

Intra-partum Fetal Monitoring – Cardiotocograph

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INTRODUCTION

The course of labour is the first challenge one ever undertakes. The uterine contractions during labour exposes the fetus to a possible risk of hypoxic brain injury due to repeated cord compression or reduction of utero-placental perfusion¹. Intrapartum fetal surveillance evolved with the principal aim of preventing adverse perinatal outcomes arising from fetal metabolic acidosis / cerebral hypoxia related to labour. However, the severity of an asphyxial injury is influenced by many factors (e.g. tissue perfusion, tissue substrate availability, fetal condition prior to the insult, duration of the insult and the severity of the insult). Therefore, the relationship between metabolic acidosis and cerebral injury is complex. Furthermore, it is clear that very often damage is actually sustained during pregnancy, prior to labour, rather than arising *de novo* during labour and delivery². In spite of this, intrapartum fetal surveillance for early detection of fetal hypoxia has become a key component of modern maternity care. Intrapartum fetal surveillance was traditionally carried out by intermittent auscultation (IA) of the fetal heart rate (FHR). This approach would be adequate to monitor a fetus at low risk of compromise, but may be inadequate for high-risk pregnancies. Therefore, the use of intrapartum electronic fetal monitoring (EFM) with cardiotocography (CTG), has steadily increased over the last three decades in an attempt to reduce the incidence of intrapartum fetal morbidity and mortality.

For low-risk women in labour, it has

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Table 1: Antenatal risk factors which would justify continuous intrapartum cardiotocography -adapted from RANZCOG Guidelines 2014 (2)

Abnormal antenatal CTG
Abnormal Umbilical Artery Doppler velocimetry
Suspected or confirmed fetal growth restriction
Oligohydroamnios or polyhydroamnios
Prolonged pregnancy ≥ 41 weeks
Multiple pregnancy
Breech presentation
Antepartum haemorrhage
Prolonged rupture of membranes
Known fetal abnormality requiring monitoring
Uterine scar
Hypertensive pregnancy
Hyperglycaemia in pregnancy requiring medication, or if it is poorly controlled, or if associated with macrosomia
Current or previous conditions which constitute a risk of fetal compromise e.g. cholestasis, isoimmunisation, substance abuse
Significantly reduced fetal movements preceding labour
Morbid obesity BMI ≥ 40
Maternal age ≥ 40

Table 2: Intrapartum factors which would justify continuous cardiotocography (2).

Abnormality detected on IA or CTG
Induction of labour with prostaglandin / oxytocin
Oxytocin augmentation
Regional anesthesia and paracervical block
Abnormal vaginal bleeding in labour
Maternal pyrexia ≥ 38 c
Absent liquor following amniotomy
Meconium or blood-stained liquor
Prolonged first stage of labour
Prolonged second stage of labour
Pre-term labour
Tachysystole
Uterine hypertonus
Uterine hyperstimulation

been suggested that the only clinically significant benefit from the use of routine continuous EFM in comparison to IA was in the reduction of neonatal seizures but with no statistically significant improvement in long-term outcomes such as cerebral palsy, although it increased the caesarean section and operative vaginal delivery rates³. Therefore, the use of continuous CTG for low risk pregnancies is not recommended⁴. It is also widely appreciated that there are still shortcomings in interpretation of CTG which is evident by the reviews of cases with poor perinatal outcomes⁵.

In order to improve the interpretation of CTG, it is important to understand the control of the fetal heart rate by the sinoatrial node, sympathetic and para-sympathetic autonomic nervous systems, baroreceptors, chemoreceptors, catecholamines and cardio-regulatory center. In addition to that, the physiology of fetal oxygenation and how the fetus reacts to hypoxia, the types of intra partum hypoxia (gradual, subacute, acute, chronic), the pathophysiological basis for CTG abnormalities, and the principles of EFM should also be clearly understood for decision making on intrapartum CTG^{6,7}. Because of the inherent limitations of the CTG, especially its poor specificity, newer techniques such as analysis of the ST segment of the fetal electrocardiograph (STAN), the calculation of the Fetal Physiological Score (FPS), and computer assisted interpretation of CTG are being studied⁸.

