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Intrapartum Fetal Heart Rate Monitoring

Early in gestation the fetal heart rate is predominately under the control of the sympathetic nervous system and arterial chemoreceptors [1]. As the fetus develops its heart rate decreases in response to parasympathetic (vagal stimulation) nervous system maturation and variability becomes more pronounced [2]. Standardized guidelines for the interpretation of the fetal heart rate have been suggested by the National Institute of Child Health and Human Development [3]and are adopted in the following discussion.

The interpretation of the fetal heart rate tracing should follow a systematic approach with a full qualitative and quantitative description of the following:

- Baseline rate
- Baseline fetal heart rate (FHR) variability
- Presence of accelerations
- Periodic or episodic decelerations
- Changes or trends of FHR patterns over time
- Frequency and intensity of uterine contractions

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Baseline Fetal Heart Rate (FHR):

The baseline FHR is the heart rate during a 10 minute segment rounded to the nearest 5 beat per minute increment excluding periods of marked FHR variability, periodic or episodic changes, and segments of baseline that differ by more than 25 beats per minute.

The minimum baseline duration must be at least 2 minutes. If minimum baseline duration is < 2 minutes then the baseline is indeterminate.

Bradycardia :Mean FHR < 110 BPM

- A rate of 100-119 BPM in the absence of other non reassuring patterns is not usually a sign of compromise[4]
- Etiologies: Heart block (little or no variability), occiput posterior or transverse position, serious fetal compromise.

Tachycardia: Mean FHR>160 BPM

- In the presence of good variability tachycardia is not a sign of fetal distress [4]
- Etiologies: Maternal fever, fetal hypoxia, fetal anemia, amnionitis, fetal tachyarrhythmia (usually > 200 BPM with abrupt onset little to no variability) SVT (200-240 BPM) [5] , fetal heart failure, drugs (beta sympathomimetics, vistaril, phenothiazines) , rebound (transient tachycardia following a deceleration accompanied by decreased variability) [4]

Baseline change: The decrease or increase in heart rate lasts for longer than 10 minutes.

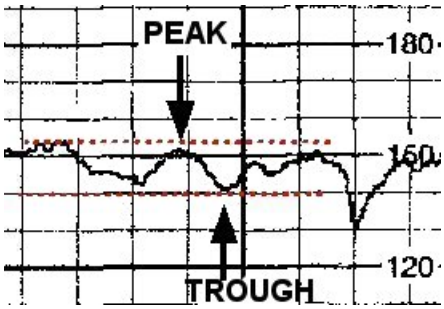
Baseline FHR Variability

Baseline variability is defined as fluctuations in the fetal heart rate of **more than 2 cycles per minute**. **No distinction is made between short-term variability** (or beat-to-beat variability or R-R wave period differences in the electrocardiogram) and long-term variability.

Grades of fluctuation are based on **amplitude range (peak to trough)**:

- Absent variability = Amplitude range undetectable
- Minimal = < 5 BPM
- Moderate = 6 to 25 BPM
- Marked = > 25 BPM

The tracing to the right shows an amplitude range of ~ 10 BPM (moderate variability).



A sinusoidal pattern has regular amplitude and frequency and is excluded in the definition of variability. A **sinusoidal pattern** has a smooth, undulating pattern, lasting at least 10 minutes with a fixed period of three to five cycles per minute and an amplitude of 5-15 bpm. Short-term variability is usually absent. [6]

- **Persistently minimal or absent FHR variability appears to be the most significant intrapartum sign of fetal compromise [32].** On the other hand the presence of good FHR variability may not always be predictive of a good outcome. [33].

  - Etiologies of decreased variability: Fetal metabolic acidosis [7], CNS depressants[8,9], fetal sleep cycles[10], congenital anomalies, prematurity [11,12], fetal tachycardia, preexisting neurologic abnormality [13], normal [14], betamethasone[15].

Accelerations

An acceleration is an abrupt increase in FHR above baseline with onset to peak of the acceleration less than < 30 seconds and less than 2 minutes in duration. The duration of the acceleration is defined as the time from the initial change in heart rate from the baseline to the time of return to the FHR to baseline.

