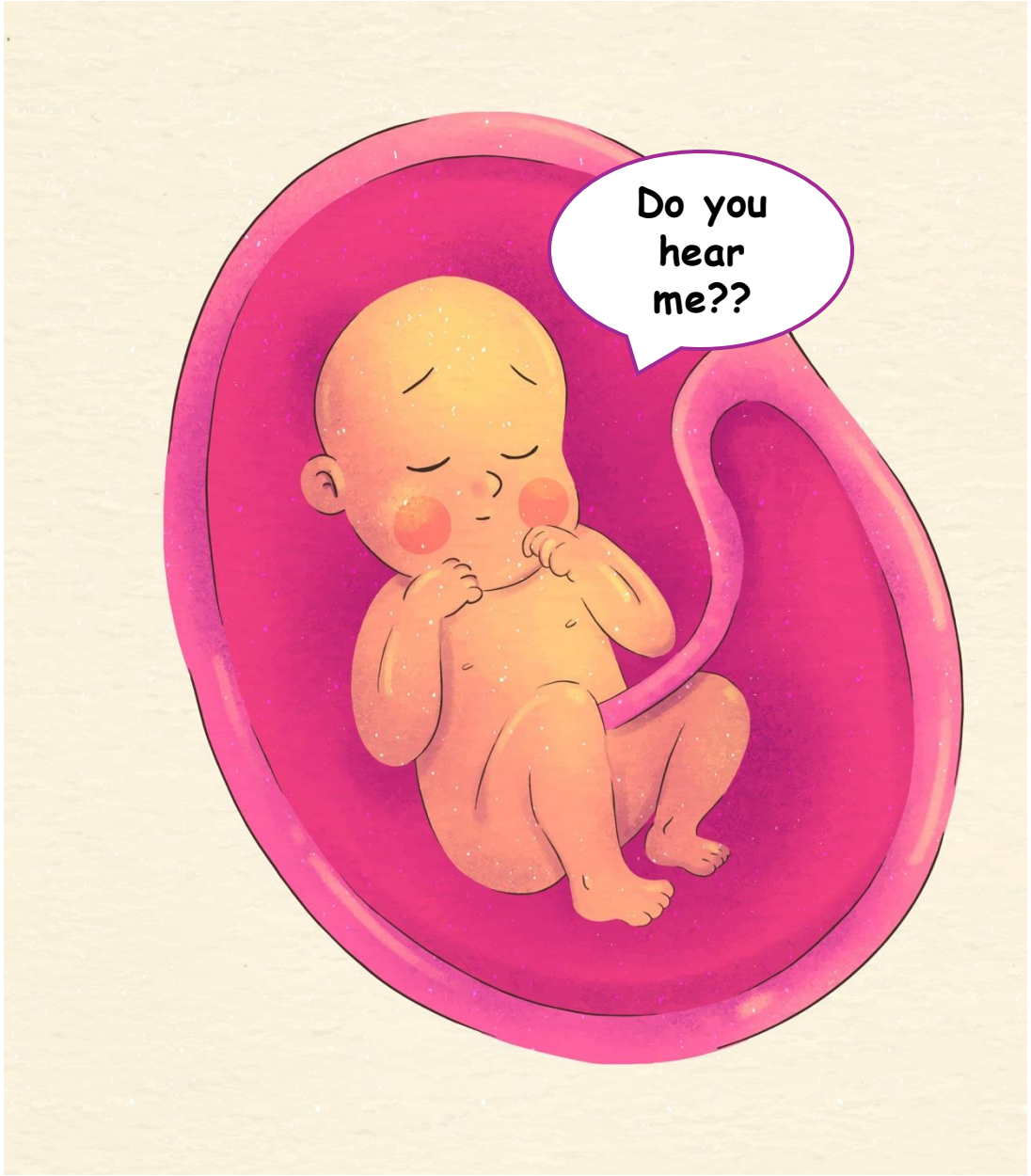
Fetal Physiological-Based CTG Classification



OUTLINE

- What is the important of intrapartum fetal monitoring?
- Why is the “traditional practice” of CTG has been questioned?
- How does fit fetal response to hypoxic condition?
- How does unfit fetal response to hypoxic condition?
- Does the fetus fit enough to take the hypoxic journey of labor?
- How is the proper way to take umbilical cord blood sample?

Oxygen pathway

Environment

Lungs

Heart

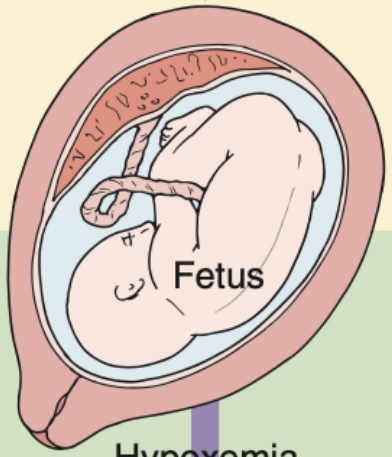
Vasculature

Uterus

Placenta

Cord

Fetal oxygenation involves transfer of oxygen from the environment to the fetus along a pathway that includes the maternal lungs, heart, vasculature, uterus, placenta, and umbilical cord.



Fetal response

Hypoxemia

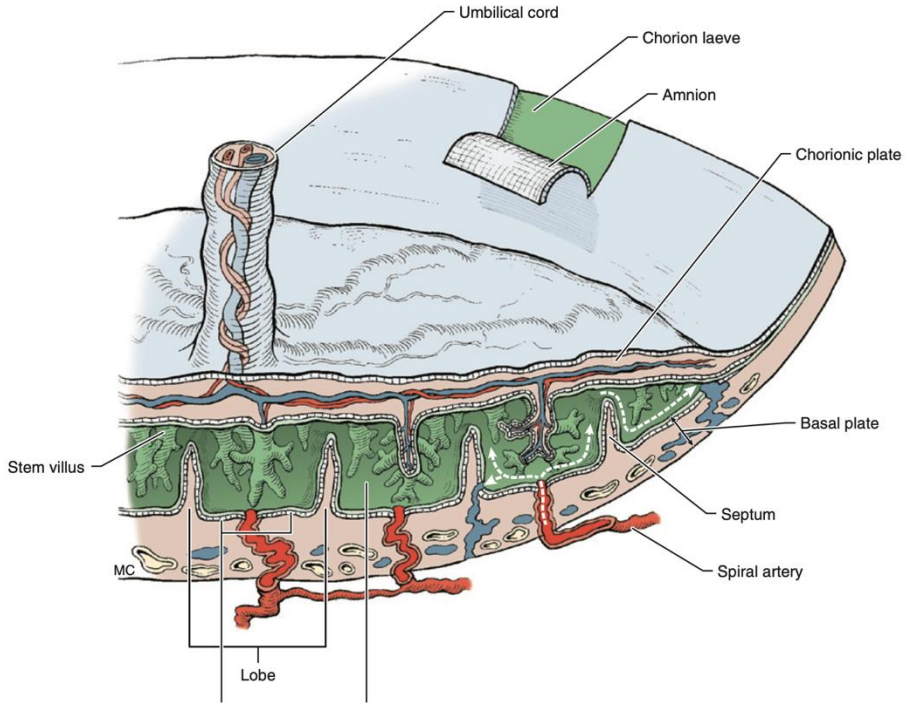
Hypoxia

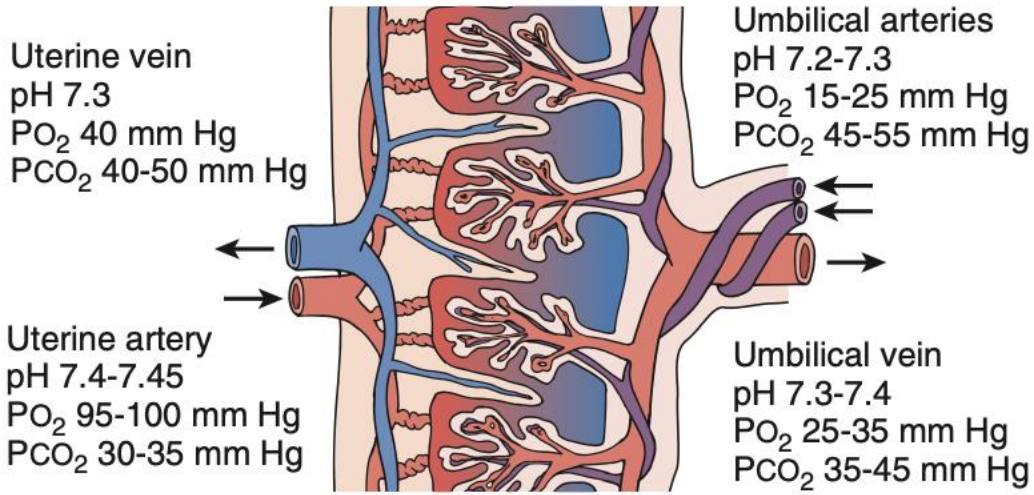
Metabolic acidosis

Metabolic acidemia

Fetal oxygenation also involves the consequences of interruption of fetal oxygenation, including hypoxemia, hypoxia, metabolic acidosis, metabolic acidemia, and potential injury.

Potential injury





Gabbe. 7th ed

What is the important of intrapartum fetal monitoring?

- Intrapartum fetal monitoring is intended to **assess the adequacy of fetal oxygenation during labor**.
- Intrapartum fetal monitoring is to **identify situations that precede hypoxia/acidemia to avoid fetal injury**
- Intrapartum fetal monitoring **determine the frequency, duration, and severity of interrupted oxygenation** so that **appropriately informed decisions can be made regarding the optimal timing and method of delivery**

“Traditional CTG”

FIGO	NICE	ACOG
Normal pattern • Baseline heart rate between 110 and 150 bpm • Amplitude of heart rate variability between 5 and 25 bpm	Normal (a CTG where all of the following four reassuring features are present) • Baseline rate: 110–160 bpm • Variability: ≥5 bpm • No decelerations • Accelerations: present	Category I (category I FHR tracings include all of the following) • Baseline rate: 110–160 bpm • Baseline variability: 6–25 bpm • Late or variable decelerations: absent • Early decelerations: present or absent • Accelerations: present or absent
Suspicious pattern • Baseline heart rate between 150 and 170 bpm or between 100 and 110 bpm • Amplitude of variability between 5 and 10 bpm for more than 40 min • Increased variability above 25 bpm • Variable decelerations	Suspicious (a CTG where one of the following features is present and all others fall into the reassuring category) • Baseline rate – 100–109 bpm – 161–180 bpm • Baseline variability – <5 bpm for 40–90 min • Decelerations – Typical variable decelerations with >50% of contractions occurring for >90 min – Single prolonged deceleration for up to 3 min • Accelerations – The absence of accelerations with an otherwise normal trace is of uncertain significance	Category II (Category II FHR tracings include all FHR tracings not categorised as Category I or Category III. Examples of Category II FHR tracings include any of the following) • Baseline rate – Bradycardia not accompanied by absent baseline variability – Tachycardia • Baseline variability – Minimal variability – Absent variability with no recurrent decelerations – Marked variability • Accelerations – Absence of induced accelerations after fetal stimulation • Periodic or episodic decelerations – Recurrent variable decelerations accompanied by minimal or moderate baseline variability – Prolonged deceleration 2–10 min – Recurrent late decelerations with moderate baseline variability – Variable decelerations with other characteristics such as slow return to baseline, overshoots or shoulders
Pathological pattern • Baseline heart rate <100 or >170 bpm • Persistence of heart rate variability of <5 bpm for >40 min • Severe variable decelerations or severe repetitive early decelerations • Prolonged decelerations • Late decelerations: the most ominous trace is a steady baseline without baseline variability and with small decelerations after each contraction • A sinusoidal pattern	Pathological (a CTG with one or more of the following features or two or more features in the previous category) • Baseline rate – <100 bpm – >180 bpm – Sinusoidal pattern ≥ 10 min • Baseline variability – <5 bpm for ≥90 min • Decelerations – Atypical variable decelerations with >50% contractions for >30 min – Late decelerations for >30 min – Prolonged deceleration >3 min	Category III (Category III FHR tracings include either) • Absent baseline FHR variability and any of the following: – Recurrent late decelerations – Recurrent variable decelerations – Bradycardia • Sinusoidal pattern

Why is the “traditional practice” of CTG has been questioned?

