Università degli Studi di Padova
Dipartimento di Salute della Donna e del Bambino SDB
U.O.C. Clinica Ginecologica Ostetrica
Scuola di Specializzazione di Ginecologia e Ostetricia - A.A. 2015-2016
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DECELERO,

ERGO SUM

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FETAL MONITORING: WHY?

- predict significant intrapartum fetal asphyxia and institute timely intervention
- prevent fetal hypoxic and ischaemic injury
- avoid unnecessary operative delivery of non-acidotic babies

1) increase costs of healthcare without benefits
2) expose mother&baby to iatrogenic harm
3) perpetuate defensive practice

In addiction to hypoxia, there are other causes of fetal damage..

HYPOXAEMIA

- activation of chemoreceptors
- increase O_2 extraction
- reduced FM
- energy balance maintained

HYPOXIA

- surge of stress hormone
- upregulation of β-receptors (gluconeogenesis + anaerobic metabolism)
- redistribution of blood to central organ
- supply are ok, no neonatal effects

ASPHYXIA

- marked output of stress hormones
- glycogenolysis + anaerobic metabolism in central organs
- risk of central organ failure
- rapid collapse

FETUS & OXYGEN: Hb

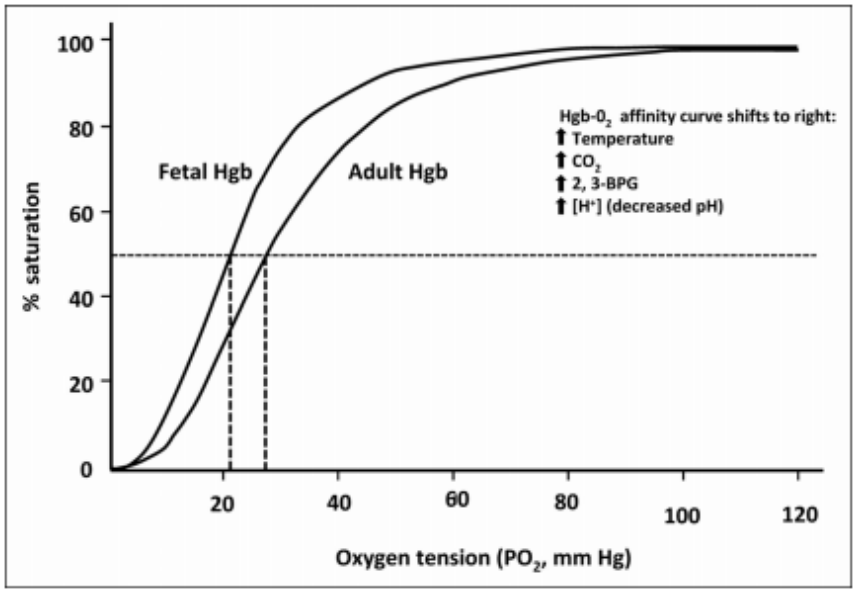
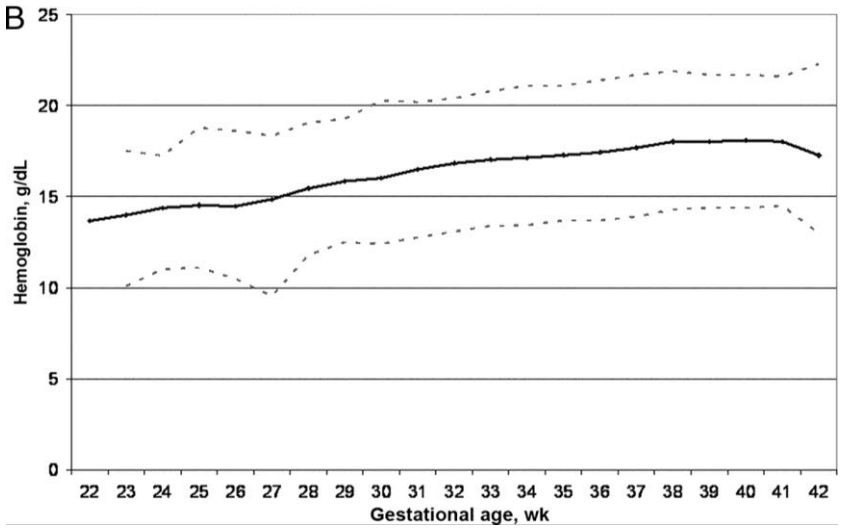


Figure 6. Hemoglobin–oxygen dissociation curve. The curve representing fetal hemoglobin is on the left, and the curve representing adult hemoglobin is on the right. The P_{50} is shown as a hatched line for each curve.



J.Jopling et al.Reference ranges for hematocrit and blood hemoglobin concentration during the neonatal period: data from a multihospital health care system Pediatrics Feb 2009, 123 (2) e333-e337

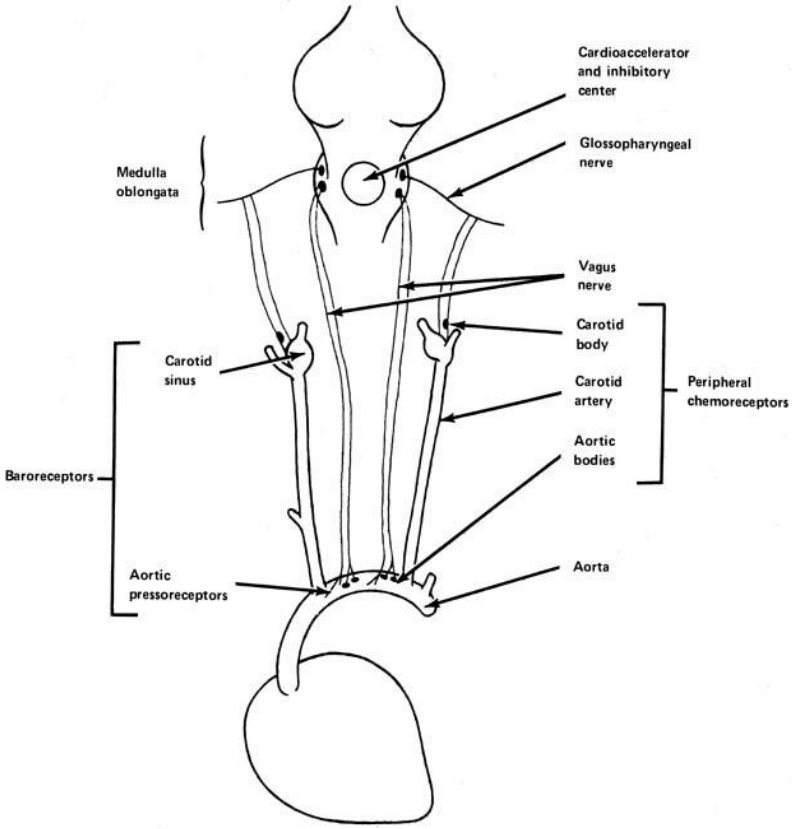
FETUS HEART RATE REGULATION

(..)The carotid sinus **chemoreceptors** are active as demonstrated by direct nerve recordings in fetal sheep(..) the transient bradycardia in fetuses in utero, especially during labor and delivery, is related to the activity of the arterial chemoreceptors in part because of the relatively low fetal blood pressure.

Wood CE, Tong H. Central nervous system regulation of reflex responses to hypotension during fetal life. American Journal of Physiology Dec 1999, 277 (6) R1541-R1552

When blood pressure increases, impulses are sent from the **baroreceptors** by way of the vagus and glossopharyngeal nerve to the midbrain, resulting in feedback impulses to the heart, tending to slow it. This is an extremely rapid response.