- Adequate accelerations are defined as:
  - <32 weeks' : ≥10 BPMabove baseline for ≥10 seconds [3]
  - >32 weeks' : ≥15 BPM above baseline for ≥ 15 seconds[3].

- **Prolonged acceleration**: Increase in heart rate lasts for 2 to 10 minutes.

The absence of accelerations for more than 80 minutes correlates with increased neonatal morbidity [38,39].

Fetal scalp stimulation can be used to induce accelerations. There is about a 50% chance of acidosis in the fetus who fails to respond to stimulation in the presence of a nonreassuring pattern [17]. This technique should not be used to verify the absence of acidemia during a deceleration of the FHR since there is insufficient literature to support its use during a deceleration.

REACTIVITY[16]*

- An increase of 15 BPM above baseline for 15 second duration (from baseline to baseline) twice in a 20 minute period.
- Since the amplitude of accelerations is inversely proportional to the rate premature fetuses often do not meet criteria for reactivity.
- Only 65% of fetuses at 28 weeks are reactive by this criteria.
- By 34 weeks 95% of fetuses are reactive.

*Reactivity (**a term used in antenatal testing**) is not defined by the NIHCD guidelines.

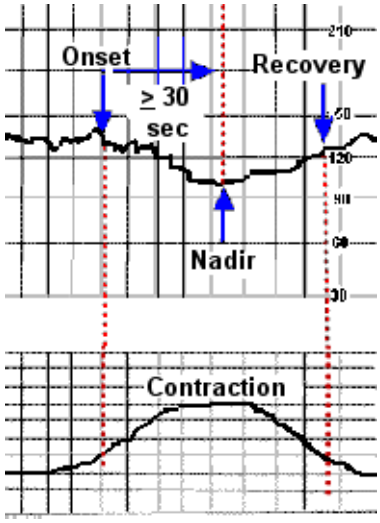
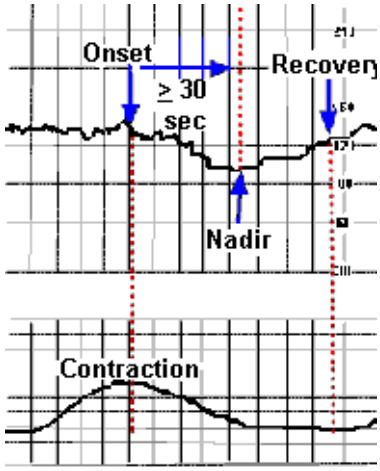
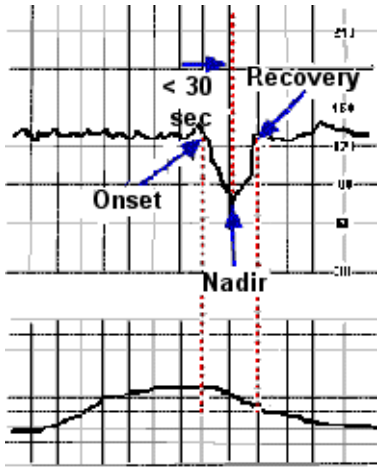
Periodic or episodic decelerations

- **Episodic patterns** are those not associated with uterine contractions .
- **Periodic patterns** are those associated with uterine contractions.
  - Early and late decelerations (with some exceptions-i.e., supine hypotension) are periodic.
  - Variables can also be periodic.

Quantitated by the depth of the nadir in BPM below the baseline.The duration is quantitated in minutes and seconds from the beginning to the end of the deceleration. (Accelerations are quantitated similarly.)

The type of the deceleration is distinguished on the basis of its waveform.

- **Gradual** decrease and return to baseline with time from onset of the deceleration to nadir ≥30 seconds**.**
  - Further subclassified based on their relation to the contraction.
- **Abrupt** decrease in FHR of ≥ 15 beats per minute with onset of deceleration to nadir < 30 seconds.