CARDIOTOCOGRAPHY

An Intrapartum CTG provides information about the fetal condition. A normal trace indicates a well-oxygenated fetus but an abnormal trace has poor specificity with upto 60% false positive rates being reported⁹. It has been suggested that FHR accelerations are a sign of a neurologically responsive fetus that does not have hypoxia/acidosis. However, the absence of accelerations in an otherwise normal CTG is of uncertain significance but it is unlikely to indicate hypoxia/acidosis¹⁰.

A mistake which could occur with intrapartum CTG is the recording of maternal heart rate, especially in the second stage of labour. A change in the pattern, sudden change in baseline rate and

accelerations coinciding with contractions may help in the differentiation from FHR.

TACHYSYSTOLE AND UTERINE HYPERTONUS

More than five uterine contractions in 10 minutes without FHR abnormalities is defined as tachysystole. Contractions lasting >2 minutes in duration or contractions occurring within 60 seconds of each other, without FHR abnormalities is referred to as uterine hypertonus². The management of these conditions includes reduction or cessation of oxytocin infusion and consideration of tocolysis.

UTERINE HYPERSTIMULATION

Tachysystole or uterine hypertonus in the presence of FHR abnormalities is defined as uterine hyperstimulation². If conservative measures such as cessation of oxytocin and tocolysis fail, urgent delivery needs to be considered. Commonly used protocols for acute tocolysis include terbutaline 250mcg iv or subcutaneous, salbutamol 100mcg iv, and glyceryl tri nitrate spray 400mcg sublingual.

REDUCED VARIABILITY

A bandwidth amplitude < 5 bpm for > 50 minutes in baseline segments, or for more than 3 minutes during decelerations indicates reduced variability. It can occur due to central nervous system hypoxia/acidosis, but it can also be seen with any condition which causes central nervous system (CNS) depression namely; deep fetal sleep, previous cerebral injury, infection, administration of CNS depressants or parasympathetic blockers, prematurity, and fetal abnormalities¹⁰.

INCREASED VARIABILITY (SALTATORY PATTERN)

A bandwidth of > 25 bpm lasting > 30 minutes indicates increased variability. Although its pathophysiology is poorly understood, it is thought to be due to a hyperactive fetal autonomic nervous system. It may be seen in fetal hypoxia associated with decelerations (10).

FETAL TACHYCARDIA

A baseline FHR > 160 bpm lasting > 10 minutes is considered as fetal tachycardia but is not associated with hypoxia in the presence of accelerations or with normal variability and absent decelerations¹⁰. Maternal fever and tachycardia, sympathomimetic medications (eg. terbutaline and salbutamol) chorioamnionitis, fetal tachyarrhythmias, a high inherent rate due to prematurity are some of the known causes of fetal tachycardia apart from fetal hypoxia.

FETAL BRADYCARDIA

A baseline value < 110 bpm lasting > 10 minutes is considered as fetal bradycardia. However a baseline FHR between 100 and 109 bpm with normal baseline variability and no variable or late decelerations should not prompt any further action¹¹. The causes of fetal bradycardia include low inherent rate, medications (e.g. local anesthetics), maternal hypothermia, maternal hypotension, fetal heart conduction defects, prolonged umbilical cord compression, and sustained hypoxia¹⁰.