Parer J. Glob. libr. women's med.(ISSN: 1756-2228) 2008; DOI 10.3843/GLOWM.10194

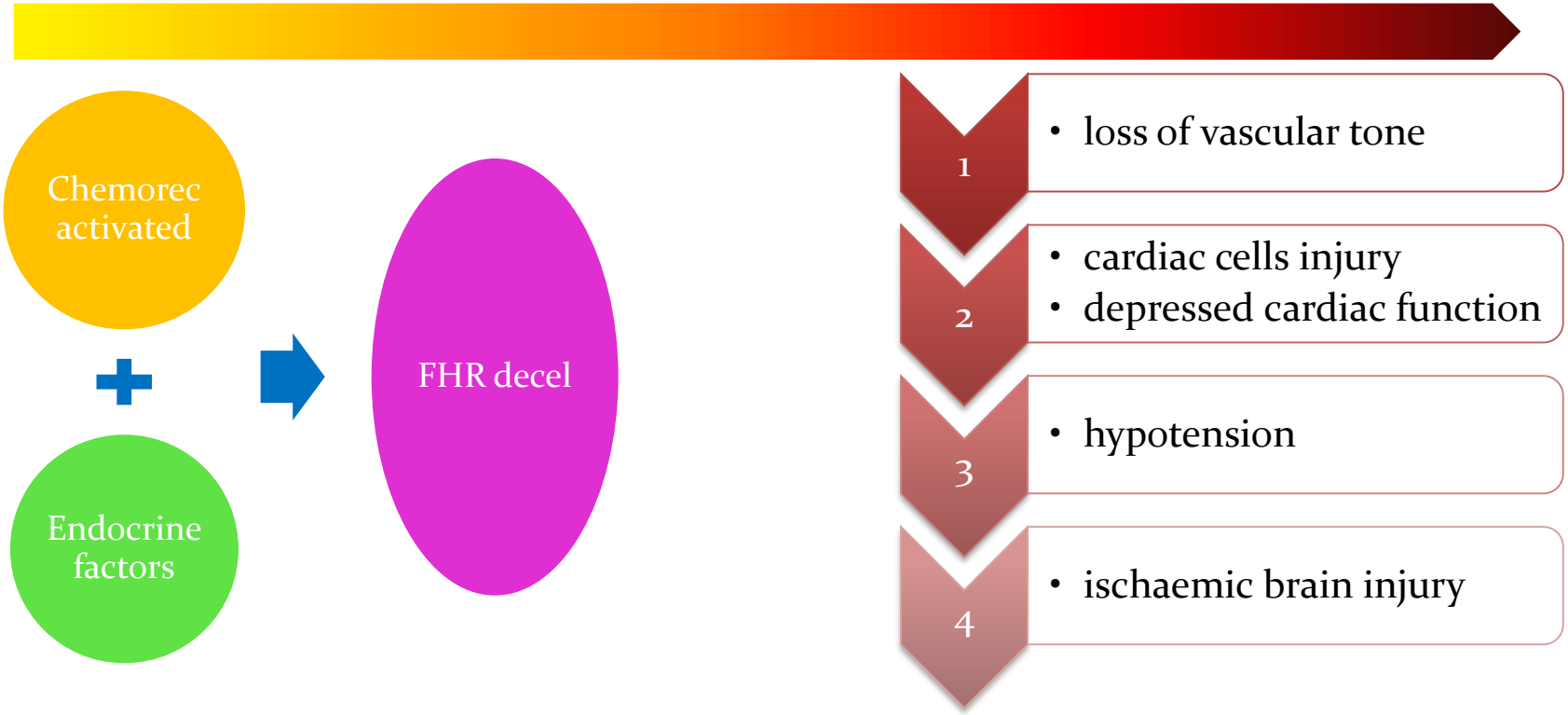


FETUS & OXYGEN & HR REGULATION

Hypoxia starts

Progressively developing asphyxia

Acidemia



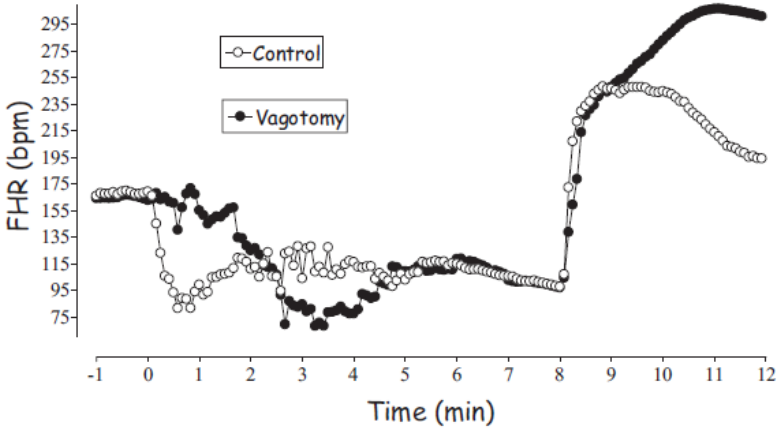
Ugwumadu A. Understanding cardiotocographic patterns associated with intrapartum fetal hypoxia and neurologic injury. Best Practice & Research Clinical Obstetrics and Gynaecology 27 (2013) 509–536

FETAL RESPONSE TO HYPOXIA

" (...) a fetus is **not** exposed to atmospheric oxygen, so it is unable to increase the rate and depth of respiration to oxygenate the myocardium (...) the only way a fetus could immediately protect the energy balance within the myocardium is by rapidly **slowing its own heart rate**."

Fletcher AJ et al. Development of the ovine fetal cardiovascular defense to hypoxemia towards full term. Am J Physiol Heart Circ Physiol 2006; 291: H3023–H3034

FIGURE 1
Examples show the contribution of the parasympathetic system to bradycardia during 8 minutes of severe asphyxia that was induced by complete occlusion of the umbilical cord in near-term sheep fetuses

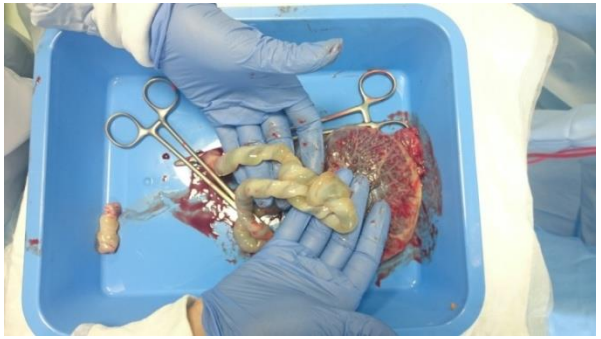


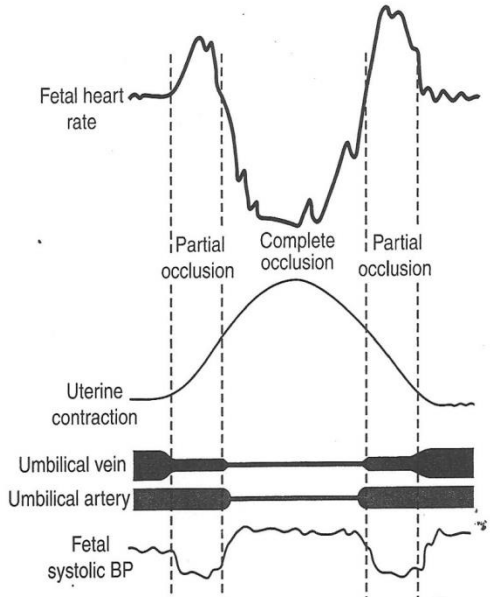
"The initial fetal response to an episode of low oxygen is bradycardia which is a **vagally mediated chemoreceptor response**, seen as decelerations on the CTG. If the degree of hypoxia is severe and prolonged (typically >3 minutes), the initial vagal bradycardia is sustained by myocardial hypoxia."

Westgate JA et al. The intrapartum deceleration in center stage: a physiologic approach to the interpretation of fetal heart rate changes in labor. AJOG 2007, 197, 3, 236.e1–236.e11

HYPOXIA MAIN CAUSE

The most common etiology of fetal hypoxia during labor is **umbelical cord occlusion**, evidenced as a FHR decelerations.





