



# Physiological

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# CTG Interpretation

## Intrapartum Fetal Monitoring Guideline

Published February 2018

### Disclaimer

This guideline describes fetal monitoring using physiology-based CTG interpretation. It has been developed by the editorial board based on the experience gained from maternity units where a reduction in the emergency caesarean section rate and/or an improvement in perinatal outcomes was demonstrated after the implementation of physiology-based fetal monitoring.

It is important to stress that fetal monitoring is only part of the overall clinical assessment of both mother and fetus, aimed mainly at the detection of fetal hypoxia. This guidance must be used within the context of the whole clinical picture, taking into account other non-hypoxic factors causing fetal injury. This is particularly important when events are evolving rapidly necessitating interventions irrespective of fetal monitoring.

This guidance is based on the evidence available to the editorial board at the time of creating this document, which are listed in the reference section of this document. We recognise that it is impossible for any guideline to cover every clinical scenario, hence it is important for clinicians using this guidance to apply it in accordance with their clinical expertise and logic, and to seek a second opinion whenever required.



## Acknowledgement

We would like to take this opportunity to express our gratitude to the fetal wellbeing team and all the maternity staff at St George's Hospital, Lewisham and Greenwich NHS Trust and Kingston Hospital. This guidance is built upon the foundation laid by their collective experiences, contributions and hard work. We dedicate this guideline to help improve the outcome of mothers and babies all over the world.

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The editorial board wishes to thank the international consensus panel of expert reviewers from 14 countries, who have embraced a physiological approach to CTG interpretation in their daily clinical practice. We are honoured to have Prof Sir Arulkumaran as a special invited expert reviewer of the physiology-based guideline on CTG interpretation. The editorial board would like to take this opportunity to acknowledge his immense contribution to intrapartum fetal monitoring, and especially, for disseminating the knowledge on fetal physiological response to intrapartum hypoxic stress through several of his publications.

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## Glossary of Abbreviations Used

AVD	Assisted Vaginal Delivery
APH	Antepartum Haemorrhage
bpm	Beats Per Minute
CEFM	Continuous Electronic Fetal Monitoring
CQC	Commission of Quality Control
CS	Caesarean Section
CSF	CerebroSpinal Fluid
CTG	Cardio-TocoGraph
DOB	Date of Birth
FBS	Fetal Scalp Blood sample
FH	Fetal Heart
FHR	Fetal Heart Rate
FIGO	International Federation of Gynaecology and Obstetrics
FSE	Fetal Scalp Electrode
FSS	Fetal Scalp Stimulation
GCP	Good Clinical Practice
IA	Intermittent Auscultation
IUGR	Intra-Uterine Growth Restriction
MAS	Meconium Aspiration Syndrome
MSL	Meconium Stained Liquor
NCC-WCH	National Collaborating Centre for Women's and Children's Health
NICE	National Institute of Clinical Excellence
PET	Pre-eclampsia
PPROM	Preterm Pre-labour Rupture Of Membranes
SFH	Symphysial Fundal Height
STAN	ST-segment Analysis
TENS	Transcutaneous Electrical Nerve Stimulation
WHO	World Health Organisation

## Introduction

This is the first fetal monitoring guideline that solely relies on physiology-based interpretation for the assessment of fetal wellbeing. Previous guidance has been mainly based on pattern recognition. We aim to encompass a pathophysiological approach to explain how a fetus defends itself against intrapartum hypoxic ischaemic insults and highlight the signs that suggest progressive loss of compensation.

The purpose of intrapartum surveillance, in general, is a timely detection of babies who may be hypoxic, so that additional assessments of fetal wellbeing may be used or the baby be delivered by caesarean or instrumental vaginal birth, to prevent perinatal/neonatal morbidity or mortality. <sup>NICE 2014, FIGO 2015</sup>

As a result of a greater understanding and incorporation of physiology into the interpretation we expect to see a reduction in unnecessary intervention as well as a reduction in fetal hypoxic neurological injury, stillbirth and early neonatal death.

## Definitions

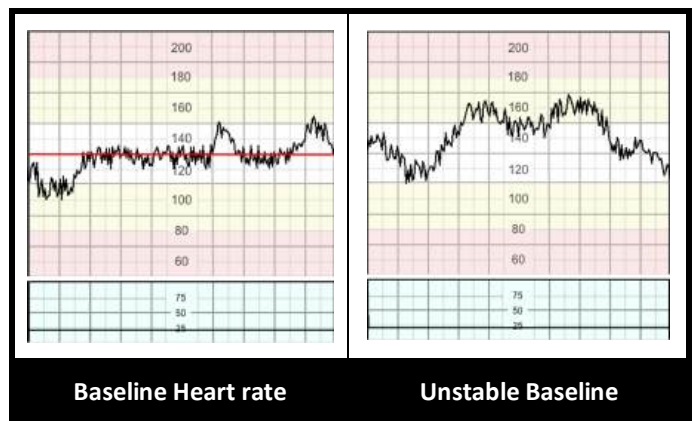
For the reason of simplicity, the editorial board have used the definitions below. These definitions were developed by other professional bodies and guidelines and have been referenced accordingly.

### CTG Features

- 1- **Baseline heart rate:** The mean fetal heart rate rounded to increments of five beats per minute during a ten-minute segment, excluding accelerations, deceleration and periods of marked FHR variability. The baseline must be for a minimum of 2 minutes in a ten-minute segment. Otherwise, the baseline for that segment is described as *indeterminate*. <sup>Macones et al. 2008</sup>

In tracings with unstable FHR signals, review of previous segments and evaluation of longer time periods may be necessary to determine the baseline. <sup>FIGO 2015</sup>

- **Normal baseline:** A value between 110 and 160 bpm. Preterm fetuses tend to have values toward the upper end of this range and post-term fetuses towards the lower end. Some experts consider the normal baseline values at term to be between 110-150 bpm. <sup>FIGO 2015</sup> It is important to note the normal baseline range for the individual fetus, by reviewing previous fetal heart rate traces if available or antenatal records. <sup>GCP</sup>
- **Tachycardia:** a baseline value above 160 bpm lasting more than 10 minutes.
- **Bradycardia:** a baseline value below 110 bpm lasting more than 10 minutes. Values between 90 and 110 bpm may occur in a normal fetus, especially in a postdate pregnancy. It is mandatory to confirm that this is not the maternal heart beat and that the trace shows normal baseline variability. <sup>NICE 2014</sup> A senior obstetric review is required before classifying the trace as normal. <sup>GCP</sup>





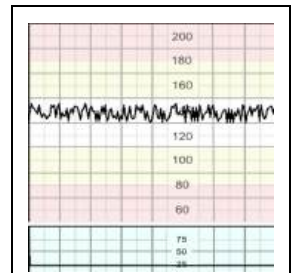
**2- Variability:** This refers to the oscillation in the FHR signal, evaluated as the average bandwidth amplitude of the signal in 1-minute segments; <sup>FIGO 2015</sup> the fluctuations should be irregular in amplitude and frequency. <sup>Macones 2008</sup> Variability is documented in beats per minute.

- **Normal:** bandwidth amplitude of 5 – 25 bpm.
- **Reduced:** a bandwidth amplitude below 5 bpm for more than 50 minutes in baseline segments, or for more than 3 minutes during decelerations. <sup>FIGO 2015, Hamilton et al 2012</sup>
- **Absent Variability:** Amplitude range undetectable with or without fetal decelerations. <sup>Macones 2008</sup>
- **Increased Variability (Saltatory Pattern):** a bandwidth value exceeding 25 bpm lasting more than 30 minutes. The pathophysiology of this pattern is incompletely understood, but it may be seen linked with recurrent decelerations, when hypoxia/acidosis evolves very rapidly. It is presumed to be caused by fetal autonomic instability/hyperactivity. <sup>FIGO 2015</sup> Intervention may be required sooner if this pattern is seen during the second stage or during decelerations. A saltatory pattern for more than 30 minutes may indicate hypoxia even without decelerations.
- **Sinusoidal pattern:** A regular, smooth, undulating signal, resembling a sine wave, with an amplitude of 5–15 bpm, and a frequency of 3–5 cycles per minute. This pattern lasts more than 30 minutes and coincides with absent accelerations.

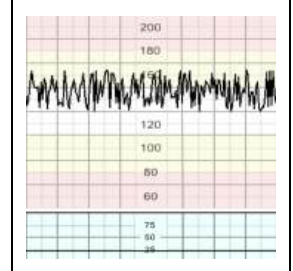
The pathophysiological basis of the sinusoidal pattern is incompletely understood, but it occurs in association with severe fetal anaemia, as is found in anti-D alloimmunisation, fetal-maternal haemorrhage, twin-to-twin transfusion syndrome, and ruptured vasa praevia. It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus, and gastroschisis. <sup>FIGO 2015</sup>

- **Pseudo-sinusoidal pattern:** A pattern resembling the sinusoidal pattern, but with a more jagged “saw-tooth” appearance, rather than the smooth sine-wave form. Its duration seldom exceeds 30 minutes and it is characterized by normal patterns before and after. <sup>FIGO 2015</sup>

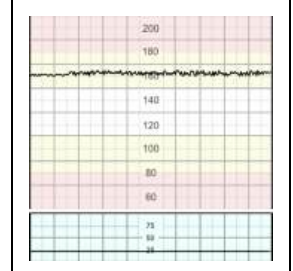
Some authorities consider a “pseudo-sinusoidal pattern” as the presence of accelerations with sinusoidal patterns. The presence of “saw toothed” or “Poole shark-teeth” pattern, is termed “atypical sinusoidal pattern” by some authorities, caused by fetal hypotension occurring secondary to acute fetomaternal haemorrhage and conditions such as ruptured vasa praevia. <sup>Yanamandra and Chandrahara 2014</sup> This pattern has been described after analgesic administration to the mother, and during periods of fetal sucking and other mouth movements. It is sometimes difficult to distinguish the pseudo-sinusoidal pattern from the true sinusoidal pattern, leaving the short duration of the former as the most important variable to discriminate between the two. <sup>FIGO 2015</sup>