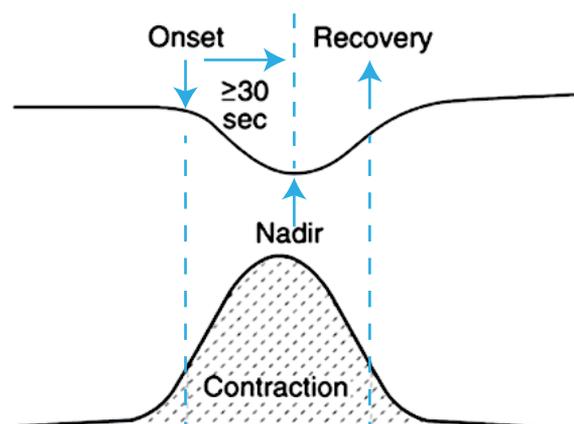


Figure 01: Early deceleration

DECELERATIONS

Decelerations are defined as decreases in the FHR of > 15 bpm below the baseline, and lasting > 15 seconds. Early decelerations are uniform in shape and they start and finish with the contraction. They probably occur due to head compression and usually the decrease is < 20 beats from baseline. They do not indicate fetal hypoxia or acidosis.

VARIABLE DECELERATIONS

Variable decelerations represent a baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression. They are variable in shape, depth, duration and timing with the contractions and exhibit a good variability within the deceleration. They typically have a rapid onset (onset to nadir < 30 seconds) and a rapid recovery to baseline. Variable decelerations constitute the majority of decelerations during labour. They are seldom associated with an important degree of fetal hypoxia/acidosis. Increases in FHR immediately before and after a variable deceleration have been referred to as “shoulders.” These increases can be visually similar to accelerations, and this led to speculations that they had a similar predictive value. However, there is inadequate evidence to support the notion that “shoulders” have the same predictive value as accelerations¹².

Variable decelerations were formerly categorized as ‘typical’ if they were considered to be normal and not indicating fetal hypoxia and ‘atypical’ if they were considered to be abnormal and indicating probable fetal hypoxia. Although the terms ‘typical’ and ‘atypical’ are not currently used, the non-reassuring features in variable decelerations which require appropriate action, as they indicate the likelihood of fetal hypoxia, have been clearly described^{2,6,11}. These non-reassuring features which are also referred to as ‘complicated variable decelerations’ include a persistently large amplitude (> 60 bpm in depth) and / or long duration (> 60 seconds duration), a slow return to the baseline after the contraction, smooth (with no baseline variability) post-deceleration shoulders (“overshoots”) often associated with a rising baseline or a baseline tachycardia and a reduced baseline variability⁶.

