mmol/L)	20.5±2.9
Ph	7.20±0.12
pH <7.1	17 (17%)
Lactate (mmol/L)	5.5±3.0
Lactate ≥8.0 mmol/L	18 (18%)
Base deficit (mmol/L)	6.8±4.6
Base deficit ≥12.0 mmol/L	12 (12%)

Table 5. Association between overall cardiotocograph (CTG) interpretation and low Apgar scores

This table shows that there was no significant correlation between Apgar scores and CTGs.

Apgar score at 1 minute:	<7 (n=9)	≥7 (n=90)	P value
CTG: Normal Non-reassuring Abnormal	 1 2 6	 8 19 63	1.000
Apgar score at 5 minutes:	<9 (n=7)	≥9 (n=92)	P value
CTG: Normal Non-reassuring Abnormal	 1 2 4	 8 19 65	0.530

Following fetal distress in the latent phase of labour (n=51), the mean pH was 7.23, while after the active phase of labour (n=48), the mean pH was 7.1 (p=0.017). There was no significant association between cord blood pH and CTG interpretation, either in latent or active phase of labour (Table 6).

Table 6. Mean cord blood pH ± standard deviation by overall cardiotocograph (CTG) interpretation in latent and active phases of labour.

CTG:	**Normal**	**Non-reassuring**	**Abnormal**	p-value
Latent phase (n=51)	n=7; 7.20±0.13	n=10; 7.24±0.06	n=34; 7.24±0.11	0.719
Active phase (n=48)	n=2; 7.09±0.11	n=11; 7.12±0.15	n=35; 7.20±0.13	0.234
All women in labour (n=99)	n=9; 7.18±0.13	n=21; 7.18±0.13	n=69; 7.22±0.12	0.381

None of the individual elements of the fetal heart rate interpretation on CTG showed any significant association with fetal cord blood acidaemia (pH<7.1) (Table 7). However, the presence of abnormal contractions was positively associated with cord blood acidaemia (p=0.04; odds ratio 3.72 with 95% confidence interval 0.99-13.10).

For decelerations, normal, non-reassuring and abnormal patterns were followed by cord blood pH values of 7.19, 7.21 and 7.21 respectively (p=0.879). For neonates with a cord blood pH of 7.1 or more (n=82), 58 CTGs (71%) were interpreted as abnormal (false positives).

Table 7. Interpretation of separate cardiotocograph (CTG) elements and overall interpretation, associated with cord blood acidaemia (pH<7.1).

Element	Classification	pH<7.1 (n=17)	pH≥7.1 (n=82)	P-value
Contractions	Normal	10	69	0.040
	Abnormal	7	13	
Baseline fetal heart rate	Normal	13	61	1.000
	Non-reassuring	3	14	
	Abnormal	1	7	
Baseline fetal heart rate variability	Normal	12	63	0.453
	Non-reassuring	4	17	
	Abnormal	1	2	
Accelerations	Present	3	25	0.382
	Absent	14	57	
Decelerations	Normal	3	10	0.656
	Non-reassuring	4	27	
	Abnormal	10	45	
Overall interpretation	Normal	2	7	0.754
	Non-reassuring	4	17	
	Abnormal	11	58	

5. DISCUSSION

In our study we used the NICE guidelines to interpret all CTGs, the registrars had interpreted all CTGs as abnormal and that is the reason they had booked the patients for caesarean section for fetal distress. The senior obstetrician found 9 CTGs to be normal therefore 9 CS were actually not warranted, if the only reason was fetal distress. But in this study some caesarean sections had more indications than fetal distress.

The Apgar scores correlated with the pH values. In this study the mean pH was 7.20±0.12, this is comparable to a number of studies. Modarressnejad et al, defined acidosis as a cord blood pH of <7.1.[42] Also in our study we described acidosis as cord blood pH of < 7.1. Kaban et al described acidosis as pH of <7.2.[43] Only 17% of cases had neonatal acidosis in our study, in a similar study by Aboulghar et al 34% neonates were acidotic.[31] This shows that the CTG is not a good marker of fetal acidosis.