The latest Cochrane Systematic Review on electronic fetal heart rate monitoring concluded that the use of continuous CTG for fetal assessment using “normal, suspicious, pathological” or “category I,II & III” classification systems have not resulted in a reduction in the incidence of cerebral palsy or perinatal deaths, but has led to an increase in the rate of cesarean sections and operative vaginal births.

Cochrane Database Syst Rev. 2017 Feb 3;2017(2):CD006066. doi: 10.1002/14651858.CD006066.pub3

A recent meta-analysis evaluating the three-tiered system of the American College of Obstetrics and Gynecology for fetal heart rate monitoring to predict adverse neonatal acidosis. The authors found that there was no difference in the incidence of hypoxic–ischemic encephalopathy between categories I and II; but most importantly, that almost 98% of fetuses with category II tracings did not present acidosis at birth.

AJOG. Volume 229, Issue 4, October 2023, Pages 377-387. https://doi.org/10.1016/j.ajog.2023.04.008

Kita memahami apa yang membuat menjadi kategori 2 atau sedapat mungkin memaksa jadi kategori I?

Over-reliance on deceleration **based on morphology** to determine the severity of intrapartum hypoxic stress

Many **abnormal CTG** patterns are suspected intrapartum, **not related to fetal acidemia**

Interobserver reliability in interpreting cardiotocographs (CTGs) using traditional categorization into "normal," "suspicious," and "pathological" is typically **very low**, ranging from Kappa 0.3 to 0.6

Urgent need to move from pattern recognition to fetal physiology – based interpretation

Each Baby Counts Reports in the UK from 2016-2021 have consistently highlighted that 33% of intrapartum related ***perinatal deaths and severe hypoxic ischaemic injuries*** *in the UK were due to* ***CTG misinterpretation***

Year 2018: 44 CTG experts from 14 countries

Contents lists available at ScienceDirect

European Journal of Obstetrics & Gynecology and Reproductive Biology

journal homepage: www.journals.elsevier.com/european-journal-of-obstetrics-and-gynecology-and-reproductive-biology

Full length article

International expert consensus statement on physiological interpretation of cardiotocograph (CTG): First revision (2024)

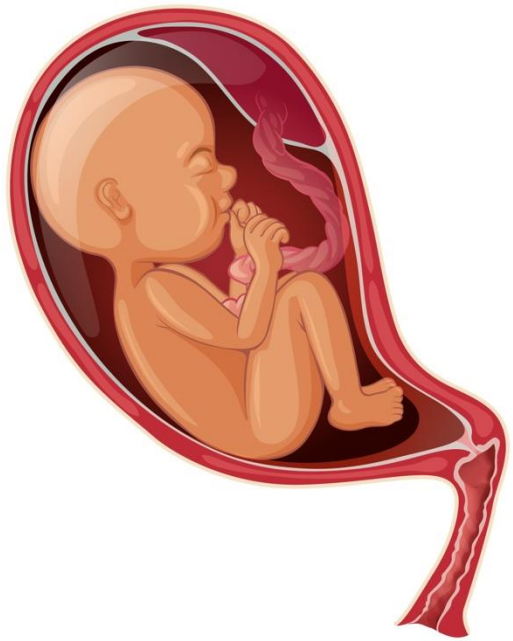
Edwin Chandraharan [a,*], Susana Pereira [b], Tullio Ghi [c], Anna Gracia Perez-Bonfils [d], Stefania Fieni [e], Yan-Ju Jia [f], Katherine Griffiths [g], Suganya Sukumaran [h], Caron Ingram [i], Katharine Reeves [j], Mareike Bolten [k], Katrine Loser [l], Elena Carreras [m,n], Anna Suy [m], Itziar Garcia-Ruiz [m], Letizia Galli [o], Ahmed Zaima [p,1,2]

have been implemented in more than 20 maternity units in the UK, and several hospitals in Spain, Belgium, France, Italy, Australia, Denmark, Estonia, Switzerland, Lithuania, Romania, Sri Lanka, China, Singapore, Oman and the United Arab Emirates

Purpose:

Attain a **better** understanding of the **adaptive changes of the fetus** when facing **intrapartum hypoxic stress**

HOW FIT FETAL RESPONSES TO HYPPOXIC CONDITION IN LABOR



Human Labor

↓

Repetitive uterine contraction

- Repetitive compression of umbilical cord
- Repetitive compression of fetal head → >50 % reduction of Uteroplacental perfusion → Hypoxemia, hypoxic and ↑ oxidative stress
- Repetitive compression of spiral arteries

Compensatory mechanism → ✖

↓

No Hypoxic ischemic injury

- ✓ ↑ Hb concentration
- ✓ HbF has greater affinity for O2
- ✓ Cardioprotector reflex → reduced myocardial workload
- ✓ Cathecolamin – mediated → Fetal heart rate (FHR) increase
- ✓ Effective redistribution to protect fetal central organs
- ✓ Extra placental blood reserve
- ✓ Glycogen stores

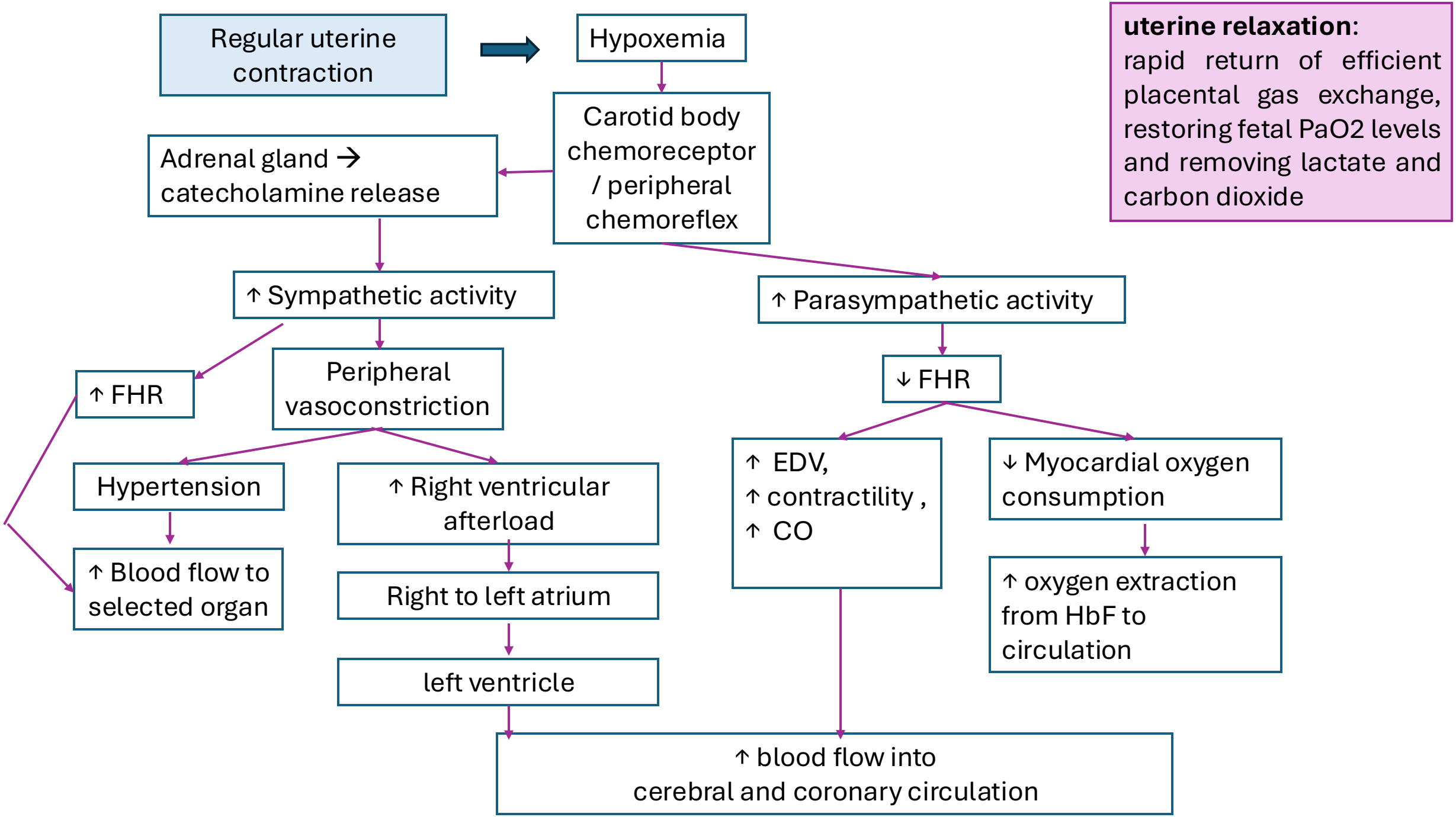
Ghi T. Acta Obstet Gynecol Scand. 2024;103:1910–1918. DOI: 10.1111/aogs.14937

Fetus Response to Hypoxic condition

Hypoxic stress

Reduce myocardiac workload

Lowering the fetal heart rate


Regular uterine contraction
Hypoxemia
Carotid body chemoreceptor / peripheral chemoreflex
Adrenal gland → catecholamine release
↑ Sympathetic activity
↑ Parasympathetic activity
↑ FHR
Peripheral vasoconstriction
↓ FHR
Hypertension
↑ Right ventricular afterload
↑ EDV, ↑ contractility , ↑ CO
↓ Myocardial oxygen consumption
↑ Blood flow to selected organ
Right to left atrium
↑ oxygen extraction from HbF to circulation
left ventricle
↑ blood flow into cerebral and coronary circulation
uterine relaxation: rapid return of efficient placental gas exchange, restoring fetal PaO2 levels and removing lactate and carbon dioxide

HOW UNFIT FETAL RESPONSES TO HYPPOXIC CONDITION IN LABOR

Maternal compromise

Preexisting compromise

Coexisting compromise

Structural abnormality or functional abnormality

Onset of regular uterine activity

Effect on the compensatory mechanism

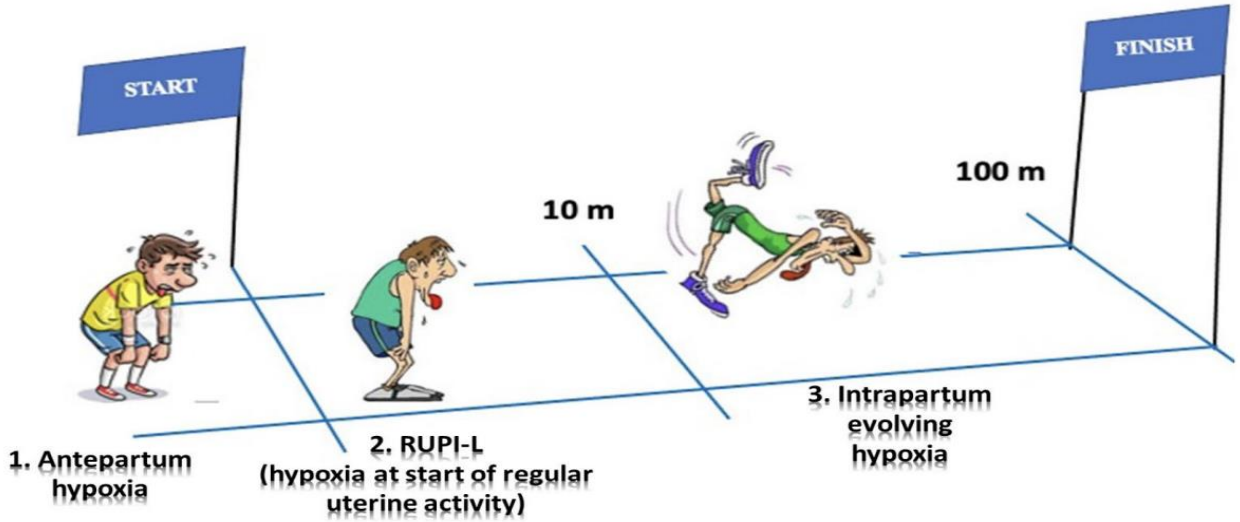
Stress to injury cell/ death shortened

Less energy reserve To maintain aerobic metabolism

Hypoxic condition

Signs on CTG

?



