

## Get in Touch -

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# Fetal Physiology in relation to Electronic Fetal Monitoring (EFM)



Produced by the East Midlands Maternity Clinical Network & Senate

Last Updated: **February 2019**

## Guidance vs Physiology

Guidance is readily available (NICE, FIGO, Local) correlating different FHR patterns to the percentage likely to be acidotic.

The problem is two fold – fetuses do not conveniently provide a trace that easily falls into one category and these guidance tables are generic, not bespoke to the individual fetal reserve.

In addition to this is the complexity of time continuum, human interpretation and interaction.

Description	Feature		Decelerations
	Baseline (beats/minute)	Baseline variability (beats/minute)	
Reassuring	110 to 160	5 to 25	None or early Variable decelerations with no concerning characteristics* for less than 30 minutes
Non-reassuring	100 to 109† OR 161 to 180	Less than 5 for 30 to 50 minutes OR More than 25 for 15 to 25 minutes	Variable decelerations with no concerning characteristics* for 30 minutes or more OR Variable decelerations with any concerning characteristics* in up to 50% of contractions for 30 minutes or more OR Variable decelerations with any concerning characteristics* in over 50% of contractions for less than 30 minutes OR Late decelerations in over 50% of contractions for less than 30 minutes, with no maternal or fetal clinical risk factors such as vaginal bleeding or significant meconium
Abnormal	Below 100 OR Above 180	Less than 5 for more than 50 minutes OR More than 25 for more than 25 minutes OR Sinusoidal	Variable decelerations with any concerning characteristics* in over 50% of contractions for 30 minutes (or less if any maternal or fetal clinical risk factors [see above]) OR Late decelerations for 30 minutes (or less if any maternal or fetal clinical risk factors) OR Acute bradycardia, or a single prolonged deceleration lasting 3 minutes or more

Abbreviation: CTG, cardiotocography.  
\* Regard the following as concerning characteristics of variable decelerations: lasting more than 60 seconds; reduced baseline variability within the deceleration; failure to return to baseline; biphasic (W) shape; no shouldering.  
† Although a baseline fetal heart rate between 120 and 100 beats/minute is a non-reassuring feature, continue usual care if there is normal baseline variability and no variable or late decelerations.

Intrapartum care: NICE guideline CG190 (February 2017)

	Normal	Suspicious	Pathological
<b>Baseline</b>	110-160 bpm		< 100 bpm
<b>Variability</b>	5-25 bpm	Lacking at least one characteristic of normality, but with no pathological features	Reduced variability. Increased variability. Sinusoidal pattern.
<b>Decelerations</b>	No repetitive* decelerations		Repetitive* late or prolonged decelerations for > 30 min (or > 20 min if reduced variability). Deceleration > 5 min
<b>Interpretation</b>	No hypoxia/acidois	Low probability of hypoxia/acidois	High probability of hypoxia/acidois
<b>Clinical management</b>	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or adjunctive methods	Immediate action to correct reversible causes, adjunctive methods, or if this is not possible expedite delivery. In acute situations immediate delivery should be accomplished

## References

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# Summary

- Changes in CTG cause anxiety and require close observation and interpretation within the clinical context unique to that fetus and mother
- Labour is a stressful event for the fetus and most mount a successful compensatory mechanism to mechanical or hypoxic stress without sustaining any lasting HIE (Hypoxic Ischemic Encephalopathy)
- Timely interventions and judicious use of oxytocin to ensure the uterine environment is optimal for fetal oxygenation
- When properly interpreted (physiology), assessment of the FHR changes in most case proves of equal value to pH measurement (FBS) in predicting fetal outcome (Parer 1982)
- Accelerations are the hallmark of fetal health
- Baseline variability is a good indicator of fetal well being
- Fetal acidosis is more common where there is a loss of baseline variability with tachycardia or late decelerations

## Ask Yourself

- Does this fetus have good reserve?
- Is the fetus compensating to intra-uterine stress?
- Is there evidence of decompensating?

**Remember** – each baby is different, each cord is different, each contraction is different – so read the physiology

CTG interpretation and response – <u>Intrapartum</u> monitoring of high risk cases (FIGO 2015)			
		By definition, all CTG's with these features require further evaluation. The total clinical picture must always be considered when interpreting CTG's – see MOTHERS acronym	
	Normal	Suspicious	Pathological
Baseline (bpm)	110 – 160	Lacking at least one feature of normality but with no pathological features	Above 180bpm or below 100bpm
Baseline variability (bpm)	5-25		Less than 5 (reduced) More than 25 (salutary) Or sinusoidal for >30 minutes
Decelerations	No repetitive* Decelerations		Repetitive late or prolonged decelerations with any concerning characteristics >30 minutes or >20 minutes if there is reduced variability also <ul style="list-style-type: none"> <li>• Reduced variability within the deceleration</li> <li>• Gradual or failure to return to baseline after contraction</li> <li>• Biphasic (W) shape</li> <li>• No shouldering</li> </ul> Or A single prolonged deceleration (below 100 bpm) lasting 5 minutes or more
Accelerations	The presence of fetal heart rate accelerations, even with reduced baseline variability, is generally a sign the baby is healthy. The loss of accelerations alone (in labour) is unlikely to be associated with fetal acidosis.		
Management and Actions	No hypoxia. No intervention necessary to improve fetal oxygenation	Actions to reverse possible causes if identified. Close, continued observation and escalation as required. Consider adjunctive monitoring methods.	High probability of hypoxia / acidosis Involvement of the senior MDT. Immediate actions to reverse possible causes if possible or if this is not possible, expedite delivery.