Williams Obstetrics (2010)

"..a **biphasic cardiovascular response:**
-Phase I (1st min of cord compression):
a fast, early bradycardia;
-Phase II (minutes 2–5 of cord compression)
a delayed fetal bradycardia
(secondary cardiovascular response).
Repeated compression of the umbilical cord abolishes the primary, but not the secondary, cardiovascular responses"

Giussani DA et al. Dynamics of cardiovascular responses to repeated partial umbilical cord compression in late-gestation sheep fetus. Am J Physiol Heart Circ 273:H2351-H2360,1997

CTG CLASSIFICATION NICE 2014
pattern recognition

Table 2 Classification of FHR trace features

Feature	Baseline (bpm)	Variability (bpm)	Decelerations	Accelerations
Reassuring	110–160	≥ 5	None	Present
Non-reassuring	100–109 161–180	< 5 for 40–90 min	Typical variable decelerations with over 50% of contractions, for over 90 min Single prolonged deceleration for up to 3 min	The absence of accelerations with otherwise normal trace is of uncertain significance
Abnormal	< 100 > 180 Sinusoidal pattern ≥ 10 min	< 5 for 90 min	Either atypical variable decelerations with over 50% of contractions or late decelerations, both for over 30 min Single prolonged deceleration for more than 3 min	

Table 1 Definition of normal, suspicious and pathological FHR traces

Category	Definition
Normal	All four features are classified as reassuring
Suspicious	One feature classified as non-reassuring and the remaining features classified as reassuring
Pathological	Two or more features classified as non-reassuring or one or more classified as abnormal

CTG CLASSIFICATION STAN 2014
pattern recognition

Classification of CTG

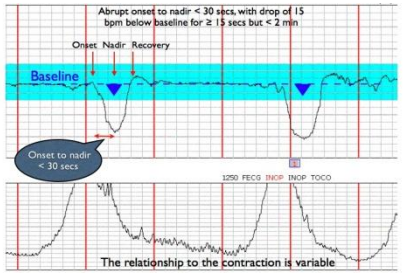
Composed by the Danish and Norwegian reference group according to FIGO guidelines, December 2007

	Baseline heart frequency	Variability Reactivity	Decelerations
Normal CTG	• 110–150 bpm	• Accelerations • 5–25 bpm	• Early uniform decelerations • Uncomplicated variable decelerations (loss of <60 beats)
Intermediary CTG	• 100–110 bpm • 150–170 bpm • Short bradycardia episode <100 bpm for >3 min <80 bpm for >2 min	• >25 bpm (saltatory pattern) • <5 bpm >40 min	• Uncomplicated variable decelerations (loss of >60 beats)
	• A combination of 2 or several intermediary observations will result in an abnormal CTG		
Abnormal CTG	• >170 bpm • Persistent bradycardia <100 bpm for >10 min <80 bmp for >3 min (without an increasing tendency)	• <5 bpm for >60 min • Sinusoidal pattern	• Complicated variable decelerations with a duration of >60 sec • Repeated late uniform decelerations
Preterminal CTG	• Total lack of variability (<2 bpm) and reactivity with or without decelerations or bradycardia		

FHR DECELERATIONS

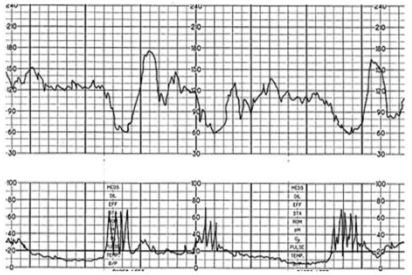
Transient decrease of the FHR of >15 bpm lasting for >15 s.
According to their relation to uterine contractions: early, late and variable.

TYPICAL or UNCOMPLICATED(baroreceptor)
1. initial slight rise in the FHR ('shouldering')
2. a sharp fall from the baseline (<60 bpm)
3. quick recovery with a second 'shouldering'
4. final recovery to the baseline within 60 sec



VARIABLE DECELERATIONS

ATYPICAL or COMPLICATED (baro-/chemo-)
1. neither first nor second 'shouldering'
2. a sharp fall from the baseline (>60 bpm)
3. abnormal shape (U, V, W)
4. delayed recovery (>60 sec)



CONTROVERSIES..

..combination of decelerations may occur as uterine contractions may compress the fetal head and the umbilical cord **at the same time**...

"Specific abnormal findings on electronic monitoring of the FHR were associated with an increased risk of cerebral palsy. However, the **false positive rate** was extremely high. Since C/S is often performed when such abnormalities are noted (...) many cesarean sections would be performed without benefit and with the potential for harm."

Nelson KB et al. Uncertain value of electronic fetal monitoring in predicting cerebral palsy. N Engl J Med 1996;334:613-8

CONTROVERSIES..

J Perinat Med. 2006;34(4):298-302.

Intrapartum cardiotocography -- the dilemma of interpretational variation.

Palomäki O[1], Luukkaala T, Luoto R, Tuimala R.

Curr Opin Obstet Gynecol. 2012 Mar;24(2):84-8. doi: 10.1097/GCO.0b013e3283505b3c.

Human factors affecting the interpretation of fetal heart rate tracings: an update.

Santo S[1], Ayres-de-Campos D.

J Biomed Inform. 2014 Oct;51:72-9. doi: 10.1016/j.jbi.2014.04.010. Epub 2014 Apr 16.

Analysis of obstetricians' decision making on CTG recordings.

Spilka J[1], Chudáček V[2], Janků P[3], Hruban L[3], Burša M[2], Huptych M[2], Zach L[2], Lhotská L[2].

J Matern Fetal Neonatal Med. 2016 Apr 26:1-2. [Epub ahead of print]

Impediments to a unified international approach to the interpretation and management of intrapartum cardiotocographs.

Parer JT[1], Ugwumadu A[2].

Author information

Abstract

There is much inconsiste
consensus include disag
speed, and the necessity

KEYWORDS: Fetal acidem

Obstet Gynecol. 2014 Sep;124(3):507-13. doi: 10.1097/AOG.0000000000000424.

Diagnostic accuracy of fetal heart rate monitoring in the identification of neonatal encephalopathy.

Graham EM[1], Adami RR, McKenney SL, Jennings JM, Burd I, Witter FR.

Author information

Abstract

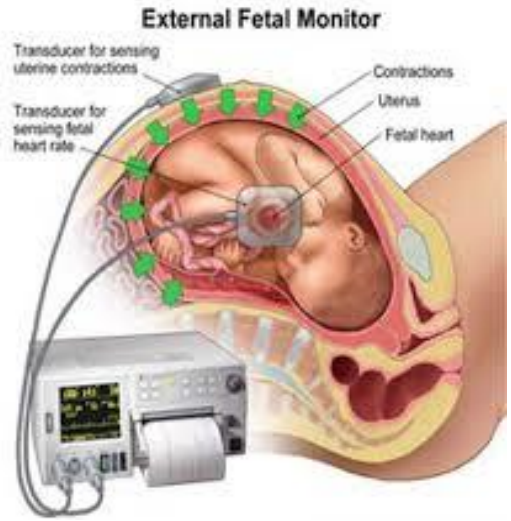
OBJECTIVE: To estimate the diagnostic accuracy of electronic fetal heart rate abnormalities in the identification of neonates with encephalopathy treated with whole-body hypothermia.

METHODS: Between January 1, 2007, and July 1, 2013, there were 39 neonates born at two hospitals within our system treated with whole-body hypothermia within 6 hours of birth. Neurologically normal control neonates were matched to each case by gestational age and mode of delivery in a two-to-one fashion. The last hour of electronic fetal heart rate monitoring before delivery was evaluated by three obstetricians blinded to outcome.

RESULTS: The differences in tracing category were not significantly different (neonates in the case group 10.3% I, 76.9% II, 12.8% III; neonates in the control group 9.0% I, 89.7% II, 1.3% III; P=.18). Bivariate analysis showed neonates in the case group had significantly increased late decelerations, total deceleration area 30 (debt 30) and 60 minutes (debt 60) before delivery and were more likely to be nonreactive. Multivariable logistic regression showed neonates in the case group had a significant decrease in early decelerations (P=.03) and a significant increase in debt 30 (.01) and debt 60 (P=.005). The area under the receiver operating characteristic curve, sensitivity, and specificity were 0.72, 23.1%, and 94.9% for early decelerations; 0.66, 33.3%, and 87.2% for debt 30, and 0.68, 35.9%, and 89.7% for debt 60, respectively.

CONCLUSION: Abnormalities during the last hour of fetal heart rate monitoring before delivery are poorly predictive of neonatal hypoxic-ischemic encephalopathy qualifying for whole-body hypothermia treatment within 6 hours of birth. LEVEL OF EVIEDENCE: II.

CTG CLASSIFICATIONS AGREEMENT



NORMAL CTG ABOVE DIFFERENT CLASSIFICATIONS:

-a baseline heart rate within the normal range (110-160 beats/min)

- moderate fetal heart rate variability (5-25 beats/min)

- absent decelerations

And before admission??