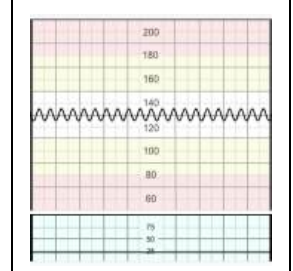
Normal Variability



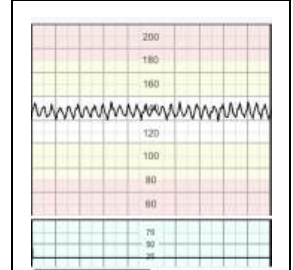
Saltatory Pattern



Reduced Variability



Sinusoidal Pattern



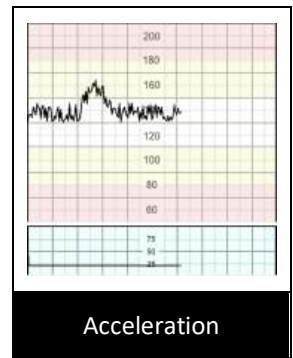
Pseudo-sinusoidal





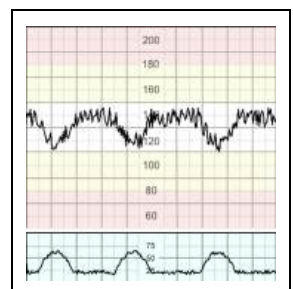
- 3- Accelerations:** Abrupt (onset to peak in less than 30 seconds) increases in FHR above the baseline, of more than 15 bpm in amplitude, and lasting more than 15 seconds but less than 10 minutes. Before 32 weeks of gestation, amplitude and duration of accelerations may be lower (10 seconds and 10 bpm of amplitude).  
Macones 2008 An acceleration must start from and return to a stable baseline. GCP

Accelerations coinciding with uterine contractions, especially in the second stage of labour, suggest possible erroneous recording of the maternal heart rate, since the FHR more frequently decelerates with a contraction, while the maternal heart rate typically increases. Nurani et al 2012

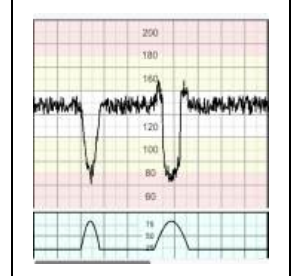


- 4- Decelerations:** Decreases in the FHR below the baseline, of more than 15 bpm in amplitude, and lasting more than 15 seconds. Decelerations are considered to be a reflex response to protect the myocardial workload when a fetus is exposed to a hypoxic or a mechanical stress, to help maintain an aerobic metabolism within the myocardium.

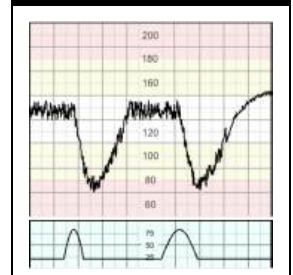
- **Early Decelerations:** Decelerations that are gradual (onset to nadir  $\geq 30$ s) that return to the baseline. They coincide with contractions, Macones et al 2008 and show normal variability within the deceleration. They are likely to be seen in the late first stage and second stage of labour and are believed to be caused by fetal head compression. They do not indicate fetal hypoxia/acidosis. FIGO 2015
- **Variable Decelerations:** V-shaped decelerations that exhibit a rapid drop (onset to nadir in  $< 30$ s) followed by a rapid recovery to the baseline. The precipitous fall and rise of the baseline due to cord compression means there is not time to exhibit good variability within the trough of the deceleration. These decelerations vary in size, shape, and relationship to uterine contractions.



Variable decelerations constitute the majority of decelerations during labour, and they translate a baroreceptor-mediated response to increased arterial pressure, as occurs with umbilical cord compression. FIGO 2015 They are believed to occur secondary to baro-receptor and/or peripheral chemoreceptor stimulation. They are seldom associated with fetal hypoxia/acidosis, unless they evolve to exhibit a *U-shaped* component ("sixties" criteria) with a reduced or an increased variability within the deceleration (see late decelerations below), and/or their individual duration exceeds 3 minutes FIGO 2015, Hamilton et al 2012 (see prolonged decelerations below).

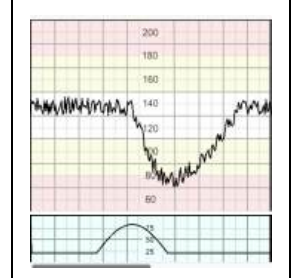


Variable decelerations meet the "sixties" criteria if two or more of the following are present: drops by 60bpm or more, reaches 60bpm or less, for the duration of 60 seconds or longer. Hamilton et al 2012



- **Late Decelerations:** Decelerations with a gradual onset and/or a gradual return to the baseline and/or reduced or increased variability within the deceleration. Gradual onset and return occurs when more than 30 seconds elapses between the beginning/end of a deceleration and its nadir. When contractions are adequately monitored, late decelerations start more than 20 seconds after the onset of a contraction, have a nadir after the acme, and a return to the baseline after the end of the contraction. FIGO 2015

These decelerations are indicative of a chemoreceptor-mediated response to fetal hypoxaemia. Hamilton et al 2012 In a trace showing no accelerations and reduced variability, the definition of late decelerations also includes those with an amplitude of 10–15 bpm (shallow decelerations).





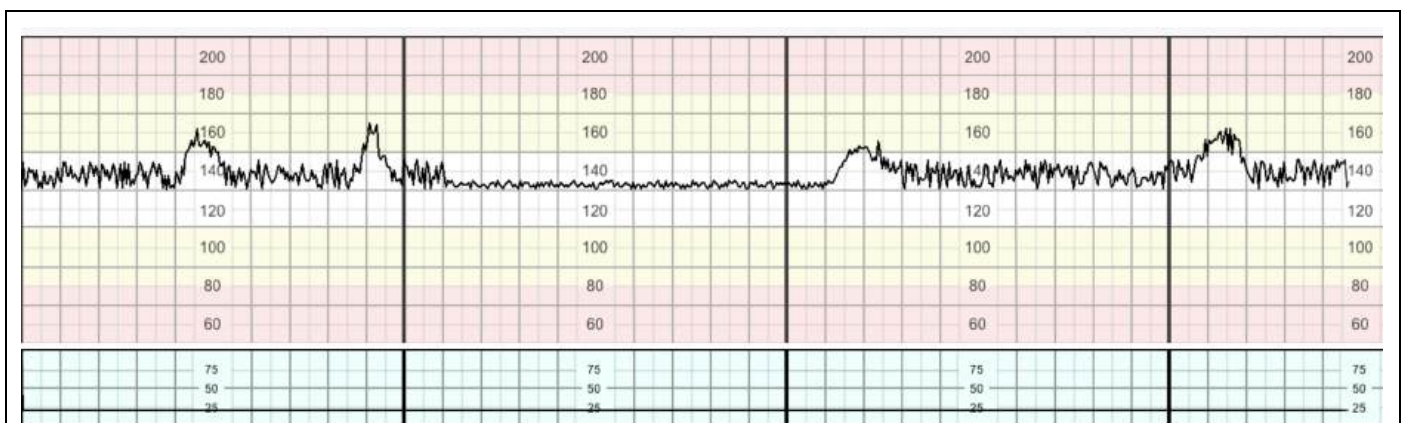
- **Prolonged decelerations:** Decelerations lasting more than 3 minutes. These are likely to include a chemoreceptor-mediated component and thus to indicate hypoxemia. Decelerations exceeding 5 minutes, with FHR maintained at less than 80 bpm and reduced variability within the deceleration, are frequently associated with acute fetal hypoxia/acidosis and require urgent intervention <sup>FIGO 2015</sup> (see “3 – minute rule”).
- 5- **Contractions:** These are recorded as bell-shaped gradual increases in the uterine activity signal followed by roughly symmetrical decreases. With the tocodynamometer, only the frequency of contractions can be reliably evaluated. <sup>FIGO 2015</sup> The intensity and duration of contractions may be assessed by manual palpation. If frequency of contractions cannot be assessed reliably by the tocodynamometer, manual palpation for 10 minutes every 30 minutes is required. <sup>GCP</sup>
- **Tachysystole** represents an excessive frequency of contractions and is defined as the occurrence of more than five contractions in 10 minutes, <sup>Peebles et al 1994</sup> in two successive 10-minute periods or averaged over a 30-minute period.
  - **Hyperstimulation** refers to an exaggerated response to uterine stimulants, presenting as an increase in frequency of the contractions, strength of uterine contraction, increased uterine tone between contractions and/or prolonged contractions for over 2 minutes. These may lead to fetal heart rate changes. Therefore, any increased uterine activity (frequency, duration or strength) associated with CTG changes should be considered as uterine hyperstimulation. This described picture of hyperstimulation may occasionally be seen in spontaneous labour without the use of uterine stimulants. (To avoid over complication, the term hyperstimulation will be used to include both iatrogenic and spontaneous increased uterine activity.)

### Fetal behavioural states <sup>Pillai and James 1990</sup>

This refers to periods of:

- 1- **Fetal quiescence** reflecting deep sleep (no eye movements): Deep sleep can last up to 50 minutes and is associated with a stable baseline, very rare accelerations, and borderline variability
- 2- **Active sleep** (rapid eye movements): This is the most frequent behavioural state and is represented by a moderate number of accelerations and normal variability.
- 3- **Wakefulness:** Active wakefulness is rarer and represented by a large number of accelerations and normal variability. In this pattern, accelerations may be so frequent as to cause difficulties in baseline estimation (confluence of accelerations).

The alternation of different behavioural states (**cycling**) is a hallmark of fetal neurological responsiveness and absence of hypoxia/acidosis. Transitions between the different patterns become clearer after 32–34 weeks of gestation, consequent to fetal nervous system maturation.



Cycling

# Physiology of Hypoxia in Labour

During labour the fetus employs various adaptive mechanisms in response to hypoxia, these generally follow a similar pathway as the physiological response to exercise. Intrapartum hypoxia generally follows one of three pathways:

## 1. Acute Hypoxia Kamoshita et al. 2010, Leung et al. 2009, Cahil et al. 2013

- **Presents** as a prolonged deceleration lasting for more than 5 minutes or for more than 3 minutes if associated with reduced variability within the deceleration. FIGO 2015

- **Causes**

- 3 Accidents

- Cord prolapse
    - Placental Abruption
    - Uterine Rupture

- 2 Iatrogenic

- Maternal Hypotension (usually secondary to supine hypotension or epidural top-up)
    - Uterine hyperstimulation (by oxytocin / PGs) or spontaneous increased activity

- **Fetal pH drops** at a rate of 0.01/min during the deceleration Gull et al. 1996

- **Management** follows the **3-Minute Rule**:

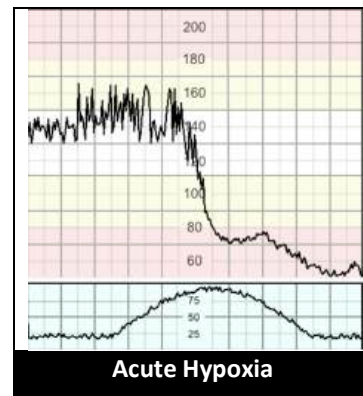
- 0 – 3: If a deceleration is noted for more than 3 minutes with no signs of recovery the emergency alarm must be raised to summon the on-call team

- 3 – 6: Attempt to diagnose the cause of the deceleration

- If an accident is diagnosed the aim would be for immediate delivery as soon as safely possible in the fastest route possible (AVD/CS)
    - If an iatrogenic cause is diagnosed immediate measures must be utilized to correct the changes. This includes avoiding supine position, stopping uterine stimulants, starting IV fluids, and administering tocolytics.