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>	<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 indications: reduced/fetal movements, thick meconium, bleeding, evidence of oligohydramnios, 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>Chronic Hypoxia</b>	<p><b>Compensated</b></p> <p>Low in the baseline with normal variability and stable baseline preceded by decelerations and loss of accelerations</p> <p><b>Decompensated</b></p> <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>	<ul style="list-style-type: none"> <li>• Likely to require to continue or interventional (see below)</li> <li>• Regular review every 30-60 minutes to assess for signs of further hypoxia change, and that the intervention reached is improvement</li> <li>• Other causes such as reduced placental reserve MUST be considered and addressed accordingly.</li> </ul>
<b>Gradually Evolving Hypoxia</b>		<p><b>First Stage</b></p> <ul style="list-style-type: none"> <li>• Remove prostaglandins/stop oxytocin infusion</li> <li>• If no improvement, needs urgent facilities</li> <li>• If still no evidence of improvement within 30-35 minutes, review situation and expedite delivery</li> </ul> <p><b>Second Stage</b></p> <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 facilities if delivery is not imminent or expedite delivery to operative vaginal delivery</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 salutary pattern (increased variability)</li> </ul>	<p><b>Preceded by reduced variability and lack of cycling or reduced variability within the first 3 minutes</b></p> <p>Immediate delivery by the safest and quickest route</p> <p><b>Preceded by normal variability and cycling and normal variability during the first 3 minutes of the deceleration (see 3 minute rule above)</b></p> <ul style="list-style-type: none"> <li>• Exclude the 3 minute rule, and if hypoxia, proceed to delivery, unless together - if an accident is expected prepare for immediate delivery</li> <li>• Correct reversible causes</li> <li>• If no improvement to 5 minutes or any of the accidents, proceed, immediate delivery by the safest and quickest route</li> </ul>
<b>Acute Hypoxia</b>	Prolonged Deceleration (> 3 minutes)	<p><b>Unable to Ascertain fetal wellbeing</b></p> <p>(Poor signal quality, uncertain baseline, possible recording of the maternal heart rate)</p> <ul style="list-style-type: none"> <li>• Escalate to senior team</li> <li>• Consider Adjunctive Techniques, if appropriate</li> <li>• Consider the application of FBS to improve signal quality</li> </ul>

## The Fetal Reserve

- Early placentation (first 4 weeks since implantation) utero-placental sinuses are developed for subsequent supply of nutrients and oxygen to the fetus and removal of waste products
- If placental reserve is low (smaller sinuses) the fetus may have restricted growth antenatally. This is seen in hypertension and pre-eclampsia
- In labour, as the myometrial fibres contract, this low fetal reserve is likely to develop hypoxia more rapid than a fetus with a larger reserve
- In diabetic pregnancies there are fewer placental pools for gaseous exchange – hence more rapid fetal hypoxia

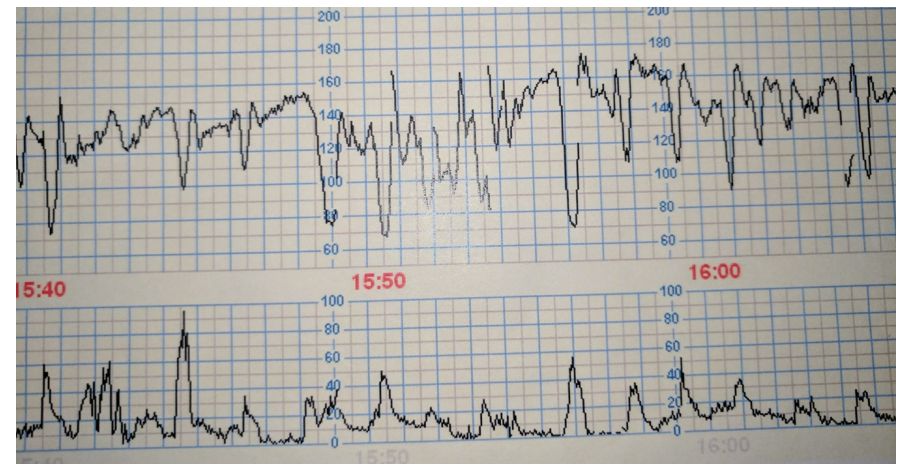
## Recognising and Managing Sub-Acute Hypoxia

- Defined as when the FHR is spending <30 seconds on a stable baseline and >90 seconds in deceleration
- Hence more time protecting its myocardium than at the baseline exchanges gases and protects its brain

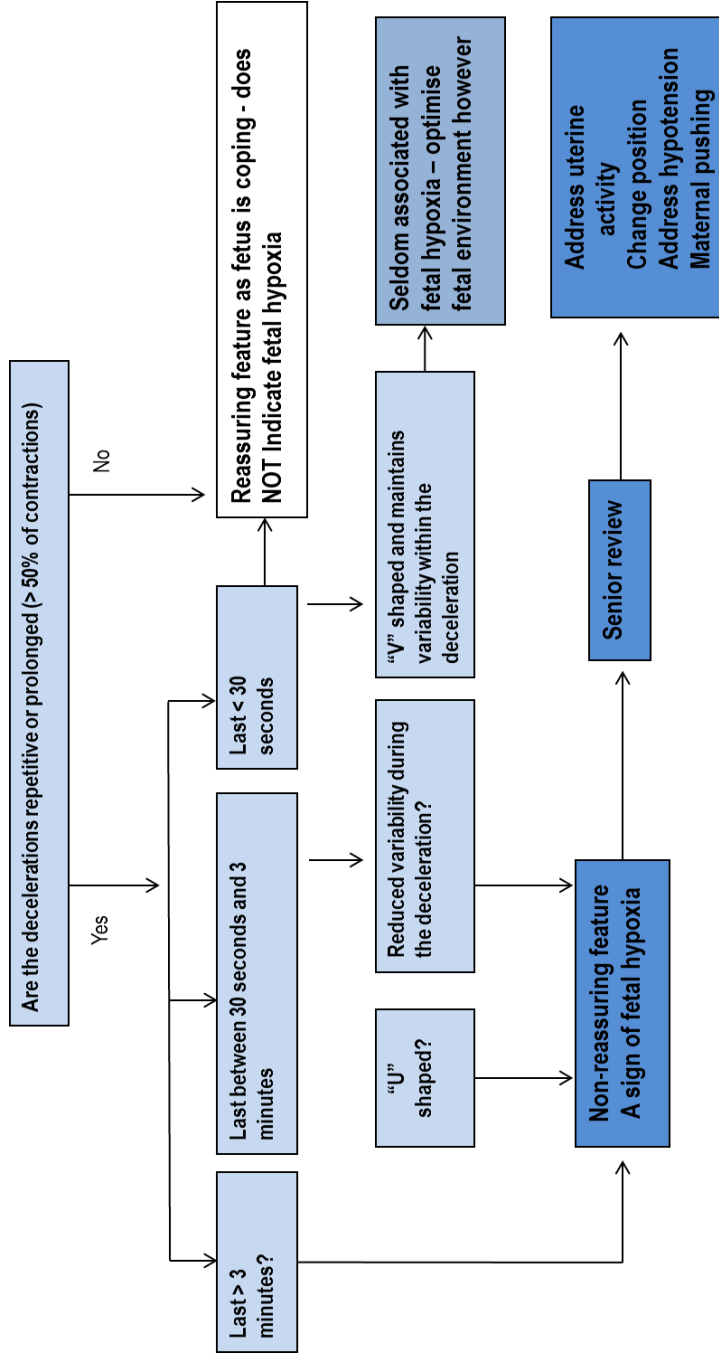
### Common causes of subacute hypoxia – hyperstimulation with oxytocin and second stage