NORMAL CTG = NO HYPOXIA
(CNS /CVS integrity, good reaction to further hypoxic events, low probability of asphyxia)

Parer JT. Handbook of fetal heart rate monitoring. Philadelphia: WB Saunders, 1983

CTG CLASSIFICATION FIGO 2015 pathophysiology pattern

	Normal	Suspicious	Pathological
Baseline	110–160 bpm	Lacking at least one characteristic of normality, but with no pathological features	<100 bpm
Variability	5–25 bpm		Reduced variability for >50 min, increased variability for >30 min, or sinusoidal pattern for >30 min
Decelerations	No repetitive[a] decelerations		Repetitive late or prolonged decelerations during >30 min or 20 min if reduced variability, or one prolonged deceleration with >5 min
Interpretation	Fetus with no hypoxia/acidosis	Fetus with a low probability of having hypoxia/acidosis	Fetus with a high probability of having hypoxia/acidosis
Clinical Management	No intervention necessary to improve fetal oxygenation state	Action to correct reversible causes if identified, close monitoring or additional methods to evaluate fetal oxygenation (Chapter 4)	Immediate action to correct reversible causes, additional methods to evaluate fetal oxygenation (Chapter 4), or if this is not possible expedite delivery. In acute situations (cord prolapse, uterine rupture or placental abruption) immediate delivery should be accomplished.

[a] Repetitive decelerations refers to occurrence of decelerations for at least 50% of uterine contractions.

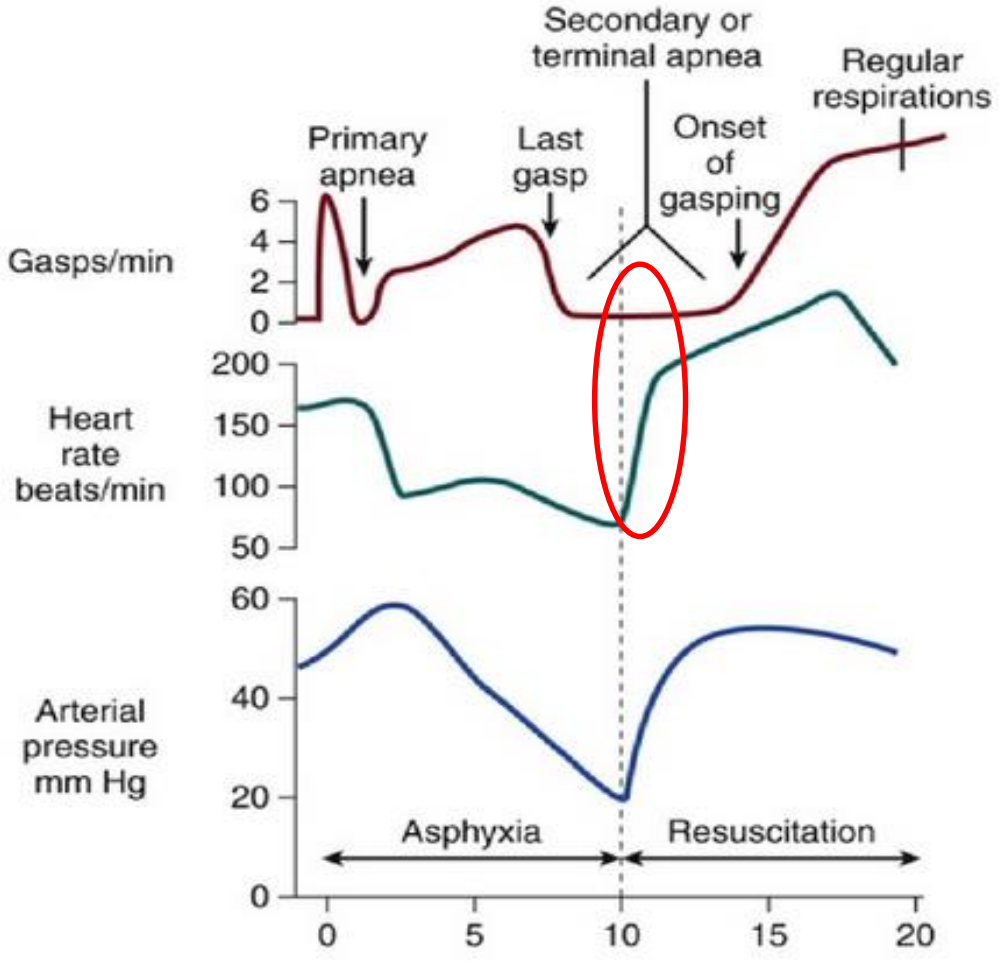
Diogo Ayres-de-Campos et FIGO intrapartum fetal monitoring consensus panel. International Journal of Gynecology and Obstetrics 133 (2016)130

TYPES OF INTRAPARTUM HYPOXIA

ACUTE HYPOXIA	GRADUALLY EVOLVING HYPOXIA	SUBACUTE HYPOXIA
• Prolonged deceleration <80 bpm for > 3 minutes • Sudden baseline drop >60bpm following gradually evolving or subacute hypoxia	• Commences with the onset of decelerations followed by **ABCDE** • **A**ccelerations disappear • **B**aseline heart rate increases • **C**ompensated stress • **D**ecompensation (loss of baseline variability, unstable baseline, 'saltatory' pattern • **E**nd stage (myocardial failure leading to 'step-wise' pattern to death)	• A fetus typically spends < 30 seconds at the baseline and > 90 seconds below the baseline (1:3 ratio) • Recurrent 'atypical' variable or late decelerations with or without 'Overshoots' and 'Saltatory' pattern in between decelerations
• pH drop at the rate of 0.01/min • If variability prior to deceleration and within the first three minutes of deceleration is normal and 3 accidents are absent, 95% recover by 9 minutes	• Rate of fall in pH depends on intensity and duration of hypoxia and fetal reserve to compensate for ongoing hypoxic stress	• pH drops at the rate of 0.01/2-3 min • Usually seen during active maternal pushing or with the use of oxytocin to augment labour

Pinas A, Chandraharan E. Best Practice & Research Clinical Obstetrics and Gynaecology 30 (2016) 33e47

ASPHYXIATED NEWBORNS



Dawes GS, Jacobson HN et al. The treatment of asphyxiated, mature foetal lambs and rhesus monkeys with intravenous glucose and sodium carbonate. J Physiol. 1963 Nov;169:167-84

CONFIRMATION OF HYPOXIA

DEFINITION: impaired respiratory gas exchange accompanied by the development of metabolic acidosis. The pH of umbilical cord blood is determined by presence of *respiratory* and metabolic acids. Carbon dioxide diffuses readily across the placenta. Fixed acids such as **lactic acid** and **b-hydroxybuty-rate**, which account for the majority of the metabolic load, have a relatively slow passage across the placenta.

pH <7.0

BE>- 12 mmol/L

In the clinical context FETAL ASPHYXIA is progressive hypoxaemia and hypercapnia with a significant **metabolic acidaemia**.

pH is not an ideal parameter because it is a logarithmic term

International Cerebral Palsy Task Force. A template for defining a causal relation between acute intrapartum events and cerebral palsy: international consensus statement. BMJ 1999;319:1054–9.

BE and PH IN FETUSES

In cases of excessive contraction such as during uterine hyperstimulation or prolonged second stage, incomplete restitution may result in cumulative acidosis, as placenta flow is not enough to grant the gas exchange.

Armstrong L, Stenson BJ. Use of umbilical cord blood gas analysis in the assessment of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007; 92: F430-F434.

Among uncomplicated pregnancies (18 and 38 weeks), mean BE was – 2.3± 0.6 mmol/L and pH 7.39 ± 0.05.

→ the normal fetus may be expected to enter labor with a BE of approximately **– 2 mmol/L**

→ - normal second stage of labour changes it by **1 mmol/l per h**

- prolonged deceleration period change it by **2 mmol/l per h**

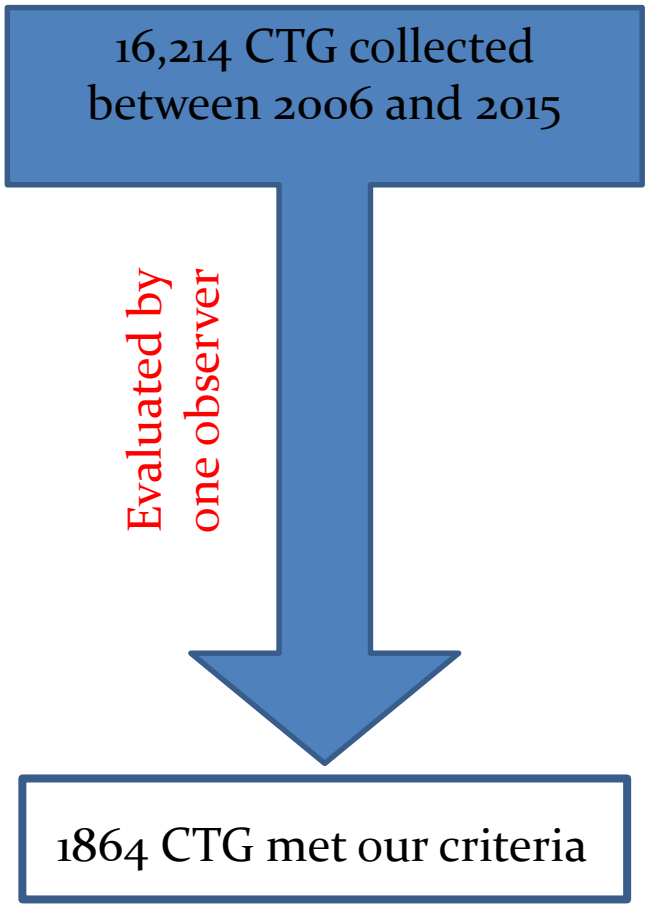
- acute terminal bradycardia change it by **1 mmol/l per 2–3 min**