Variable Deceleration

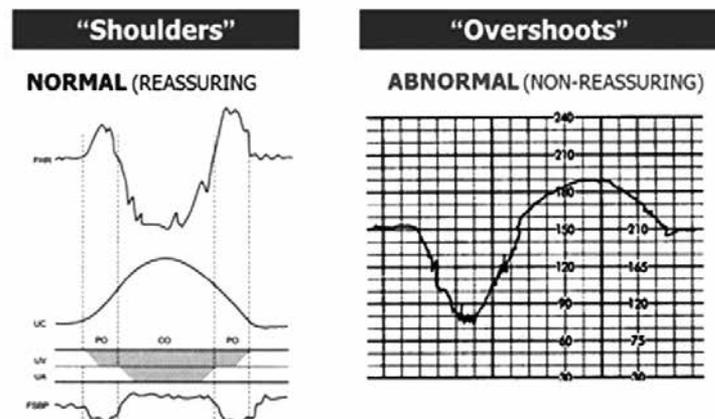


Figure 02: Shoulders and Overshoots in variable decelerations

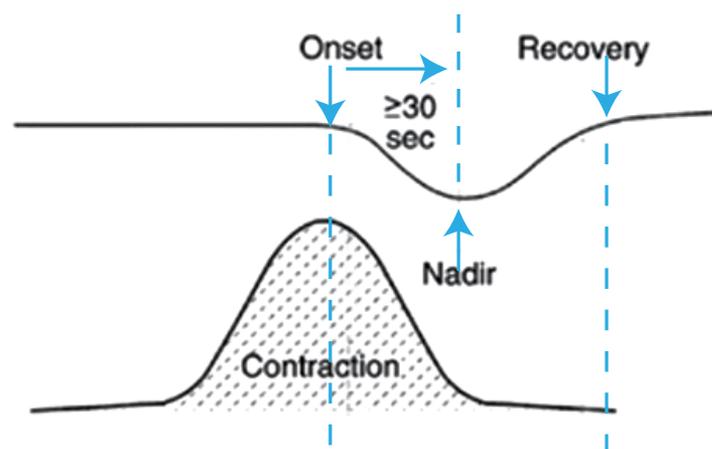


Figure 03: Late deceleration

LATE DECELERATIONS

Late decelerations are caused by contractions in the presence of fetal hypoxia. These are uniform and repetitive. Late decelerations start after the start of the contraction and the nadir of the deceleration is more than 30 seconds after the peak of the contraction. They return to the baseline after the contraction has finished. Late decelerations of any depth are significant and should be immediately attended to. In fact shallow decelerations (with decreases of < 10 bpm) with reduced baseline variability are particularly dangerous and indicate significant fetal hypoxia. These can even be detected with careful IA of FHR immediately after uterine contractions, but not in between uterine contractions when the FHR has returned to its normal baseline

PROLONGED DECELERATIONS

Decelerations lasting > 3 minutes are defined as prolonged decelerations. They may indicate chemoreceptor-mediated hypoxaemia. Decelerations exceeding 5 minutes, with FHR less than 80 bpm and reduced variability within the deceleration are frequently associated with acute fetal hypoxia/acidosis and require urgent intervention¹⁰.

SINUSOIDAL PATTERN

This is an oscillating pattern resembling a sine wave (very smooth with a regular cycle rate). It has a relatively fixed period of 3-5 cycles per minute and typically an amplitude of 5-15 beats. Its pathophysiology is poorly understood but

it may be seen with severe fetal anemia (fetal-maternal haemorrhage, twin-to-twin transfusion syndrome, anti-D alloimmunization and vasa previa) It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus, and gastroschisis¹⁰.

There is agreement about baseline FHR and fetal tachycardia, with FIGO describing a time frame too. Although NICE Guidelines describe FHR of 160–180 bpm as non-reassuring and FHR > 180 bpm as pathological, FIGO Guidelines do not sub categorize fetal tachycardia in this manner. A fetal tachycardia (FHR > 160 bpm) may be secondary to fetal compensatory response to evolving hypoxia. A mere increase in