- 6 – 9: Signs of recovery should be noted (return of variability and improvement in heart rate). If no signs of recovery are noted, preparation for immediate delivery **MUST** be started.

- 9 – 12: By this point in time the deceleration has either recovered, or preparation for an assisted vaginal delivery / caesarean section is in progress aiming for a delivery of the fetus by 12 – 15 minutes.



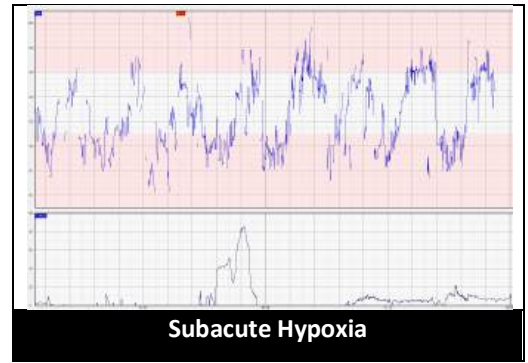
### Important Notes:

- Do not follow the 3-minute rule if the deceleration is preceded by reduced variability and lack of cycling, immediate preparation should be made to expedite delivery by the safest and fastest route possible. Williams and Galerneau 2002
- If normal variability and cycling before and during the first 3 minutes of the deceleration, it is likely that 90% will recover within 6 minutes, and 95% in 9 minutes, if acute accidents have been excluded.



**2. Subacute Hypoxia** Albertson et al. 2016

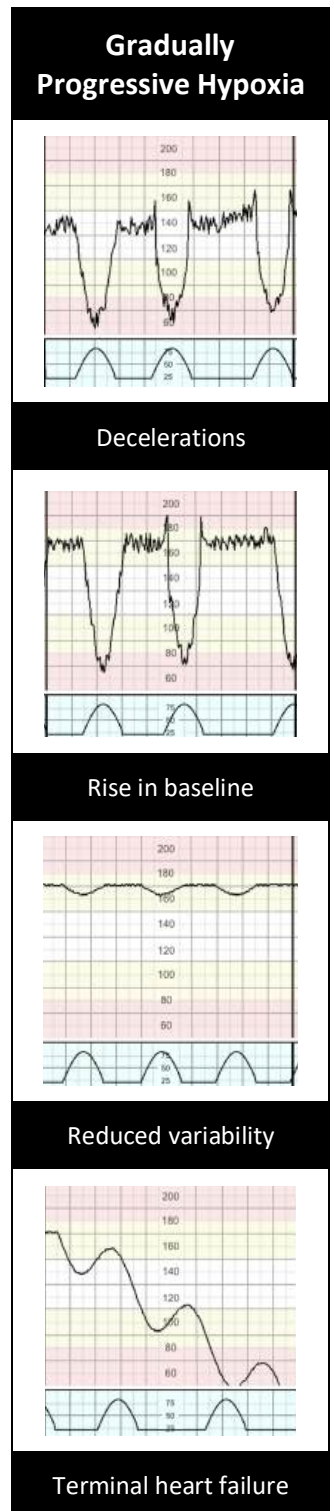
- **Presents** on the CTG by the fetus spending most of the time in decelerations.
- This is almost invariably **caused** by uterine hyperstimulation.
- **Fetal pH drops** at a rate of 0.01 / 2-3 minutes
- **Management** is by:
  1. Stopping / reducing uterotonics
  2. Avoiding supine position
  3. Starting IV fluids
  4. Administering tocolytics if hyperstimulation persists despite previous measures
  5. Expediting delivery if hypoxia persists despite tocolysis (AVD / CS)



If encountered in the second stage of labour, ask the mother to stop pushing to allow the recovery of the fetal status. If no improvement is seen within 10 minutes, expedite delivery. Once stable recommence pushing. If subacute hypoxia recurs, expedite delivery.

**3. Gradually Evolving Hypoxia** Richardson et al 1996

- This is the most common type of hypoxia in labour
- During this process, the fetus undergoes the same changes that a normal adult would be expected to show during exercise
- This tends to **present** with the following order:
  1. Evidence of hypoxic stress (decelerations)
  2. Loss of accelerations and lack of cycling
  3. Exaggerated response to hypoxic stress (decelerations become wider and deeper)
  4. Attempted redistribution to perfuse vital organs facilitated by catecholamines (first sign noted is a rise in baseline)
  5. Further redistribution with vasoconstriction affecting the brain (reduced baseline variability)
  6. Terminal heart failure (unstable/ progressive decline in the baseline - “step ladder pattern to death”)



**Important Notes:**

- Stages 1 – 4 represent evidence of stress with maintained fetal **compensation**.
- Stages 5 & 6 represent evidence of stress with fetal **decompensation**.
- Stages 4 & 5 may be reversible although prolonged episodes of hypoxia can lead to fetal organ damage.

- **Management** of gradually progressive hypoxia is by improving fetal conditions with the first signs of redistribution to avoid internal organ damage. (stage 4).

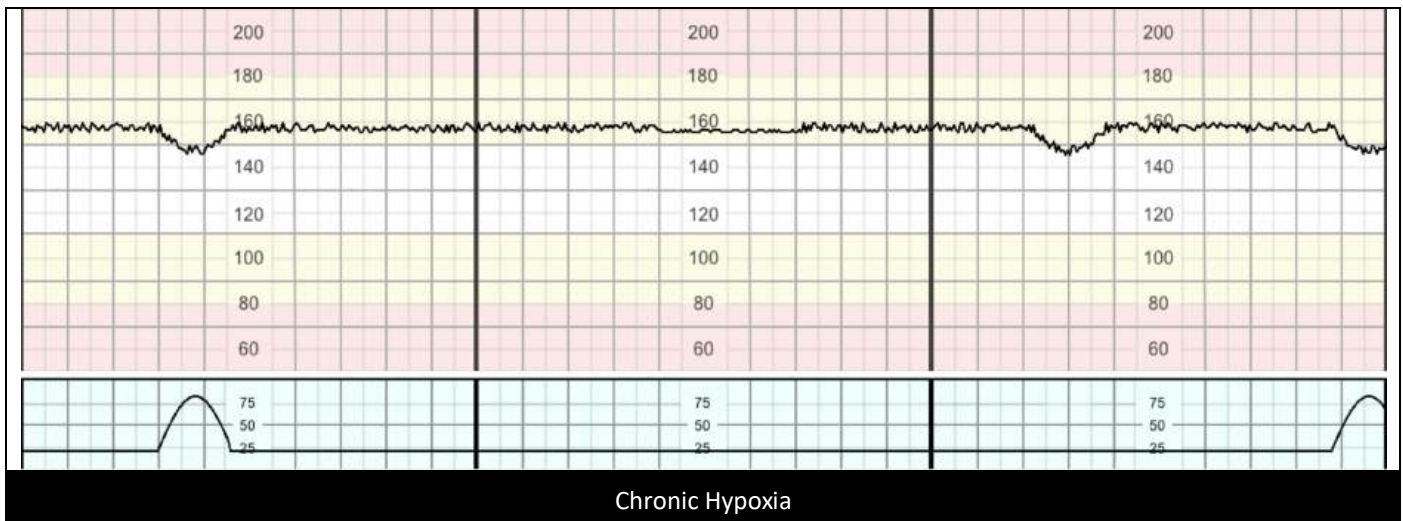




#### 4. Chronic Hypoxia Pulgar et al 2007

(This is an antenatal type of hypoxia with implications for intrapartum care)

- **Presents** as a baseline rate at the upper end of normal associated with reduced variability and blunted responses (infrequent accelerations and lack of cycling) and is frequently associated with shallow decelerations.
- This represents a fetus with reduced reserve and increased susceptibility to hypoxic injury during labour.
- Careful consideration should be given when planning interventions potentially increasing the risk of hypoxia, with low threshold for surgical intervention.
- A useful checklist that helps exclude signs of chronic hypoxia is presented below (see table 3).



# Intermittent Auscultation

All women that have existing medical or obstetric conditions should have an obstetric review during pregnancy with a full plan of care formulated for labour and birth. This should include the suitability for different birth settings and the type of fetal monitoring required when in labour. The care plan should be explained to and agreed by the woman.

## 1 Inclusion Criteria

Continuous electronic fetal monitoring (CEFM) in low risk women is associated with an increased level of intervention without any improvement in outcome. <sup>Maude et al 2014</sup> Women who are healthy and have had an uncomplicated pregnancy should be offered and recommended intermittent auscultation to monitor fetal well-being. This should be performed using a Doppler ultrasound or pinard stethoscope. <sup>NICE 2014</sup> A woman must be fully informed of the risks and benefits of intermittent auscultation (IA) and CEFM. If during labour, she chooses not to be monitored by the recommended method a full discussion of the potential impact on her and the fetus should be undertaken and the labour ward coordinator and senior obstetrician should be informed. This discussion must be clearly documented in the patient's records.