FETAL DECOMPENSATION

↓Myocardial responsiveness

↓Systemic BP

↓Cerebral perfusion

Myocardial hypoxic damage

Neuronal Damage

HYPOXIC ISCHEMIC ENCEPHALOPATHY

If hypoxia prolonged → decompensate → maintain myocardial integrity by balancing energy → catecholamine - induced glycogenolysis (glycogen to glucose) → generate energy → maintain integrity of myocardial cell membrane

Ghi T. Acta Obstet Gynecol Scand. 2024;103:1910–1918. DOI: 10.1111/aogs.14937

RUPI-L: Relative utero plasental insufficiency

Baseline Fetal Heat Rate

Reflects the number of times the fetal heart chambers must beat to meet the ongoing metabolic demands of the fetus

Stable: sufficient glycogen reserves and remains in an aerobic metabolism → reflects good oxygenation and myocard is in a good aerobic balance

Stability, actual rate ~ gestational age, changes compared with previous recording

Unstable: negative myocardial energy balance

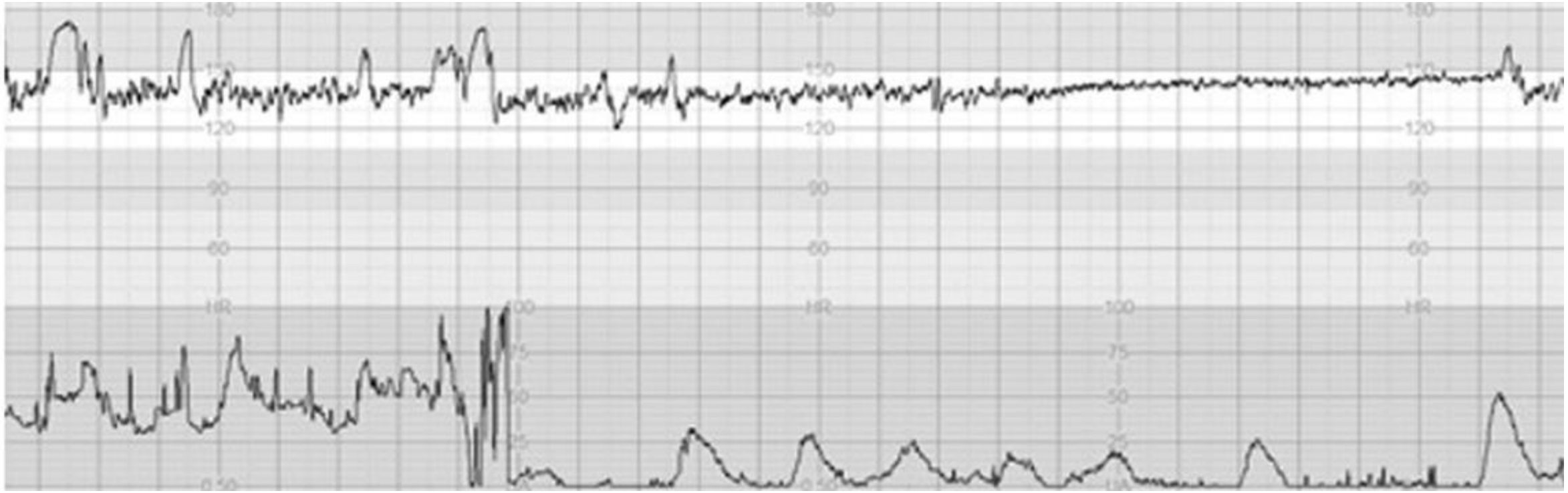
- >10% **sustained increase in the baseline FHR** without any preceding decelerations → a high index of suspicion for **ongoing fetal inflammatory response**
- Progressive maturity of the parasympathetic nervous system as gestation advances → a gradual and progressive reduction in the baseline FHR with advancing gestational age

Baseline Fetal Heart Rate Variability

- Between 28 and 32 weeks of gestation rest-activity cycles start coupling to specific parameters of the fetal heart rate
- With advancing gestation, the maturation connections → steady decrease of the fetal heart baseline, the increase in the short- and long-term variability, and the association between the accelerations of the FHR with active movements
- Integrity of the autonomic nervous system centers→ sympathetic center attempts to increase the heart rate while parasympathetic center attempts to reduce it
- **Normal and stable baseline FHR variability** indicates a nondepressed fetal central nervous system (CNS), sufficient myocardium glycogen reserves and aerobic metabolism
- Several **nonhypoxic causes** (eg, fetal deep sleep cycle; maternal hypoglycemia; medications, such as opiates and magnesium sulfate; and fetal neuroinflammation in chorioamnionitis) can depress the fetal CNS

“Cycling”

- “cycling” defined as the **physiological alternating epochs** of normal and reduced heart rate variability
- Absense of cycling → consider neuroinflammation secondary to chorioamnionitis



Up and down fluctuation

- An erratic up and down fluctuation of >25 bpm **usually** occurs during labor for at least one minute, called the“Zig-Zag” pattern
- 1 to 10 minutes “Zig-Zag” pattern – may occur in cases of rapidly evolving hypoxic stress. The zig zag preceded late deceleration
- when it is uniformly increased and persists for >30 minutes: **Saltatory pattern** (usually occurs during the antenatal period when a fetus recovers from an acute insult).
- Increased variability is associated with abnormal umbilical arterial pH

- If **the ZigZag pattern** with an **increase in the baseline FHR** without repetitive decelerations → should raise the suspicion of **fetal neuroinflammation** in the context of chorioamnionitis
- An increase in the baseline FHR by > 10 % without preceding deceleration and/or a baseline FHR >10 % higher than what is expected for the gestational age should be considered as **suggestive of fetal inflammation (SOFI)**

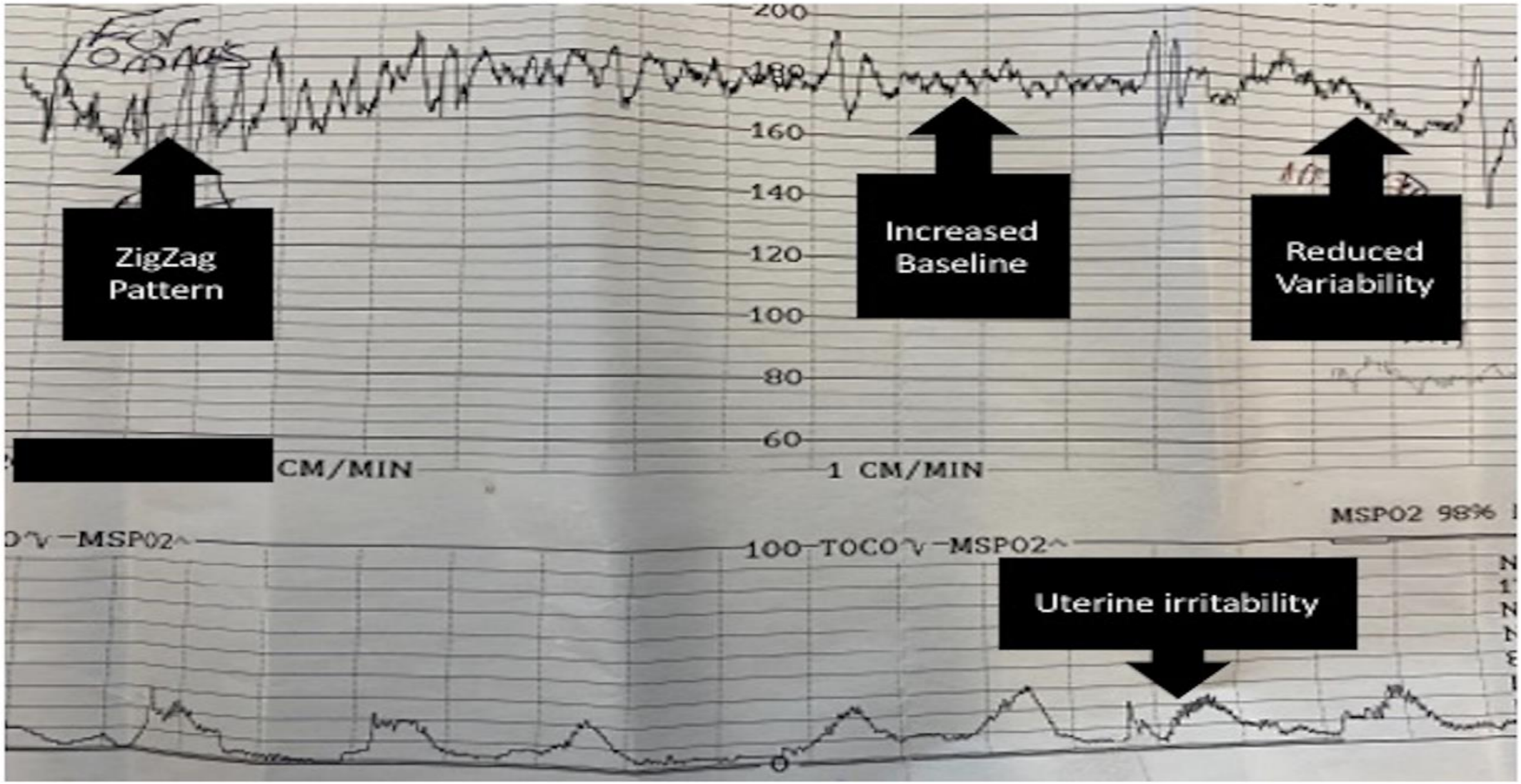


Fig. 2. ZigZag pattern, increased baseline, reduced variability and uterine irritability.

Neonatal outcome characteristics when CTG changes are present or absent

Variables	ZigZag Pattern present	ZigZag Pattern absent	*P* value	Late Decelerations present	Late Decelerations absent	*P* value
Number	582 (11.7)	4406 (88.3)		2045 (41.0)	2943 (59.0)	
UA pH	7.19 (± 0.08)	7.28 (± 0.09)	<.001*	7.25 (± 0.10)	7.27 (± 0.09)	<.001*
UA BE (meq/L)	−5.8 (± 3.1)	−3.1 (± 3.0)	<.001*	−4.1 (± 3.0)	−3.9 (± 3.0)	<.001*
UA pO_2 (kPa)	2.9 (± 1.0)	3.1 (± 0.9)	<.001*	3.0 (± 1.0)	3.1 (± 0.9)	.004*
UA acidosis						
UA pH <7.10	57 (9.8)	89 (2.0)	<.001*	102 (5.0)	44 (1.5)	<.001*
UA BE <−12.0 (meq/L)	21 (3.6)	18 (0.4)	<.001*	31 (1.5)	8 (0.3)	<.001*
5 min Apgar score <7	7 (1.2)	4 (0.1)	<.001*	10 (0.5)	1 (0.03)	<.001*
Hypoglycemia, P-gluc <2.6 mmol/L	48 (8.2)	45 (1.0)	<.001*	76 (3.7)	17 (0.6)	<.001*
Intubation for resuscitation	10 (1.7)	7 (0.2)	<.001*	14 (0.7)	3 (0.1)	<.001*
NICU admission	45 (7.7)	179 (4.1)	<.001*	140 (6.8)	84 (2.9)	<.001*
Neonatal encephalopathy	0 (0.0)	2 (0.05)	.06	0 (0.0)	2 (0.07)	.24

Variables	Bradycardia present	Bradycardia absent		Reduced Variability present	Reduced Variability absent	
Number	2639 (52.9)	2349 (47.1)		1831 (36.7)	3157 (63.3)	
UA pH	7.27 (± 0.09)	7.26 (± 0.09)	<.001^	7.26 (± 0.19)	7.26 (± 0.09)	.51
UA BE (meq/L)	−4.0 (± 1.2)	−4.2 (± 2.9)	<.001^	−4.2 (± 4.4)	−4.1 (± 2.7)	.27
UA pO_2 (kPa)	3.2 (± 1.0)	3.1 (± 1.0)	<.001^	3.2 (± 0.9)	3.2 (± 0.9)	.72
UA acidosis						
UA pH <7.10	75 (2.8)	71 (3.0)	.42	67 (3.7)	79 (2.5)	.06
UA BE <−12.0 (meq/L)	17 (0.6)	22 (0.8)	.63	21 (1.1)	18 (0.6)	.08
5-min Apgar score <7	6 (0.2)	5 (0.2)	.60	4 (0.2)	7 (0.2)	.94
Hypoglycemia, P-glucose <2.6 mmol/L	36 (1.3)	57 (2.4)	.006^	47 (1.5)	46 (2.6)	.009^
Intubation for resuscitation	7 (0.3)	10 (0.4)	.80	7 (0.3)	10 (0.3)	.90
NICU admission	121 (4.6)	103 (4.4)	.21	114 (6.2)	110 (3.5)	<.001*
Neonatal encephalopathy	0 (0.0)	2 (0.09)	.84	2 (0.1)	0 (0.0)	.16