Early deceleration: Gradual decrease in FHR **with onset of deceleration to nadir ≥30 seconds.** The nadir occurs with the peak of a contraction.	
Late Deceleration: Gradual decrease in FHR **with onset of deceleration to nadir ≥30 seconds. Onset of the decleration occurs after the beginning of the contraction, and the nadir of the contraction occurs after the peak of the contraction.**	
Variable: Abrupt decrease in FHR of ≥ 15 beats per minute measured from the most recently determined baseline rate. **The onset of deceleration to nadir is less than 30 seconds.** The deceleration lasts ≥ 15 seconds and less than 2 minutes. A shoulder, if present, is not included as part of the deceleration.	

- **Recurrent decelerations (variable, early, or late):** Decelerations occur with ≥ 50% of uterine contractions in any 20 minute segment.
- **Prolonged deceleration :** A decrease in FHR of ≥ 15 beats per minute measured from the most recently determined baseline rate. The deceleration lasts >= 2 minutes but less than 10 minutes.

- Etiologies: Maternal hypotension [18] , uterine hyperactivity, cord prolapse, cord compression, abruption, artifact (maternal heart rate) , maternal seizure [19]

Although umbilical cord compression is often responsible for a prolonged deceleration a pelvic examination should be performed to rule out umbilical cord prolapse or rapid descent of the fetal head.[4]

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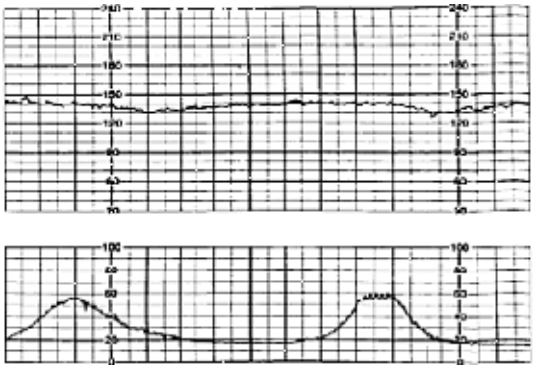
Late Decelerations



Late decelerations associated with preservation of beat-to beat variability

- These decelerations appear to be mediated by arterial chemo receptors in mild hypoxia.
- When the level of oxygen in the fetal blood is below a pO2 of 15-20 mm Hg chemoreceptors are triggered causing reflex alpha adrenergic stimulation which constricts blood vessels in nonvital peripheral areas such as the arms and legs to divert more blood flow to vital organs such as the heart and brain. Constriction of peripheral blood vessels leads to hypertension.
- The hypertension stimulates a baroreceptor mediated vagal response that slows the heart rate. [20] .

Late decelerations associated with no variability (where loss of variability has not been caused by drug administration)

- If the supply of oxygen continues to be limited (hypoxia) , the peripheral tissues cannot completely break down glucose and instead convert it to lactic acid. Significant levels of acid in the blood (acidemia) may suppress the fetal nervous system which becomes evident as decreased variability.
- As acidosis develops the brain stem reflexes become blunted and direct myocardial depression causes shallow decelerations [20,22].
- If myocardial depression is severe enough, lates may be absent all together [22].

Etiologies of Late Decelerations

- Excessive uterine contractions, maternal hypotension, or maternal hypoxemia.
- Reduced placental exchange as in hypertensive disorders, diabetes, IUGR, abruption.

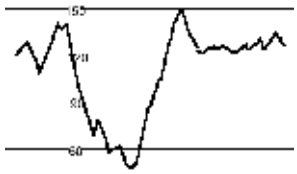
Management of Late Decelerations

These maneuvers are primarily intended to alleviate "reflex" lates.

