Cardiotocography (CTG)

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Cardiotocography (CTG) monitors fetal well-being, by continuously recording the fetal heart rate (FHR) as well as maternal uterine contractions. The FHR is recorded by using either an ultrasound transducer or a fetal scalp electrode whereas uterine contractions are measured by a pressure transducer placed on the mother's abdomen. It is imperative that obstetric anesthetists have a good grasp on the fundamentals of CTG. This will enable better communication with their obstetric colleagues, who often base the decisions for urgent delivery of the fetus on CTG changes.

Indications: Although it is common practice to use continuous CTG monitoring for all laboring women across many centers around the world, the 2017 NICE (National Institute for Health and Care Excellence) guidelines [1] do not endorse its use in mothers who are otherwise at low risk of complications. It is recommended in the following high-risk intrapartum conditions (this list is not exhaustive and may also include preexisting antepartum indications):

- suspected chorioamnionitis, sepsis, or temperature ≥ 38 °C
- severe hypertension ($\geq 160/110$ mmHg)
- oxytocin use
- significant meconium
- fresh vaginal bleeding.

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R. Fernando et al. (eds.), *Quick Hits in Obstetric Anesthesia*,
https://doi.org/10.1007/978-3-030-72487-0_28

Interpretation:

CTG recording (Fig. 28.1) conventionally consists of a tracing of FHR (top of the graph) and a second tracing of uterine activity (bottom of the graph) plotted on a paper that is moving at a rate of 1 cm/min (3 cm/min in North America). Each large square is equal to one minute. The average FHR and number of uterine contractions are observed over a 10-min window averaged over 30 min. The interpretation of an antepartum CTG includes evaluation of the following [2]:

- timing and quality of the recording
- baseline FHR
- baseline FHR variability
- accelerations and decelerations
- uterine activity.

Baseline FHR: Normal FHR baseline: 110–160 beats/min.

Tachycardia: FHR baseline >160 beats/min. The most important cause to consider with fetal tachycardia is fetal hypoxia. Fetal tachycardia may also be observed with maternal fever or stress, excessive fetal movement, prematurity and uterine stimulation.

Bradycardia: FHR baseline <110 beats/min. Fetal bradycardia is commonly attributable to umbilical cord compression, fetal head compression and prolonged fetal hypoxia. It may also be associated with fetal post-maturity.

Baseline variability: FHR normally undergoes beat-to-beat fluctuations in amplitude and frequency, which is referred to as baseline variability. It is due to activation of the sympathetic and parasympathetic nervous systems. Variability can be classified as follows:

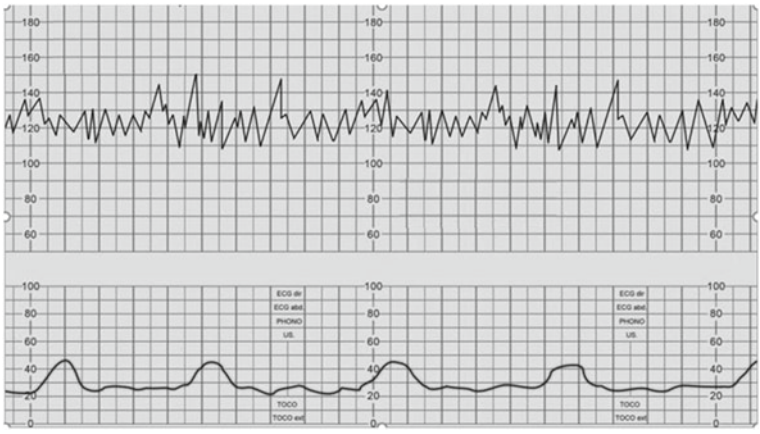

Fig. 28.1 Normal CTG. Figure reproduced from: Antepartum and intrapartum fetal evaluation. Jacquemyn Y, Kwee A. In Oxford Textbook of Obstetric Anaesthesia. Eds. Clark V, Van de Velde M, Fernando R. Oxford University Press 2016. Published with permission from Oxford University Press through PLSclear

- normal: 6–25 beats/min
- increased: more than 25 beats/min
- decreased: 3–5 beats/min
- absent: less than 3 beats/min.

Although the presence of normal baseline variability reflects adequate oxygenation of the central nervous system (CNS) and dependably predicts the absence of detrimental degrees of hypoxia-induced metabolic acidemia at the time it is observed [3], minimal or absent variability alone is a poor predictor of hypoxia. Other potential causes of reduced variability may include fetal sleep states, extreme prematurity, preexisting fetal neurologic anomalies and decreased CNS activity due to opioids or magnesium sulfate.

