Cardiotocography

CTG

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DEVELOPMENT COOPERATION

Why is CTG important?

- Alpha and omega of the assessment of fetal condition
- Learn = understand
- Understand: what is happening to the fetus, the mother, why it is happening, if it is okay and what we can do about it

A matter of life and death/health

Cardiotocography

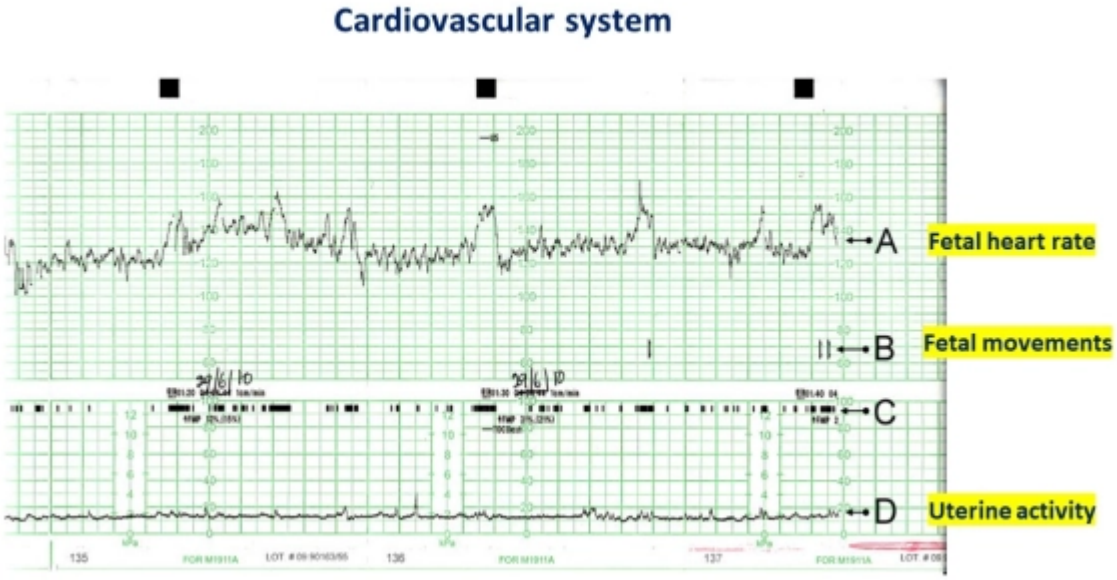
Method of fetal monitoring:

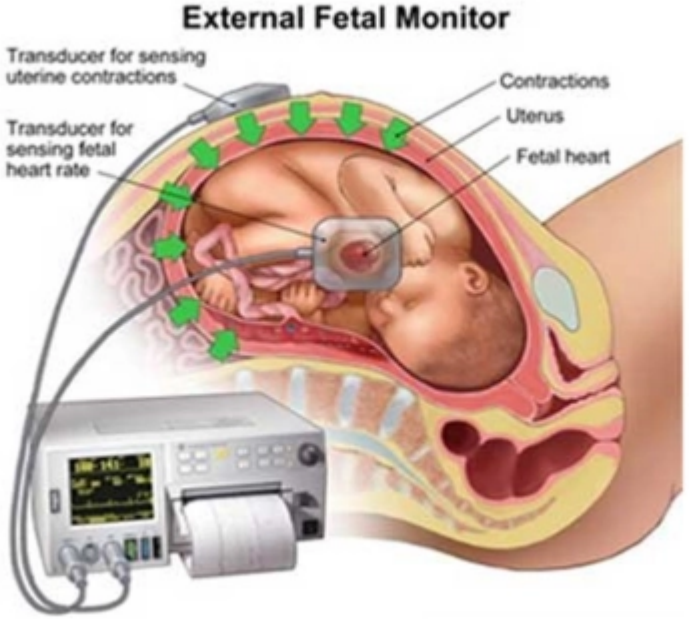
- Fetal heart rate recording (cardiotachogram)

- Recording of uterine contractions (tocogram)
- Recording of fetal movements

Principle:

- hypoxic changes affect fetal haemodynamics, and changes in uteroplacental circulation are manifested by a change in the frequency of fetal echoes