- Place patient on side [23,24]
- Discontinue oxytocin.
- Correct any hypotension
- IV hydration.
- If decelerations are associated with tachysystole consider terbutaline 0.25 mg SC [26,27]
- Administer O2 by tight face mask [25, 40]
- If late decelerations persist for more than 30 minutes despite the above maneuvers, fetal scalp pH is indicated.
- Scalp pH > 7.25 is reassuring, pH 7.2-7.25 may be repeated in 30 minutes.
- Deliver for pH < 7.2 or minimal baseline variability with late or prolonged decelerations and inability to obtain fetal scalp pH [28,29]

The observation of **recurrent late decelerations** with minimal or absent variability should lead to consideration of expeditious delivery unless the abnormal results are believed to be the result of a reversible maternal condition such as diabetic ketoacidosis or pneumonia with hypoxemia.

Variable Decelerations



- Vagally mediated through chemoreceptors or baroreceptors.
- Accelerations "shoulders" before and after a variable deceleration are thought to be caused by partial cord occlusion .Decreased venous return causes a baroreceptor-mediated acceleration.
- Hypertension and decreased arterial oxygen tension secondary to complete cord occlusion results in deceleration.
- Variables occur with head compression secondary to vagal nerve activation, and with movement in the premature fetus[30]
- The timing of the deceleration may occur periodically either with or after the contraction [31].

Management of Variables

- Change position to where FHR pattern is most improved. Trendelenburg may be helpful.
- Discontinue oxytocin.
- Check for cord prolapse or imminent delivery by vaginal exam.
- Consider amnioinfusion[35-37]
- Administer 100% O2 by tight face mask [4].

Uterine Contractions [41]

Uterine contractions are quantified as the number of contractions present in a 10-minute window, averaged over 30 minutes.

Normal: 5 or less contractions in 10 minutes, averaged over a 30-minute window.

Tachysystole: More than 5 contractions in 10 minutes, averaged over a 30-minute window. Applies to both spontaneous or stimulated labor. Tachysystole should always be qualified as to the presence or absence of associated FHR decelerations.

The terms hyperstimulation and hypercontractility are not defined and should no longer be used.

Three-Tier Fetal Heart Rate Interpretation System [41]

Category I : Normal.

The fetal heart rate tracing shows **ALL** of the following:

Baseline FHR 110-160 BPM, moderate FHR variability, accelerations may be present or absent, no late or variable decelerations, may have early decelerations.

Strongly predictive of normal acid-base status at the time of observation. Routine care.

Category II : Indeterminate.

The fetal heart rate tracing shows **ANY** of the following:

Tachycardia, bradycardia without absent variability, minimal variability, absent variability without recurrent decelerations, marked variability, absence of accelerations after stimulation, recurrent variable decelerations with minimal or moderate variability, prolonged deceleration $\geq$ 2minute but less than 10 minutes, recurrent late decelerations with moderate variability, variable decelerations with other characteristics such as slow return to baseline, and "overshoot".

Not predictive of abnormal fetal acid-base status, but requires continued surveillance and reevaluation.

Category III: Abnormal.

The fetal heart rate tracing shows **EITHER** of the following:

Sinusoidal pattern **OR** absent variability with recurrent late decelerations, recurrent variable decelerations, or bradycardia.

Predictive of abnormal fetal-acid base status at the time of observation. Depending on the clinical situation, efforts to expeditiously resolve the underlying cause of the abnormal fetal heart rate pattern should be made.

SEE ALSO

- The use of electronic fetal monitoring: the use and interpretation of cardiotocography in intrapartum fetal surveillance. Evidence-based clinical guideline number 8. Clinical Effectiveness Support Unit. London (UK): RCOG Press; 2001. Available at: www.rcog.org.uk/resources/public/pdf/efm_guideline_final_2may2001.pdf.
- 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 [published erratum appears in J Obstet Gynaecol Can 2007;29:909]. J Obstet Gynaecol Can 2007;29 suppl:S3–56.