## 2 Method

There does not appear to be any good evidence from trials to recommend any particular frequency and duration of IA. Therefore, it is more a 'custom of practice' than 'evidence-based approach'. <sup>Walsh 2008</sup> Auscultation is carried out every 15 minutes in the first stage and every 5 minutes in the second stage - a practice adopted from the randomized controlled trials comparing IA and CEFM. On assessing the woman and establishing that she is low risk and is suitable for IA the method is as follows:

- Ask about fetal movements over the preceding 24 hours.
- Perform a full abdominal palpation to determine the lie, presentation and position of the baby.
- At the initial assessment use a pinard stethoscope on the mother's abdomen in line with the fetal scapula to establish the real sounds of the fetal heart (FH). <sup>Munro and Jokinen 2012</sup>
- On first auscultation listen for at least one full minute in between contractions when the baby is at rest to establish a baseline FH rate. <sup>Maude et al 2014</sup>
- If in early labour, auscultate during fetal movements or following stimulation of the baby. An acceleration should be noted, and the presence of chronic hypoxia can be excluded. This is more difficult to demonstrate later in labour
- The maternal pulse should be palpated simultaneously while auscultating FH to differentiate between the two, as it is possible to inadvertently pick up maternal heart rate from surrounding vessels. This should be done on the initiation of each auscultation and throughout if a FH abnormality is detected. <sup>NCC-WCH 2007</sup>
- During the first stage and passive\* second stage of labour FH should be auscultated immediately after a contraction for at least 1 minute every 15 minutes.
- During active+ second stage of labour, the FH should be auscultated every 5 minutes. <sup>Liston et al 2007</sup>
- Count the FH and document as a single number, and not as an average. If using a Doppler do not rely on the range shown on the screen, as there have been instances where the machine has miscalculated the FH rate. <sup>NICE 2014, MHRA 2010</sup>
- Record acceleration and decelerations, if heard. <sup>NICE 2014</sup>
- None of the literature suggests that variability can be determined by IA. <sup>Munro and Jokinen 2012</sup>
- An observed rise in baseline rate, slow recovering decelerations or persistent accelerations (overshoot) after contractions should be confirmed by listening throughout the next 3 contractions to clarify the suspected pattern. Confirmation of an abnormality warrants a move to CEFM and transfer to obstetric-led care (see section criteria for change). <sup>FIGO 2015</sup>



- Although a CTG machine utilizes the same technology as the handheld Doppler, it should not be used in a low risk labour for intermittent auscultation, as this is inappropriate use of resources.<sup>GCP</sup> The handheld doppler has a narrow beam and is unlikely to easily pick up the maternal sound. It gives a swishing noise when tracked to a blood vessel compared with the electronic heartbeat sounds of the US transducer of a CTG machine, hence the handheld doppler device is preferred.

\* Passive second stage is defined as the finding of full dilatation of the cervix prior to or in the absence of involuntary expulsive efforts or active maternal effort.

+ Active second stage is defined as the onset of involuntary expulsive efforts or active maternal effort following confirmation of full dilatation.

### 3 Documentation

- Initial risk assessment should be documented in the maternal notes on admission in labour.
- The FH should be documented in the notes as a single number counted as beats over one minute.
- Maternal pulse should be documented every hour as a single number.
- Women not in established labour should be encouraged to go home. A clear plan should be established and documented explaining when to return.
- For women in the latent phase of labour with strong regular contractions, showing signs of progress or that have a history of precipitate labour, commence monitoring every 15min and reassess in 2 hours. If still not in labour, reconsider the management plan. It is important to remain alert to possible transitions between different phases of labour and adjust frequency of monitoring accordingly.
- When labour is confirmed a partogram must be started. This will act as a visual prompt to identify any changes from the norm. Blood pressure, pulse, temperature and urine output should also be documented on the partogram.

### 4 Conversion Criteria for changing from IA to CEFM

During the course of pregnancy or labour the clinical circumstances may change, increasing risk to mother and/or fetus (see table-1). In this situation, the mother should be informed of the rationale for changing the method of auscultation and should also be clearly documented in the notes.

If CEFM has been commenced due to concerns arising during IA but the CTG is normal after a minimum of 30 minutes, it is deemed suitable to return to IA. <sup>Maude et al 2014</sup> If concerns arise again, CEFM would be recommended until delivery.

If conversion to CEFM is advised but declined, the risks of not continuously monitoring should be explained, and the midwife in charge and obstetric team informed. All discussions must be clearly documented in the notes.


**Table 1 – Risk factors indicating conversion from Intermittent Auscultation to Continuous Electronic Fetal Monitoring**

<b>Maternal</b>	<b>Fetal</b>
*Pulse over 120 beats/minute on 2 occasions 30 minutes apart	Undiagnosed breech presentation; transverse or oblique lie (review mode of delivery)
* A single reading of diastolic blood pressure $\geq 110$ mmHg or systolic blood pressure $\geq 160$ mmHg	Free-floating head in a nulliparous woman
*Diastolic blood pressure 90 to 109 mmHg or systolic blood pressure of 140 to 159 mmHg on 2 consecutive readings taken 30 minutes apart	Recurrent Accelerations (immediately following a contraction i.e. overshoot)
Maternal pyrexia (defined as $\geq 38.0$ °C once or $\geq 37.5$ °C on two occasions 1 hour apart)	Fetal heart rate below 110 or above 160 beats/minute, or if it is perceived as inappropriate for gestational age.
Any vaginal blood loss other than a show	Evidence of a rising baseline on the partogram
The presence of meconium if birth is not imminent NICE 2014	2 x decelerations in fetal heart rate heard on intermittent auscultation after 2 successive contractions
Persistent pain in between contractions	
Epidural analgesia	

\* measured between contractions



# Continuous Electronic Fetal Monitoring

CEFM could potentially reduce mobility. However, every effort should be made to facilitate the normal physiology of labour by encouraging the woman to adopt upright positions and mobilise. This can be facilitated by the use of wireless telemetry or the encouragement to move within the constraints of being connected to the monitor.

CEFM is a screening tool for hypoxia and does not replace the need for accurate clinical observations on which decisions should be made in conjunction with the CTG. <sup>FIGO 2015</sup>

## 1 Inclusion criteria

<b>Maternal indication</b>	<b>Fetal indication</b>
Gestation <37 or > 42 weeks	Abnormal Doppler artery velocimetry
Induced labour	Known or suspected IUGR
Administration of oxytocin	Oligohydramnios or polyhydramnios
Ante/Intrapartum haemorrhage	Malpresentation
Maternal illness (e.g. diabetes, cardiac, renal, hyperthyroidism). *	Meconium stained liquor
Pre-eclampsia	Multiple pregnancy (all babies to be monitored)
Previous uterine scar (caesarean section or myomectomy)	Suspected small for gestational age or macrosomia
Contractions > 5:10 or lasting for more than 90 seconds	Reduced fetal movements in the last 24 hours reported by the woman
During / following insertion of an epidural block	Two-vessel cord
Prolonged rupture of membranes > 24 hours unless delivery is imminent.	A rise in baseline, repeated decelerations or slow to recover decelerations, or overshoots
Maternal request	Fetal structural abnormalities diagnosed during the antenatal period and planned for CEFM

\* Monitoring as per consultant formulated plan.

The table above is not exhaustive, any condition which is thought to increase the risk of fetal hypoxia mandates CEFM.

## 2 Interpretation of CTG

- **Step 1:** The clinical setting
  - Current gestational age
  - Antenatal events (e.g. IUGR, PET, medications)
  - Previous CTG traces and their clinical scenario to assess whether they can be used as a baseline for the current monitoring
- **Step 2:** Current clinical situation, and indication for CTG
- **Step 3:** Set the limits acceptable as normal for THIS trace BEFORE starting the assessment
- **Step 4:** Assess the trace:
  - Identified risks
  - Contractions (is the inter-contraction interval more than 90 seconds?)
  - **Baseline Heart rate:**
    - It is the most important feature on a CTG trace.
    - Consider whether the baseline is appropriate for G.A.



- Compare baseline rates on previous CTGs.
    - A change in baseline (by >10%) signifies a need for further attention. (In the presence of chronic hypoxia, more subtle changes to the baseline should also be considered significant).
  - Variability and Cycling:
    - Cycling is a sign of fetal well-being. It signifies normal fetal physiology.
    - In the presence of decelerations and increase in baseline rate, episodes of reduced variability must be managed promptly, and not assumed to be cycling. Reduced variability in this situation is caused by CNS inhibition secondary to hypoxia.
  - Accelerations:
    - The presence of accelerations is generally considered to be a reassuring feature.
    - **An acceleration starts from, and returns to the baseline.**
    - It is important to differentiate accelerations from overshoots (rebound increase in heart rate caused by brief accumulation of CO<sub>2</sub> during hypoxic episodes) and shouldering (increase in heart rate preceding and/or following decelerations commonly occurring with cord compressions).
    - **If accelerations are coinciding with contractions, especially in the second stage of labour, exclude maternal heart rate.**
  - Decelerations:
    - It is important to realise that although the presence of decelerations does not in itself reflect that the fetus is unwell, it may signal the need to try and alter fetal conditions. Such as:
      - Repeated chemoreceptor decelerations (late, prolonged, or reduced variability within deceleration) signify that the placental stores are being depleted. Frequently, this can be corrected by changing maternal position, or increasing circulating volume by hydration, reducing the stress by reducing/stopping oxytocin. If this does not correct the situation it is important to monitor closely for any rise in baseline rate or reduction of variability.
      - Prolonged decelerations (>5 minutes or >3 minutes with reduced variability) need to be managed according to the 3-minute rule (refer to page 11)
      - An isolated deceleration in non-labouring women is acceptable, however, repeated unprovoked decelerations in non-labouring women are a need for further investigation and assessment.
  - **Overall Assessment of Presence of Hypoxia and management plan** (see table below)

Table 3 - Checklist to exclude chronic hypoxia and pre-existing fetal injury <small>Pereira and Chandraharan 2017</small>			
1	Baseline fetal heart rate appropriate G.A.	Yes	No
2	Normal variability and cycling	Yes	No
3	Presence of accelerations (not in labour or latent phase of labour)	Yes	No
4	No shallow/ late decelerations	Yes	No
5	Consider the wider clinical picture: meconium, temperature, fetal growth, reduced fetal movements	Yes	No
<b>Overall Impression:</b> Normal / Chronic Hypoxia / Other:			
<b>Management Plan:</b>			