Variables	Tachycardia present	Tachycardia absent		Uterine Tachysystole present	Uterine Tachysystole absent	
Number	694 (13.9)	4294 (86.1)		228 (4.6)	4760 (95.4)	
UA pH	7.27 (± 0.08)	7.26 (± 0.09)	.01^	7.28 (± 0.09)	7.26 (± 0.09)	.18
UA BE (meq/L)	−3.9 (± 2.7)	−4.2 (± 2.9)	.001^	−4.1 (± 2.8)	−4.2 (± 2.8)	.45
UA pO_2 (kPa)	3.2 (± 0.9)	3.1 (± 1.0)	.005^	3.2 (± 1.1)	3.2 (± 0.9)	.20
UA acidosis						
UA pH <7.10	22 (3.2)	124 (2.9)	.68	9 (3.9)	137 (2.9)	.42
UA BE <−12.0 (meq/L)	6 (0.9)	33 (0.8)	.80	3 (1.3)	36 (0.8)	.47
5-min Apgar score <7	3 (0.4)	8 (0.2)	.05	0 (0.0)	11 (0.2)	.001^
Hypoglycemia, P-glucose <2.6 mmol/L	19 (2.7)	74 (1.7)	.12	5 (1.7)	88 (1.8)	.72
Intubation for resuscitation	1 (0.1)	16 (0.4)	.18	1 (0.4)	16 (0.3)	.86
NICU admission	67 (9.7)	157 (3.7)	<.001*	10 (4.4)	214 (4.5)	.43
Neonatal encephalopathy	0 (0.0)	2 (0.09)	.57	0 (0.0)	2 (0.04)	.76

Data are mean ± SD or number (%). * Significant when present. ^ Significant when absent.
BE, base excess; NICU, neonatal intensive care unit; UA, umbilical artery;

Tarvonen M, Hovi P, Sainio S, Vuorela P, Andersson S, Teramo K. Intrapartum zigzag pattern of fetal heart rate is an early sign of fetal hypoxia: a large obstetric retrospective cohort study. Acta Obstetricia et Gynecologica Scandinavica. 2021 Feb;100(2):252-62.

Intrapartum zigzag pattern of fetal heart rate is an early sign of fetal hypoxia: A large obstetric retrospective cohort study

Mikko Tarvonen[1] | Petteri Hovi[2,5] | Susanna Sainio[3] | Piia Vuorela[4] | Sture Andersson[5] | Kari Teramo[1]

Neonatal outcome characteristics when ZigZag pattern is compared with other CTG changes present

Variables	ZigZag pattern present	Late decelerations present	*P* value	Bradycardia episodes present	*P* value	Reduced variability present	*P* value	Tachycardia present	*P* value	Uterine tachysystole present	*P* value
n	582 (11.7)	2045 (41.0)		2639 (52.9)		1831 (36.7)		694 (13.9)		228 (4.6)	
UA pH	7.19 (± 0.08)	7.25 (± 0.10)	<.001	7.27 (± 0.09)	<.001	7.26 (± 0.19)	<.001	7.27 (± 0.08)	<.001	7.28 (± 0.09)	<.001
UA BE (meq/L)	−5.8 (± 3.1)	−4.1 (± 3.0)	<.001	−4.0 (± 1.2)	<.001	−4.2 (± 4.4)	<.001	−3.9 (± 2.7)	<.001	−4.1 (± 2.8)	<.001
UA pO_2 (kPa)	2.9 (± 1.0)	3.0 (± 1.0)	<.001	3.2 (± 1.0)	<.001	3.2 (± 0.9)	<.001	3.2 (± 0.9)	<.001	3.2 (± 1.1)	<.001
UA acidosis											
UA pH <7.10	57 (9.8)	102 (5.0)	<.001	75 (2.8)	<.001	67 (3.7)	<.001	22 (3.2)	<.001	9 (3.9)	<.001
UA BE <−12.0 (meq/L)	21 (3.6)	31 (1.5)	.011	17 (0.6)	<.001	21 (1.1)	<.001	6 (0.9)	.001	3 (1.3)	.034
5-min Apgar score <7	7 (1.2)	10 (0.5)	.06	6 (0.2)	.028	4 (0.2)	.002	3 (0.4)	.14	0 (0.0)	.008
Hypoglycemia, P-glucose <2.6 mmol/L	48 (8.2)	76 (3.7)	<.001	36 (1.3)	<.001	47 (1.5)	<.001	19 (2.7)	<.001	5 (1.7)	<.001
Intubation for resuscitation	10 (1.7)	14 (0.7)	.014	7 (0.3)	<.001	7 (0.3)	<.001	1 (0.1)	.003	1 (0.4)	.02
NICU admission	45 (7.7)	140 (6.8)	.46	121 (4.6)	.008	114 (6.2)	.20	67 (9.7)	.23	10 (4.4)	.09
Neonatal encephalopathy	0 (0.0)	0 (0.0)	1.00	0 (0.0)	1.00	2 (0.1)	.43	0 (0.0)	1.00	0 (0.0)	1.00

Data are mean ± SD or number (%).

BE, base excess; CTG, cardiotocography; NICU, neonatal intensive care unit. UA, umbilical artery.

- late decelerations occurred in 91.2% (531/582) of the CTG recordings together with ZigZag pattern.
- ZigZag pattern preceded late decelerations in 88.7% of the cases.
- A normal FHR preceded the ZigZag pattern in 90.4% (526/582) of the cases
- After ZigZag episodes, a normal FHR pattern was observed in only 0.9% (5/582).

the ZigZag pattern is
an early sign of fetal hypoxia

Accelerations

Abrupt and transient increase in the baseline heart rate, approximately 15 bpm lasting for approximately 15 seconds in a term fetus

Reflect the integrity of the fetal somatic nervous system.

In hypoxia or acidosis→fetus converse oxygen and nutrients →restricts the somatic body movements

True accelerations arise from a stable baseline FHR with a normal variability and should return to the same stable baseline

Deceleration

Quicklie Deceleration

- Quick reflex response/ quick recovery to the baseline
- mediated by baroreceptor reflex
- Repetitive compression of the umbilical cord
- Resultant transient hypoxemia
- Not related to acidosis
- Changing maternal position might restore it to normal

Tardy Deceleration

- Gradual/ slow recovery to the baseline
- Structural abnormality of the placenta or functional reduction in oxygenation of the placental pool
- low oxygen tension, high carbon dioxide concentration, and high hydrogen ion concentration→ stimulate the chemoreceptor
- When the myometrium relaxes, fresh oxygenated blood has to come into the placental venous sinuses from the maternal spiral arterioles to gradually“wash out” the accumulated chemicals from their respective chemoreceptors
- Past term: late deceleration
- Related to acidosis

QUICKLIE

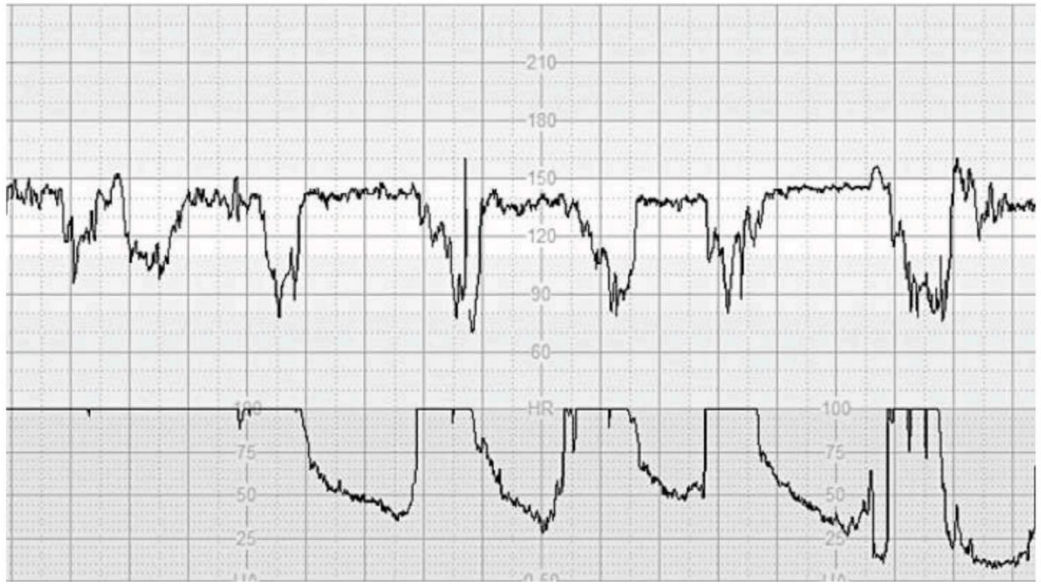


Fig. 1. "Quicklie" Deceleration.

TARDY

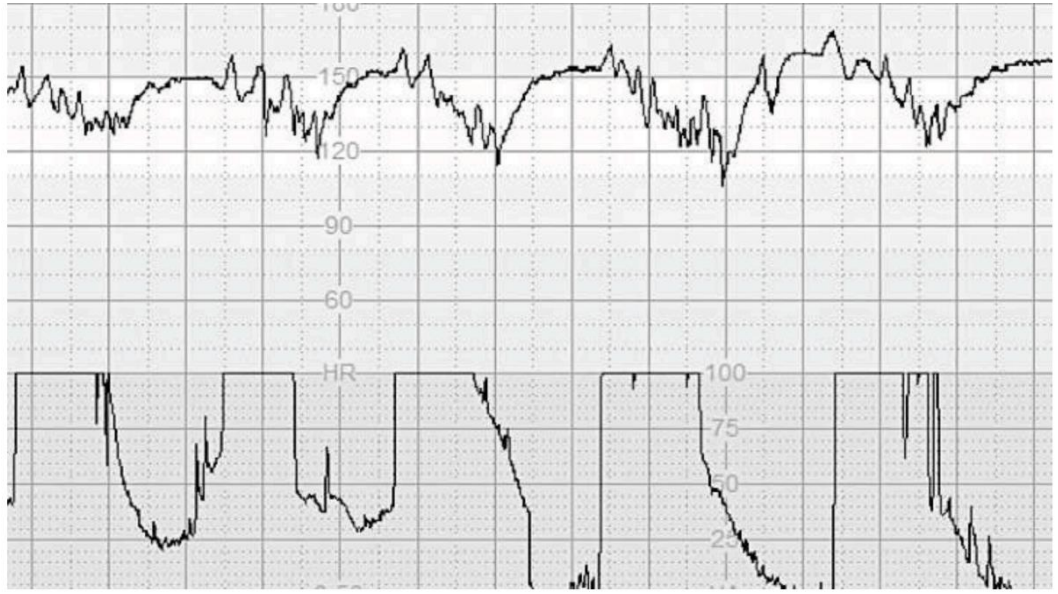


Fig. 2. "Tardy" Deceleration.

1. excluding features of chronic hypoxia and preexisting fetal compromise on the CTG trace at the beginning of recording

The chronic hypoxia checklist: Is this fetus fit to undertake the journey of labor?

	CTG feature	Yes	No
1	Is the baseline FHR "stable," and "appropriate" for the gestational age?		
2	Is there normal variability and cycling?		
3	Are there accelerations?		
4	Are shallow or late decelerations (ie, uteroplacental insufficiency) "absent"?		
5	No evidence of risk factors which may increase fetal compromise (meconium, maternal pyrexia, chorioamnionitis, vaginal bleeding, fetal growth restriction, etc.)		

If the answers to all 5 questions are "yes," then continue CTG monitoring.
If not, immediately seek senior input.

The Fetal Monitoring Checklist" Is THIS Fetus FIT to undertake the progressive hypoxic journey of labour?"

Antenatal CTG Tool The CAUTION checklist to detect Antenatal Fetal Compromise.