REFERENCES

1. Renou P, Warwick N, Wood C :Autonomic control of fetal heart rate. Am J Obstet Gynecol 105:949,1969 PUBMED

2. Gagnon R, Campbell K, Hunse C, Patrick J Patterns of human fetal heart rate accelerations from 26 weeks to term. Am J Obstet Gynecol. 1987 Sep;157(3):743-8. PUBMED

3.Electronic fetal heart rate monitoring: research guidelines for interpretation. National Institute of Child Health and Human Development Research Planning Workshop. Am J Obstet Gynecol. 1997 Dec;177(6):1385-90. PUBMED

4.American College of Obstetricians and Gynecologists. Fetal Heart Rate Patterns: Monitoring, Interpretation, and Management. ACOG Technical Bulletin 207. Washington, DC: ACOG, 1995

5. Hobel CJ Intrapartum clinical assessment of fetal distress. Am J Obstet Gynecol. 1971 Jun 1;110(3):336-42. PUBMED

6. Modanlou HD, Freeman RK. Sinusoidal fetal heart rate pattern: its definition and clinical significance. Am J Obstet Gynecol 1982;142:1033-1038

7. Paul RH, Suidan AK, Yeh S, Schifrin BS, Hon EH Clinical fetal monitoring. VII. The evaluation and significance of intrapartum baseline FHR variability. Am J Obstet Gynecol. 1975 Sep 15;123(2):206-10. PUBMED

8.Petrie RH, Yeh SY, Murata Y, Paul RH, Hon EH, Barron BA, Johnson RJ The effect of drugs on fetal heart rate variability. Am J Obstet Gynecol. 1978 Feb 1;130(3):294-9. PUBMED

9. Babaknia A, Niebyl JRThe effect of magnesium sulfate on fetal heart rate baseline variability. Obstet Gynecol. 1978 Jan;51(1 Suppl):2s-4s. PUBMED

10. Visser GH, Goodman JD, Levine DH, Dawes GSDiurnal and other cyclic variations in human fetal heart rate near term. Am J Obstet Gynecol. 1982 Mar 1;142(5):535-44. PUBMED

11. Devoe LD Antepartum fetal heart rate testing in preterm pregnancy. Obstet Gynecol. 1982 Oct;60(4):431-6. PUBMED

12.Assali NS, Brinkman CR 3d, Woods JR Jr, Dandavino A, Nuwayhid B Development of neurohumoral control of fetal, neonatal, and adult cardiovascular functions. Am J Obstet Gynecol. 1977 Dec 1;129(7):748-59. PUBMED

13. van der Moer PE, Gerretsen G, Visser GH Fixed fetal heart rate pattern after intrauterine accidental decerebration. Obstet Gynecol. 1985 Jan;65(1):125-7. PUBMED

14. Smith JH, Dawes GS, Redman CW Low human fetal heart rate variation in normal pregnancy. Br J Obstet Gynaecol. 1987 Jul;94(7):656-64. PUBMED

15. Derks JB, Mulder EJ, Visser GH The effects of maternal betamethasone administration on the fetus. Br J Obstet Gynaecol. 1995 Jan;102(1):40-6. PUBMED

16. Evertson LR, Gauthier RJ, Schifrin BS, Paul RH Antepartum fetal heart rate testing. I. Evolution of the nonstress test.Am J Obstet Gynecol. 1979 Jan 1;133(1):29-33. PUBMED

17. Smith CV, Nguyen HN, Phelan JP, Paul RH. Intrapartum assessment of fetal well-being: a comparison of fetal acoustic stimulation with acid-base determinations. Am J Obstet Gynecol 1986;155:726-728 PUBMED

18. Hon EH, Reid BL, Hehre FW: The electronic evaluation of the fetal heart rate II. Changes with maternal hypotension. Am J Obstet Gynecol 79:209, 1960

19.Boehm FH, Growdon JH Jr The effect of eclamptic convulsions on the fetal heart rate. Am J Obstet Gynecol. 1974 Nov 15;120(6):851-2. PUBMED

20. Harris JL, Krueger TR, Parer JT Mechanisms of late decelerations of the fetal heart rate during hypoxia. Am J Obstet Gynecol. 1982 Nov 1;144(5):491-6. PUBMED

21. Murata Y, Martin CB Jr, Ikenoue T, Hashimoto T, Taira S, Sagawa T, Sakata H Fetal heart rate accelerations and late decelerations during the course of intrauterine death in chronically catheterized rhesus monkeys. Am J Obstet Gynecol. 1982