Lazarevic B et al. Respiratory gases and acid base parameter of the fetus during the second and third trimester. Clin Exp Obstet Gynecol. 1991;18:81–84

Ross MG, Gala R. Use of umbilical artery base excess: algorithm for the timing of hypoxic injury Am J Obstet Gynecol 2002; 187:1–9

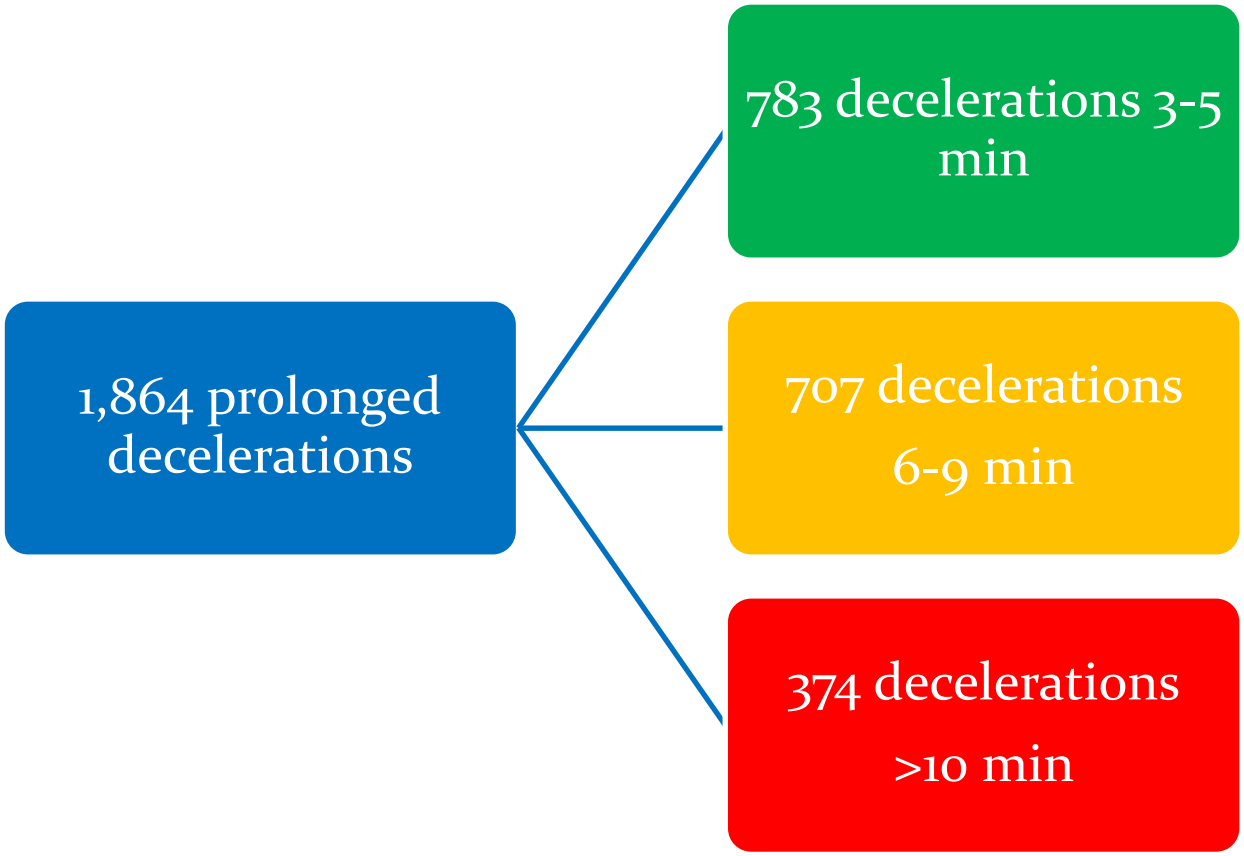
DATABASE

St George's Maternity Service delivers almost 5,000 patients/year, but less than one third are available on STANviewer.



- singleton pregnancy
- >37 weeks
- patient ID available
- good quality of registration
- at least 40 min of CTG before deceleration

DATABASE


1,864 prolonged decelerations
783 decelerations 3-5 min
707 decelerations 6-9 min
374 decelerations >10 min

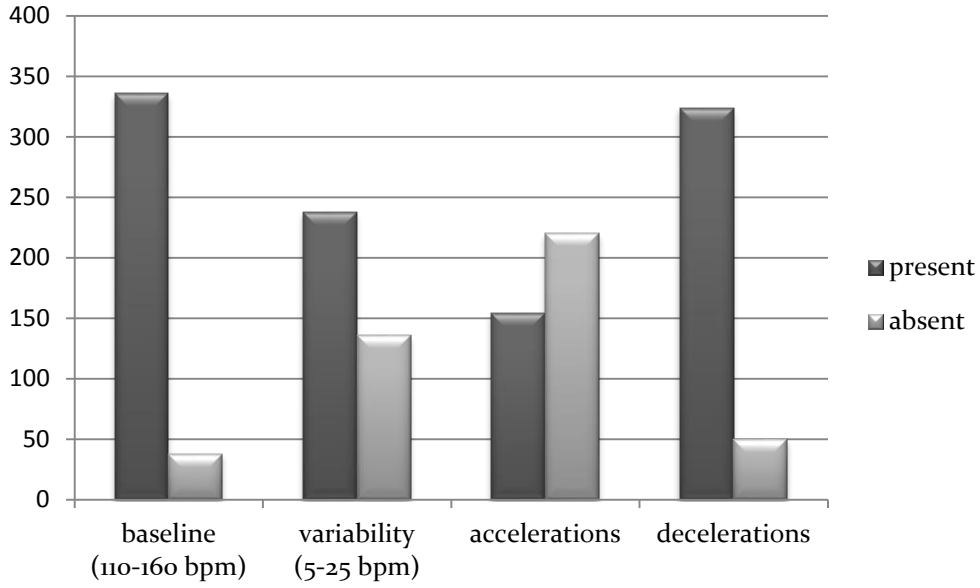
RESULTS: POPULATION

We have selected 374 CTG showing prolonged decelarations lasting for more than 10 minutes.

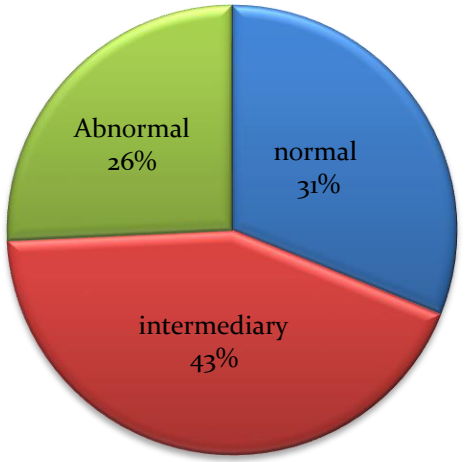
	YES	NO
37-39+6 weeks	35%	65%
PO	69.5%	30.5%
VBAC	0.3%	99.7%
Spontaneous labour	62.3%	37.7%
Meconium	24%	76%
Epidural	64.2%	35.8%
Syntocinon	43%	57%