Antenatal History:				**Sig 2**
Cycling absent	**YES**	**NO**	Depression of the CNS	
Accelerations absent	**YES**	**NO**	Depression of the somatic NS	
Unstable baseline	**YES**	**NO**	Myocardial decompensation	
Tardy recovery (late decelerations)	**YES**	**NO**	Utero-placental insufficiency	
Irritability of the uterus/ Inappropriate baseline for gestational age	**YES**	**NO**	Potential abruption or chorioamnionitis	
Obvious history: vaginal bleeding, PPROM, reduced fetal movement, abdominal pain	**YES**	**NO**	Underlying pathology that may contribute to fetal compromise	
Non-hypoxic features: Zig-zag pattern or sinusoidal	**YES**	**NO**	Feto-maternal haemorrhage, chronic fetal anaemia or CNS irritability	
Date and time				
Print name and sign	1)		2)	

2. Subtypes of Fetal Hypoxia Ante and Intrapartum

The frequency of composite adverse outcome seems related to the duration and the type of the hypoxic injury, being higher in fetuses showing CTG features of antepartum chronic hypoxia

Before labor

- Chronic hypoxia

Labor starts

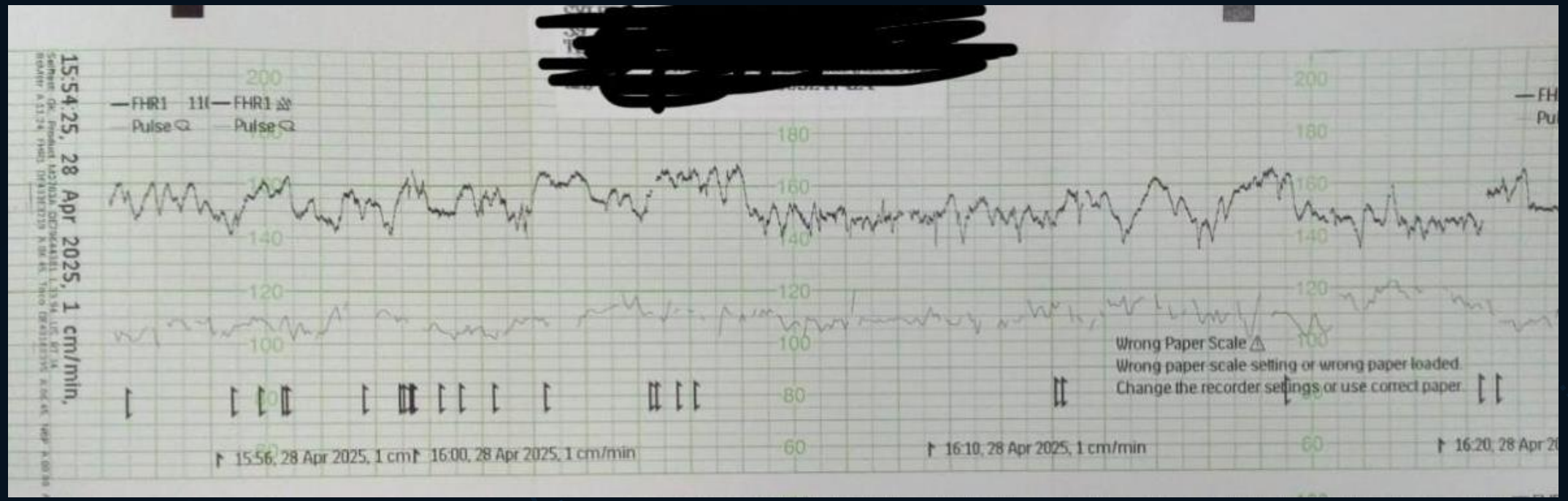
- RUPI-L

Advanced labor

- Acute hypoxia
- Subacute hypoxia
- Gradually evolving hypoxia

Pasquo ED. BJOG. 2022;129:1916–1925. DOI: 10.1111/1471-0528.17133

Ghi T. Acta Obstet Gynecol Scand. 2024;103:1910–1918. DOI: 10.1111/aogs.14937



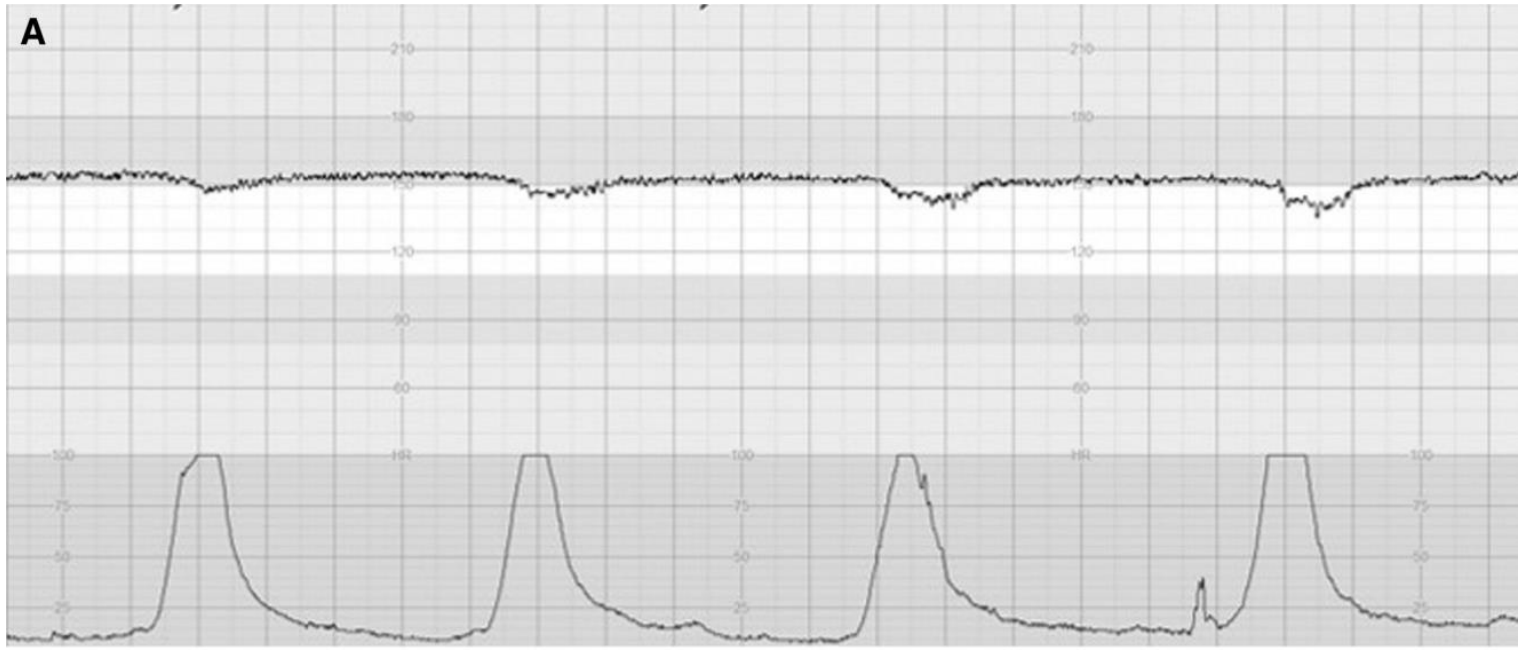
NO HYPOXIA

- Baseline appropriate for gestational age
- Stable
- Normal variability
- Presence of cycling
- No repetitive deceleration

CHRONIC HYPOXIA

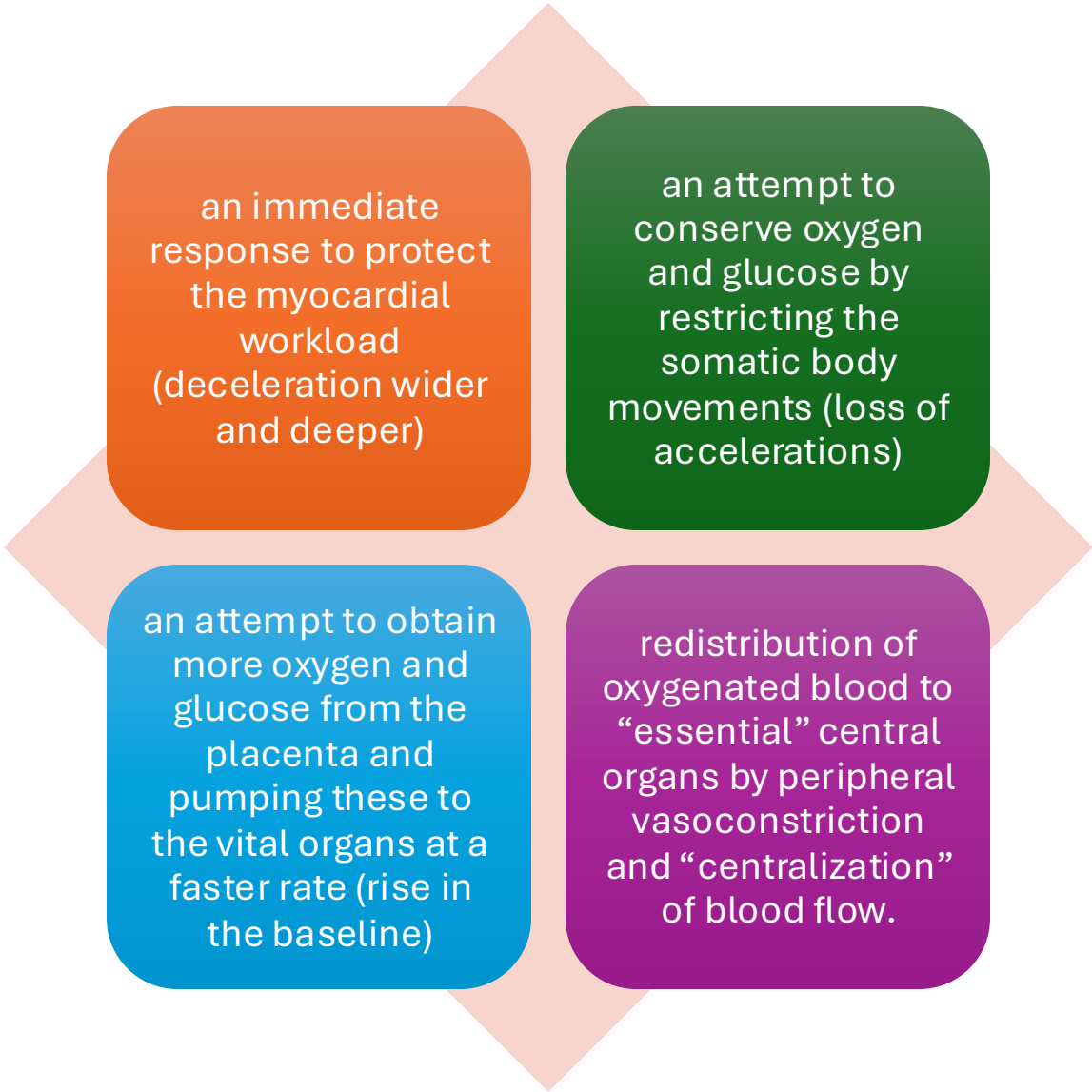
corresponding to exposure of the fetus over a prolonged period to hypoxia →the fetus is unable to withstand any further hypoxic stress → low glycogen reserves & CNS depression

- Higher baseline than expected for gestational age
- Reduced variability and/or absence of cycling
- Absence of accelerations
- Shallow deceleration



E. Chandraharan et al. https://doi.org/10.1016/j.ejogrb.2024.09.034

Gradually Evolving Hypoxia



https://doi.org/10.1016/j.ajog.2022.05.023

Gradually Evolving Hypoxia Pattern

Compensated:	**Decompensated:**
Rise in the baseline (with normal variability and stable baseline) **preceded** by decelerations **and** loss of accelerations, with **inter-deceleration interval greater** than the time spent during decelerations	• **Reduced or increased variability** • Unstable/ progressive **decline in the baseline FHR (step ladder pattern to death)**

E. Chandraharan et al. https://doi.org/10.1016/j.ejogrb.2024.09.034

TABLE 5

Evolution of CTG changes in a gradually evolving hypoxic stress "ABCDE"[7]