In our study we concentrated more on the arterial cord blood pH, but we did document base deficit and lactate measurements. The pH, base deficit and lactate measurements correlated well. The choice of using pH was decided on consensus with the researcher's supervisors and a neonatologist in Rahima Moosa Hospital.

We wanted to see if there is a good correlation between CTG findings and neonatal outcomes. In our study there was poor correlation between CTGs and neonatal outcomes, there were no HIE cases and there were only 18% admissions for observation. This is not surprising at all given the poor specificity of CTGs as shown in a study by Nelson et al, where in the false positive rate of CTGs was shown to be as high as 98%.[35] This shows that the CTG is not the best predictor of neonatal outcome.

We currently don't know what could have happened if the CSs were not done after recognition of abnormal CTGs, the outcomes could have been the same or not. But, given that some infants had low pH, it seems that not doing CTGs and not doing CSs could well have resulted in a small number being damaged or dying. Only a randomised trial, where in patients are randomised to receive caesarean section vs not getting caesarean section for abnormal CTGs, (which would be unethical) could solve that problem.

Other intrapartum tests for fetal wellbeing including STAN has not helped that much, nor has fetal scalp blood sampling. Both are difficult and/or unaffordable. A meta-analysis was done by Potti et al which showed no difference in perinatal outcomes between STAN with CTG or CTG alone.[61]

CTG compared to IA hasn't been shown to be superior as it was shown in a systemic review in 2017.The review included 13 randomized trials with over 37 000 high and low-risk pregnancies. There were no significant differences between the two methods, in terms of acidaemia after delivery, Apgar scores at 5 minutes, neonatal ICU admission, hypoxic-ischaemic encephalopathy, perinatal mortality and neurodevelopmental impairment.[53] This clearly indicates that a different test is worth investigating or researching, which can detect fetal acidaemia.

Baseline beat to beat variability has been shown to be a good predictor of poor acid-base status of the fetuses, this was shown in a study by Williams et al, who showed that in the presence of minimal/absent variability (amplitude <5) for at least an hour, the incidence of significant acidemia (pH <7.0) was increased.[63] In our study decreased variability was poorly associated with neonatal acidosis, only 1 out of 17 (5.8%) had abnormal variability in the acidotic group.

Sometimes CTG might appear normal but the fetus at that time is acidotic, in our study there were only 2 out of 17 (12%) of CTGs that were interpreted as normal but the neonates had acidosis, this shows the low false-negative rate of CTGs in detection of acidosis. This correlated with the study by Steer et al, where only 4 out of 24 (17%) of neonates who had severe acidosis had normal CTGs.[25] In our study 75 out of 82 (CTGs were interpreted as reassuring or abnormal but the pH was normal (>7.1), this shows a false-positive rate of 91%.

We also wanted to assess how long it takes for a caesarean section to be done once the diagnosis of fetal distress was made at Rahima Moosa Mother and Child hospital. In this study some of the cases took very long to be delivered following the recognition of CTG abnormalities. This may have caused bias because acidosis progressively worsens during

labour. Had these operations been started earlier, the number of acidotic neonates would have likely been smaller.

The decision to delivery interval varied in our cases with an average of 87 minutes which is well beyond the recommended interval, this can be explained by lack of resources and increased number of patients that are seen at Rahima Moosa Mother and child hospital. In a study in South Africa by Hall et al, the mean DDI was 52.9 which is slightly better than what we had.[3]

Conclusion

The CTG remains a very important tool in obstetrics practice, in our study some neonates were severely acidotic and if the caesarean sections were not performed when they did the outcomes could have been detrimental. Our study is limited by small numbers, larger studies similar to this one are needed in order to give a better view of these problems, and hopefully shed some light on what could be done to improve the sensitivity of CTGs.