Hypoxia	Features	Management
Subacute Hypoxia	<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 in noted, consider tocolysis if delivery is not imminent or expedite delivery by operative vaginal delivery</li> </ul>

## Hyperstimulation



# Understanding Decelerations as a sign of Hypoxia



Taken from Norfolk and Norwich University Hospitals (2016) Clinical Guideline for the Use of Intrapartum Fetal Monitoring and Fetal Blood Sampling

- The fetus lives in a relatively hypoxic environment (arterial oxygen sats at the start of labour are 70%)
- This can drop to 30% with uterine activity
- The placenta is the respiratory organ for the fetus
- Unlike adults, a fetus has 18-22g of fetal haemoglobin (HbF) which help increase the oxygen carrying capability of fetal blood
- HbF binds with oxygen molecules at higher partial pressures and releases oxygen at very low tensions- allowing the fetus to maintain adequate oxygenation of its organs
- In metabolic acidosis the increased HbF also acts as a buffering system to help avoid fetal neurological damage
- The fetal circulatory system consists of the ductus venosus and foramen ovale – both preferentially shunt oxygenated blood from the umbilical vein to the heart and brain (the most vital organs)
- The ductus arteriosus diverts blood from the pulmonary artery to the descending aorta – bypassing the non-functioning lungs
- Through this arrangement of its vascular system, the fetus is able to supply its central organs with relatively well-oxygenated blood as compared to its peripheries
- The fetal heart has to beat faster to rapidly distribute blood to its organs (110-160bpm)

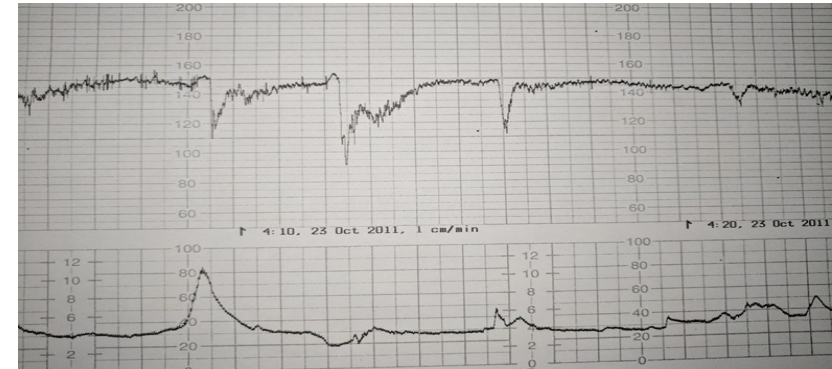


## Fetal Response to Hypoxia

- In response to hypoxic stress, the fetus attempts to protect its heart (myocardium) above everything else
- A fetus cannot rapidly increase its oxygen levels through increasing its respiratory rate like we do as adults
- It can decrease the myocardium workload by a reflex slowing of the FHR. This is a **deceleration**
- If this reflex response is insufficient at oxygenating the vital organs, the fetus also conserves non-essential activity by stopping movements

### This is seen as a loss of accelerations on the CTG

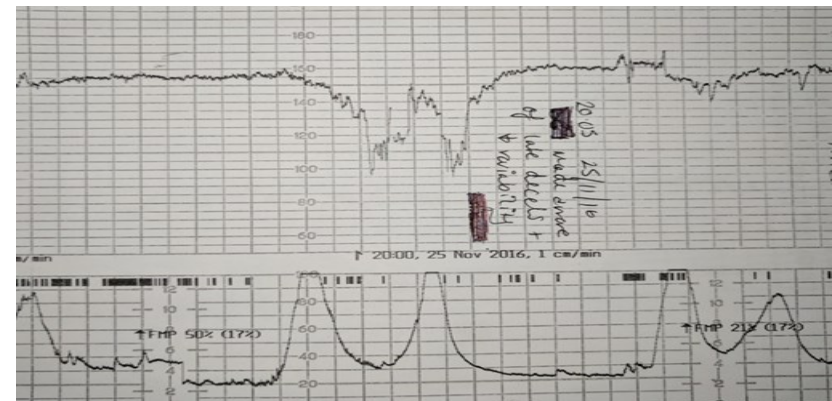
- If the hypoxia continues, stress hormones (catecholamines) are released (adrenaline and noradrenaline) which increases the heart rate to increase oxygenation. This requires energy – glycogen is broken down to glucose which also maintains positive energy balance in the heart (further protection)