RESULTS: CTG PRIOR TO DECELERATION

CTG features before decelerations



STAN classification of CTG before deceleration



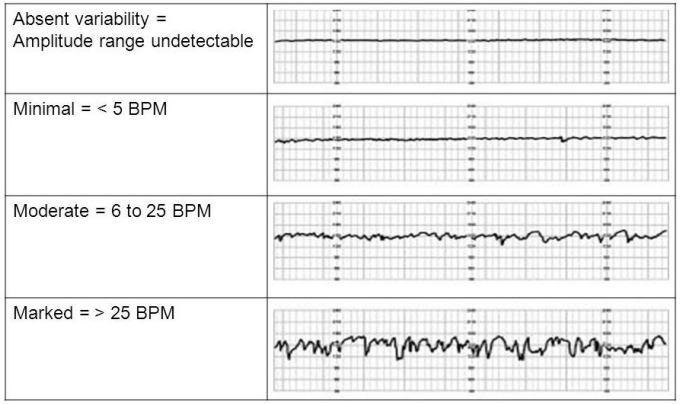
THE VARIABILITY BEFORE BRADYCARDIA

RESULTS: In the presence of minimal/absent variability (amplitude <5) for at least an hour, the incidence of significant acidemia (pH <7.0) ranged from 12% to 31%. CONCLUSION: The most significant intrapartum fetal heart rate parameter to predict the **development of acidemia** is the presence of minimal/absent variability for at least 1 hour as a solitary abnormal finding or in conjunction with late decelerations in the absence of accelerations

KP Williams et al.Intrapartum fetal heart rate patterns in the prediction of neonatal acidemia. Am J Obstet Gynecol 2003;188:820-3

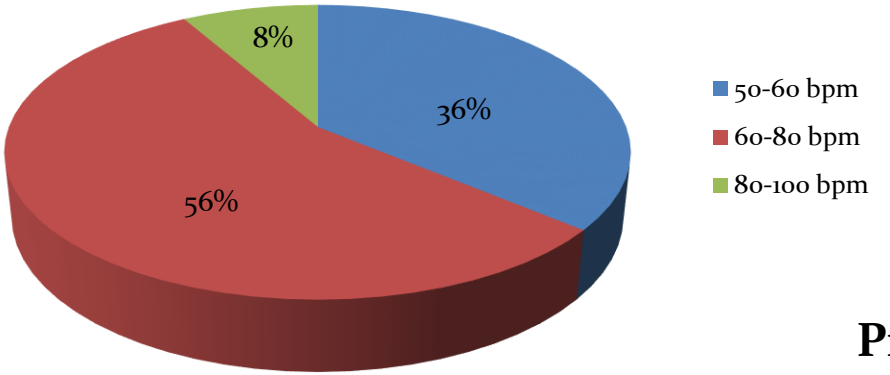
The following relationships were observed: (1) Moderate FHR variability was strongly associated (98%) with an umbilical pH>7.15 or newborn vigor (5-minute Apgar score>7). (2) Undetectable or minimal FHR variability in the presence of late or variable decelerations was the most consistent predictor of **newborn acidemia**.

Parer JT, et al. Fetal acidemia and electronic fetal heart rate patterns: is there evidence of an association? J Matern Fetal Neonatal Med 2006; 19: 289–294

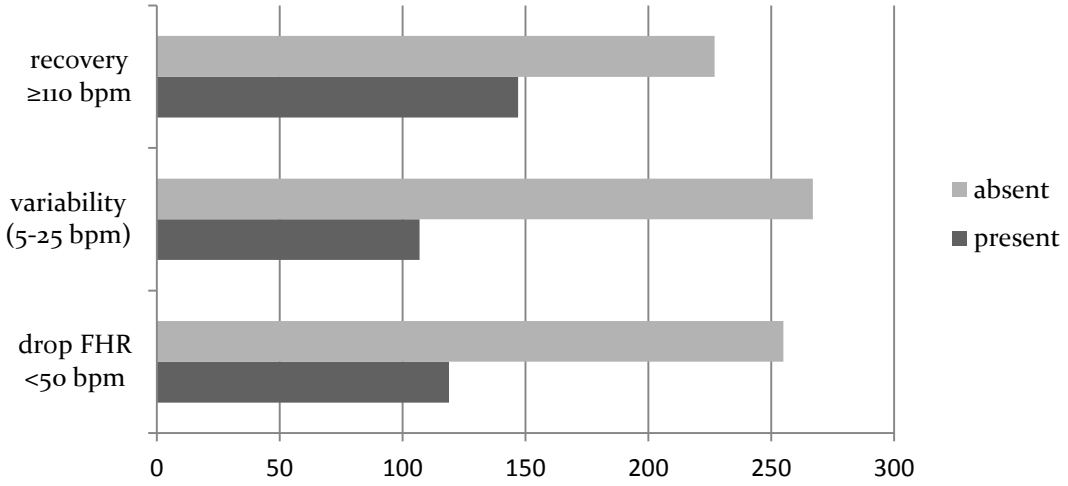


RESULTS: CTG DURING DECELERATION

Nadir FHR during deceleration



Prolonged deceleration features



THE VARIABILITY WITHIN BRADYCARDIA

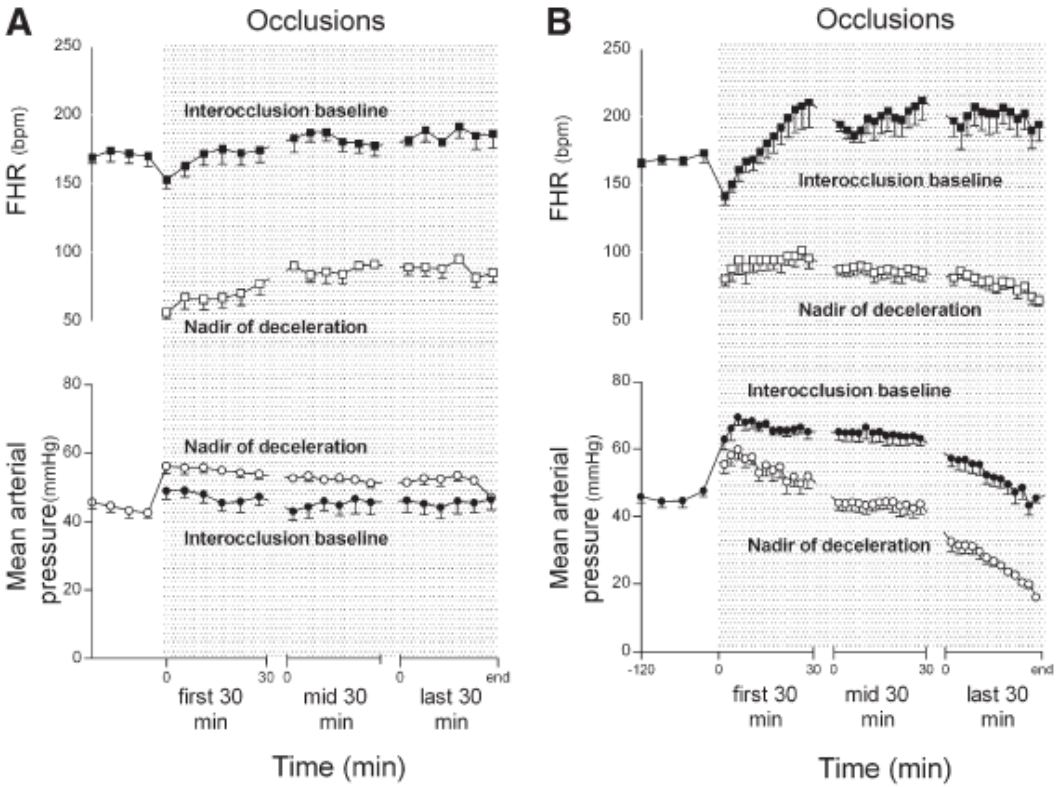
The reduction in fetal heart rate variability in the presence of bradycardia appears to be a rather late sign of severe acidosis, and is associated with reduced cerebral oxygen consumption and **poor fetal outcome**. It appears that a large proportion of fetal heart rate variability may be explained by centrally mediated fluctuations in the autonomic nervous system.

SM Siira et al.Marked fetal acidosis and specific changes in power spectrum analysis of fetal heart rate variability recorded during the last hour of labour. BJOG: An International Journal of Obstetrics & Gynaecology, 2005,112: 418–423

Results: Only 3 subtypes of variable decelerations showed significant discrimination to detect **babies with abnormal gases**: those with a prolonged duration (Area Under Curve 0.6109 P< 0.0001), loss of internal variability (AUC 0.5694 P< 0.0001) or with "sixties" criteria (AUC 0.5997 P< 0.0001). Among variable decelerations, only those with the atypical feature of loss of internal variability both alone or with another atypical feature had a statistically significance.