Neonatal death and neonatal encephalopathy, are rare events, so that using only 99 subjects might miss devastating outcomes and these patients were not later followed up to see if some ended up as late neonatal deaths. It easy for us to miss HIE and fetal death cases because of these small numbers in our study.

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7.APPENDIX

Appendx A: NICE guidelines of CTG interpretation:

Feature		Definition
1. Baseline FHR • Moderate bradycardia • Moderate tachycardia • Abnormal bradycardia • Abnormal tachycardia		The mean level of the FHR in bpm when it is stable over 5-10 minutes • 100-109bpm • 160-179bpm • <100bpm • >180bpm
2. Baseline Variability		The difference in bpm between the highest peak and the lowest trough of fluctuation in a 1-minute segment.
3. Accelerations		Transient increases in FHR of 15bpm or more for 15 seconds/more
4.Decelerations		Transient decreases in FHR of 15bpm or more for 15 second/more
	Early	➢ Uniform, repetitive decelerations with onset and recovery that Coincide with the beginning and end of a contraction.
	Late	➢ Uniform, repetitive decelerations with onset mid to end of the contraction, nadir more than 20 seconds after the peak of the contraction and ending after the contraction.
	Typical variable	➢ Intermittent with variable shapes and /or time relationships with contractions and a rapid recovery.
	Atypical Variable	➢ Variable decelerations with loss of primary or secondary rise in baseline FHR, slow recovery or prolonged secondary rise in baseline or continuation of baseline at a lower level.
5. Sinusoidal pattern		A regular oscillation of the FHR resembling a sine wave that is smooth, lasts for at least10 minutes, and has a fixed period of 3-5 cycles per minute amplitude of 5-15bpm, absent variability. If normal variability is present, the pattern is called ‘pseudo-sinusoidal’.

CATEGORISATION OF FHR FEATURES:

Category	Baseline FHR (bpm)	Variability (bpm)	Decelerations	Accelerations
Normal OR reassuring	110-160	5 or more	None or early	Present
Non-reassuring	100-109 161-180	<5 for 30-90 minutes	Variable decelerations: 1. Dropping from baseline by 60 bpm/less and taking over 60 seconds to recover. 2. present for over 90 min 3. Occurring with over 50% of contractions **OR** Variable decelerations: 1. Dropping from baseline by >60bpm or taking over 60 seconds to recover 2. Present for up to 30 minutes 3. Occurring with over 50% of contractions **OR** Late decelerations: 1. Present for over 30 min 2. Occurring with over 50% of contractions	Absence of accelerations is of uncertain significance in an otherwise normal CTG
Abnormal	<100 >180	<5 for >90 minutes	Non-reassuring variable decelerations(see row above): 1.Still observed 30 min after starting conservative measures 2. Occurring with over 50% of contractions **OR** Late decelerations - Present for over 30 min -Do not improve with conservative measures - Occurring with over 50% of contractions **OR** Bradycardia or a single prolonged deceleration lasting 3 minutes or more.	

Appendix B: Data Sheet

1. Study number:
2. Age in completed years at delivery:
3. Parity before delivery:
4. Gravidity after delivery:
5. Gestational age at delivery in completed weeks:
6. Was labour: Spontaneous Yes/No

   Induced Yes/No
7. Date of delivery:
8. Last cervical dilatation before delivery:
9. Interval between the end of CTG and delivery in minutes:
10. Maternal fever (>380C in labour): Yes/No
11. Cord Prolapse: Yes/No
12. Ruptured Uterus: Yes/No
13. Antepartum haemorrhage: Yes/No
14. Diabetes: Gestational DM Yes/No

    Type 1 Yes/No

    Type 2 Yes/NO
15. Hypertension: Pre-eclampsia (hypertension arising after 20 weeks of gestation and returning to normal within 3 months postpartum with one or more of the following: 1proteinuria 2. Renal dysfunction 3. Liver disease 4. Neurological problems 5 heamatological disturbances 6fetal growth restriction): Yes/No