## Concerning Chemical Induced Decelerations Suggest Compensation

Loss of the features that reassure us

- ⇒ Loss of shouldering
- ⇒ Drop below 60 bpm
- ⇒ Delayed return to baseline
- ⇒ Overshoot present
- ⇒ Not 'V' shaped – may be 'U' shaped or 'W' shaped
- ⇒ If these decelerations continue, there may be a component of acidosis within time delivery

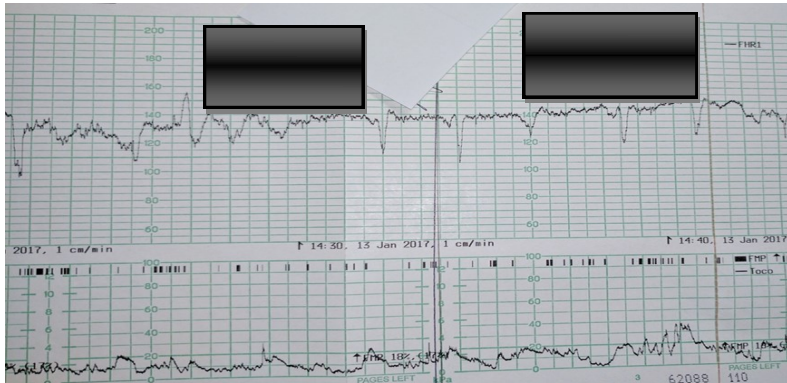


## What are “variable” Decelerations?

- **ALL** decelerations are technically variable!

Because they **vary** in shape, length, size and timing to contractions. Most commonly seen in labour

- Caused by cord compression – baroreceptor mechanism
- Features that reassure us – shouldering, sharp fall <60bpm, quick rise, second shouldering and final recovery to baseline. They are V-shaped



## “Late” Decelerations

- Occurs late in relation to contraction
- Related to fetal hypoxemia, hypercarbia and fetal acidosis – which stimulate chemoreceptors

**Any change in the baseline FHR (rise) and/or variability, with preceding late decelerations requires immediate delivery**

## Glycogenolysis

- The release of adrenaline stimulate **glycogenolysis**
- When fetal oxygen supply is no longer sufficient to maintain energy requirements, **glucose is released from glycogen stores** and metabolised anaerobically (without oxygen)
- During anaerobic metabolism, stores of glycogen in the heart, muscle and liver are broken down to provide energy
- Lactate is the by-product of anaerobic metabolism, which eventually causes the pH of the fetal blood to fall further (metabolic acidosis)

## Gas Exchange at the Placenta

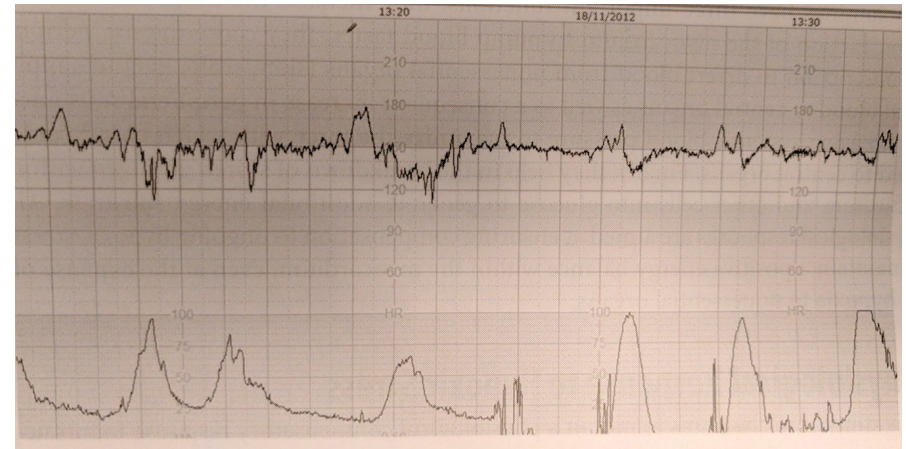
- Gas exchange is impaired during contractions (blood flow into the intervillous space is interrupted) causing retention of CO<sub>2</sub> and lowering the pH of the fetal blood
- The frequency, duration and strength of uterine contractions must be considered when interpreting the CTG
- **This should be resolved when placental perfusion is restored in between contractions, provided there is:**
  - ⇒ Sufficient time between contractions
  - ⇒ A healthy placenta
  - ⇒ The cord isn't occluded
  - ⇒ The mother isn't hypotensive
- The onset of active second stage decreases maternal oxygenation even further.
- Aim for a 90 second inter-contraction period (3-4 contractions in 10 min)

**Uterine hyper-contraction is the most frequent cause of a pathological CTG**

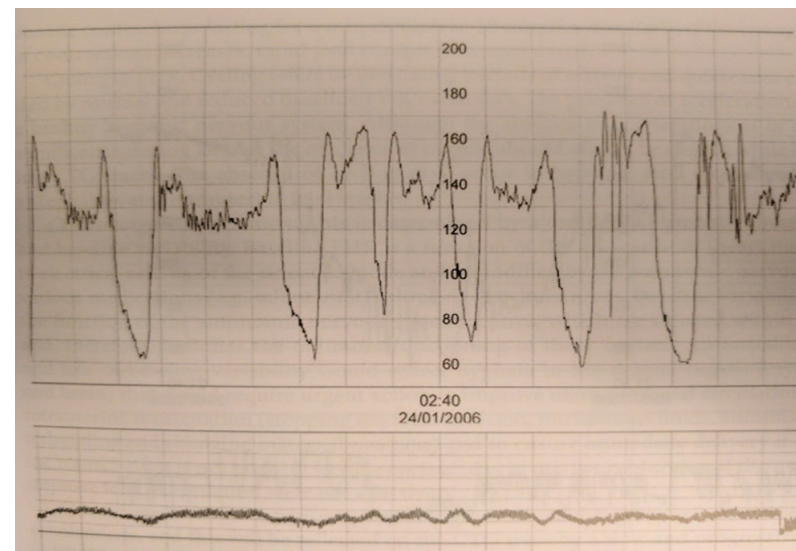
## Think!