Hypoxia	Features	Management
<b>No Hypoxia</b>	<ul style="list-style-type: none"> <li>• Baseline appropriate for G.A.</li> <li>• Normal variability and cycling</li> <li>• No repetitive decelerations</li> </ul>	<ul style="list-style-type: none"> <li>• Consider whether the CTG needs to continue.</li> <li>• If continuing the CTG perform routine hourly review. (see CTG Assessment Tool below)</li> </ul>
<b>Evidence of Hypoxia</b>		
<b>Chronic Hypoxia</b>	<ul style="list-style-type: none"> <li>• Higher baseline than expected for G.A.</li> <li>• Reduced variability and/ or absence of cycling</li> <li>• Absence of accelerations</li> <li>• Shallow decelerations</li> <li>• Consider the clinical indicators: reduced fetal movements, thick meconium, bleeding, evidence of chorioamnionitis, postmaturity, IUGR</li> </ul>	<ul style="list-style-type: none"> <li>• Avoid further stress</li> <li>• Expedite delivery, if delivery is not imminent</li> </ul>
<b>Gradually Evolving Hypoxia</b>	<b>Compensated</b>	<ul style="list-style-type: none"> <li>• Likely to respond to conservative interventions (see below)</li> <li>• Regular review every 30-60 minutes to assess for signs of further hypoxic change, and that the intervention resulted in an improvement.</li> <li>• Other causes such as reduced placental reserve MUST be considered and addressed accordingly.</li> </ul>
	Rise in the baseline (with normal variability and stable baseline) preceded by decelerations and loss of accelerations	
	<b>Decompensated</b>	<ul style="list-style-type: none"> <li>• Needs urgent intervention to reverse the hypoxic insult (remove prostaglandin pessary, stop oxytocin infusion, tocolysis)</li> <li>• Delivery should be expedited, if no signs of improvement are seen</li> </ul>
<ul style="list-style-type: none"> <li>• Reduced or increased variability</li> <li>• Unstable/ progressive decline in the baseline (step ladder pattern to death)</li> </ul>		
<b>Subacute Hypoxia</b>	<ul style="list-style-type: none"> <li>• More time spent during decelerations than at the baseline</li> <li>• May be associated with saltatory pattern (increased variability)</li> </ul>	<b>First Stage</b>
		<ul style="list-style-type: none"> <li>• Remove prostaglandins/stop oxytocin infusion</li> <li>• If no improvement, needs urgent tocolysis</li> <li>• If still no evidence of improvement within 10-15 minutes, review situation and expedite delivery</li> </ul>
		<b>Second Stage</b>
<ul style="list-style-type: none"> <li>• Stop maternal active pushing during contractions until improvement is noted.</li> <li>• If no improvement is noted, consider tocolysis if delivery is not imminent or expedite delivery by operative vaginal delivery</li> </ul>		
<b>Acute Hypoxia</b>	Prolonged Deceleration (> 3 minutes)	<b>Preceded by reduced variability and lack of cycling or reduced variability within the first 3 minutes</b>
		Immediate delivery by the safest and quickest route
		<b>Preceded by normal variability and cycling and normal variability during the first 3 minutes of the deceleration (see 3-minute rule above)</b>
		<ul style="list-style-type: none"> <li>• Exclude the 3 accidents (i.e. cord prolapse, placental abruption, uterine rupture - if an accident is suspected prepare for immediate delivery)</li> <li>• Correct reversible causes</li> <li>• If no improvement by 9 minutes or any of the accidents diagnosed, immediate delivery by the safest and quickest route</li> </ul>
<b>Unable to Ascertain fetal wellbeing</b> (Poor signal quality, uncertain baseline, possible recording of the maternal heart rate)		<ul style="list-style-type: none"> <li>• Escalate to senior team</li> <li>• Consider Adjunctive Techniques, if appropriate</li> <li>• Consider the application of FSE to improve signal quality</li> </ul>



**Please Note:**

- If fetal well-being cannot be determined, timely advice and management needs to be sought.
- **Be aware that if the CTG parameters of baseline fetal heart rate and baseline variability are normal, the risk of fetal acidosis is low.** NICE 2014, FIGO 2015
- Even if the CTG trace is classified as pathological, if the baseline fetal heart rate is stable, and the variability is reassuring, according to both NICE and FIGO guidelines, no intervention is required other than careful observation.
- In many trusts, it would still be advisable to classify the CTG trace according to the trust agreed national guideline (FIGO / NICE). If the trace is classified as suspicious or pathological while no evidence of hypoxia is noted, this has to be clearly documented in the notes justifying the management plan.

Table 4 - CTG Assessment Tool							
Baseline	bpm	Variability	bpm	Accelerations		Decelerations	
Rise in Baseline ( $\geq 10\%$ )				Yes		No	
Inter-contraction interval > 90 sec				Yes		No	
Maintained Cycling				Yes		No	
Abnormal Variability (<5 or >25)				Yes		No	
Features of Hypoxia				Yes		No	
		Type					
Central Organs well oxygenated				Yes		No	
Other risk factors noted							
Recommended Management							

### 3 Actions in situation of suspected fetal hypoxia

Identify reversible causes as alleviating them can lead to subsequent recovery of adequate fetal oxygenation and the return to a normal trace.

When CTG changes develop, it is important to address underlying causes before hypoxia occurs. The midwife caring for the woman should escalate to a senior midwife/obstetric team for review without delay.

#### 3.1 Excessive uterine activity (most frequent cause)

- Could be detected by palpating the uterine fundus assessing the frequency, strength and duration of contractions and the tone in between.
- It can usually be reversed by
  - Reducing or stopping oxytocin infusion
  - Removing administered prostaglandins
  - Starting acute tocolysis with beta-adrenergic agonists (terbutaline) or nitro-glycerine
  - During the second stage of labour, maternal pushing efforts can also contribute to fetal hypoxia/acidosis and the mother can be asked to stop pushing until the situation has improved. FIGO 2015 If this does not improve the trace, delivery should be expedited.

**Important note:**

Due to the longer half-life of prostaglandins, hyperstimulation usually requires the removal of the pessary and administration of tocolytics at the same time, especially when dealing with acute hypoxia.

**3.2 Aorto-caval compression** can occur in supine position. Turning the mother to lateral or upright positions may relieve compression.

**3.3 Transient cord compression** (variable decelerations) can sometimes be relieved by changing maternal position



**3.4 Sudden maternal hypotension** most frequently occurs after spinal or epidural administration. This is reversible by rapid fluid administration  $\pm$  I.V. ephedrine bolus (by the anaesthetic team).

### 3.5 ACTION WITH NO SUPPORTING EVIDENCE

- Oxygen administration to the mother in a well oxygenated mother, this does not alleviate fetal hypoxia and may actually be more harmful. Fawole and Hofmeyr 2012
- I.V. fluids in normotensive, well hydrated women. Although, some may consider IV fluids to improve the placental flow, administration of IV fluids in chronic hypoxia and chorioamnionitis may provide a false sense of reassurance without improving perinatal outcomes. ***Good clinical judgement is required to diagnose the underlying cause of the changes on the CTG, to judge the reversibility of the conditions with which it is associated, and to determine the timing of delivery. The objective is to avoid prolonged fetal hypoxia / acidosis, as well as unnecessary obstetric interventions. Additional methods such as fetal scalp stimulation and STAN monitoring may be used to evaluate fetal oxygenation.***

## 4 Quality, Documentation and Storage

### 4.1 Documentation

- It is the responsibility of every clinician using the CTG machine to perform the following initial checks prior to commencing the trace:
  - Correct Date and Time
  - Correct scale in use
  - Paper specific for the machine in use, with correct orientation
- Every trace should start by clearly documenting
  - The name, DOB, and hospital number of the patient
  - Machine Number
  - Indication for CEFM
  - Maternal observations
- Ongoing documentation of
  - Maternal heart rate every hour
    - Simultaneous maternal heart rate monitoring should be considered in:
      - Fetal heart block
      - Fetal heart rate similar to maternal heart rate
      - Maternal tachycardia
      - During 2<sup>nd</sup> stage of labour
      - Trace shows accelerations coinciding with contractions/ expulsive efforts
  - Maternal BP and temperature is to be measured every 4 hours unless more frequent observations are indicated clinically
  - Relevant intrapartum events for example
    - Vaginal examination
    - Siting of an epidural
    - Review of CTG

### 4.2 Quality

- External FHR monitoring is the recommended initial method, provided that a recording of acceptable quality is obtained i.e. that the basic CTG features can be identified. If the trace is of poor quality early recourse to FSE is advised if no contraindications exist. Such instances include increased maternal BMI and poor recording during second stage (thus avoiding monitoring maternal heart rate).
- Monitoring of Twins:
  - CEFM should preferably be performed with dual channel monitors.
  - Clearly identify which trace is allocated to which twin on the CTG and in the notes.



- Consider offsetting twin 2 by 20 beats in order to clearly identify each twin separately.
- External monitoring of both twins is acceptable for as long as distinct traces of good quality are obtained.
- There should be a low threshold for internal monitoring of the presenting twin in the absence of contraindications as it is often superior in quality especially in the second stage.

#### **4.3 Storage**

CTG must be stored for 25 years. Given that thermal paper deteriorates and is only legible for about 10 years, storage should ideally be in electronic form.

# Adjunctive Techniques to Assess Fetal Wellbeing

Be aware that if the CTG parameters of baseline fetal heart rate and baseline variability are stable, the risk of fetal acidosis is low. NICE 2014, FIGO 2015

It is important to attempt to understand the physiological events behind changes in fetal heart rate pattern (see above). This can provide reassurance of fetal status without the need to perform further testing. However, in situations where changes cannot be explained, it is important to seek senior advice and plan further testing accordingly.

## 1 Fetal Scalp Stimulation

- **Supporting Evidence**

There are many observational studies supporting the use of fetal scalp stimulation (FSS) compared to fetal blood sampling. However, the evidence grading for the trials behind the use of FSS is predominantly moderate to low. Skupski et al 2002

- **Limitation**

There is no consensus on the clinical situation for FSS to be used. FIGO 2015

- **Method**

FSS involves stimulating the fetal scalp by rubbing it with the examiner's fingers. Other techniques involve using forceps to clasp the fetal skin, or alternatively using vibroacoustic stimulation applied to the mother's abdomen. However, these are not locally available. Elimian 1997 Digital scalp stimulation is the most widely used as it is the easiest to perform, least invasive, and appears to have a similar predictive value for fetal hypoxia/acidosis to the other alternatives. FIGO 2015

- **Interpretation**

If an acceleration is noted at FSS the likelihood of fetal hypoxia is <2.5%, while in the absence of an acceleration, the likelihood of fetal hypoxia is > 38%. Skupski et al 2002 The risk of hypoxia is increased if the lack of acceleration is associated with reduced variability. Elimian 1997 The information should be considered in the context of the entire clinical picture. FIGO 2015, NICE 2014

## 2 Combined Continuous Fetal monitoring with ST-Analysis (STAN)

- **Supportive evidence**

There have been six meta-analyses assessing the effectiveness of STAN in ascertainment of fetal wellbeing. Five of these have shown that although STAN reduces the need for fetal scalp blood sampling and operative vaginal delivery, there was no improvement in number of births by caesarean section, babies with low APGAR scores at 5 minutes, severe fetal metabolic acidosis, or the number of babies with neonatal encephalopathy. Adalina et al 2015 The sixth meta-analysis claimed to have found a mistake within the other five, and after correcting for this, it showed reduction of rates of metabolic acidosis. Olofsson et al 2014 The latest meta-analysis which included the largest US randomised trial on STAN Blix et al, 2016 has reported 36% statistically significant reduction in neonatal metabolic acidosis and 8% reduction in operative vaginal birth. Therefore, in the opinion of the authors, STAN remains the only additional test of fetal wellbeing with robust scientific evidence. Bhide et al, 2016, Chandraran E, 2018.