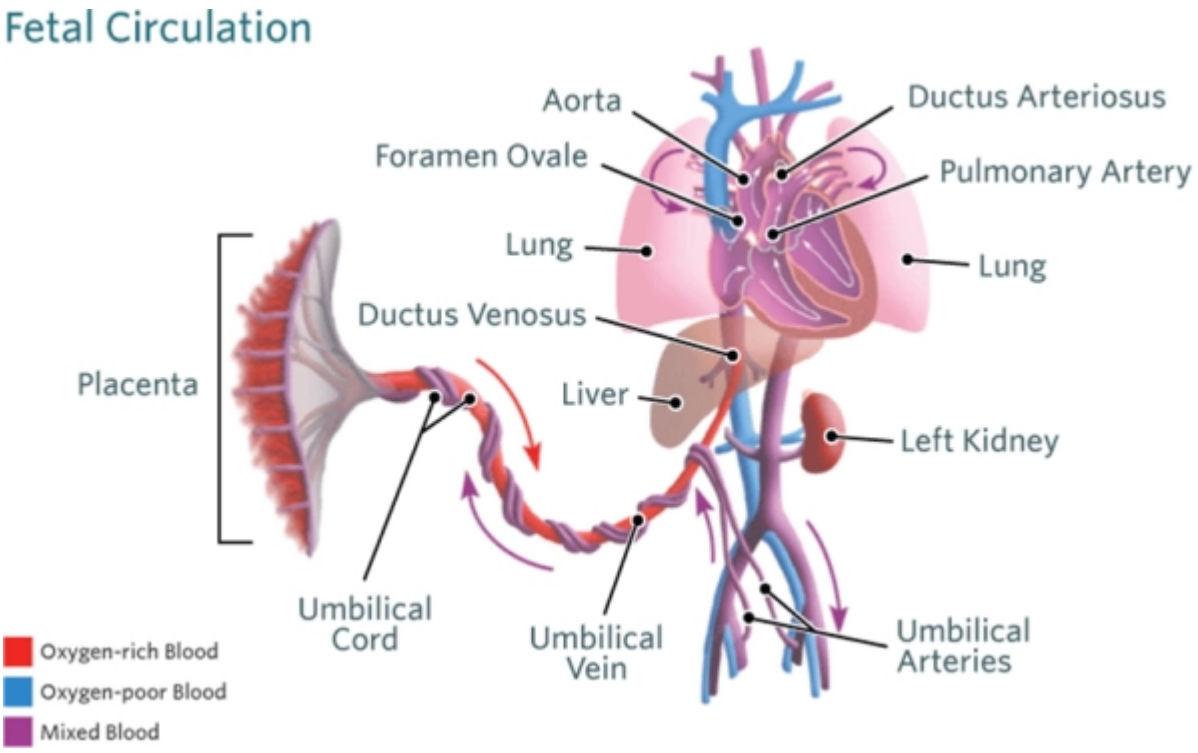
Technical aspects of CTG

- The device works on the principle of ultrasound (external probe)
- The internal probe directly senses electrical potentials (like an ECG)
- **Pay attention to the speed of paper movement (in the Czech Republic, the standard is 1 cm/min)**

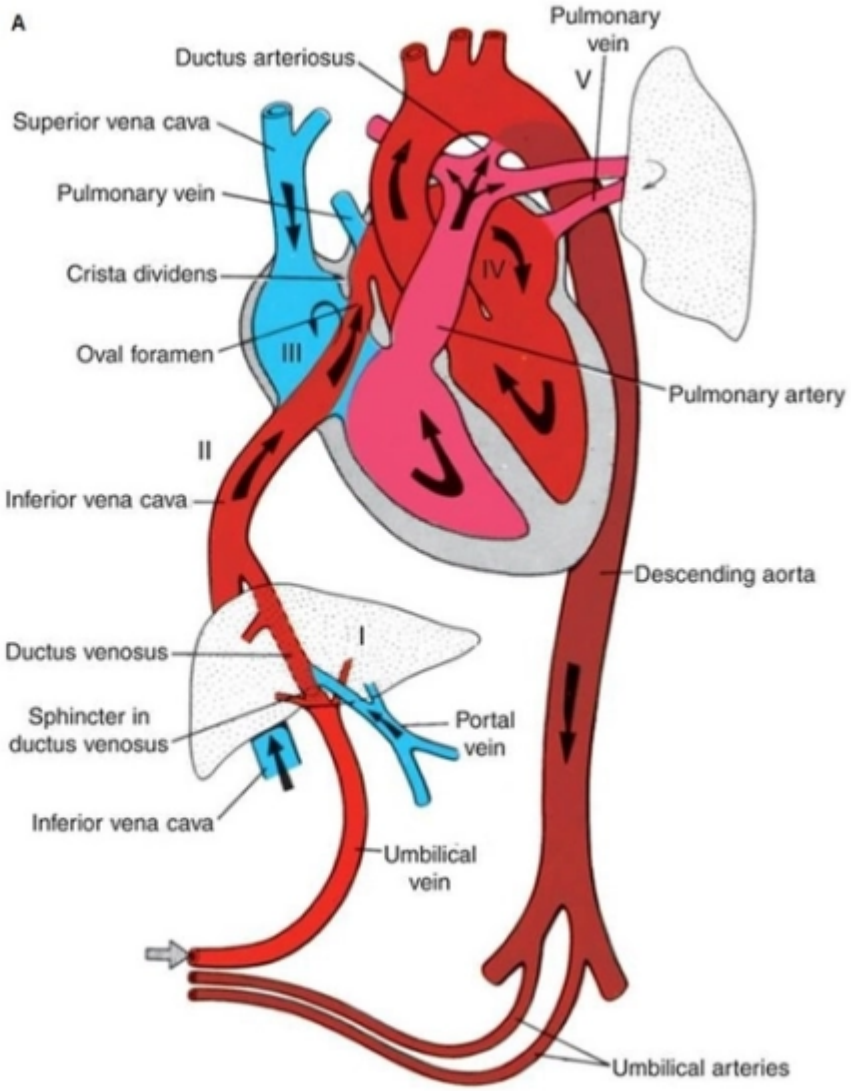
Fetal adaptation to hypoxia

- Intrauterine environment = permanent relative hypoxia
  - -> the fetus is ready

	Mother/Adult	Fetus
Saturation	97%	70% (childbirth >30%)
Haemoglobin	+- 120g/l	180-220 g/l
	Low affinity to O2	High affinity to O2
	Lower buffering capacity	Higher buffering capacity
P	60-80‘	110-160‘
Capillary density	low	high
		Anatomical proportions



1. heart
2. brain



Regulation of FHR

- The heart is a semi-autonomous organ
- Affected by sympathetic (increases FHR) and parasympathetic (decreases FHR)

BF (basal frequency) – gradually decreases with the length of pregnancy (sympathetic matures earlier, parasympathetic later)

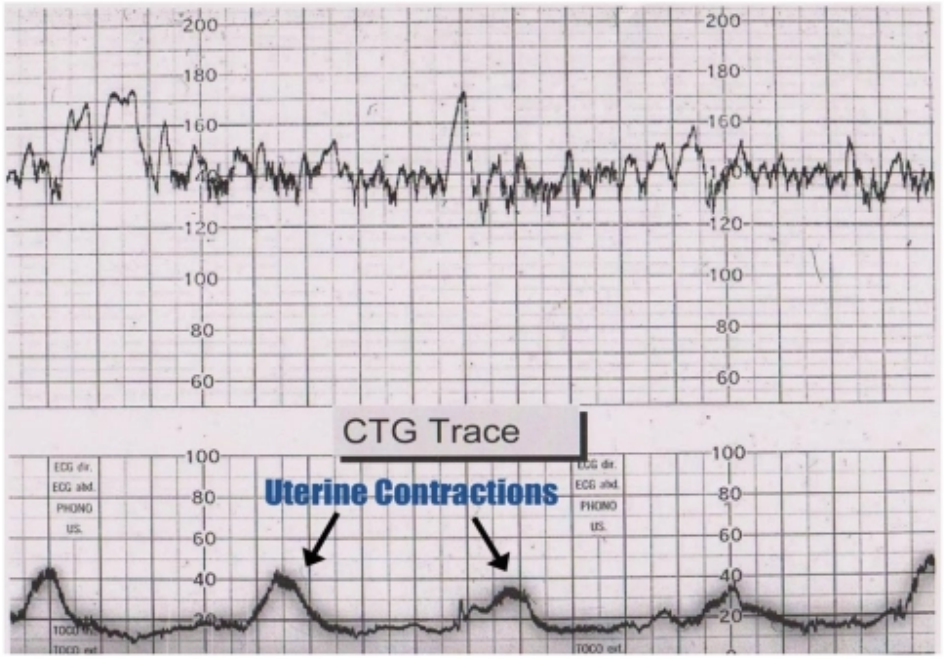
Variability – see above, around term balanced action – „struggle for power“ – constant regulation – BF amplitude