What can you do to improve the fetus uterine environment?

## 8. Baroreceptors to protect the Myocardium



## 9. These are still baroreceptors – why?





## Remember...

- Baseline and variability are the most important features on a CTG – they are indicative of hypoxia
- Remember a fetus will protect its heart muscle as a priority.....the other organs and the brain will suffer the hypoxia first
- It is during relaxation of the heart muscle (not contraction ) that the muscle gets its own supply of oxygen and nutrients - that's why the rising baseline is so significant
- Also interpret the CTG in the full clinical context and understanding of the fetal reserve

## Decelerations

- Transient decrease in FHR >15bpm lasting longer than 15 seconds
- We need to rethink decelerations not as “fetal compromise / distress” but as fetal response to ongoing stress
- Reflex response of the fetus to the on-going hypoxia or mechanical stresses – the team must decide!

**Baroreceptor Decelerations** – occur secondary to an increase in fetal systemic blood pressure (occlusion of umbilical arteries during compression of the cord)

⇒ Rapid fall then rapid recovery to baseline

**Chemoreceptor Decelerations** –occur secondary to build up of carbon dioxide and metabolic acids during hypoxia (caused by utero-placental insufficiency, repeated and sustained contractions or prolonged cord compression)

## A Compensated Response

- **Following these physiological changes** (reflex response, stopping un-necessary movements and releasing stress hormones) , the fetus is showing **compensation:**

Seen as a **stable baseline, reassuring variability**, albeit with **continuing decelerations (non-reassuring)** and a **rise in baseline**

- Recognition and subsequent changes to the uterine environment at this crucial time can reverse this hypoxia. Remember that chemoreceptor-mediated decelerations (the non-reassuring ones) take longer to recover as fresh oxygenated blood from the mother is required to wash out the stress hormones – hence why they are slower to recover to the baseline
- **Continuing hypoxia causes decompensation** in the central nervous system (inadequate oxygen reaching the fetal brain):

Seen as a **loss of baseline, variability** and ultimately **myocardial hypoxia** – unstable baseline and **progressive reduction in FHR (bradycardia)**

## Recognition and Management of Evolving Hypoxia

- Fetal physiology is so very different to that of an Adult
- If you can't increase your supply -you decrease your demand!**

**EFM is the art of reading the physiology within the clinical context to observe for signs of hypoxia, whilst improving the uterine environment to allow for adequate oxygenation of the vital organs**

Features	Management
<p><b>Compensated</b></p> <p>Rise in the baseline (with normal variability and stable baseline) preceded by decelerations and loss of accelerations</p>	<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 improvement.</li> <li>Other causes such as reduced placental reserve MUST be considered and addressed accordingly.</li> </ul>
<p><b>Decompensated</b></p> <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>	<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>

Taken from Hypoxia Classification Table – Physiological CTG.com

## Recognising and Managing Sinusoidal Patterns

### 6. Sinusoidal (Saw-tooth)

In the antenatal CTG – an abnormal pattern distinguishable from variability as the fluctuations from baseline are regular in amplitude and in frequency

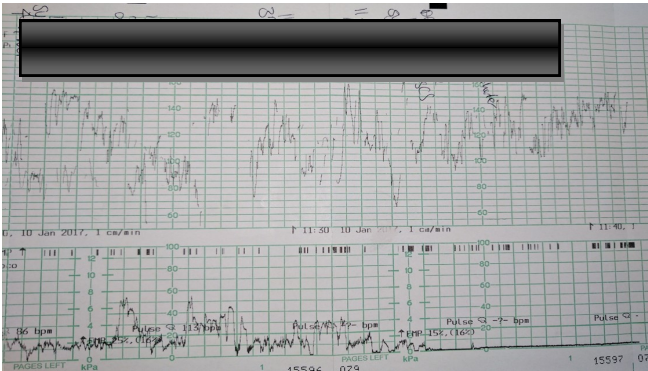


### 7. “Pseudo” Sinusoidal CTG

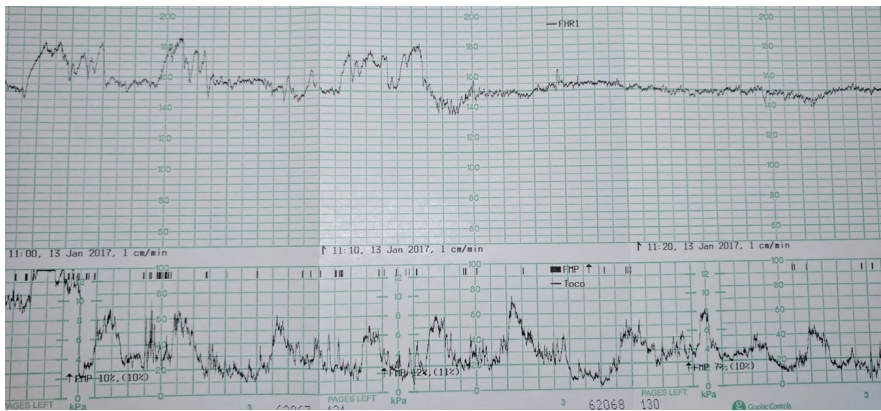
Note normal variability and accelerations prior and after. Does not normally exceed 30 minutes.