    Chronic Hypertension Yes/No

    Gestational Hypertension Yes/No

16. Maternal collapse: Yes/No

17. Previous Caesarean section: Yes/No

18. HIV status: Positive/negative/unknown

If positive: ARV Yes/No

CD4

19. Apgar Score: 1 min:

5 min:

Birth weight:

20. Neonatal resuscitation: Bag-mask Yes/No

Intubation Yes/No

CPR Yes/No

21. Meconium stained liquor: Present/Absent

If present: Grade 1, Grade 2, Grade 3, Offensive

22. Admission: NICU Yes/No

Neonatal High care Yes/No

For observation Yes/No

For cooling: Yes/No

23. Cord Blood gas: PH

pCO2

pO2

Lactate

Bicarbonate

Base excess

24. Time interval between delivery and cord blood gas in minutes:

25. Prolonged pregnancy (GA >42 completed weeks): Yes/No

26. Augmentation of labour: Yes/No

27. CTG photos interpreted by senior obstetrician using the NICE guidelines:

Appendix C

R14/49 Dr Khathutshelo A. Tshikanda

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL)

CLEARANCE CERTIFICATE NO. M161020

NAME: (Principal Investigator)	Dr Khathutshelo A. Tshikanda
DEPARTMENT:	Obstetrics and Gynaecology Rahima Moosa Mother and Child Hospital
PROJECT TITLE:	Correlating Cardiotocography, Apgar Score and Cord Blood PH After Caesarean Section for Fetal Distress at Rahima Moosa Hospital
DATE CONSIDERED:	28/10/2016
DECISION:	Approved unconditionally
CONDITIONS:	
SUPERVISOR:	Prof E. Buchmann
APPROVED BY:	Professor P Cleaton-Jones, Chairperson, HREC (Medical)
DATE OF APPROVAL:	08/02/2017

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and **ONE COPY** returned to the Research Office Secretary in Room 301, Third Floor, Faculty of Health Sciences, Phillip Tobias Building, 29 Princess of Wales Terrace, Parktown, 2193, University of the Witwatersrand. I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to resubmit the application to the Committee. **I agree to submit a yearly progress report**. The date for annual re-certification will be one year after the date of convened meeting where the study was initially reviewed. In this case, the study was initially reviewed in October and will therefore be due in the month of October each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).

Principal Investigator Signature

Date 10/02/2017

PLEASE QUOTE THE PROTOCOL NUMBER IN ALL ENQUIRIES

Appendix D

GAUTENG PROVINCE
HEALTH
REPUBLIC OF SOUTH AFRICA

RAHIMA MOOSA MOTHER AND CHILD HOSPITAL

Enquiries : Dr Edward Hank
Tel : (011) 470 9030/1
Fax : (011) 477 4117
Email : Edward.Hank@gauteng.gov.za

Department of Obstetrics and Gynaecology
University of the Witwatersrand

Dear Dr Tshikanda,

RE: CORRELATING CARDIOTOCOGRAPHY, APGAR SCORES AND CORD BLOOD PH AFTER CEASAREAN SECTIONS FOR FETAL DISTRESS IN RAHIMA MOOSA MOTHER AND CHILD HOSPITAL

Permission is granted for you to conduct the research as indicated in the title above.

The terms under which this permission is granted is contained in the Researcher Declaration form that you have signed. Failure to comply with these conditions will result in the withdrawal of such permission.

It is crucial for you to inform the Research Coordinator, Karen Marshall of the actual start and end dates of your study. This could be done by e-mail.

Should the study commence more than 12 months after receipt of this approval letter you will have to go through the process of applying again.

You are strongly advised to keep a signed copy of the declaration form so as to ensure that the terms of this agreement are complied with at all times.

Yours sincerely,

DR EDWARD HANK
Clinical Manager
2016:11:24

ADDRESS: Cnr. FUEL & OUDSTHOORN STREET CORONATIONVILLE 2093 / PRIVATE BAG X20 NEWCLARE 2112 JHB