Hamilton E et al. Variable decelerations: do size and shape matter?Journal of maternal-fetal and neonatal medicine 2012; 25(6): 648–65

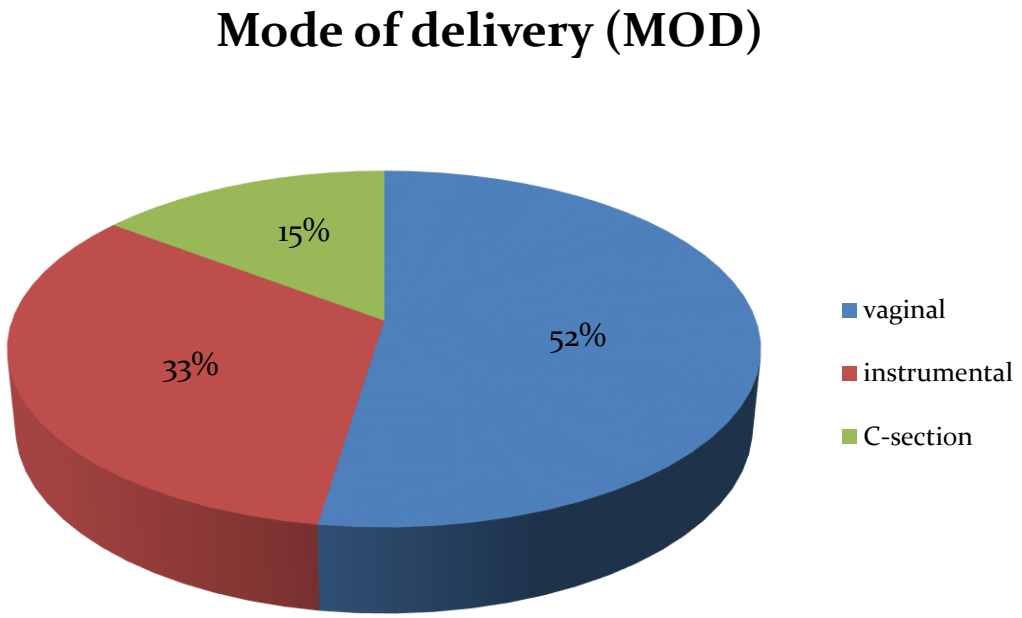
THE FREQUENCE OF CONTRACTIONS



A, In the 1:5 group (1-minute umbilical cord occlusion that was repeated every 5 minutes for 4 hours until fetal MAP fell <20 mm Hg), note that there was no significant change in interocclusion baseline FHR and MAP was higher during occlusions. The FHR decelerations were uniform in size. **B,** In the 1:2.5 group (1-minute occlusions repeated every 2.5 minutes until fetal MAP fell <20 mm Hg), note that interocclusion baseline FHR was higher in the first and mid 30 minutes. In the first 30 minutes, minimum MAP transiently rose to greater than baseline values, but fell progressively in the last 30 minutes. The FHR decelerations appeared to become much larger because of both a small fall in the nadir and a rise in the interocclusion baseline FHR. Data modified from Westgate et al.[47,49,71]

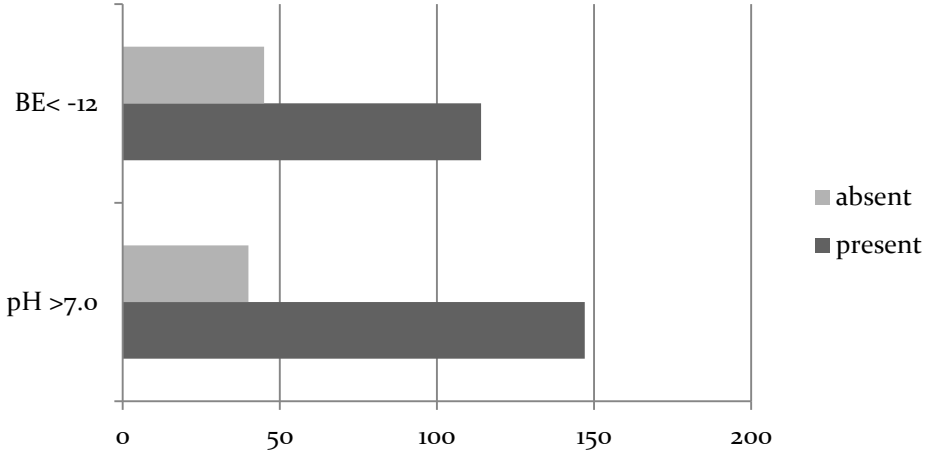
Westgate JA et al.
The intrapartum deceleration in center stage: a physiologic approach to the interpretation of fetal heart rate changes in labor.
Am J Obstet Gynecol 2007;197:236.e1-236.e11.

RESULTS: OBSTETRIC OUTCOME

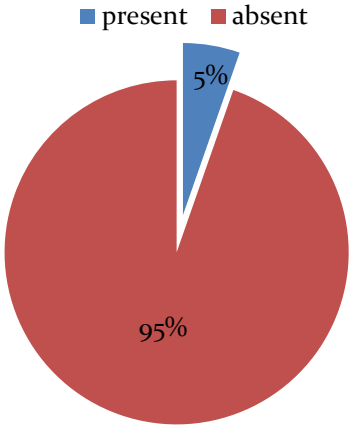


RESULTS: NEONATAL OUTCOME

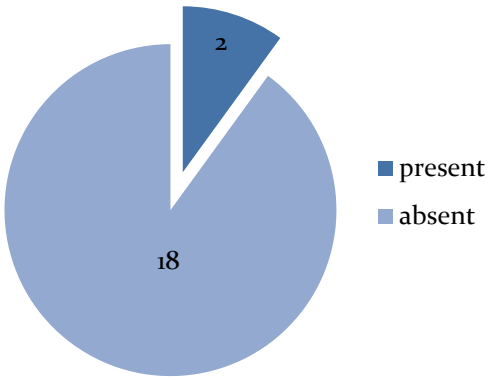
Neonatal outcome



NNU admission



HIE

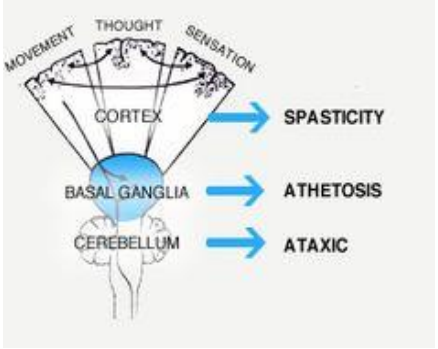


BAD NEONATAL OUTCOME & CTG DURING DECELERATION

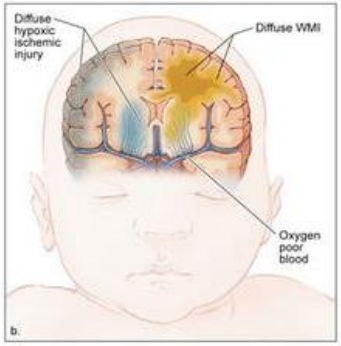
Outcome	*Variability*	*Prolonged deceleration*	*Nadir FHR*	*Number of events*
HIE	<5	>10 minutes	50-60	1
	<5	>10 minutes	60-80	1
NNU	<5	>10 minutes	50-60	3
	<5	>10 minutes	60-80	3
	5-20	>10 minutes	50-60	6
	5-20	>10 minutes	60-80	7
	5-20	>10 minutes	80+	1

NEUROLOGICAL CONSEQUENCES

"A **redistribution of blood flow** within the brain was evident both during and after cord occlusion, with the increase in blood flow more pronounced in the **subcortex and brainstem structures** relative to that of cortical structures. The lowest increase in blood flow is to the corpus striatum and hippocampus, and it is primarily this structure that shows neuronal loss in the near-term ovine fetus after repetitive cord occlusion of a severe degree."



Mallard EC et al. Transient umbilical cord occlusion causes hippocampal damage in the fetal sheep. Am J Obstet Gynecol 1992;167:1423-30



(…)Despite the marked increase in cerebral blood flow, **oxygen delivery** to the cerebral cortex was still decreased by approximately 35% when measured at 2 minutes of the first cord occlusion. However, although oxygen delivery was decreased similarly to the cerebellum during cord occlusion, oxygen delivery to the subcortex and brainstem structures was in fact well maintained, with the differential increase in regional blood flows within the brain."