Cardiotocography

- Antepartum (FIGO 1986) – „stricter“
- Intrapartum (FIGO 2015)

Tocography

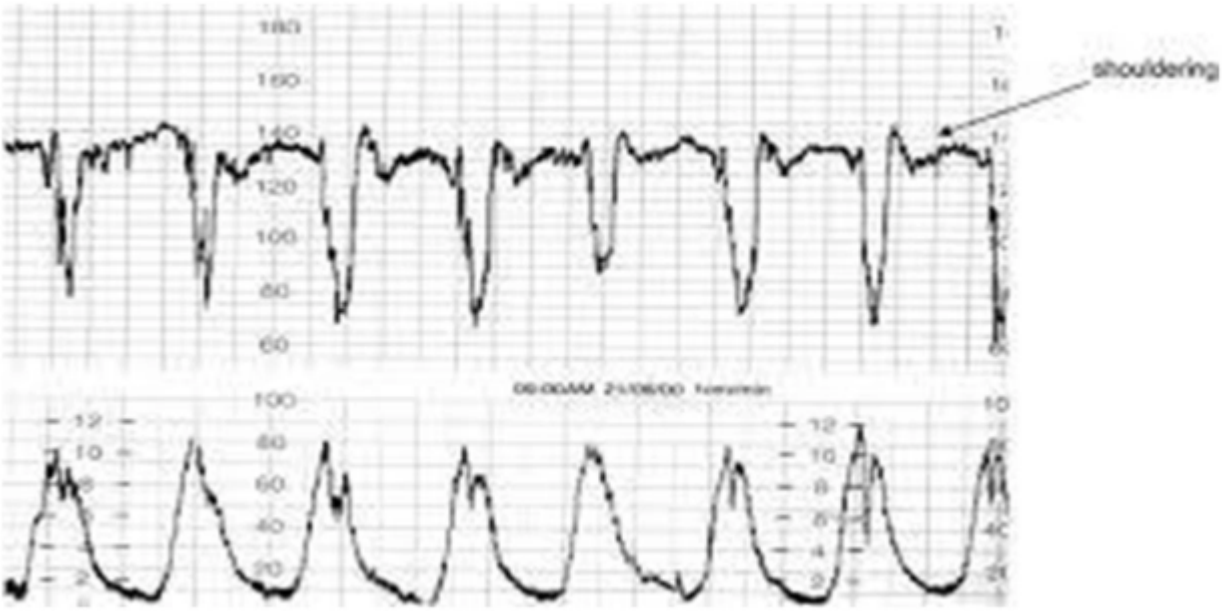
- Record of uterine contractions (tocogram)
- Measured mostly by an external sensor (rarely internal – rather experimental)
- The absolute pressure values are indicative only.
- The length, intensity and frequency of uterine contractions can be estimated on the tocogram.
- It is important for the evaluation of medium-term phenomena in the cardiotachogram.



Tachysystole

- Excessive uterine activity

>5 contractions per 10 minutes for at least 30 minutes or in two consecutive periods lasting 10 minutes



Hypertonus

- Uterine contractions lasting more than 2 min (normally 45-120s)
- Increase in the basal tone of the uterus
- Contractions are necessary for the progress of labour, but perfusion in the placenta occurs/can occur during them

Excessive uterine activity can lead to intrauterine fetal distress
Causes:

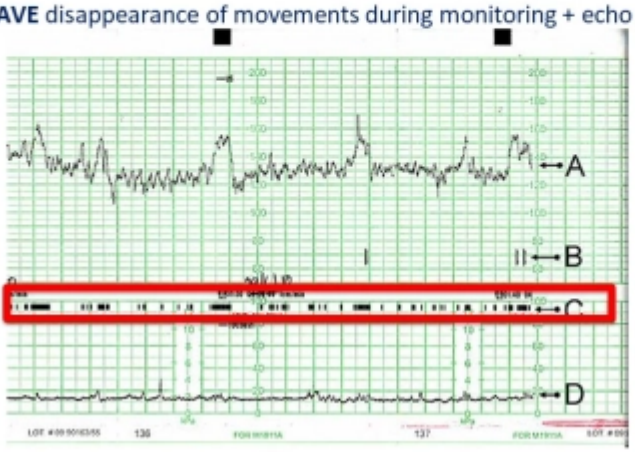
- Induction (prostaglandins)
- Oxytocin
- **CAVE:** placentalabruption, uterinerupture

Therapy: EDA, tocolysis, give birth (in case of abruption/rupture) -> C-section

Evaluation of fetal movements

- Presence of movements = positive
- Absence of movements = not necessarily a negative

(**CAVE** disappearance of movements during monitoring + echo alteration)



Cardiotachogram

- Key component of CTG recording
- Fetal heart rate assessment
- **hypoxic changes affect fetal haemodynamics, and changes in the uteroplacental circulation are manifested by a change in the frequency of fetal echoes**