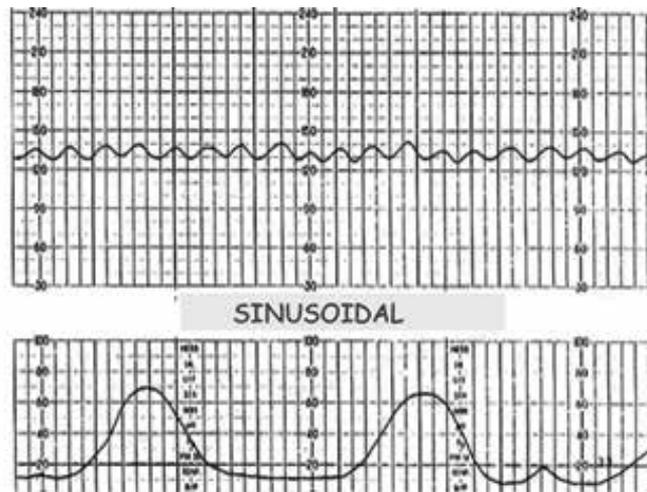


Figure 04: Sinusoidal pattern

Table 3. Comparison of guidelines on cardiotocography

Category	FIGO	NICE	RANZCOG
Baseline heart rate	110-160 bpm	110-160 bpm	110-160 bpm
Tachycardia	>160 bpm for more than 10 minutes	>160 bpm	>160 bpm
Bradycardia	<110 bpm for more than 10 minutes	<110 bpm	<110 bpm
Normal Baseline variability	5 – 25 bpm	5 bpm or more	6 – 25 bpm
Reduced variability	<5 bpm for more than 50 minutes	<5 bpm for 30 – 90 minutes (non-reassuring) <5 bpm for more than 90 minutes (abnormal)	3 – 5 bpm
Absent variability	no comment	no comment	< 3 bpm
Increased variability	>25 bpm for more than 30 minutes	no comment	>25 bpm
Accelerations	The presence of accelerations denotes a fetus that does not have hypoxia/acidosis, but their absence during labor is of uncertain significance	The presence of fetal heart rate accelerations is generally a sign that the baby is healthy. The absence of accelerations in an otherwise normal cardiotocograph does not indicate acidosis	Absence of accelerations in isolation is unlikely to be associated with fetal compromise
Prolonged decelerations	Deceleration >3 minutes	Deceleration >3 minutes	Decelerations > 90 seconds but less than 5 minutes

the baseline FHR > 160 bpm by itself is not associated with fetal compromise as it could be physiological (preterm due to the immaturity of the parasympathetic nervous system), or could occur secondary to maternal dehydration, drugs (eg. sympathomimetics). However, any increase in the fetal heart rate associated with changes in baseline variability is considered to be abnormal. In addition, if the original baseline fetal heart rate was 105 bpm, even an increase in FHR to 145 bpm should be considered abnormal for that fetus.

The FIGO Guidelines consider FHR < 100 bpm as pathological and RANZCOG Guidelines also describe prolonged bradycardia of FHR < 100 bpm for more than 5 minutes as a likely feature of fetal compromise. It has been suggested that a stable baseline fetal heart rate between 90 and 99 bpm with normal baseline variability (having confirmed that this is not the maternal heart rate) may not be pathological¹¹. However, it is essential that patients with such CTGs are evaluated properly by an experienced senior obstetrician.

There is no significant difference in the definitions of normal baseline variability and reduced variability, between the different guidelines. However, the NICE Guidelines further categorize reduced baseline variability being non-reassuring or abnormal according to the duration, which would be more useful in clinical decision making.

INTRAPARTUM FHR INTERPRETATION AND MANAGEMENT; A STEP-WISE PHYSIOLOGIC APPROACH

An Intrapartum CTG, just like all investigations / tests in clinical practice, should be interpreted in the background of the total clinical picture which should include the patient's clinical details, indication for EFM, previous CTGs, and results of all previous other investigations including sonological assessments. All the other information such as medications, oxytocin infusions, stage of labour and progress of labour must also be considered. **MOTHERS** is a mnemonic which could be useful when considering the clinical picture; **M**econium stained liquor, use of **O**xytocin, maternal **T**emperature

(infection), **H**yperstimulation / **H**aemorrhage, **E**pidural, **R**ate of progress in labour, and **S**car³.

STEP 1—THE NORMAL AND THE ABNORMAL INITIAL CTG

If the CTG is normal the fetus is very likely to be neurologically intact, normoxic, without acidaemia or acidosis, at low risk of intrapartum asphyxia, and is able to react and defend itself against intrapartum hypoxia. Surveillance may continue depending on the situation or the woman may be monitored by IA¹³.

STEP 2—RECOGNITION OF THE COMPENSATED AND THE DECOMPENSATING FETUS

An intact fetus with a previously normal CTG will exhibit predictable patterns of FHR responses if exposed to hypoxic ischaemic insults during labour. Based on the intensity and duration of hypoxic stress during labour, three types of intrapartum hypoxia had been described, namely: gradually evolving hypoxia; subacute hypoxia; and acute hypoxia. The management should be tailed according to the type of hypoxia to optimize fetal outcome⁷.

GRADUALLY EVOLVING HYPOXIA

The hypoxic stress evolves over time (hours) giving the fetus time to use its compensatory mechanisms effectively in order to prevent hypoxic damage. The first feature on CTG is the presence of decelerations with contractions. If the hypoxic insult continues, the decelerations will be followed by ABCDE.