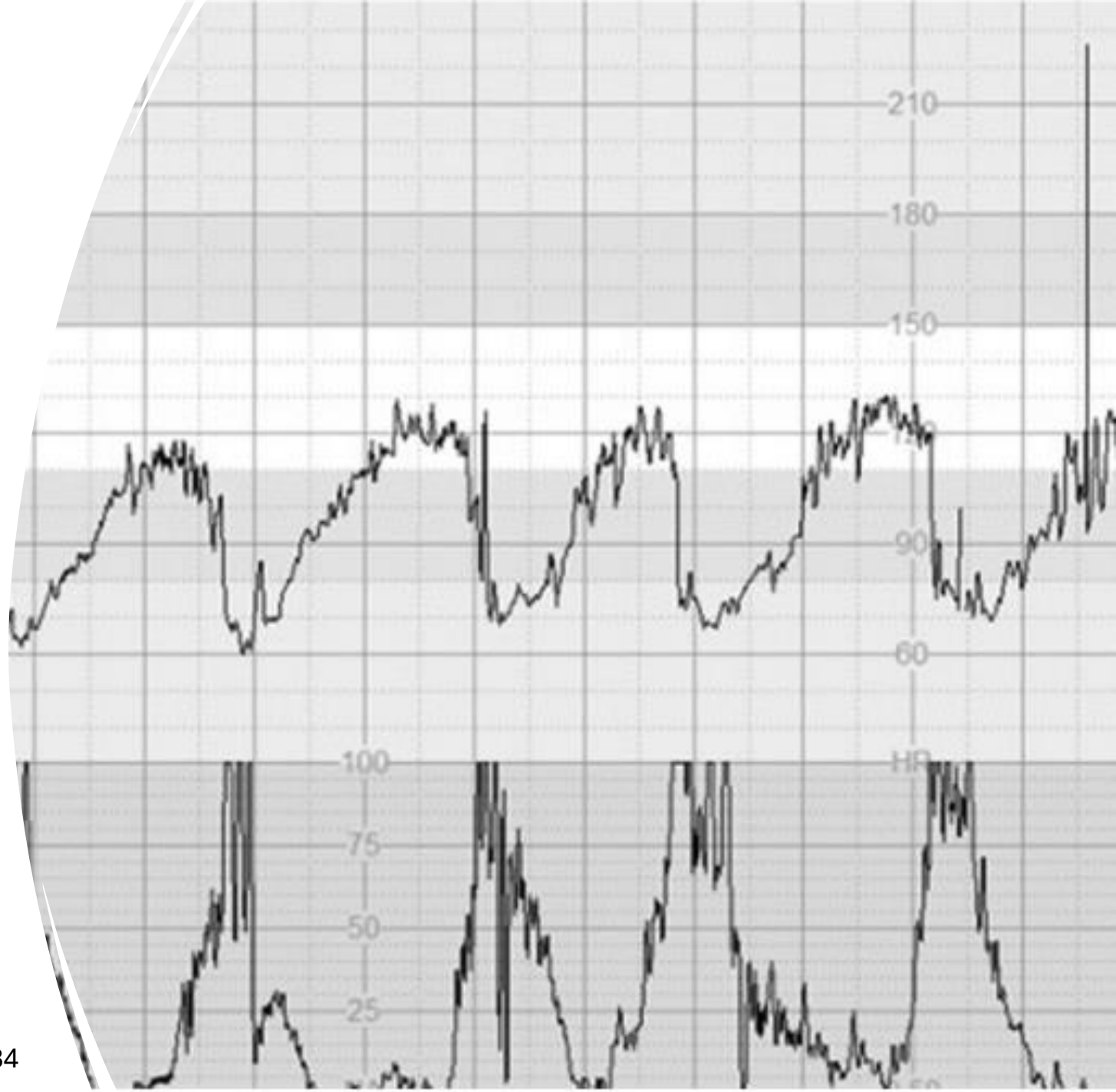
CTG change	Mechanism
Onset of decelerations (and progressive widening and deepening of decelerations as hypoxic stress progressively worsens)	Cardioprotective reflex response to protect the myocardial workload to avoid anaerobic metabolism when oxygen supply is intermittently interrupted
A = loss of accelerations	Restriction of somatic body movements to conserve oxygen and nutrients to ensure continuous supply to "essential" central organs
B = baseline heart rate increases	Release of catecholamine to increase the heart rate to obtain more oxygen and nutrients from the placenta and to perfuse the central organs at a higher rate between the decelerations
C = compensated stress response	Increase in the tissue perfusion and effective redistribution through the catecholamine-mediated compensatory response is sufficient to maintain aerobic metabolism in central organs
D = decompensation	Unstable baseline FHR (myocardial anaerobic metabolism) and/or an abnormal variability (hypoxia to the autonomic nervous system centers in the brain stem)
E = end stage	Myocardial acidosis leading to the "step-ladder" pattern to death

CTG, cardiotocography; *FHR*, fetal heart rate.

Jia. Fetal heart rate tracing interpretation in clinical practice. Am J Obstet Gynecol 2023.

Sub acute hypoxia

- Fetus **spends less time at the normal baseline than during deceleration** (typically <30s at the baseline and > 90s during decelerations)
- Frequently associated with increased variability (the zigzag pattern > 1 minute)
- characterized by the deepening and widening of ongoing decelerations



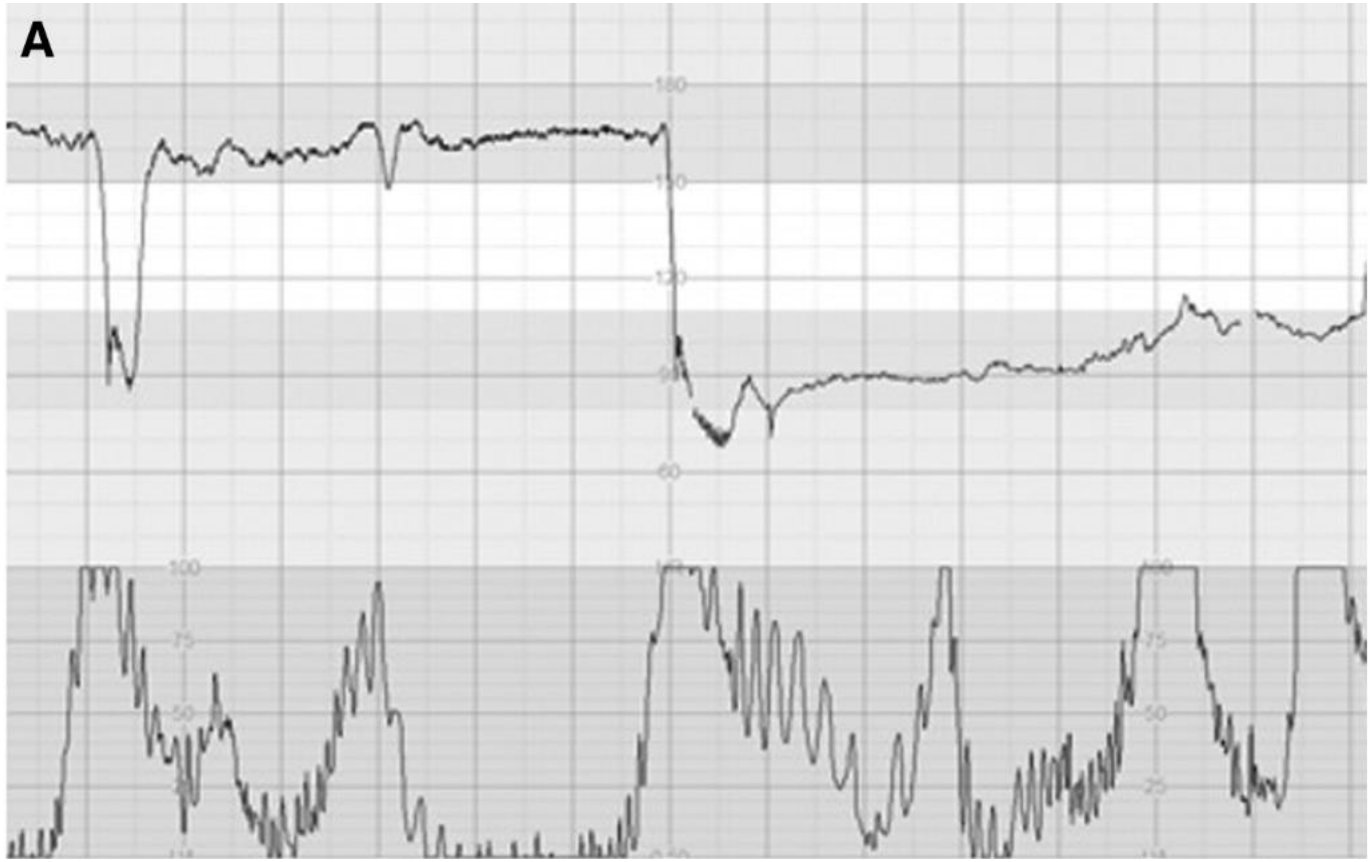
E. Chandraharan et al. https://doi.org/10.1016/j.ejogrb.2024.09.034

Acute Hypoxia

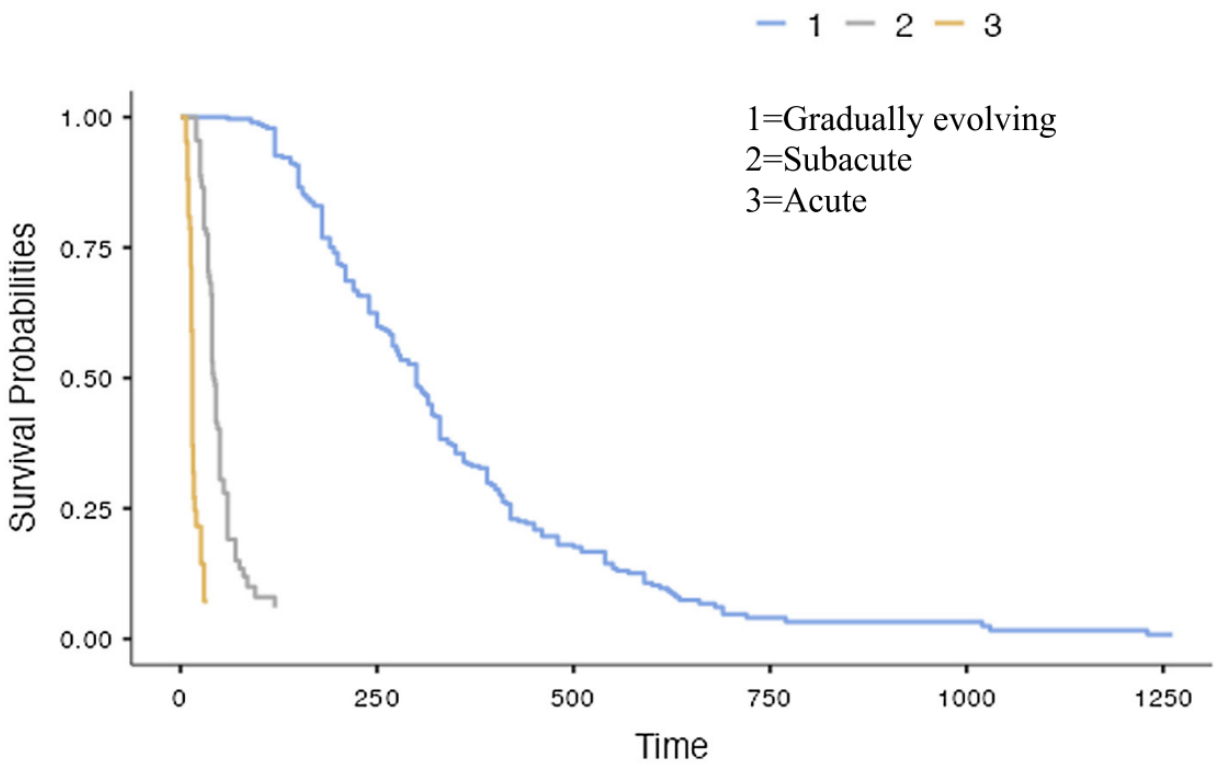
- **Sudden prolonged deceleration** (>3 min)
- Caused by sudden cessation of oxygenation to the fetus and the attempt by the fetus to immediately drop the heart rate to prevent myocardial hypoxia and acidosis
- Preceded by reduced variability and lack of cycling **or** reduced variability within the first 3 minutes → immediate delivery
- Preceded by normal variability & cycling → in 3 minutes, exclude 3 irreversible, correct 3 reversible causes:
  - 3 intrapartum irreversible accidents (umbilical cord prolapse, placental abruption, uterine rupture – if such an accident is suspected prepare for immediate delivery)
  - Correct 3 reversible causes (uterine hyperstimulation, maternal hypotension, sustained umbilical cord compression)
  - If no improvement by 9 min or any of the accidents diagnosed, immediate delivery by the safest and quickest route. Some require birth within 15 min