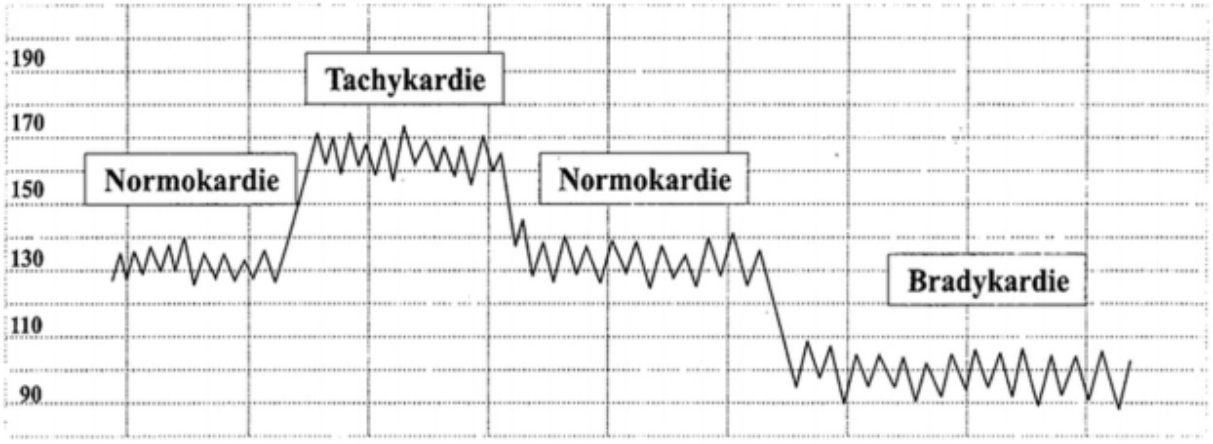
Evaluationoftherecordfromthe point ofview:

- Long-term phenomena
- Medium-term phenomena
- Short-term phenomena

Long-term phenomena – basal frequency

- stable heart rate during a period of 10 minutes, from which medium- and short-term phenomena deviate.
- **Normal BF** (normocardia) - 110–160/min (antepartum 110-150/min)
- **Tachycardia** - >160/min (preterm fetuses +- 160/min)

- **Bradycardia** - <110/min (post-term fetuses physiol. 100-110/min)

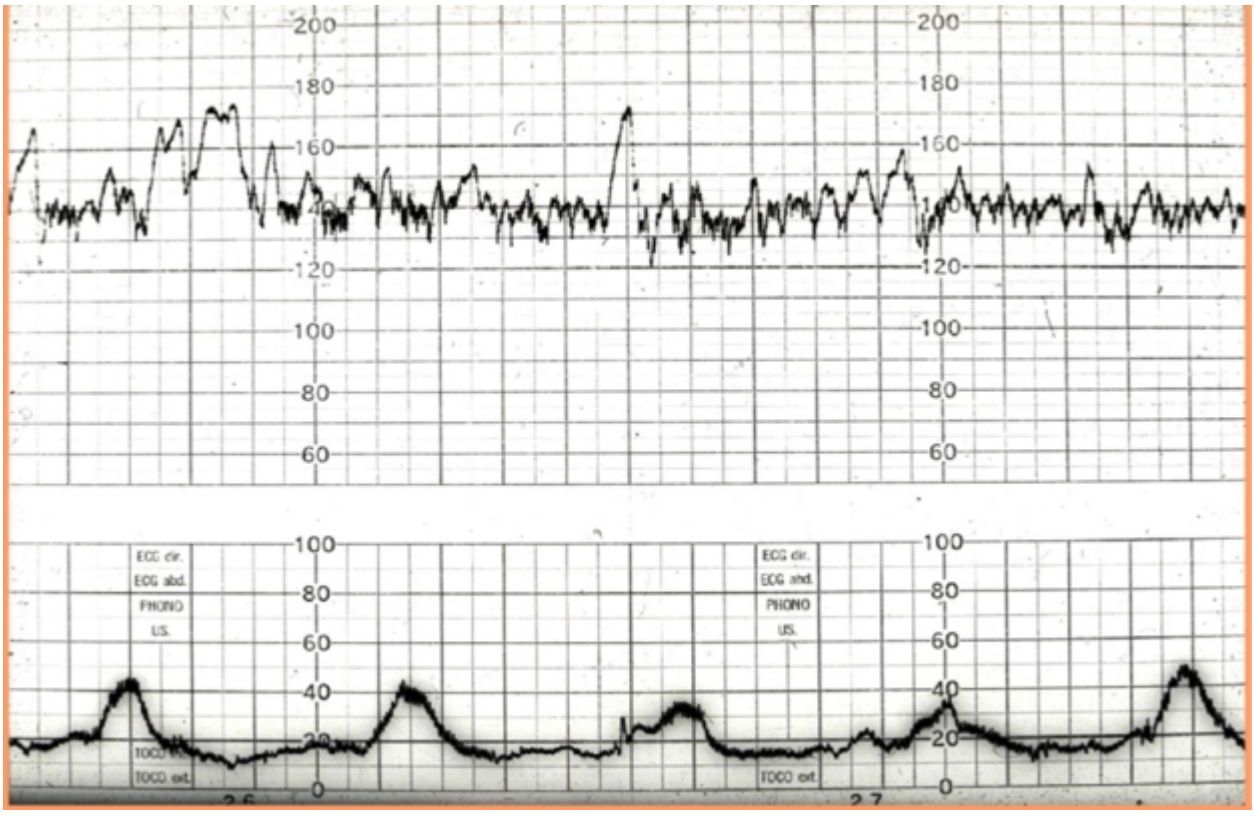


Short-term phenomena – variability

- deviations of the fetal heart action from the basal rate of less than 15 sec
- **Normal variability** – 5-25/min
- **Increased variability** - >25/min for more than 30 minutes
- **Reduced variability** - < 5/min for more than 50 minutes