- **Principles**

- When the CTG is normal, ST events should be ignored as in this setting they do not indicate fetal hypoxia/acidosis. They occur in about 50% of well oxygenated fetuses. The generation of ST events on a normal CTG may be secondary to fetal catecholamine-mediated cardiac glycogenolysis or changes in the vector of the ECG complexes during fetal movements.
- A few cases have been described in which CTG traces have gradually changed to show signs of hypoxia, without the appearance of ST events. Westerhuis et al 2007 However, these cases illustrated the erroneous use of STAN in preterminal CTG Traces and in fetal infection (i.e. not suitable for STAN monitoring). For this reason, any abnormal CTG lasting more than 60 minutes, or less if the CTG



pattern deteriorates rapidly, requires assessment by a senior obstetrician whether or not ST events occur. <sup>FIGO 2015</sup>

- With a CTG showing persistently reduced variability or a pattern indicating a severe or acute hypoxic event, intervention is always required irrespective of ST data. <sup>Amer-Wahlin et al 2007</sup>
- Everybody using STAN must have undergone adequate training and assessment.
- In the presence of sepsis, the analysis becomes unreliable. The fetal brain may get injured by pathways other than hypoxia and is even more susceptible to injury by hypoxia which may not be reflected by ST events. This needs to be taken into account if initiating STAN on uncomplicated fetal tachycardia.
- **Prerequisites**
  - A CTG with a stable baseline, normal variability and accelerations is required at the start of monitoring for a confident evaluation of ST data. <sup>FIGO 2015</sup> However, even in an abnormal CTG Trace, if the baseline fetal heart rate remains stable and the variability is reassuring (i.e. good central organ oxygenation), STAN can be commenced <sup>Preti et al, 2013.</sup>
  - The ST technology has not been extensively evaluated for gestational ages below 36 weeks. Repetitive Biphasic ST Events may be noted due to the immaturity of fetal myocardium.
  - No existing contraindications to the application of FSE
  - TENS has to be discontinued as it interferes with the acquisition of the ECG signal
- **Contraindications**
  - It should not be initiated in active second stage, spontaneous pushing or in a precipitate labour.
  - Pre-terminal trace or acute hypoxia
  - Suspected chorioamnionitis – whilst STAN may be used to identify hypoxia, clinicians should be aware of the alternative (i.e. inflammatory) pathway of brain damage. Therefore, the management decisions should be based on the progress of labour, parity, presence of meconium and observed CTG features, irrespective of the absence of STAN Events
  - Active genital Herpes infection
  - Women seropositive to hepatitis B, C, D, E, or to HIV
  - Suspected fetal blood disorders
  - Uncertainty about the presenting part
  - When artificial rupture of membranes is inappropriate
  - Structural or functional cardiac abnormality in the fetus preventing reliable monitoring
  - If immediate delivery is required for any other indication
- **Method**

It is a combined assessment of the standard fetal heart rate trace with an automated analysis of the fetal electrocardiogram. This is obtained by placing a spiral electrode on the fetal scalp. The machine initially analyses the ST segments (required 20 'x's) to obtain a baseline shape of the ECG complex, which usually takes the first 4 minutes. Subsequently, it compares the complex every 30 beats for changes indicating the presence of possible ischaemia. With every successful comparison made, an 'x' is indicated at the bottom of the trace.
- **Interpretation**

Trace interpretation needs to take into account the CTG pattern classification and the degree of ST changes. The system's automatic warnings of ST events occur when it detects changes in ECG morphology when compared with the previous state. (table 5). A Flow Chart to aid management when using STAN for fetal monitoring is provided in the Appendix. <sup>Townsend and Chandrharan 2015</sup>
- **Signal Quality Check**
  - The effectiveness of STAN depends on the quality and continuity of the signal obtained by the scalp electrode. If the fetal ECG signal is lost for 4 minutes or longer (i.e. no crosses underneath the CTG for 4 minutes), or if less than 10 successful checks are completed within 10 minutes, then the ECG signal can no longer be compared to the baseline ECG. An alarm will sound if this has occurred. In such situations, it is recommended that if the CTG has remained normal in the period of loss then monitoring can be continued. If there is suspicion of fetal hypoxia based on the interpretation of the





CTG during the gap, STAN should not be considered reliable.

○ Recording the maternal ECG

The scalp electrode may pick up the maternal ECG if it is applied to the cervix or if there is no fetal heartbeat. For this reason, the ECG complex is displayed on the screen and should be reviewed every time monitoring is commenced and whenever there is suspicion that a trace is maternal and not fetal. A maternal ECG will appear different – the P wave will usually appear blunted or absent as it is not transmitted so far from the maternal heart to be picked up by the fetal electrode, and the QRS complex is wider. It will coincide with the maternal pulse.

○ Fetal heart rate >170 bpm

As the fetal heart rate increases, so does the possibility that the repolarising activity of the ventricle (the T wave) will occur simultaneously with the P wave of the next atrial contraction. As a result, the morphology of the T wave may theoretically be altered in such a way as to either mask significant changes or to trigger STAN events of no other significance. Therefore, caution should be exercised in the presence of fetal tachycardia secondary to chorioamnionitis. STAN monitoring is not recommended in the presence of cardiac arrhythmias.

**Table 5 - Classification of CTG**

Table 5 - Classification of CTG				
	Baseline Rate	Variability and Reactivity	Decelerations	
<b>Normal CTG</b>	<ul style="list-style-type: none"> <li>110 – 150 bpm</li> </ul>	<ul style="list-style-type: none"> <li>Accelerations</li> <li>Variability 5 - 25</li> </ul>	<ul style="list-style-type: none"> <li>Early uniform decelerations</li> <li>Uncomplicated variable decelerations with duration &lt; 60s and loss of &lt; 60 beats from Base rate</li> </ul>	
<b>Intermediary CTG</b>	<ul style="list-style-type: none"> <li>100 – 110 bpm</li> <li>150 – 170 bpm</li> <li>Decelerations &lt; 100 bpm for ≤ 3 minutes</li> </ul>	<ul style="list-style-type: none"> <li>&gt; 25 bpm (Saltatory pattern)</li> <li>&lt; 5 bpm &gt; 40 min with absence of accelerations</li> </ul>	<ul style="list-style-type: none"> <li>Uncomplicated variable decelerations with duration of &lt; 60s and loss of &gt; 60 beats</li> </ul>	
<b>The combination of several intermediary factors results in an abnormal CTG</b>				
<b>Abnormal</b>	<ul style="list-style-type: none"> <li>150 – 170 bpm and reduced variability</li> <li>&gt; 170 bpm</li> <li>prolonged deceleration &lt; 100 bpm for &gt; 3 minutes</li> </ul>	<ul style="list-style-type: none"> <li>&lt; 5 bpm for &gt; 60 minutes</li> <li>sinusoidal pattern</li> </ul>	<ul style="list-style-type: none"> <li>Complicated variable deceleration with a duration of &gt; 60s</li> <li>Repeated late uniform decelerations</li> </ul>	
<b>Pre-terminal</b>	Total lack of variability (<2bpm) and reactivity with or without decelerations			
ST Analysis				
ST Event	Normal CTG	Intermediary	Abnormal	Pre-terminal
Episodic T-QRS rise	<ul style="list-style-type: none"> <li>Expectant management</li> </ul>	> 0.15	> 0.10	Immediate delivery
Baseline T-QRS rise		> 0.10	> 0.05	
Biphasic ST	<ul style="list-style-type: none"> <li>Continued Observation</li> </ul>	3 biphasic log messages	2 biphasic log messages	

The above guideline was based on FIGO CTG Guidelines of 1987 as ST-Analyser was validated using these guidelines. Based on the experience and perinatal outcomes of the maternity units which have implemented STAN after training staff on fetal physiology, the Editorial Board recommends the use of the STAN guideline in combination of deeper understanding of fetal physiology.

○ Presentation

If a STAN electrode is applied to a fetus in the breech presentation, the ECG recording will be inverted. This can give the impression of a negative ST segment and false biphasic events may be recorded.



There is a 'breech mode' function which can be used to invert the ECG. This was evaluated in an observational study, <sup>Stein et al 2006</sup> which found no increase in adverse neonatal outcomes when compared to using STAN in the vertex presentation.

#### Practice Points when using STAN for fetal Monitoring

1. Always consider the 'Wider Clinical Picture' (Meconium, Oxytocin, Temperature, Haemorrhage, Hyperstimulation, Rate of Progress or the presence of uterine Scar – MOTHERS) whilst using STAN without solely relying on 'Black Boxes' (ST Events).
2. Even in the presence of significant STAN Events in the first or second stage of labour, first take action to improve fetal oxygenation (i.e. stopping oxytocin, changing maternal position and/or administration of terbutaline or stopping active maternal pushing). If the CTG improves and central organs are well oxygenated with no evidence of a subacute or saltatory pattern, it is appropriate to continue labour, based on the observed clinical picture.
3. Conversely, even in the absence of significant ST Events, in the presence of an unstable baseline heart rate, reduced baseline variability with preceding decelerations and an increase in the baseline fetal heart rate or saltatory pattern or absence of cycling or saltatory pattern, urgent action has to be taken to improve fetal oxygenation, irrespective of the presence of STAN Events. If fetal condition cannot be improved, urgent delivery should be accomplished, and one should not await STAN Events.
4. In clinical or subclinical chorioamnionitis or in the presence of thick meconium stained amniotic fluid, the pathway for fetal neurological damage may be non-hypoxic and therefore, management should depend on the clinical situation, including the rate of progress and the requirement for oxytocin augmentation, irrespective of the absence of STAN Events. STAN is a test of fetal hypoxia and not of inflammatory neurological and myocardial damage.
5. STAN should not be commenced in cases of chronic hypoxia as the fetus had most likely exhausted all the glycogen reserves in the myocardium and therefore, may not be able to generate any ST Events.

### 3 Fetal Scalp Blood Sampling

The Cochrane systematic review in 2013 has demonstrated that there is no available evidence of a correlation between fetal scalp pH and improvement in long term outcomes. In addition, the review has also demonstrated that contrary to the erroneous belief in the past, current evidence suggests that FBS may increase the number of caesarean sections and operative vaginal births. Further review of evidence has shown rare, but, potentially serious fetal complications. Therefore, as current scientific evidence does not support the use of FBS in clinical practice as its benefits no longer outweigh its risks, the authors of this guideline do not support the use of FBS as an adjunctive technique for the assessment of fetal well-being.