E. Chandraharan et al. https://doi.org/10.1016/j.ejogrb.2024.09.034

Acute Hypoxia with sudden prolonged deceleration



The intact survival rates according to the duration and the type of intrapartum hypoxic insult



	Median (min)	Range (min)	Time of 100% survival rate (min)
Acute	15	14-17	12
Subacute	42	40-50	20
Gradually evolving	300	275-320	145

Pasquo ED. BJOG. 2022;129:1916–1925. DOI: 10.1111/1471-0528.17133

A 100% intact survival rate was observed below 12 minutes of acute hypoxia,

20 minutes of subacute hypoxia and 145 minutes of gradually evolving hypoxia

THE JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE
2022, VOL. 35, NO. 25, 8498–8506
https://doi.org/10.1080/14767058.2021.1986479

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ORIGINAL ARTICLE

Diagnostic capacity and interobserver variability in FIGO, ACOG, NICE and Chandraharan cardiotocographic guidelines to predict neonatal acidemia

ABSTRACT

Objective: Despite its routine use in intrapartum care, the technique of fetal cardiotocography has some limitations. The aim of this study is to analyze the predictive capacity and interobserver agreement in the latest versions of four international cardiotocography guidelines: Federation of Gynecology and Obstetrics (FIGO), American College of Obstetrics and Gynecology (ACOG), the National Institute for Health and Care Excellence (NICE) and Chandraharan, used to predict neonatal acidemia.

Study design: The last 30 min of 150 cardiotocographic records were analyzed over all the pH ranges and were blindly evaluated by three independent reviewers. The sensitivity, specificity, positive predictive value, negative predictive value, and area under the receiver operating characteristic curve (AUC) were calculated to assess the predictive capacity of each fetal cardiotocographic guideline. The degree of interobserver agreement was evaluated with the Fleiss Kappa coefficient.

Results: Observers found fetal cardiotocography guidelines to have a variable sensitivity and specificity. The Chandraharan classification reached the highest sensitivity (78.79%), while ACOG had the highest specificity (95.73%). On average for the three observers, Chandraharan had the highest discrimination capacity for neonatal acidemia, although this was only moderate (AUC 0.66; 95%CI, 0.55–0.77) and did not differ significantly from the remaining guidelines. The degree of agreement among the three observers, assessed according to the Fleiss Kappa coefficient, was generally acceptable or moderate for all items and classifications, being highest with the FIGO classification ($\kappa = 0.35$; 95%CI, 0.28–0.41) and lowest with the ACOG ($\kappa = 0.23$; 95%CI, 0.16–0.30).

Conclusion: Although all the guidelines have a moderate capacity to predict neonatal acidemia, the Chandraharan guideline has the highest capacity. This follows a different approach from the others in that it relies on interpretations of cardiotocographic traces based on fetal physiology. The degree of interobserver agreement is, in general, acceptable for the four guidelines, and is the highest for FIGO.

Umbilical cord blood gas sampling

"hidden acidosis" occurring within a few seconds after birth

Lactate level increases significantly within 5 minutes unless the syringe is placed in an ice slurry → constant for up to 20-30 minutes

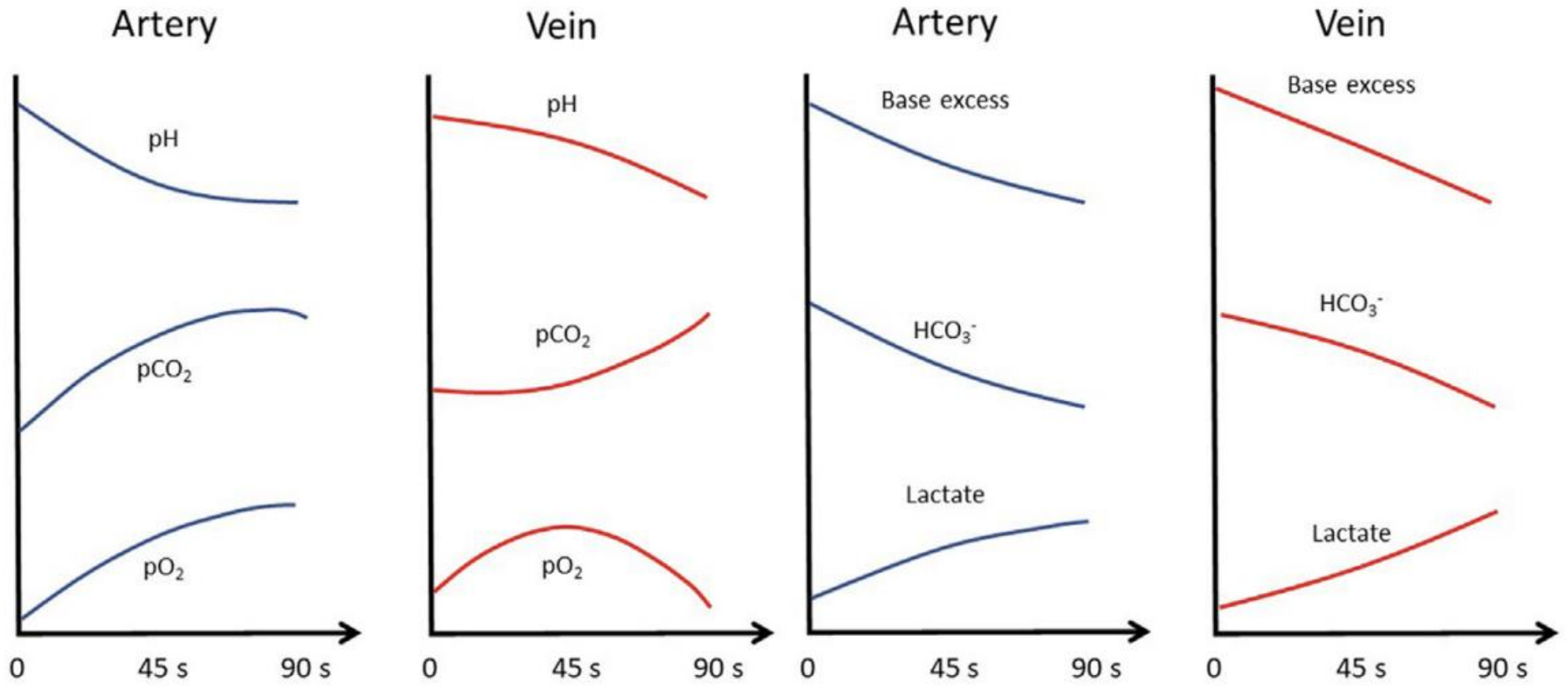
Needle puncture of intact cord vessels immediately after birth and do not postponed analysis longer than 15 minutes

Lactates have been proposed as predictors of the neonatal outcome. Arterial lactates >3.9 mmol/L were significantly more predictive of neonatal morbidity compared with the pH of the cord blood.

Parameter	Umbilical Artery (UA)	Umbilical Vein (UV)
Normal pH	7.20–7.30	7.30–7.40
PaO_2	15–25 mmHg	30–40 mmHg
$PaCO_2$	45–55 mmHg	35–45 mmHg
Base Deficit	0 to–12 mmol/L	0 to–8 mmol/L
Primary Use	Gold standard for fetal acidosis	Reflects placental function

BJOG. 2013 Jul;120(8):996-1002. doi: 10.1111/1471-0528.12234.

Hidden acidosis: acidic metabolites trapped in peripheral tissues flood into cord blood after birth



Changes of umbilical cord blood gases and lactate when blood sampling is delayed with maintained flows in arteries and vein. Note that artery and vein scales are not similar.

BJOG. 2013 Jul;120(8):996-1002. doi: 10.1111/1471-0528.12234.

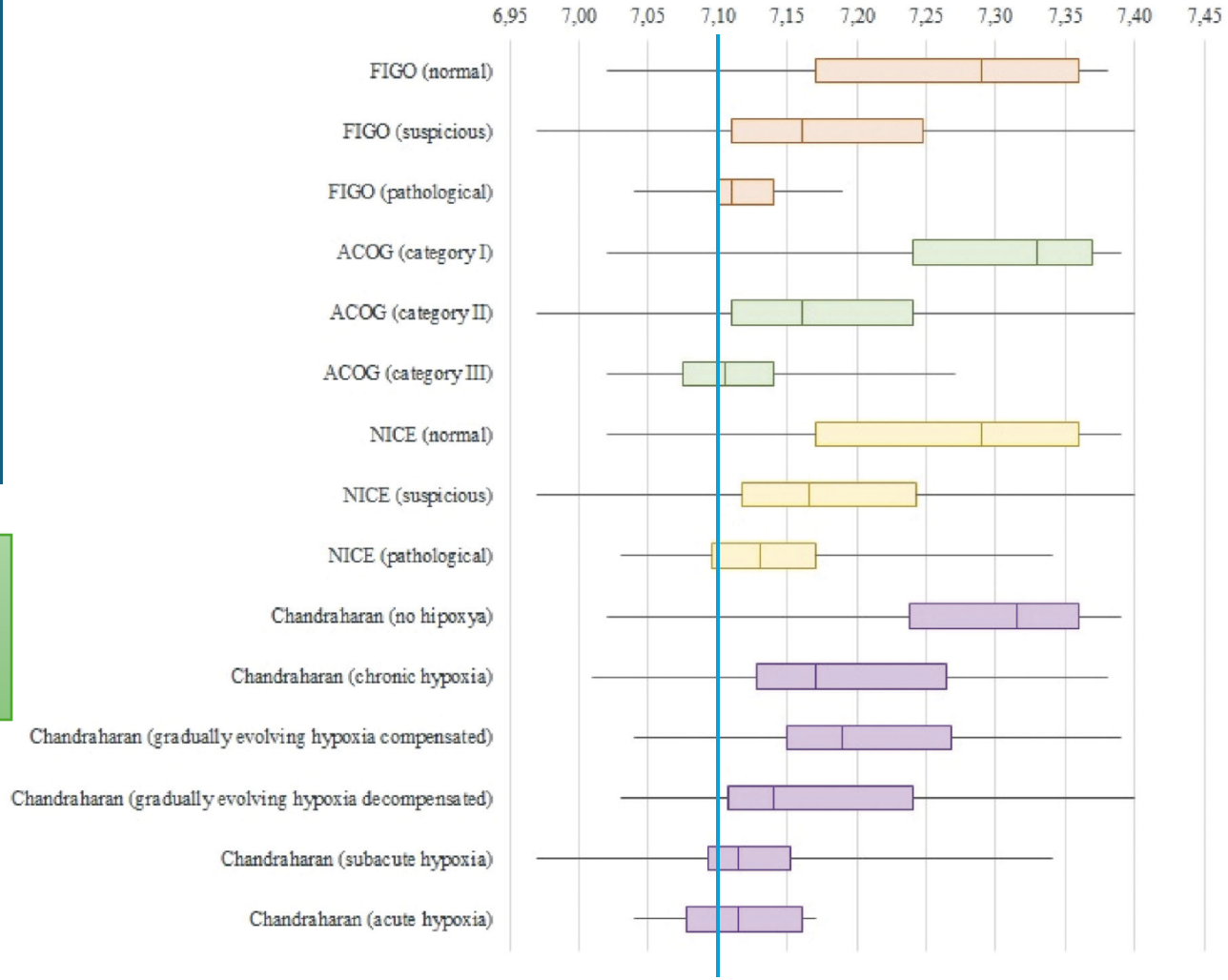
ACOG recommends umbilical cord blood acid-base analysis in the following situations:

- Cesarean delivery for fetal compromise
- Low 5-minute Apgar score
- Severe growth restriction
- Abnormal FHR tracing
- Maternal thyroid disease
- Intrapartum fever
- Multifetal gestations

The threshold PH for increased risk of adverse neurologic outcomes is 7.10
The ideal umbilical cord artery PH is 7.26 – 7.30

American College of Obstetricians and Gynecologists, American Academy of Pediatrics. Neonatal encephalopathy and neurologic outcome. Pediatrics. 2014 Mar;133(5):e1482-8.