Reduced variability < 5/min for more than 50 minutes

- **Physiologically:** low gestational age, drug suppression (EDA, opiates, psychotropic drugs...)
  - sleep! (cycles are no longer than 50 minutes)
- **Pathologically:** brain (hypothalamus) perfusion disorder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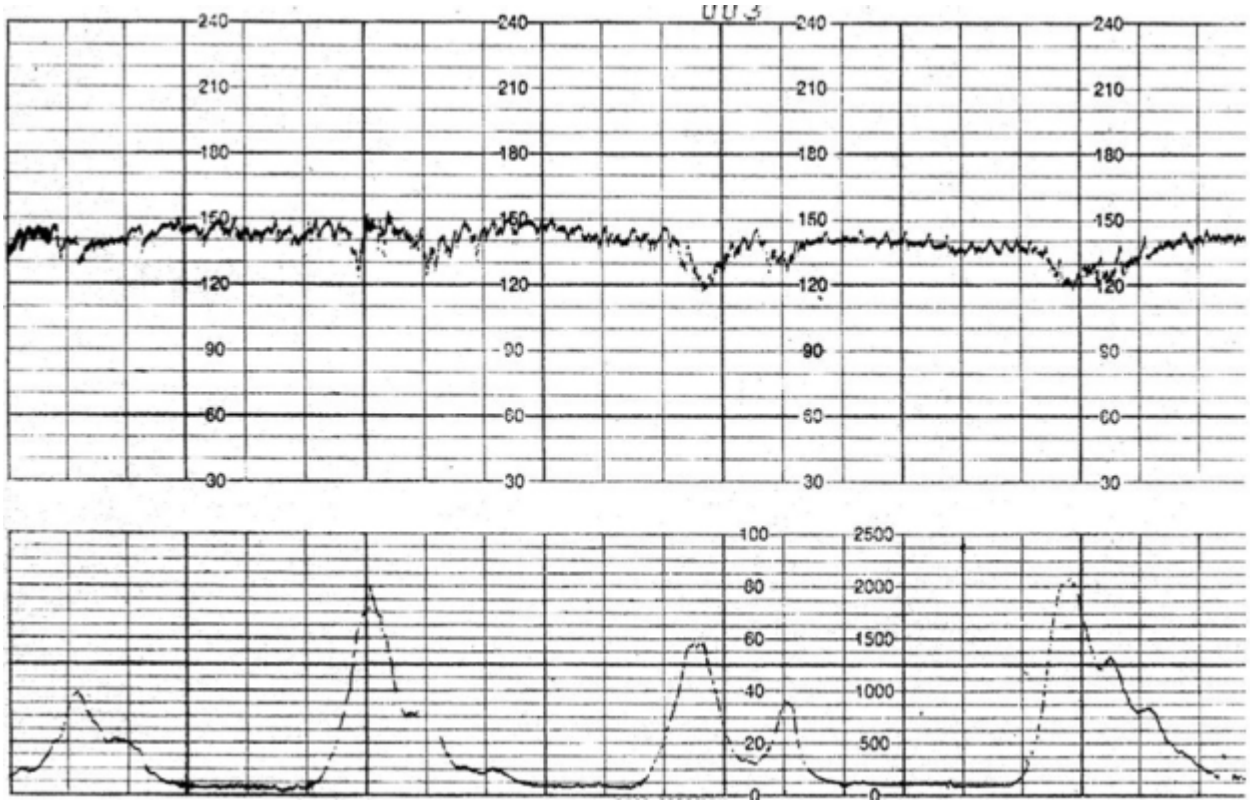
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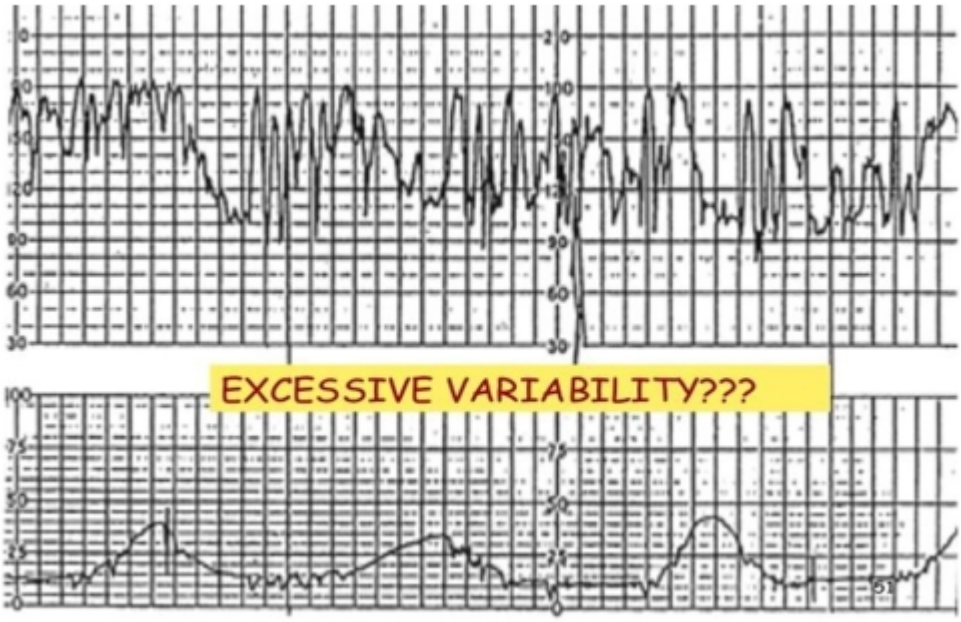


Medium-term phenomena

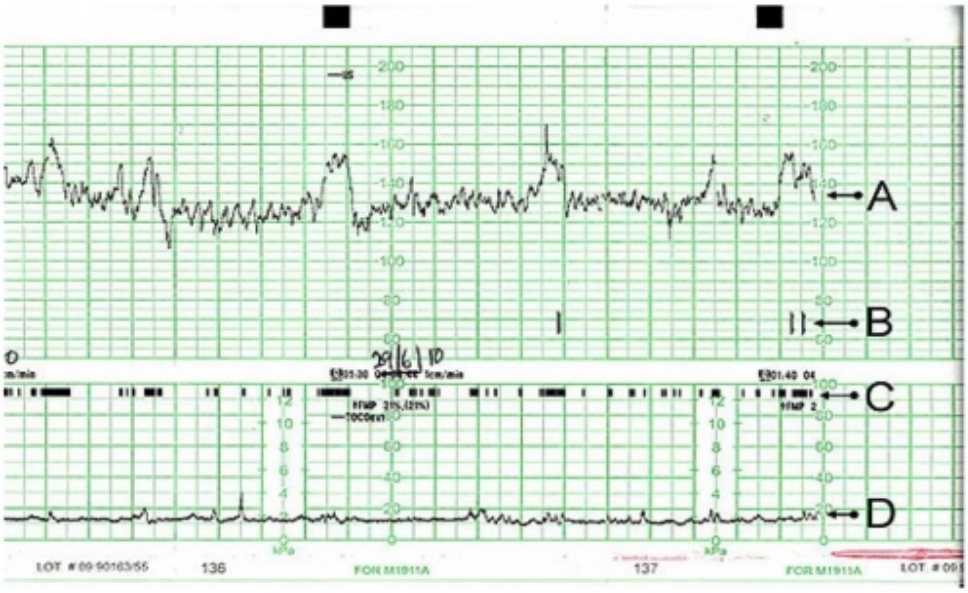
- deviations of the fetal heart action from the basal rate of less than 15 sec
- **Acceleration** - transient increase of frequency from basal frequency by 15/min lasting more than 15 sec
- **Deceleration** - transient decrease in frequency from basal frequency by 15/min lasting longer than 15 sec

In termsofevaluation, the most complicatedaspectof CTG!