Acceleration: A clear and abrupt increase (>15 beats/min) in the FHR lasting for at least 15 s, is frequently associated with fetal movement, possibly mediated by stimulation of peripheral proprioceptors. The presence of FHR accelerations indicates the absence of clinically significant fetal hypoxia and fetal metabolic acidemia at the time they are observed [3], but an absence of accelerations may not necessarily be detrimental. Causes of absent FHR accelerations include fetal sleep cycles, arrhythmia, extreme prematurity, congenital anomalies, fetal anemia, and preexisting neurologic injury.

Decelerations: A transient reduction in FHR by at least 15 beats/min from baseline for at least 15 s. Decelerations are subdivided into early, late and variable decelerations based on their relationship to uterine contractions.

- In **Early** decelerations (Fig. 28.2), FHR starts to decrease gradually with the onset of contraction, nadirs at the peak of the contractions and resolves quickly

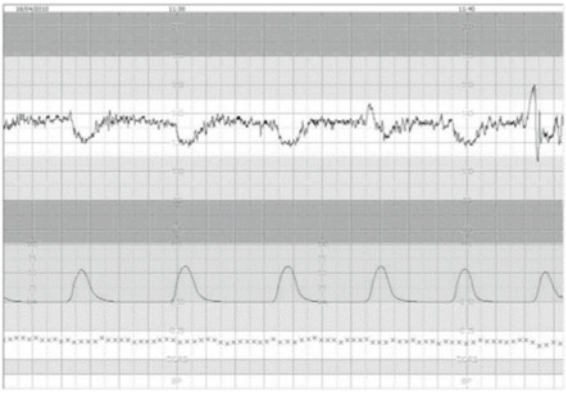

Fig. 28.2 Early Deceleration starts and nadirs at onset and peak of uterine contraction. Figure reproduced from: Antepartum and intrapartum fetal evaluation. Jacquemyn Y, Kwee A. In Oxford Textbook of Obstetric Anaesthesia. Eds. Clark V, Van de Velde M, Fernando R. Oxford University Press 2016. Published with permission from Oxford University Press through PLSclear

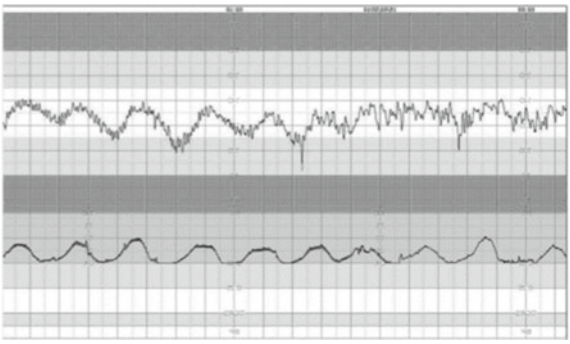

Fig. 28.3 Late Deceleration starts and nadirs after onset and peak of uterine contraction. Figure reproduced from: Antepartum and intrapartum fetal evaluation. Jacquemyn Y, Kwee A. In Oxford Textbook of Obstetric Anaesthesia. Eds. Clark V, Van de Velde M, Fernando R. Oxford University Press 2016. Published with permission from Oxford University Press through PLSclear

as the contraction subsides. They are clinically benign, occurring due to an autonomic response to changes in intracranial pressure and/or cerebral blood flow caused by fetal head compression during a uterine contraction and maternal expulsive efforts.

- In **Late** decelerations (Fig. 28.3), FHR starts to decrease gradually after the onset of contraction, nadirs after the peak of the contractions and resolves after the end of the contraction. These decelerations occur due to a chemoreceptor-mediated response to fetal hypoxemia. Late decelerations require prompt attention as they may indicate fetal acidosis.
- **Variable** decelerations (Fig. 28.4), These manifest a rapid drop, with onset to nadir less than 30 s, good variability within the deceleration, rapid recovery to

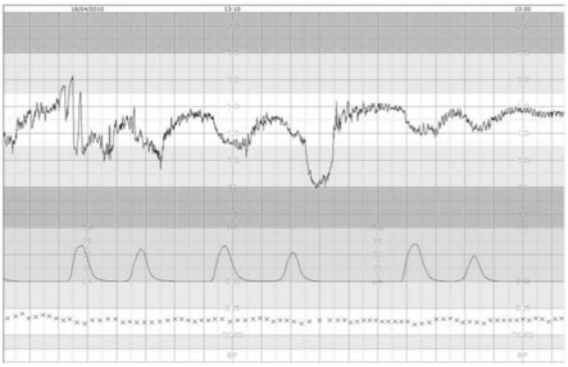

Fig. 28.4 Variable deceleration has no relation to uterine contraction. Figure reproduced from: Antepartum and intrapartum fetal evaluation. Jacquemyn Y, Kwee A. In Oxford Textbook of Obstetric Anaesthesia. Eds. Clark V, Van de Velde M, Fernando R. Oxford University Press 2016. Published with permission from Oxford University Press through PLSclear

the baseline and are varying in size, shape, and relationship to uterine contractions. Variable decelerations, with no concerning features are very common, can be a normal feature in an uncomplicated labour, and usually reflect the fetal autonomic reflex response to umbilical cord compression.