#### 4. Saltatory Variability

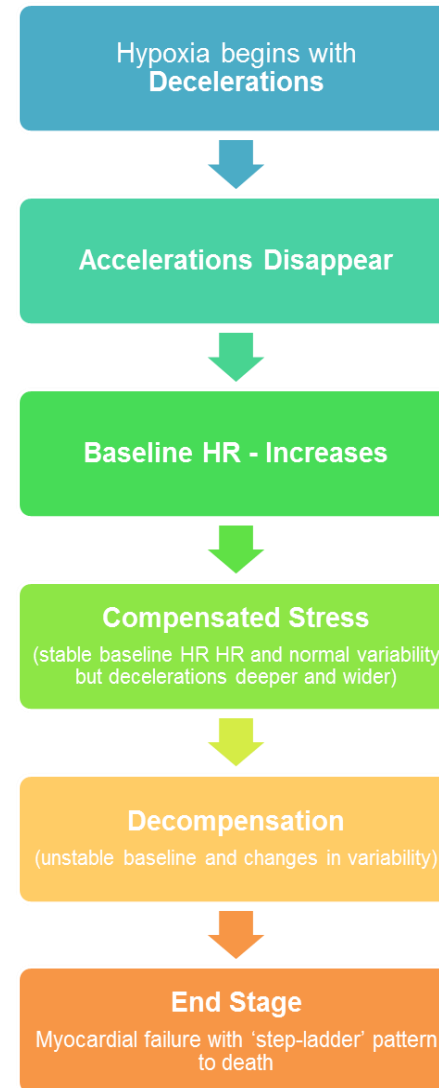


#### 5. Cycling



### Observe, Classify, Predict & Act

We must **observe** and **classify** the physiology, **predict** how it might get worse and **act**



# Lactate and FBS

- Peripheral testing should not be used in isolation but as an adjunct to the CTG and clinical picture
- **FBS estimates the capillary blood pH from peripheral scalp surface**, hence might not truly reflect the acid-base status of the fetus
- There is **no evidence that FBS reduces caesarean section rates** or improves long-term neurological outcomes for neonates (Alfirevic et al 2013)
- NICE acknowledge that FBS increases LSCS rates and instrumental vaginal deliveries
- **FBS can in fact cause delay in delivery** of the fetus that warrants it the most
- FBS does not distinguish between respiratory and metabolic acidemia
- It is **metabolic acidosis that is damaging** to the fetal tissues , where as respiratory acidosis is transitory (not permanent) , and not uncommon in low-risk labours (Gibb and Arulkumaran 2017)
- When the fetus is in compensatory mode glycogenolysis will create a **rise in lactate** and a further shift of the pH into **metabolic acidosis**

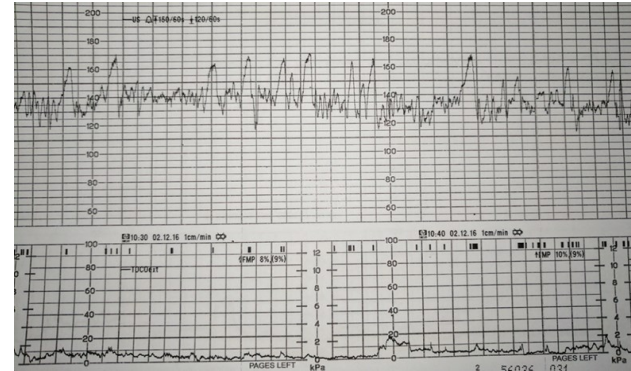
**More comprehensive blood gas analysis – one that includes PCO2, base excess and lactic acid is more desirable and predictive**

## Think!

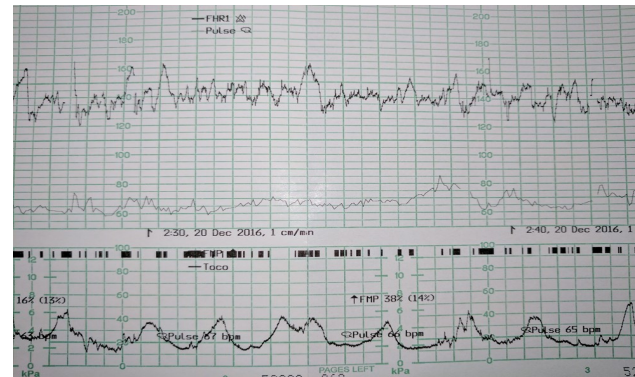
Have you experienced the phenomenon of a baby born with cord pH below 7.00 but Apgar at 1 minute >7?

This would have been respiratory acidosis, not metabolic

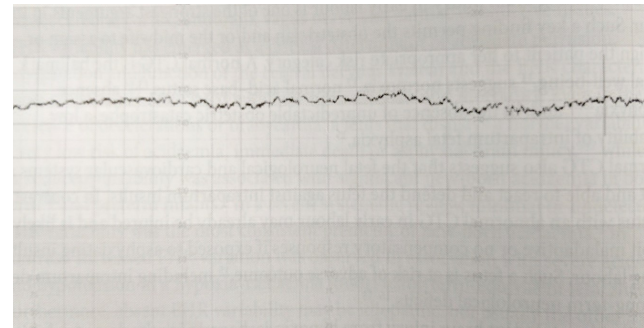
## 1. Accelerations



## 2. Normal Variability



## 3. Reduced Variability





## Accelerations

- Transient increase in baseline of >15bpm
- Presence of 2 or more in 20 minute period is reassuring
- Absent when fetus is sleeping, in chronic hypoxia, drugs and infection
- Erroneous monitoring of maternal pulse may show “accelerations” of greater magnitude and coinciding with uterine contractions

## Variability

- Bandwidth variation of the baseline – excluding accelerations and decelerations
  - ⇒ Normal 5-25bpm – a reassuring feature
  - ⇒ Reduced <5bpm – non-reassuring
  - ⇒ Saltatory >25bpm – non-reassuring
- Normal variability is unlikely to be associated with cerebral hypoxia
- Cycling – refers to alternating patterns of activity and then quiescence – seen as normal and reduced variability. Accelerations present on a CTG signify a healthy functioning of the somatic nervous system. Although the absence of accelerations is of uncertain significance, evidence of cycling should always be sought.
- Absence of cycling may occur with hypoxia, infection and fetal stroke

- **FBS in gradually evolving hypoxia** – in the absence of an acute accident (cord prolapse, abruption, scar dehiscence), it is unusual for a fetus who has shown accelerations and normal baseline variability to become hypoxic in labour
- **The presence of decelerations** – which would classify the CTG as suspicious, either indicate the presence of stress – either hypoxic or mechanical. If the baseline hasn’t started to rise and the variability remains within normal range and hence normal – there is little to be gained from FBS
- Clinicians must identify the **stress-to-distress period**, that will be a unique period of time for each fetus, depending on the fetal reserve

NICE advocate that fetal scalp stimulation can also be deployed to test for hypoxia

Indeed if the FHR reacts to the VE and start of FBS process then the FBS should be abandoned.