Accelerations

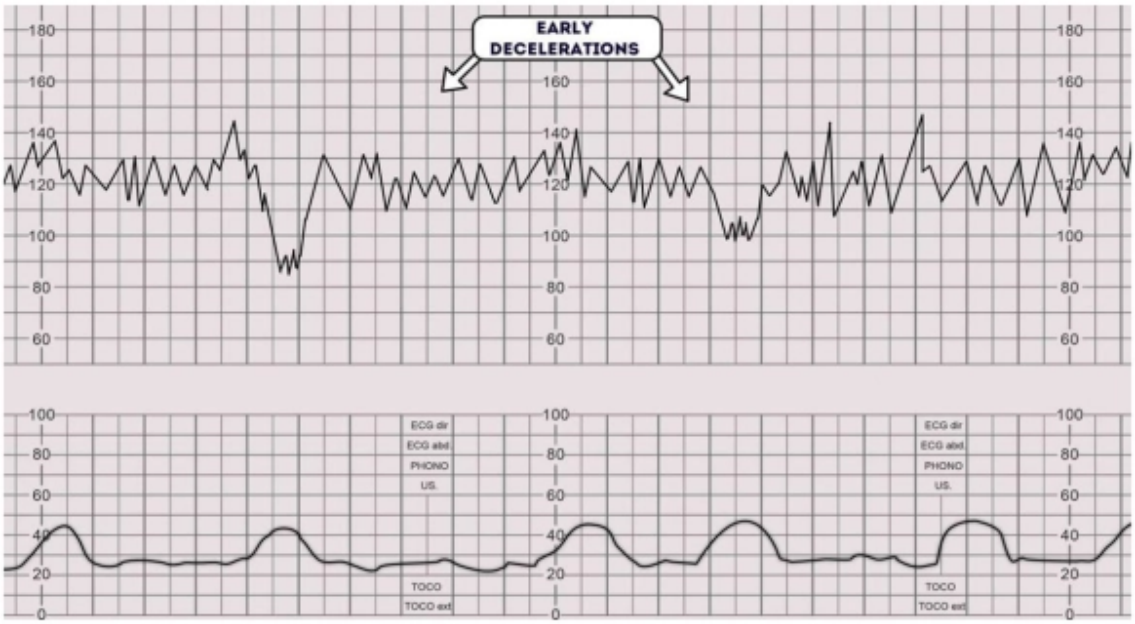
- transient increase of frequency from basal frequency by 15/min lasting more than 15 sec
- for preterm fetuses at least by 10/min each 10 sec
- Sign of neuro- a cardio-compensated fetus

- Their absence does not have to be pathological (with intrapartum recording)



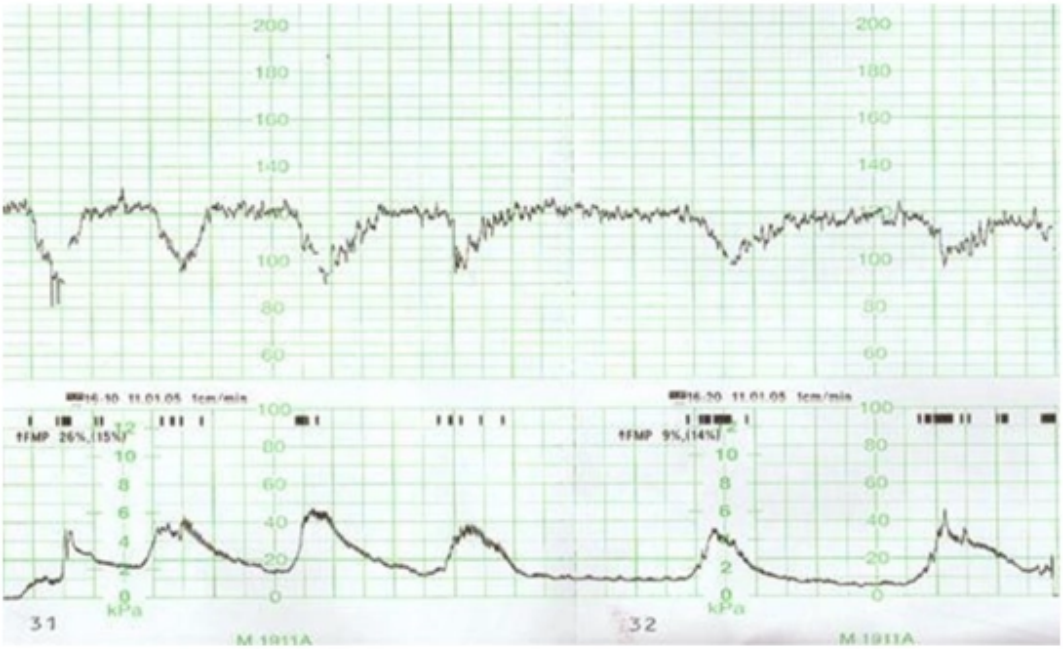
Decelerations

- Transient decrease in frequency from basal frequency by 15/min lasting longer than 15 sec
- Sign of fetal neuro- a cardio-reaction to a particular insult
- Their presence does not have to be pathological (with intrapartum recording)
- There are several types