## Special circumstances

Other factors that are present during labour such as prolonged rupture of membranes, defined as spontaneous rupture of membranes for greater than 24 hours, chorioamnionitis, anhydramnios, meconium-stained liquor, maternal infection or pyrexia, and the speed of evolution of hypoxia are likely to modify the responses of the fetus as well as affect the perinatal outcome. <sup>Sacco et al 2015</sup>

### 1 Meconium

Meconium stained liquor (MSL) can be present in a normal post term fetus without an indication that the baby has experienced hypoxia. In a preterm fetus, <34/40, the presence of meconium signifies that there is likely infection, such as listeria, ureaplasma or rotavirus. <sup>Blot et al. 1983</sup> Clear liquor has antibacterial properties, however in the presence of meconium these properties are restricted. <sup>Unsworth and Vause 2010</sup> With thick meconium, E coli has the ability to grow rapidly, whereas Group B Streptococcus proliferates even in clear liquor. <sup>Eidelman et al. 2002</sup> Fetal tachycardia ( $\geq 160$  bpm), in the presence of MSL has a Relative Risk of 51 for the development of chorioamnionitis, in comparison to clear liquor. <sup>Blot et al. 1983</sup>

MSL is associated with complications in the newborn. The most severe complication is meconium aspiration syndrome (MAS). <sup>Unsworth and Vause 2010</sup> Aspiration of meconium can occur in-utero with fetal gasping, or after birth, with the first breaths of life. <sup>Mundhra and Agarwal 2013</sup>

There is still no effective and safe treatment or prophylactic measure for MAS once the meconium has passed below the vocal cords into the lungs. <sup>Chandrarahan and McDonnell 2015</sup>

Evidence shows that when the placental oxygen supply is interrupted, the fetus attempts to breathe. Should these attempts fail to provide an alternative oxygen supply, and if hypoxia continues, the respiratory centre becomes unable to continue initiating breathing and the breathing stops, usually within 2 to 3 minutes. <sup>MOET 2014</sup>

In view of this, extra vigilance should be taken to observe for signs of hypoxia in the presence of meconium. A lower threshold for expediting delivery should be considered when there is meconium and signs of hypoxia as a CTG cannot predict if a fetus will gasp or when this would happen.

If a fetus has passed meconium, the mother should be informed that there is a risk of meconium already being present in the lungs. Most meconium will be expelled from the fetal lungs as the baby passes down the birth canal but in 1-3% of live births, the baby will develop MAS. <sup>Impey et al. 2008</sup>

### 2 Oxytocin and Hyperstimulation

Care should be taken when using prostaglandins or oxytocin to augment or stimulate labour. One of the iatrogenic causes of prolonged decelerations includes prolonged or frequent uterine contractions secondary to oxytocin. If this cause is identified, immediate action should be taken to improve utero-placental oxygenation by stopping oxytocin and changing maternal position to reduce the stress the baby is experiencing. <sup>FIGO 2015</sup> Consideration should also be given to starting acute tocolysis using a beta-adrenergic agonist such as terbutaline. <sup>NICE 2014</sup>

CEFM is necessary with oxytocin augmentation. If the fetal heart rate is normal, oxytocin should be titrated to achieve contractions at a rate of 4:10. It should be reduced if contractions occur more frequently than 5:10. If evidence/suspicion of fetal decompensation occur, oxytocin infusion should be stopped and an urgent assessment of the fetal condition should be undertaken and documented by an obstetrician. <sup>Arulkumaran et al. 2004</sup> In the event of acute hypoxia, oxytocin should be stopped, and the 3-minute rule initiated. A full assessment of the fetal condition must be undertaken and documented by an obstetrician BEFORE oxytocin is recommenced.



### 3 Pyrexia

Heat transmission during pregnancy results in fetal temperature being 0.3-0.5°C higher than maternal temperature. The umbilical circulation transfers 85% of the heat produced by the fetus to the maternal circulation. The remaining 15% is dissipated through the fetal skin to the amnion and is then transferred through the uterine wall to the maternal abdomen. <sup>Lieberman et al. 2000</sup> If there is pyrexia, the metabolic demands of the fetal tissues are increased and so the risk of hypoxia is elevated. <sup>Holt et al. 1994</sup> This should be considered especially when using oxytocin, and a prolonged labour should be avoided. The combination of maternal pyrexia with cord acidosis [indicative of fetal acidosis] greatly increases the risk of neonatal encephalopathy. Evidence suggests that acidosis and pyrexia represent two separate causal pathways of neonatal encephalopathy leading to a cumulative effect. <sup>Impey et al. 2008</sup>

There is no clear evidence to suggest when a fetus should be delivered if there is maternal or fetal infection. In view of the lack of clear evidence/guidance on a safely acceptable time frame for delivery, a clear discussion with the mother should be undertaken with agreed management plan and time frame documented from this. Measures, such as paracetamol, IV fluids and IV antibiotics, should be used to treat any pyrexia and infection. Evidence shows that an intrapartum maternal dose of 1500mg of cefuroxime IV produces effective fetal concentrations for prophylaxis, but not treatment. <sup>Holte et al. 2004</sup>

Intrapartum fever, even when unlikely to be caused by infection, is associated with a fourfold increase in the risk of unexplained, early-onset seizures in term infants. <sup>Holt 1994</sup>

### 4 Antepartum Haemorrhage

Major placental abruption is one of the 3 major intrapartum accidents and may present as a single and sudden drop in the baseline rate (acute hypoxia). In this case, delivery must be expedited as it is most likely to be the evidence of a placental abruption and is irreversible. <sup>FIGO 2015</sup> It is also important to note that the use of tocolytics in APH may aggravate placental separation causing worsening fetal hypoxia.

### 5 Epidural

This can cause a sudden drop in maternal blood pressure which causes redistribution of maternal blood away from the placenta resulting in inadequate placental perfusion. It will present as a single and sudden drop in the baseline rate (acute hypoxia). In this instance, it is reversible and should be corrected by changing the maternal position and IV fluids ± I.V. ephedrine (to be administered by the anaesthetic team). <sup>Greenwell et al. 2012</sup> This vasodilation can also cause an increase in maternal temperature as a result of altered thermoregulation. <sup>RCOG 2015</sup>

### 6 Scar rupture

If a woman has had a previous lower segment caesarean section and begins to labour, the risk of uterine scar rupture is between 0.07% <sup>Nahum and Isaac 2016</sup> – 0.5% <sup>RCOG 2015</sup> and must be considered. This is the third major intrapartum accident and may present as a single and sudden drop in the baseline rate (acute hypoxia). In this case, delivery must be expedited as it is irreversible. <sup>FIGO 2015</sup>

### 7 Subclinical Chorioamnionitis

Research has shown that only 8-12% of women with histologically confirmed chorioamnionitis would demonstrate tachycardia and pyrexia during labour. Therefore, any increase in the baseline fetal heart rate without preceding decelerations should arouse the suspicion of an ongoing subclinical chorioamnionitis. Other clinical parameters, such as presence of meconium, rate of progress of labour, history of prolonged rupture of membranes or prolonged labour and absence of cycling should be considered whilst making management decisions.



## 8 Preterm Afors and Chandraharam 2011

There is paucity of evidence/ guidelines on the use of CTG in Preterm babies. This has resulted in some authors advising against continuous monitoring in extreme prematurity (24 – 28 weeks). The key factors affecting FHR characteristics in the preterm fetus are immaturity of the central and peripheral nervous systems, reduced placental reserve, immature adrenal gland and myocardium, and reduced amount of Wharton's jelly in the umbilical cord.

CTG findings include:

- Immaturity of the autonomic nervous system will result in a higher baseline heart rate and reduced variability.
- Immaturity of the somatic nervous system may result in less accelerations being less frequent and of smaller amplitude (10bpm) and for a shorter duration (10sec). This is especially more evident at gestations before 30 weeks.
- Fetal heart rate decelerations in the absence of uterine contractions often occur in the normal preterm fetus between 20 and 30 weeks' gestation. Variable decelerations have been shown to occur in 70–75% of intrapartum preterm fetuses, in comparison to 30-50% of term fetuses.
- Immaturity of the central nervous system results in a less developed cycling pattern, this is especially more evident in extreme prematurity.

## 9 Effect of medication on the CTG

It is important to consider the effect of any medication administered to the mother during labour and anticipate the changes it may cause on the CTG trace. This is even more important when medications are given for the purpose of improving fetal conditions. In such cases, we would need to consider what to look for as signs of improvement, what may occur if our intervention did not work or if the situation is worsening, how soon to expect changes and how long should they last for.