Sep 15;144(2):218-23. PUBMED

22. Gaziano EP, Freeman DW Analysis of heart rate patterns preceding fetal death. Obstet Gynecol. 1977 Nov;50(5):578-82. PMID: PUBMED

23. Abitbol MM. Supine position in labor and associated fetal heart rate changes. Obstet Gynecol 1985;65:481-486

24. Clark SL, Cotton DB, Pivarnik JM, Lee W, Hankins GDV, Benedetti TJ, et al. Position change andcentral hemodynamic profile during normal third-trimester pregnancy and post partum. Am J Obstet Gynecol 1991;164: 883-887

25. Willcourt RJ, King JC, Queenan JT: Maternal oxygenation administration and the fetal transcutaneous PO2. Am J Obstet Gynecol. 1983 Jul 15;146(6):714-5. PUBMED

26. Arias F. Intrauterine resuscitation with terbutaline: a method for the management of acute intrapartum fetal distress. Am J Obstet Gynecol 1978;131:39-43

27. Reece EA, Chervenak FA, Romero R, Hobbins JC. Magnesium sulfate in the management of acute intrapartum fetal distress. Am J Obstet Gynecol 1984;148:104-107

28. In Freeman RK, Garite TJ, Nageotte MP, Fetal Heart Rate Monitoring, 3rd ed. ,Williams & Wilkins , 2003

29. Low JA, Victory R, Derrick EJPredictive value of electronic fetal monitoring for intrapartum fetal asphyxia with metabolic acidosis. Obstet Gynecol. 1999 Feb;93(2):285-91. PUBMED

30. Timor-Tritsch IE, Dierker LJ, Zador I, Hertz RH, Rosen MGFetal movements associated with fetal heart rate accelerations and decelerations. Am J Obstet Gynecol. 1978 Jun 1;131(3):276-80. ; PUBMED

31. Young BK, Katz M, Wilson SJ Fetal blood and tissue pH with variable deceleration patterns. Obstet Gynecol. 1980 Aug;56(2):170-5. PUBMED

32.Williams KP and Galerneau F. Intrapartum fetal heart rate patterns in the prediction of neonatal acidemia. Am J Obstet Gynecol.200;188(3):820-3. PUBMED

33. Samueloff A, Langer O, Berkus M, Field N, Xenakis E, Ridgway L.Is fetal heart rate variability a good predictor of fetal outcome? Acta Obstet Gynecol Scand.1994;73(1):39-44. PUBMED

35. Miyazaki FS, Nevarez F. Saline amnioinfusion for relief of repetitive variable decelerations: a prospective randomized study. Am J Obstet Gynecol 1985;153:301-306

36. Nageotte MP, Freeman RK, Garite TJ, Dorchester W. Prophylactic intrapartum amnioinfusion inpatients with preterm premature rupture of membranes. Am J Obstet Gynecol 1985;153:557-562

37. Strong TH Jr, Hetzler G, Sarno AP, Paul RH. Prophylactic intrapartum amnioinfusion: a randomized clinical trial. Am J Obstet Gynecol

38.Patrick J ,et al. , Accelerations of the human fetal heart rate at 38 to 40 weeks' gestational age.Am J Obstet Gynecol.1984 Jan 1;148(1):35-41 PUBMED

39.Luerti M, et al Accelerations in greater than intra-partum greater than cardiotocographic recording I. Correlation with perinatal outcome. Clin Exp Obstet Gynecol.1980;7(2):94-100. PUBMED

40.Haydon ML, et al. The effect of maternal oxygen administration on fetal pulse oximetry during labor in fetuses with nonreassuring fetal heart rate patterns. Am J Obstet Gynecol September 2006;195:735-8.PUBMED

41. Macones GA et al., The 2008 National Institute of Child Health and Human Development Workshop Report on Electronic Fetal MonitoringUpdate on Definitions, Interpretation, and Research Guidelines Obstetrics & Gynecology 2008;112:661-666 PMID:18757666

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