Early decelerations

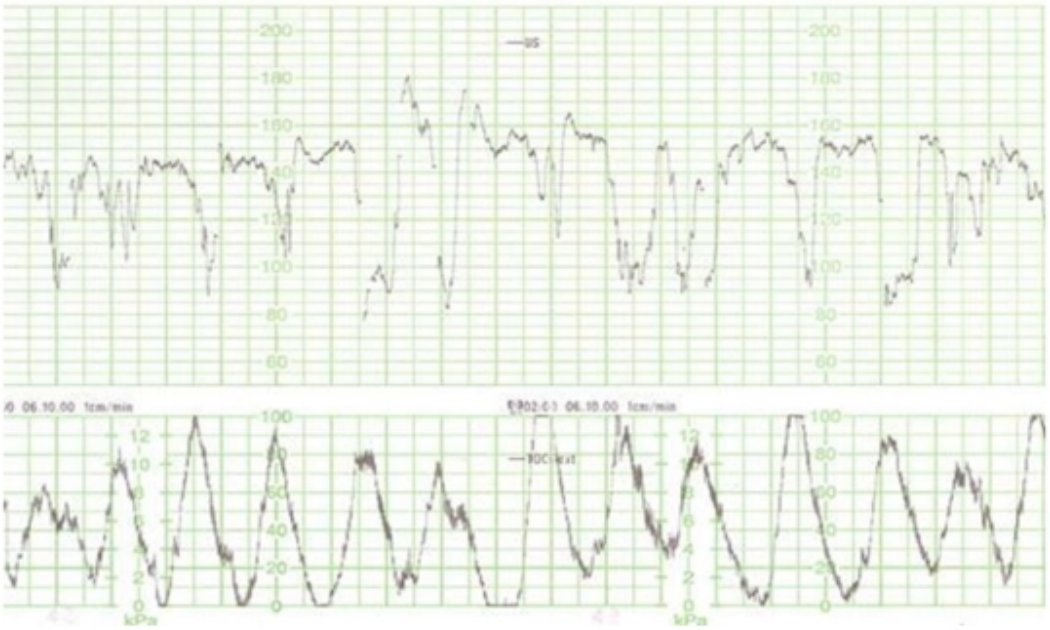
- Short-term, mostly shallow, mostly with normal variability
- Coinciding with contractions
- Caused by compression of the head (baroreceptors on the dura mater -> parasympathetic –> slowing of the heart rate)
- They do not indicate fetal acidosis/hypoxia



Decelerations variable

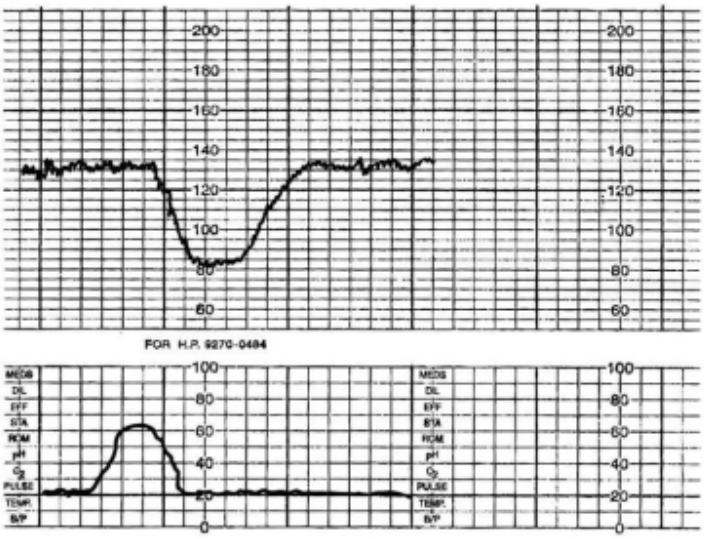
- Short-term rapid decline and rapid return, variability preserved, different shape and size
- Caused by compression of the umbilical cord and subsequent response of baroreceptors in the arch of the aorta (parasympathetic)
- **THE MOST COMMON TYPE of decelerations!!!**
- Variable dependence on contractions
- Typically “shouldering“

- In most cases, they do not lead to fetal acidosis/hypoxia



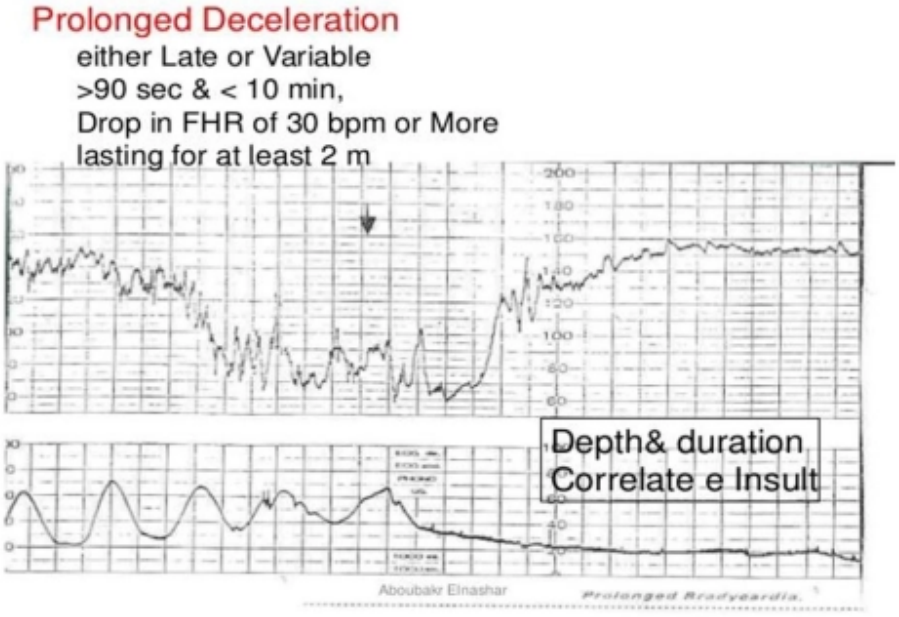
Late decelerations

- gradual decline and return (at least 30 sec), variability mostly reduced, different shape and size
- They start more than 20 sec after the start of the contraction
- Caused by a disorder of the uteroplacental circulation -> insufficient oxygenation of the blood occurs -> chemoreceptors in the aorta and carotids -> decrease of frequency -> return only after the receptor is “washed“ with oxygenated blood
- The most serious, but not necessarily pathological! (depends on frequency/variability/depth)



Prolonged decelerations

- Decelerations lasting more than 5 minutes with FHR less than 80/min
- Often associated with acute fetal hypoxia -> **require a solution!!!**