Kaneko M et al.Cerebral blood flow and metabolism in relation to electrocortical activity with severe umbilical cord occlusion in the near-term ovine fetus. Am J Obstet Gynecol 2003;188:961-72

RESEARCH ADVANTAGES

• MORE THAN 350 CASES with prolonged deceleration more than 10 min as we have analyzed each deceleration as a single case

• FIRST STUDY that evaluated so carefully each deceleration, considering the nadir FHR and the gap between baseline and nadir

• obstetric history available for all patients

• delivery notes available for all patients (induction, meconium, epidural, syntocinon)

• good quality of ctg recording before, during and after prolonged decelerations with midwife notes recorded precisely

• apgar, birthweight and info about possible NNU admission and HIE available for all newborns, while BE and pH available for most of them

RESEARCH LIMITATIONS

• both obstetric and neonatal outcome are influenced by clinical decision of consultant (interobserver difference in evaluating the CTG)

•BE and pH not available for all newborns

• statistical analysis not completed yet

• time lapse between prolonged deceleration and delivery has not been analyzed but it maybe helpful

FURTHER RESEARCH

- another database with MORE THAN 1800 CASES OF PROLONGED DECELERATIONS (classified according to their duration in 3 groups)

- comparison of obstetric and neonatal outcome between prolonged deceleration with different duration

- evaluation of time lapse between prolonged deceleration and delivery? maybe helpful to understand the neonatal outcome

- possible long term follow-up in children born after prolonged decelerations (some subclinical brain or heart damage?)

NEONATAL EVALUATION

Most infants with evidence of intrapartum asphyxia do not develop serious long-term sequelae (..) A **score** combining a measure of cardiotocographic abnormality, umbilical arterial base excess, and low 5-min Apgar score is much more strongly associated with morbidity (multiorgan impairment following perinatal asphyxia) than any individual factor.

Portman RJ et al. Predicting neonatal morbidity after perinatal asphyxia: a scoring system. Am J Obstet Gynecol 1990; 162:174–82.

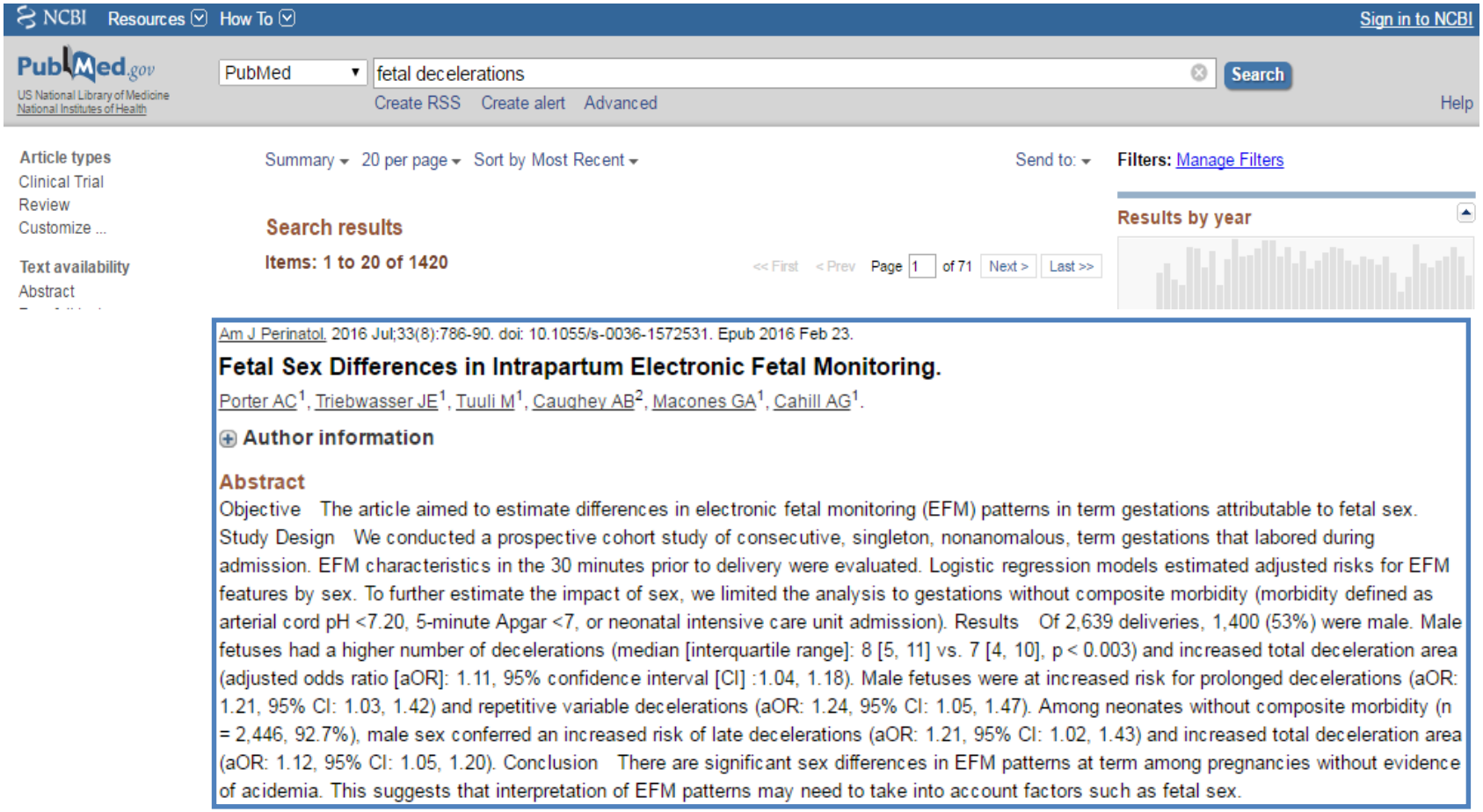
Low cord pH in infants who are **vigorous** at birth and free of cardiopulmonary compromise does not indicate an increased risk of adverse outcome. Infants with pH less than 7.0 at birth who are not vigorous are at high risk of adverse outcome.

Goldaber KG et al. Pathologic fetal acidemia.Obstet Gynecol 1991; 78: 1103–7.

If the obstruction to the umbilical vessels was sudden and complete and this persisted until the moment of delivery or until fetal death then the cord gases sampled at birth would give a snapshot of the **fetal acid–base balance prior to the obstruction**, so that normal cord venous and arterial pH do not therefore exclude acute intrapartum asphyxia

Armstrong L, Stenson BJ. Use of umbilical cord blood gas analysis in the assessment of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007; 92: F430-F434.

NEW DISCUSSIONS..



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Results by year

Am J Perinatol. 2016 Jul;33(8):786-90. doi: 10.1055/s-0036-1572531. Epub 2016 Feb 23.

Fetal Sex Differences in Intrapartum Electronic Fetal Monitoring.

Porter AC[1], Triebwasser JE[1], Tuuli M[1], Caughey AB[2], Macones GA[1], Cahill AG[1].

Author information

Abstract

Objective The article aimed to estimate differences in electronic fetal monitoring (EFM) patterns in term gestations attributable to fetal sex. Study Design We conducted a prospective cohort study of consecutive, singleton, nonanomalous, term gestations that labored during admission. EFM characteristics in the 30 minutes prior to delivery were evaluated. Logistic regression models estimated adjusted risks for EFM features by sex. To further estimate the impact of sex, we limited the analysis to gestations without composite morbidity (morbidity defined as arterial cord pH <7.20, 5-minute Apgar <7, or neonatal intensive care unit admission). Results Of 2,639 deliveries, 1,400 (53%) were male. Male fetuses had a higher number of decelerations (median [interquartile range]: 8 [5, 11] vs. 7 [4, 10], p < 0.003) and increased total deceleration area (adjusted odds ratio [aOR]: 1.11, 95% confidence interval [CI] :1.04, 1.18). Male fetuses were at increased risk for prolonged decelerations (aOR: 1.21, 95% CI: 1.03, 1.42) and repetitive variable decelerations (aOR: 1.24, 95% CI: 1.05, 1.47). Among neonates without composite morbidity (n = 2,446, 92.7%), male sex conferred an increased risk of late decelerations (aOR: 1.21, 95% CI: 1.02, 1.43) and increased total deceleration area (aOR: 1.12, 95% CI: 1.05, 1.20). Conclusion There are significant sex differences in EFM patterns at term among pregnancies without evidence of acidemia. This suggests that interpretation of EFM patterns may need to take into account factors such as fetal sex.

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