## References

1. Adalina Sacco, Javaid Muglu, Ramesan Navaratnarajah, Matthew Hogg. ST analysis for intrapartum fetal monitoring. *The Obstetrician & Gynaecologist* 2015; 17:5–12.
2. Afors K, Chandrharan E. Use of continuous electronic fetal monitoring in a preterm fetus: clinical dilemmas and recommendations for practice. *J Pregnancy*. 2011; 2011:848794.
3. Albertson A, Amer-Wåhlin I, Lowe V, Archer A, Chandrharan E. Incidence of subacute hypoxia during active maternal pushing during labour. *RCOG World Congress* 2016.
4. Amer-Wahlin I, Arulkumaran S, Hagberg H, Marsál K, Visser GH. Fetal electrocardiogram: ST waveform analysis in intrapartum surveillance. *BJOG* 2007;114(10): 1191–3.
5. Arulkumaran S, Penna LK, Bhasar Rao K [Eds] (2004) *The Management of Labour* Orient Longman P 95-96.
6. Ayres-de-Campos D, Spong CY, Chandrharan E, 'FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography', FIGO (2015).
7. Bhide A, Chandrharan E, Acharya G. Fetal monitoring in labor: Implications of evidence generated by new systematic review. *Acta Obstet Gynecol Scand*. 2016 Jan;95(1):5-8.
8. Blix E<sup>1</sup>, Brurberg KG<sup>2,3</sup>, Reiherth E<sup>4</sup>, Reinar LM<sup>2</sup>, Øian P<sup>5,6</sup>. ST waveform analysis versus cardiotocography alone for intrapartum fetal monitoring: a systematic review and meta-analysis of randomized trials. *Acta* 2016 Jan;95(1):16-27.
9. Blot P, Milliez J, Breart G, Vige P, Nessmann C, Onufryk JP, Dendrinis S, Sureau C (1983). Fetal tachycardia and meconium staining; a sign of fetal infection. *International journal of gynecology and obstetrics* 21(3) P189-194.
10. Cahill AG<sup>1</sup>, Caughey AB, Roehl KA, Odibo AO, Macones GA. Terminal fetal heart decelerations and neonatal outcomes. *Obstet Gynecol*. 2013 Nov;122(5):1070-6. doi: 10.1097/AOG.0b013e3182a8d0b0.
11. Chandrharan E and McDonnell S (2015) Fetal Heart Rate Interpretation in the Second Stage of Labour: Pearls and Pitfalls. *British Journal of Medicine and Medical Research* 7 (12) P957-970.
12. Chandrharan E. Foetal electrocardiograph (ST-analyser or STAN) for intrapartum foetal heart rate monitoring: a friend or a foe? *J Matern Fetal Neonatal Med*. 2018 Jan;31(1):123-127.
13. Debrah, L and Downe, S 'FIGO consensus guidelines on intrapartum fetal monitoring: Intermittent Auscultation', FIGO (2015).
14. Eidelman AI, Nevet A, Rudensky B, Hamnerman C, Raveh D, Schimmel MS (2002). The effect if meconium staining of amniotic fluid on the growth of *Escherchia Coli* and Group B streptococcus. *Journal of perinatology* 22(6) p467-471.
15. Elimian A, Figueroa R, Tejani N. Intrapartum assessment of fetal well-being: a comparison of scalp stimulation with scalp blood pH sampling. *Obstet Gynecol* 1997; 89(3):373–6.
16. Fawole B. and Hofmery GJ.: Maternal oxygen administration for fetal distress. *Cochrane Database Syst Rev*. 2012 Dec.
17. Greenwell EA, Wyshak G, Ringer SA, Johnson LC, Rivkin MJ, Lieberman E, (2012) Intrapartum Temperature Elevation, Epidural Use and Adverse Outcome in Term Infants, *PEDIATRICS* 129(2)
18. Hamilton E, Warrick P, O'Keeffe D. Variable decelerations: do size and shape matter? *J Matern Fetal Neonatal Med* 2012; 25(6):648–53.
19. Holt DE, Broadbent M, Spencer JA, de Louvois J, Hurley R, Harvey D (1994) The placental transfer of cefuroxime at parturition. *European Journal of Obstetric Gynecology and Reproductive Biology*. May 18;54(3):177-80.



20. Holte K, Foss NB, Svensén C, Lund C, Madsen JL, Kehlet H (2004) Epidural anesthesia, hypotension, and changes in intravascular volume. *Anesthesiology*. 100(2) p281-6.
21. Impey LWM, Greenwood CEL, Black RS, Yeh PS-Y, Sheil O, Doyle P (2008). The relationship between intrapartum maternal fever and neonatal acidosis as risk factors for neonatal encephalopathy. *Am J Obstet Gynecol* 198:49.e1-49.e6.
22. Kamoshita E1, Amano K, Kanai Y, Mochizuki J, Ikeda Y, Kikuchi S, Tani A, Shoda T, Okutomi T, Nowatari M, Unno N. Effect of the interval between onset of sustained fetal bradycardia and caesarean delivery on long-term neonatal neurologic prognosis. *Int J Gynaecol Obstet*. 2010 Oct;111(1):23-7. doi: 10.1016/j.ijgo.2010.05.022. Epub 2010 Aug 4.
23. Leung TY1, Chung PW, Rogers MS, Sahota DS, Lao TT, Hung Chung TK. Urgent caesarean delivery for fetal bradycardia. *Obstet Gynecol*. 2009 Nov;114(5):1023-8. doi: 10.1097/AOG.0b013e3181bc6e15.
24. Lewis D and Downe S FIGO consensus guidelines on intrapartum fetal monitoring: Intermittent auscultation. *International Journal of Gynecology and Obstetrics* 131 (2015) 9-12.
25. Lieberman E, Eichenwald E, Mathur G, Richardson D, Heffner L, Cohen A (2000) Intrapartum Fever and Unexplained Seizures in Term Infants *Pediatrics* 106(5).
26. Liston R, Sawchuck D, Young D, Society of Obstetrics and gynaecologists of Canada; British Columbia perinatal health program. Fetal health surveillance: Antepartum and Intrapartum consensus guideline. *Journal of Obstetrics and gynaecology Canada* 29 (9s4); s3-56 (2007).
27. Macones GA1, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines.
28. Maude RM, Skinner JP, Foureur MJ. Intelligent Structured Intermittent Auscultation (ISIA): evaluation of a decision-making framework for fetal heart monitoring of low-risk women. *BMC Pregnancy Childbirth* 2014;14:184.
29. Medicines and Healthcare products Regulatory Agency. Medical safety alert: Fetal monitor/cardiocograph (CTG) - adverse outcomes still reported (All) adverse outcomes are still reported when CTG traces appear normal - this replaces alert SN 2002(23) issued August 2002. (MDA/2010/054) Gov.co.uk 28 June 2010.
30. MOET working group. 2014. Resuscitation of the baby at birth. In: Paterson-Brown and Howe led. *The MOET course Manual*. Cambridge University Press; 3 edition, pp 139 – 156.
31. Mundhra R, Agarwal M; Fetal outcome in meconium stained deliveries. *J Clin Diagn Res*. 2013 Dec;7(12):2874-6. doi: 10.7860/JCDR/2013/6509.3781. Epub 2013 Dec 15. Ivanov VA, Gewolb IH and Uhal BD (2010) A New Look at the Pathogenesis of the Meconium Aspiration Syndrome: A Role for Fetal Pancreatic Proteolytic Enzymes in Epithelial Cell Detachment. *Pediatric Research* 68 P221–224.
32. Munro J, Jokinen M: Evidence based guidelines for Midwifery-led Care in labour. *Intermittent Auscultation (IA)*. The Royal College of Midwives Trust 2012.
33. Nahum, GG and Isaacs,C (2016). Uterine Rupture in Pregnancy accessed via <http://reference.medscape.com/article/275854-overview#a1> on 23/5/2016 @ 12.00.
34. National Collaborating Centre for Women's and Children's Health *Intrapartum care: care of healthy women and their babies during childbirth*. RCOG Press: London (2007).
35. National Institute for Health and Care Excellence. *Intrapartum care: care of healthy women and their babies during childbirth* NICE clinical guideline 190 (2014).
36. Nurani R, Chandraharan E, Lowe V, Ugwumadu A, Arulkumaran S. Misidentification of maternal



- heart rate as fetal on cardiocography during the second stage of labour: the role of the fetal electrocardiograph. *Acta Obstet Gynecol Scand* 2012; 91(12):1428–32.
37. Olofsson P, Ayres-de-Campos D, Kessler J, Tendal B, Yli BM, Devoe L. A critical appraisal of the evidence for using cardiocography plus ECG ST interval analysis for fetal surveillance in labor. Part II: the meta-analyses. *Acta Obstet Gynecol Scand* 2014;93:571–86.
  38. Peebles DM, Spencer JA, Edwards AD, Wyatt JS, Reynolds EO, Cope M, Delpy DT. Relation between frequency of uterine contractions and human fetal cerebral oxygen saturation studied during labour by near infrared spectroscopy. *Br J Obstet Gynaecol.* 1994 Jan;101(1):44-8.
  39. Pereira S and Chandraharan E. Recognition of chronic hypoxia and pre-existing foetal injury on the cardiocograph (CTG): Urgent need to think beyond the guidelines. *Porto Biomed.* Jul-Aug 2017. 4:124-9.
  40. Pillai M and James D. Behavioural states in normal mature human fetuses. *Arch Dis Child.* 1990 Jan;65(1 Spec No):39-43.
  41. Preti M, Chandraharan E, Lowe V, et al. In: Effectiveness of ‘George’s intrapartum monitoring strategy’ (fetal ECG (STAN), physiology-based training on cardiocography (CTG) and mandatory competency testing) on operative delivery and perinatal outcomes at a teaching hospital in London: a 5 year experience. COGI Conference, Vienna; 2013.
  42. Pulgar VM, Zhang J, Massmann GA, Figueroa JP. Mild chronic hypoxia modifies the fetal sheep neural and cardiovascular responses to repeated umbilical cord occlusion. *Brain Res.* 2007 Oct 24;1176:18-26.
  43. Richardson BS1, Carmichael L, Homan J, Johnston L, Gagnon R. Fetal cerebral, circulatory, and metabolic responses during heart rate decelerations and cord compression. *Am J Obstet Gynecol.* 1996 Oct;175(4 Pt 1):929-36.
  44. Royal College of Obstetricians and Gynaecologists (2015) RCOG Green-top Guideline No. 45 - Birth After Previous Caesarean Birth.
  45. Sacco A, Muglu J, Navaratnarajah R, Hogg M. ST analysis for intrapartum fetal monitoring. *The Obstetrician & Gynaecologist* 2015; 17:5–12.
  46. Skupski DW, Rosenberg CR, Eglinton GS. Intrapartum fetal stimulation tests: a meta-analysis. *Obstet Gynecol* 2002;99(1):129–34.
  47. Stein W1, Hellmeyer L, Misselwitz B, Schmidt S. Impact of fetal blood sampling on vaginal delivery and neonatal outcome in deliveries complicated by pathologic fetal heart rate: a population based cohort study. *J Perinat Med.* 2006;34(6):479-83.
  48. Townsend R & Chandraharan E. Fetal ECG (ST analysis); An evolving standard for intrapartum fetal surveillance. Chapter in: *Current Progress in Obstetrics and Gynaecology*, 2015.
  49. Unsworth J and Vause S (2010). Meconium in labour. *Obstetrics, Gynaecology and Reproductive Medicine* 20(10)p289-294.
  50. Walsh D CTG use in intrapartum care: assessing the evidence. *British Journal of Midwifery* 16(6): 367-369 (2008).
  51. Westerhuis ME, Kwee A, van Ginkel AA, Drogtróp AP, GyselaersWJ, Visser GH. Limitations of ST analysis in clinical practice: three cases of intrapartum metabolic acidosis. *BJOG* 2007;114(10):1194–201.
  52. Williams KP, Galerneau F. Fetal heart rate parameters predictive of neonatal outcome in the presence of a prolonged deceleration. *Obstet Gynecol.* 2002 Nov;100(5 Pt 1):951-4.
  53. Yanamandra N, Chandraharan E. Saltatory and sinusoidal fetal heart rate (FHR) patterns and significance of FHR ‘overshoots’. *Curr Wom Health Rev* Jan2014; 9(3): 175-182.



# Appendix