Interpreting FBS Results		
Scalp pH	Scalp lactate	Interpretation
≥ 7.25	≤ 4.1	Normal
≥ 7.21-7.24	4.2-4.8	Borderline
≤ 7.20	≥ 4.9	Abnormal

## When Not FBS

- The fetal reserve is **LOW** in **HIGH risk situations** of:

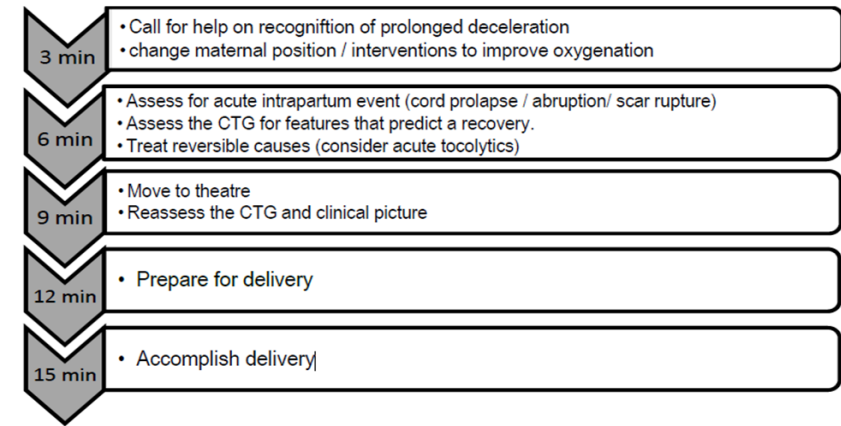
- ⇒ Postmaturity
- ⇒ UGR
- ⇒ Suspected or confirmed intrauterine infection

### Think! MEWS

- ⇒ Significant Meconium
- ⇒ Scanty amniotic fluid

- The challenge to clinicians is the timing of an FBS – when used as an adjunct to interpreting the physiology and the clinical picture

### Management of prolonged decelerations



## Recognising and Managing Acute Hypoxia

- Identify if the cause is reversible?
- Acute “accidents” – cord prolapse, scar dehiscence, abruption require immediate delivery

Hypoxia	Features	Management
Acute Hypoxia	Prolonged Deceleration (> 3 minutes)	<p>Preceded by reduced variability and lack of cycling or reduced variability within the first 3 minutes</p> <p>Immediate delivery by the safest and quickest route</p>
		<p>Preceded by normal variability and cycling and normal variability during the first 3 minutes of the deceleration (see 3-minute rule above)</p> <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>

## Bradycardia or Prolonged Deceleration?

### CTG parameters that predict recovery of prolonged decelerations

- Most prolonged decelerations with a reversible cause will respond to conservative measures to improve the utero-placental circulation, so the approach to the woman and her family should be reassuring
- The CTG features prior to the prolonged deceleration provide information on the oxygenation of the fetus prior to the onset on current insult (prolonged deceleration)
- The variability on the CTG corresponds to the integrity of the fetal autonomic nervous system – If there is normal variability in the 3 minutes before the deceleration and in the first 3 minutes of the deceleration then it is highly likely the FHR will recover – 90% in 6 minutes and 95% in 9 minutes
- If there is reduced variability before the prolonged deceleration, then even after recovery is seen there is a high probability of hypoxia and consideration should be given to delivery after consideration of the wider clinical picture

## Suspected or Confirmed Sepsis

- **Chorioamnionitis** is a significant cause of non-hypoxic fetal compromise
- **Warning** – in cases of intrauterine infection, the high metabolic rate presents a greater oxygen demand to the fetus
  - metabolic acidosis (that can do harm to fetal tissue/organs) might develop with minimal interruption of placental perfusion (seen on the CTG as decelerations)
- Coexistence of intrauterine infection and hypoxia further increase the risk of cerebral palsy
- **Do not rely on pattern recognition** of the “pathological” CTG to prompt actions in cases of suspected or confirmed intrauterine infection
- **Screen the Mother** – history, clinical exam, MEWS, bloods, involve the senior MDT
- **Clinical Features of Chorioamnionitis**
  - ⇒ Maternal pyrexia
  - ⇒ Persistent fetal tachycardia (exclude dehydration and other features of hypoxia)
  - ⇒ Maternal tachycardia
  - ⇒ Uterine tenderness
  - ⇒ Offensive liquor
  - ⇒ Purulent discharge
  - ⇒ Meconium stained liquor may also be a possible sign

## CTG Key Features: Chorioamnionitis

- Features can include- but none have been seen consistently
  - ⇒ Fetal tachycardia
  - ⇒ Reduced variability
  - ⇒ Lack of accelerations
  - ⇒ Presence of decelerations
  - ⇒ Lack of cycling
- FBS is contraindicated in suspected or diagnosed intrauterine infection – the test result is unreliable in the metabolic context of infection
- Ensure paired cord gasses are taken and the placental swab for histology
- The placenta should be sent for histopathological examination