Disappearance of Accelerations

Increase in Baseline heart rate

Compensated stress

Decompensation

End stage

The accelerations disappear in order to conserve oxygen and energy substrates in the fetus. The catecholamine release increases the FHR and cardiac output to maintain perfusion to the vital organs. Therefore, a sustained FHR tachycardia in association with uterine contractions is a sensitive marker of a compensatory

increase in fetal cardiac output. If the hypoxic insult continues, depending on the fetal reserve and the intensity and duration of hypoxia, fetal decompensation may ensue. When the perfusion to the brain is compromised, loss in the baseline variability would be observed in the CTG trace. Finally, myocardial hypoxia and acidosis may lead to a terminal bradycardia resulting in the 'step-ladder pattern to death'⁷.

The management should be tailored according to the level of hypoxia. In the presence of a stable baseline in between decelerations and normal baseline variability, labour can be continued with continuous CTG monitoring. If raised baseline and or abnormal variable or late decelerations appear on the CTG, care should be taken to improve fetal environment (intrauterine resuscitation) which may include stopping or reducing oxytocin, iv fluids and placing patient in the left lateral position. If baseline variability is reduced despite conservative measures, immediate delivery should be considered⁷.

ACUTE HYPOXIA

This is characterized by a sudden drop in the baseline heart rate, which is also known as a 'single prolonged deceleration'. This could be either suspicious (lasting for < 3 minutes and returning to the normal baseline with good variability) or abnormal (lasting for > 3 minutes)

First, it is essential to exclude three major intrapartum accidents (placental abruption, umbilical cord prolapse and uterine rupture) and two iatrogenic causes (hyper-stimulation due to oxytocin or prostaglandins and maternal hypotension usually secondary to supine hypotension or epidural analgesia). In case of intrapartum accidents, delivery should be expedited via the safest and quickest way to save the fetus.

If acute hypoxia is considered to be due to an oxytocin infusion, 'intrauterine resuscitation' should be initiated immediately. In the presence of normal variability prior to deceleration and within the first three minutes of deceleration and the three accidents mentioned above are absent, unto 90% of the prolonged decelerations have been reported to recover by 6 minutes and 95% by 9 minutes¹⁴.

In case of acute hypoxia occurring in the absence of intrapartum accidents or iatrogenic causes, the '3, 6, 9, 12 and 15-minute' rule, which includes the following, should be applied: if a normal baseline variability has been noted before the onset of deceleration and within the first 3 min of the deceleration, appropriate intrauterine resuscitation by 6 min, moving the patient to a theatre by 9 min, and if the CTG shows no signs of recovery, commencing delivery by 12 min with the aim of delivering the baby by 15 min¹⁵.

Reduced baseline variability before or within the first 3 min of the deceleration, repetitive late decelerations before the onset of the prolonged deceleration or a drop in the heart rate to >60 bpm are associated with a poor outcome. In these circumstances, the '3, 6, 9, 12 and 15-minute' rule should not be applied, and immediate delivery should be undertaken⁷.

SUBACUTE HYPOXIA

This is characterized by complicated variable decelerations, with the amplitude of the deceleration ≥ 60 bpm and lasting for ≥ 90 seconds. When the FHR returns to its baseline in subacute hypoxia, it spends less than 30 seconds at the baseline level before the onset of the next deceleration^{7,16}. Therefore, the time available at the baseline to wash off the acid and carbon dioxide and to obtain fresh oxygenated blood from the placenta becomes progressively shorter. Therefore, a rapidly cumulative build up of CO₂ takes place which results in an initial respiratory acidosis and a subsequent metabolic acidosis. The baseline FHR may remain within the normal range (110-160 bpm) as the fetus is unable to raise its baseline heart rate because of the short duration of time spent at the baseline in between two decelerations. Subacute hypoxia is associated with a rapid decline

in pH, usually at the rate of 0.01 every 2-4 minutes, in contrast to the gradually evolving hypoxia¹⁶.

Once subacute hypoxia is established, there is likely to be insufficient time to obtain, analyze and react to a fetal blood sample result without the risk of severe acidaemia. Therefore, in clinical practice, it is crucial to recognize this pattern¹⁶.