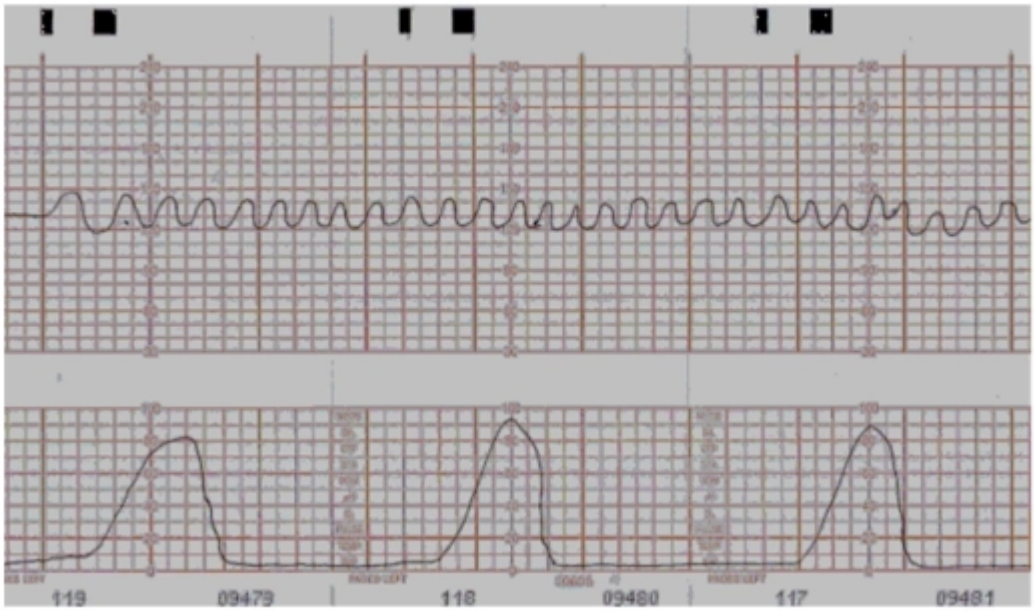


Sinusoid

- Regular smooth wavy signal resembling a sinusoid
- Amplitude 5-15/min
- 3-5 cycles per minute
- Recording lasting more than 30 min (up to 30 min pseudo-sinusoid)

Cause: fetomaternal haemorrhage, anemia...

Pathologicalrecord!!!



Decelerations

- Fetal response (physiological, not pathological) to hypoxic insult
- Cannot increase 02 supply, so has to economize – slowing of FHR
- Cannot remove metabolites, so has to produce less of them – slowing of FHR
- If the decelerations are short (below 60 sec) and with preserved variability (good hypothalamic function), then the fetus is not at risk

Absence of movements/accelerations

- The fetus saves O2, minimizes energy requirements
- So does not move
- Therefore, there are no accelerations
- The physiological reaction of the compensated fetus, does not matter in itself

Loss of variability – CAVE!

- Especially if variability disappears in connection with the above-mentioned phenomena (disappearance of movements/accelerations, decelerations, elevation of FHR)
- **-> decompensation!!!**
- **Brain perfusionmismatch, fetus isat risk**
- **Interventionneeded**

CTG assessment

1. **Basal frequency**
2. **Accelerations**
3. **Decelerations**
4. **Variability**
5. **Physiol. / Susp. / Pathol.**

Normal CTG - antepartum

- normal B-FHR (110-150)
- No decelerations
- At least 2 accelerations present (in 20-30 min)
- Normal variability (10-25/min)

➢**neuro- and cardio-compensated fetus without risk of acidosis/hypoxia**

Evaluation of the intrapartum cardiotocogram according to FIGO 1986			
	Physiological	**Suspicious**	**Pathological**
Basal frequency (bpm)	110-150	150-170 100-110	>170 <100
Amplitude of variability (bpm)	10-25	5-10 for ≥ 40 min >25	<5 for ≥ 40 min Sinusoid for ≥ 20 min
Decelerations (bpm)	Early decelerations (in the late I. stage of labour, which do not have amplitude greater than 50 bpm	• Drop in variable decelerations by not less than 60 bpm lasting less than 60 sec • Transient short-term bradycardia (below 100 bpm lasting 3 min, below 80 bpm lasting 2 min)	• Serious repetitive early decelerations (of amplitude more than 50 bpm) • Serious variable decelerations, late decelerations
Acceleration	≥ 2 during 20 min	absent for ≥ 40 min	

Normal CTG - intrapartum

- normal B-FHR (110-160) corresponding to grav.hebd. (pregnancy week)
- no severe deceleration (>60 sec), pause between decelerations at least 60 sec
- periods of reduced and increased variability (and accelerations + movements) –> cycling

➢neuro- and cardio-compensated fetus without risk of acidosis/hypoxia

Cardiotocography: classification criteria, interpretation and recommended management*			
	Normal	**Suspicious**	**Pathological**
Baseline	110-160 bpm	At least one characteristic of normality is missing but without pathological signs	<100 bpm
Variability	5-25 bpm		Reduced variability >50 min, increased variability >30 min, Sinusoid > 30 min
Decelerations	No repetitive** decelerations		Repetitive late or prolonged decelerations for period >30 min or 20 min during reduced variability or one prolonged deceleration >5 min
Interpretation	Fetus without hypoxia/acidosis	Fetus with low probability of hypoxia / acidosis	Fetus with high probability of hypoxia/acidosis
Clinical management	No intervention needed for improving of fetus oxygenation	Measures to correct avoidable causes, if identified, careful monitoring or using other methods of evaluation of the fetus's oxygenation	Immediate measures to correct avoidable causes, using of other methods of evaluation of the fetus's oxygenation or, if not possible, quick childbirth In acute situations (umbilical cord prolapse, uterine rupture or placental abruption) delivery should be immediate
* Presence of accelerations means that the fetus does not have hypoxia/acidosis but the importance of their absence during labour is uncertain. ** Decelerations are by their nature repetitive, providing that they appear in more than 50% of contractions.			

	Normal CTG[a]	**Suspicious CTG**	**Pathological CTG**
Baseline[b]	110-160 bpm	Lacking at least one of normal characteristics, but with no pathological features	<100 bpm
Variability[c,d,j]	5-25 bpm		Reduced/increased variability[c,d]; sinusoidal pattern[j]
Decelerations[e,f,g,h,i]	No repetitive* decelerations		Repetitive* late or prolonged decelerations for >30 min (or >20 min if reduced variability); one deceleration >5 min
Interpretation	No hypoxia/acidosis	Low probability of hypoxia/acidosis	High probability of hypoxia/acidosis