## Try This Exercise!

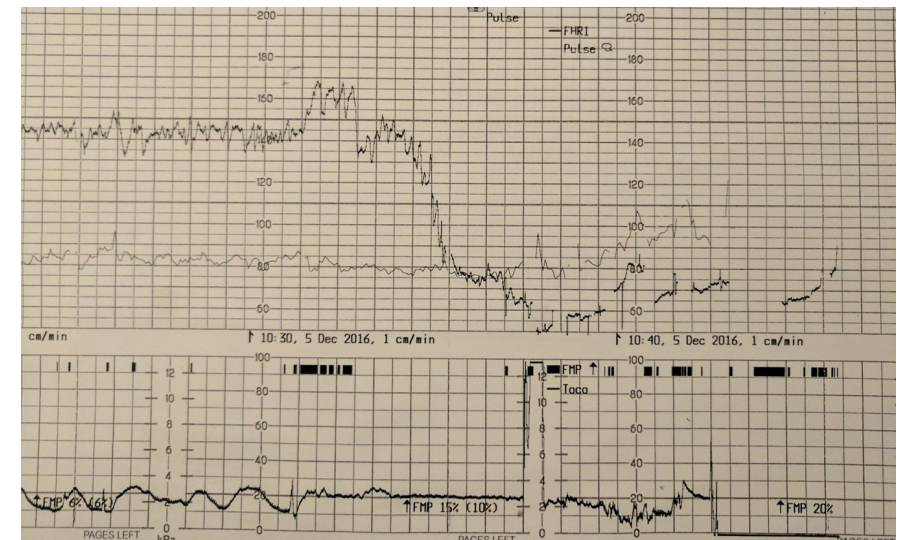
Squeeze your fist 130 times in a minute  
– this is sustainable.

Then increase this to 150 times in a minute  
– what do you notice?

Take this up to 170 squeezes and your hand starts to hurt and cramp up.

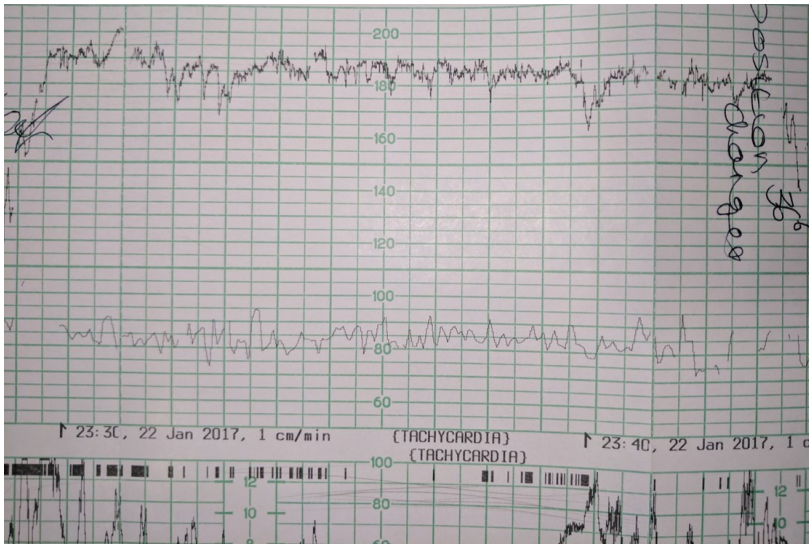
Keeping this going is difficult and eventually your hand starts to slow down and become “bradycardic”

## Bradycardia





## Tachycardia



### Rising baseline in Gradually Evolving Hypoxia

- A fetus exposed to gradually evolving hypoxia will release stress hormones (catecholamines) – this is reflected on the CTG as a slow and gradual rise in the baseline
- Importance of comparing baseline over series of hours and “fresh eyes” to notice these subtle changes
- It is vital that we notice this attempt by the fetus to compensate and take actions to improve the uterine environment (changing maternal position, stopping or reducing oxytocin)
- This continuing catecholamine surge is energy intensive and produces lactate within the myocardium
- If the environment is not improved and hypoxia continues, loss of baseline variability is seen and then terminal bradycardia.

## CTG Key Features:

### Applying an understanding of Physiology

#### Antenatal CTG (no uterine stress)

– what generates CTG features?

#### Autonomic nervous system - involuntary

- Controls HR, breathing, bowel function etc.
- Generates a **BASELINE**
- Interplay between **Sympathetic** (fight or flight) and **parasympathetic** (rest and digest) nervous systems generates **VARIABILITY**

**Decelerations** reflect hypoxic insult

- Poor placental perfusion
- Maternal complications
- Acute events – Abruption / cord prolapse



**Somatic nervous** system control **voluntary** body movements

- Transient activity causes transient changes to HR
- Generates **ACCELERATIONS**

## Recognition and Management of Chronic Hypoxia in the antenatal CTG

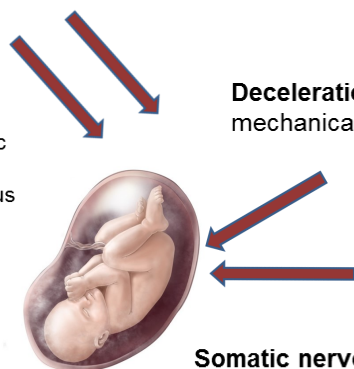
- Chronic utero-placental insufficiency and antenatal insults may lead to a hypoxia that has existed for days and weeks possibly
- Compensation attempts (increase in baseline FHR) stress hormones (shallow decelerations) and evidence of decompensation (loss of baseline variability) will be present on the CTG
- This fetus would have reduced reserve to cope with hypoxia in labour

Hypoxia	Features	Management
Chronic Hypoxia	<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>

## Intrapartum CTG – With Stress!

### Autonomic nervous system

- Generates a **BASELINE**
- Interplay between Sympathetic (fight or flight) and parasympathetic (calm) nervous systems generates **VARIABILITY**



**Decelerations** reflect mechanical or hypoxic insult?

### Somatic nervous system

- Transient activity causes transient changes to HR
- Generates **ACCELERATIONS**

## Baseline Fetal Heart Rate

- The mean level of the FHR when this is stable
- Analysed over 10 minutes
- Normal range 110 – 160bpm
- Gestational appropriateness – baseline lowers as gestational age advances and the autonomic nervous system matures
- Baseline tachycardia (>160bpm) for longer than 10 min – associated with maternal tachycardia, dehydration and pyrexia- always suspect intrauterine infection
- Maternal temp rise of 1 degree = 10% increase in baseline
- Look back at previous CTG's for an understanding of baseline
- Bradycardia is a change to the baseline (FHR <110bpm) that lasts longer than 10 minutes