The management involves in utero resuscitation and discouraging active maternal pushing for the next few contractions to ensure oxygenation of placental venous sinuses. Immediate delivery should be considered if changes are not reversed with conservative measures⁷.

CHRONIC HYPOXIA

In this situation, the fetus has been exposed to a prolonged period of hypoxia with or without resultant neurological injury during the antenatal period before the onset of labour. This happens usually secondary to chronic utero-placental insufficiency. The fetus adapts several compensatory mechanisms for survival including reduction in growth, movements and diversion of oxygenated blood and nutrients from non-vital organs to supply the vital organs⁷.

The affected fetus may be identified by the features observed on the CTG; increase in the baseline rate with reduced variability and the presence of shallow decelerations. Even though some degree of cerebral damage may have already taken place, the presence of this CTG pattern requires immediate delivery. This is because, there will be further reduction in oxygenation of the fetus with the onset of labour due to uterine contractions, intermittent umbilical cord compression and reduction in utero-placental circulation. This will eventually lead to hypoxic ischaemic encephalopathy, terminal bradycardia and

fetal death⁷.

FETAL SCALP BLOOD SAMPLING (FBS)

This is a test to assess the acid-base status of the fetus. The units employing EFM are encouraged to have access to FBS facilities to aid in the management of labours where fetus shows equivocal CTG changes. FBS can be assessed for pH and lactate levels. The incidence of false positives from an abnormal CTG can be reduced with FBS. A reduction in the total caesarean section rate from 18% to 11% and caesarean section indicated by fetal distress from 7% to 3%, when fetal scalp blood sampling was allowed, has been reported¹⁷.

The most important question is the identification of the appropriate clinical situation for FBS. Delivery must be expedited and FBS should not be undertaken, if there is clear evidence of serious and/or sustained fetal compromise. Delivery also needs to be expedited if CTG abnormalities are of a degree requiring further assessment, but FBS is unavailable or contraindicated.

CONTRAINDICATIONS TO FBS

1. Evidence of serious, sustained fetal compromise.
2. Fetal bleeding disorders (e.g. suspected fetal thrombocytopenia, haemophilia).
3. Face or brow presentation.
4. Less than 34 weeks of gestation.

Maternal infection.

(e.g. HIV, hepatitis B, hepatitis C, herpes simplex virus and suspected intrauterine sepsis).
Group B Streptococcus carrier status does not preclude FBS².

If facilities are available, for medico legal

Table 04: Normal and abnormal values of pH and Lactate on Fetal Scalp Blood Sampling (2, 11)

pH	Lactate	Management
≥ 7.25	≤ 4.1 mmol/l	Normal
7.24 – 7.21	4.2 – 4.8 mmol/l	Suspicious (Repeat in 30 minutes)
≤ 7.20	≥ 4.9 mmol/l	Abnormal (Recommend delivery)