Categories:

Evidence suggests that the use of a standardized approach using three- tier FHR patterns proposed by the UK National Institute for Health and Care Excellence (NICE) [1] coupled with therapeutic interventions may improve neonatal outcomes [4]. It is important to recognize that FHR tracing patterns provide information only about the current acid–base status of the fetus. FHR patterns may move back and forth between the categories depending on the current clinical status and management strategies used. Consideration should also be given to patient-specific factors in the interpretation and management of FHR patterns (Tables 28.1 and 28.2).

Adjuncts to CTG:

Fetal scalp blood sampling (FSBS): This procedure assesses the presence of fetal acidemia by analyzing scalp pH and lactate levels in fetal capillary blood. Although the NICE guideline [1] recommends its use, recent Cochrane systematic reviews have shown that FSBS did not reduce caesarean delivery or operative vaginal births and did not influence any neonatal outcomes [6, 7].

ST Analysis (STAN) monitor: This monitors fetal electrocardiogram (ECG) obtained via a spiral electrode attached to the fetal scalp. Its use is based on the principle that fetal hypoxemia can result in elevation or depression of the ST segment. Although there is no evidence to suggest that it improves neonatal outcome, its use has shown to reduce the rates of fetal blood sampling, operative vaginal delivery and metabolic academia [8].

Fetal Pulse Oximetry: This is a non-invasive method of measuring fetal oxygen saturation using a sensor/catheter placed on the fetal scalp or cheek. Its use is based on the principle that in the presence of an abnormal FHR pattern, a fetal $SpO_2 < 30\%$ for greater than 10 min may increase the risk of fetal acidemia. However, evidence lacks the support of its clinical usefulness [9].

Table 28.1 Interpretation and management of FHR patterns. Adapted from NICE Guideline CG190. Intrapartum Care for healthy women and babies. 2017 [1]

	Reassuring	Non-Reassuring	Abnormal
Baseline FHR (beats/min)	110–160	100–109[a] 161–180	<100 >180
FHR variability	5–25	<5 for 30–50 min or >25 for 15–25 min	<5 for >50 min or >25 for >25 min or Sinusoidal pattern
Decelerations	None Early Variable without concerning[b] features for <90 min	Early Variable without concerning[b] features >90 min *OR* Variable with concerning[b] features in up to 50% of contractions for ≥ 30 min *OR* Variable with concerning[b] features in >50% of contractions for <30 min *OR* Late decelerations in >50% of contractions for <30 min, with no maternal or fetal clinical risk factors such as vaginal bleeding or significant meconium	Variable with concerning features[b] in >50% of contractions for 30 min (or less if any maternal or fetal clinical risk factors) OR Late decelerations >30 min (or less if any maternal or fetal clinical risk factors) OR Single prolonged >3 min
Management	Continue monitoring. No specific action needed	Correct any underlying causes More frequent or additional monitoring may be needed	Start aggressive intrauterine resuscitation[c]; if there is no improvement, caesarean delivery is indicated

[a]Baseline FHR 100–109 beats/min indicates a non-reassuring feature but continue usual care if there is normal baseline variability and no variable or late decelerations

[b]Concerning features: (1) lasting >60 s, (2) reduced baseline variability within the deceleration, (3) failure to return to baseline, (4) biphasic (W) shape, (5) No shouldering (brief increase in fetal heart rate from baseline immediately before and after a deceleration)

[c]Intrauterine resuscitation [1, 5]: (1) If uterine hyperstimulation-reduce/stop oxytocin infusion and/or use a tocolytic e.g. terbutaline 0.25 mg s/c, (2) Left lateral position, (3) IV fluids/vasopressors if hypotensive

Key points

- Basic understanding of the CTG may aid communication and appropriate management when the fetus is considered at high risk.
- Continuous CTG monitoring is strongly recommended in high-risk pregnancies.
- Categorization of the CTG using three-tier FHR patterns coupled with therapeutic interventions may improve neonatal outcomes.
- Patient specific factors must be considered during management of abnormal FHR pattern.

Table 28.2 Adapted from the ACOG Practice Bulletin Number 106 [2]

	Category I	Category II	Category III
Baseline FHR (beats/min)	110–160	<110 >160	<100
FHR variability	6–25	Minimal (<5 beats/min) Marked (>25 beats/min) Absent with no recurrent decelerations	Absent Sinusoidal pattern
Accelerations	Present or absent	Absence of induced accelerations after fetal stimulation	Sinusoidal pattern
Decelerations	None Early may be present No variable or late	Recurrent variable with minimal or moderate variability Prolonged deceleration >2 but <10 min Recurrent late decelerations with moderate baseline variability	Recurrent late decelerations Recurrent variable decelerations

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