Types of Intrapartum Hypoxia

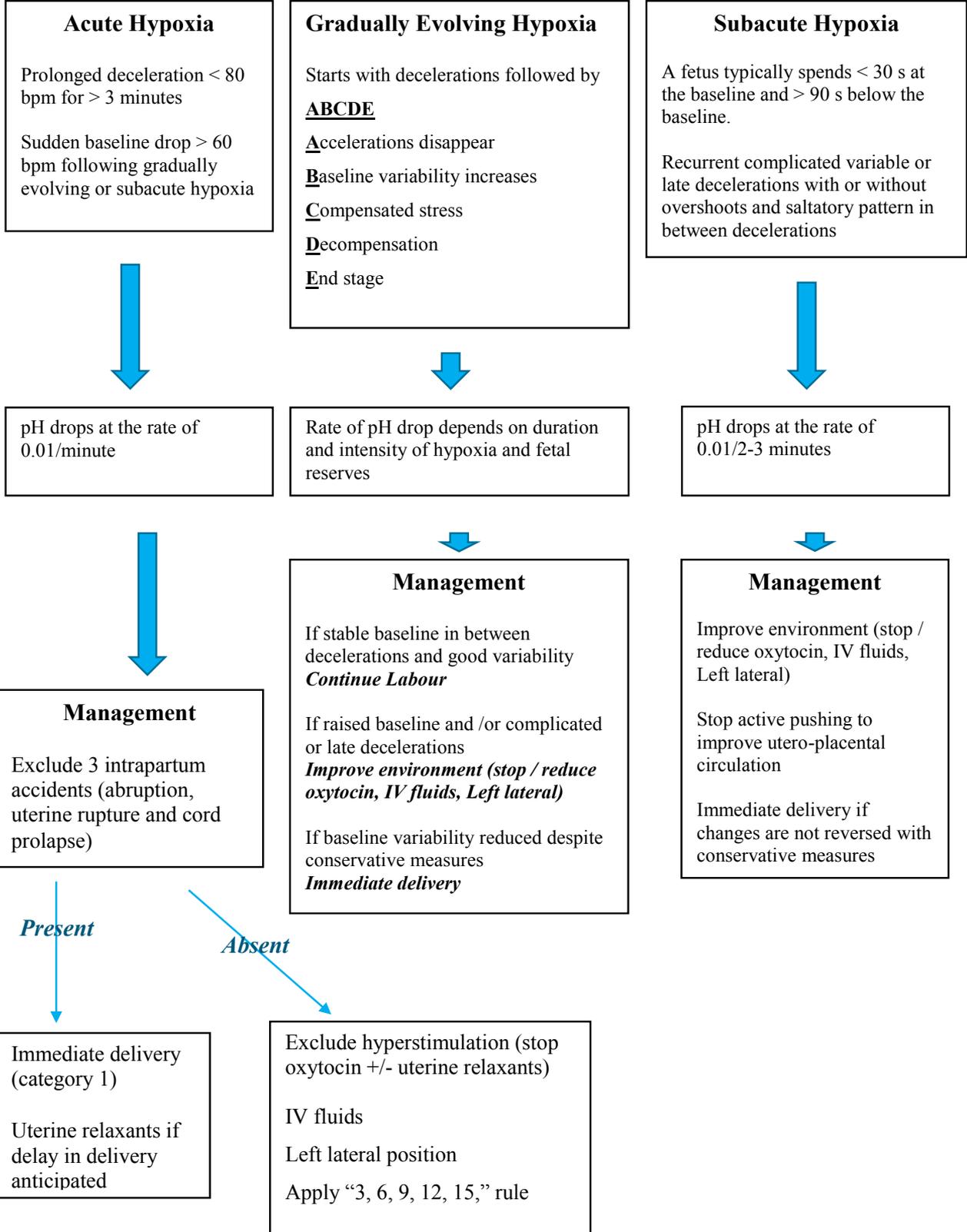


Figure 05: Types of Intrapartum Hypoxia (7)

purposes, paired (arterial and venous) umbilical cord blood gas and lactate analysis should be undertaken within one hour of delivery if any of the following are present:

- a. Apgar score < 4 at 1 minute.
- b. Apgar score < 7 at 5 minutes.
- c. Fetal scalp sampling performed in labour.
- d. Operative delivery undertaken for fetal compromise.

CONCLUSION

Intra partum care was expected to take a new turn with the introduction of EFM 30 years back. Decades later, however, the results are not too convincing. Despite questions about its efficacy and outcomes associated with its use, FHR monitoring continues to be the predominant method for intrapartum fetal surveillance. A considerable proportion of asphyxial injury is thought to occur before the start of labour and EFM may not offer additional benefit for those fetuses. The challenge of EFM is to identify early, those fetuses who are compromised and to intervene before injury occurs. The various mechanisms which control the fetal heart rate, the physiology of fetal oxygenation and how the fetus reacts to hypoxia, the types of intra partum hypoxia, the pathophysiological basis for CTG abnormalities, and the principles of EFM need to be well understood in order to improve the interpretation of CTG and facilitate intra partum decisions

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