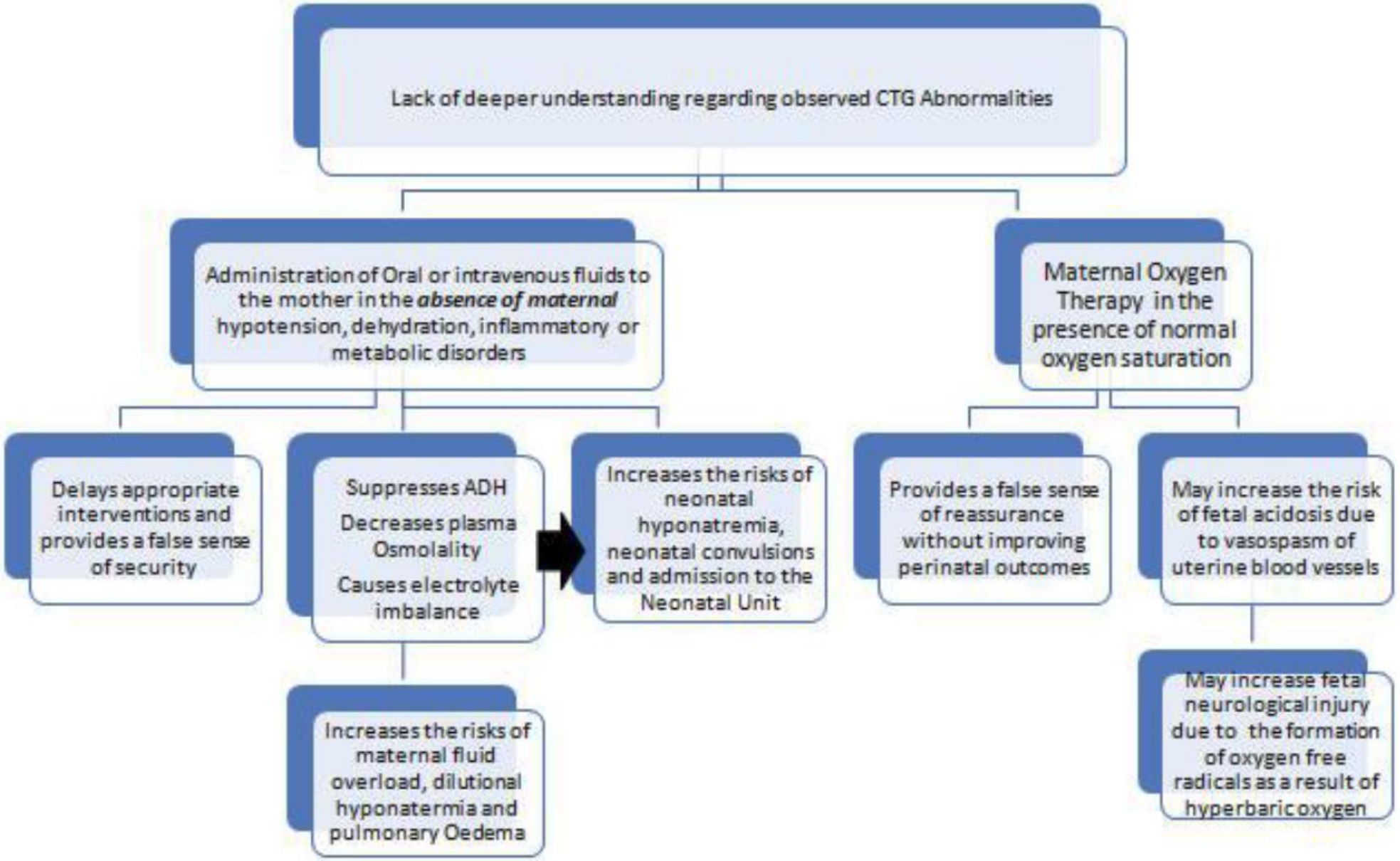
Boxplot of the median PH and its interquartile ranges of the different categories and guidelines studied



THE JOURNAL OF MATERNAL-FETAL & NEONATAL MEDICINE 2022, VOL. 35, NO. 25, 8498–8506https://doi.org/10.1080/14767058.2021.1986479

Intrauterine treatment for varian fetal heart rate patterns

Causes	Possible Resulting FHR Patterns	Corrective Maneuver	Mechanism
Hypotension (e.g., supine hypotension, conduction anesthesia)	Bradycardia, late decelerations	Intravenous fluids, position change, ephedrine	Return of uterine blood flow to normal
Excessive uterine activity	Bradycardia, late decelerations	Decrease in oxytocin, lateral position	Return of uterine blood flow to normal
Transient umbilical cord compression	Variable decelerations	Change in maternal position (e.g., left or right lateral, Trendelenburg) Amnioinfusion	Presumably removes fetal part from cord Relieves compression of cord
Head compression	Early or variable decelerations	Push only with alternate contractions	Allows fetal recovery
Decreased uterine blood flow associated with uterine contraction	Late decelerations	Change in maternal position (e.g., left lateral, Trendelenburg) Tocolytic agents (e.g., terbutaline)	Enhanced uterine blood flow toward optimum Decreased contractions or tone
Prolonged asphyxia	Decreasing FHR variability[a]	Change in maternal position (e.g., left lateral, Trendelenburg), establishment of maternal hyperoxia	Enhanced uterine blood flow toward optimum, increase in maternal-fetal oxygen gradient


Lack of deeper understanding regarding observed CTG Abnormalities
Administration of Oral or intravenous fluids to the mother in the absence of maternal hypotension, dehydration, inflammatory or metabolic disorders
Maternal Oxygen Therapy in the presence of normal oxygen saturation
Delays appropriate interventions and provides a false sense of security
Suppresses ADH
Decreases plasma Osmolality
Causes electrolyte imbalance
Increases the risks of neonatal hyponatremia, neonatal convulsions and admission to the Neonatal Unit
Increases the risks of maternal fluid overload, dilutional hyponatermia and pulmonary Oedema
Provides a false sense of reassurance without improving perinatal outcomes
May increase the risk of fetal acidosis due to vasospasm of uterine blood vessels
May increase fetal neurological injury due to the formation of oxygen free radicals as a result of hyperbaric oxygen

Administration of fluids to the mother to correct abnormal FHR changes

- Maternal fluids should only be administered to correct abnormalities in the maternal circulation and should not be administered to correct fetal heart rate abnormalities.
- Intravenous fluids should be administered to the mother only in cases of a prolonged deceleration and/or fetal bradycardia secondary to maternal hypotension to restore maternal blood volume
- They may be part of treatment of maternal sepsis or any other medical condition (e.g. diabetic ketoacidosis), which necessitates the administration of fluids to ensure maternal well-being
- Oral fluids are only indicated in a case of reactive fetal tachycardia secondary to maternal dehydration

Journal of Advances in Medicine and Medical Research

32(8): 10-16, 2020; Article no.JAMMR.57653
ISSN: 2456-8899
(Past name: British Journal of Medicine and Medical Research, Past ISSN: 2231-0614, NLM ID: 101570965)

Maternal "Oxygen and Fluids Therapy" to Correct Abnormalities in the Cardiotocograph (CTG): Scientific Principles vs Historical *(Mal)* Practices

Edwin Chandraharan[1*]

[1]*Global Academy of Medical Education and Training, London, UK.*

Maternal oxygen supplementation to treat fetal heart rate abnormalities

- It was not recommended in the first edition of the international expert consensus guidelines on physiological interpretation of CTG in 2018.
- Routine use of oxygen supplementation in individuals with normal oxygen saturation is not recommended for fetal intrauterine resuscitation
- Maternal oxygen or fluid therapy to correct fetal heart rate abnormalities is no longer recommended in clinical practice
- Maternal oxygen supplementation is recommended in all clinical situations where administration of oxygen is essential to ensure maternal wellbeing (e.g., bronchial asthma, maternal sepsis, maternal cardiopulmonary disorders etc)

Journal of Advances in Medicine and Medical Research

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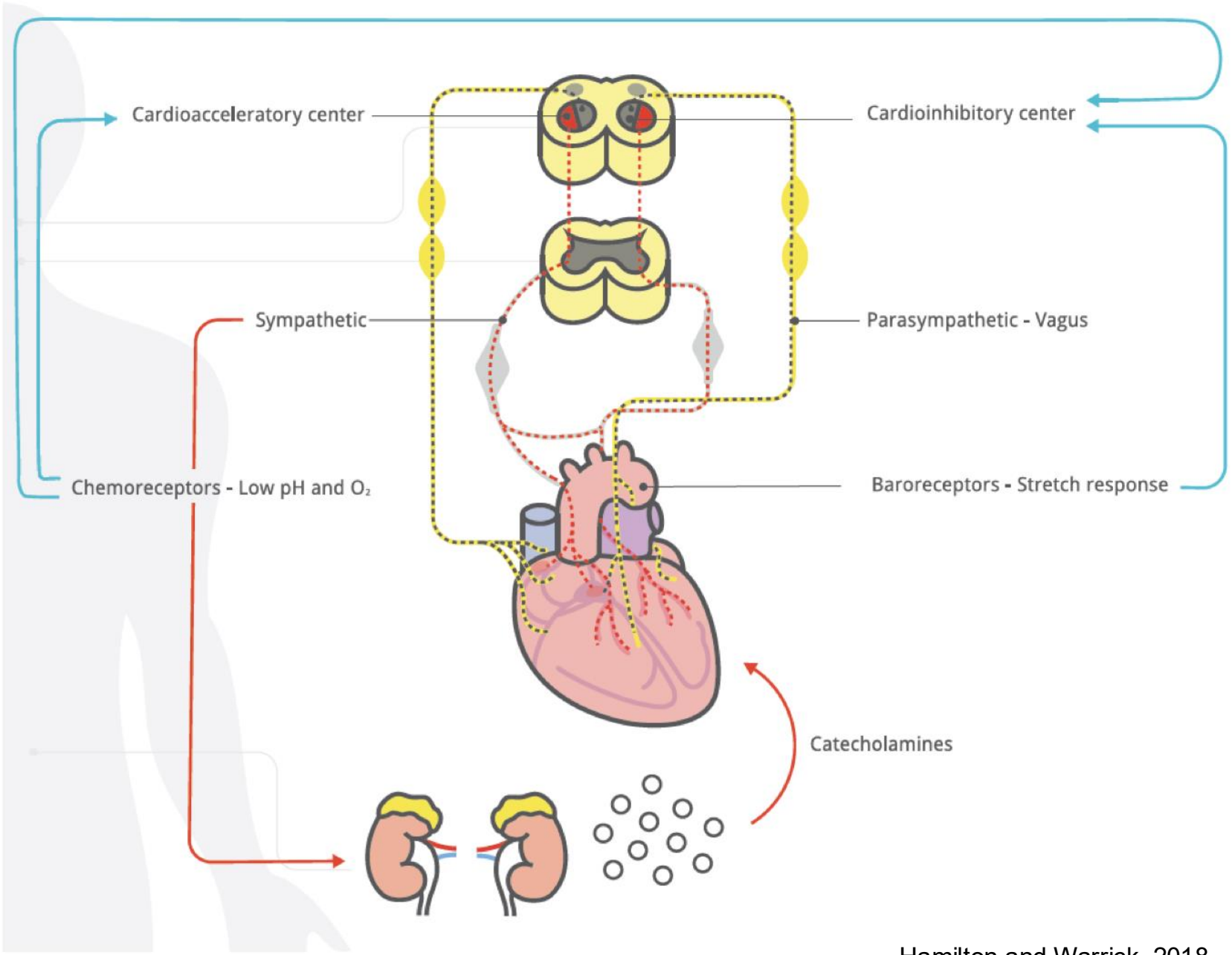
Take home message:

- *Try to understand physiology **not** just remember the pattern*
- *Do not prolong the intrapartum hypoxic insult due to waiting for the worst CTG pattern*
- *The sequence of FHR modifications was variable and could be one of the limits of FHR analysis to predict neonatal acidemia*
- *Consider the overall clinical context, including background maternal and fetal risk factors, also the progress of labor, to make a clinical decision*

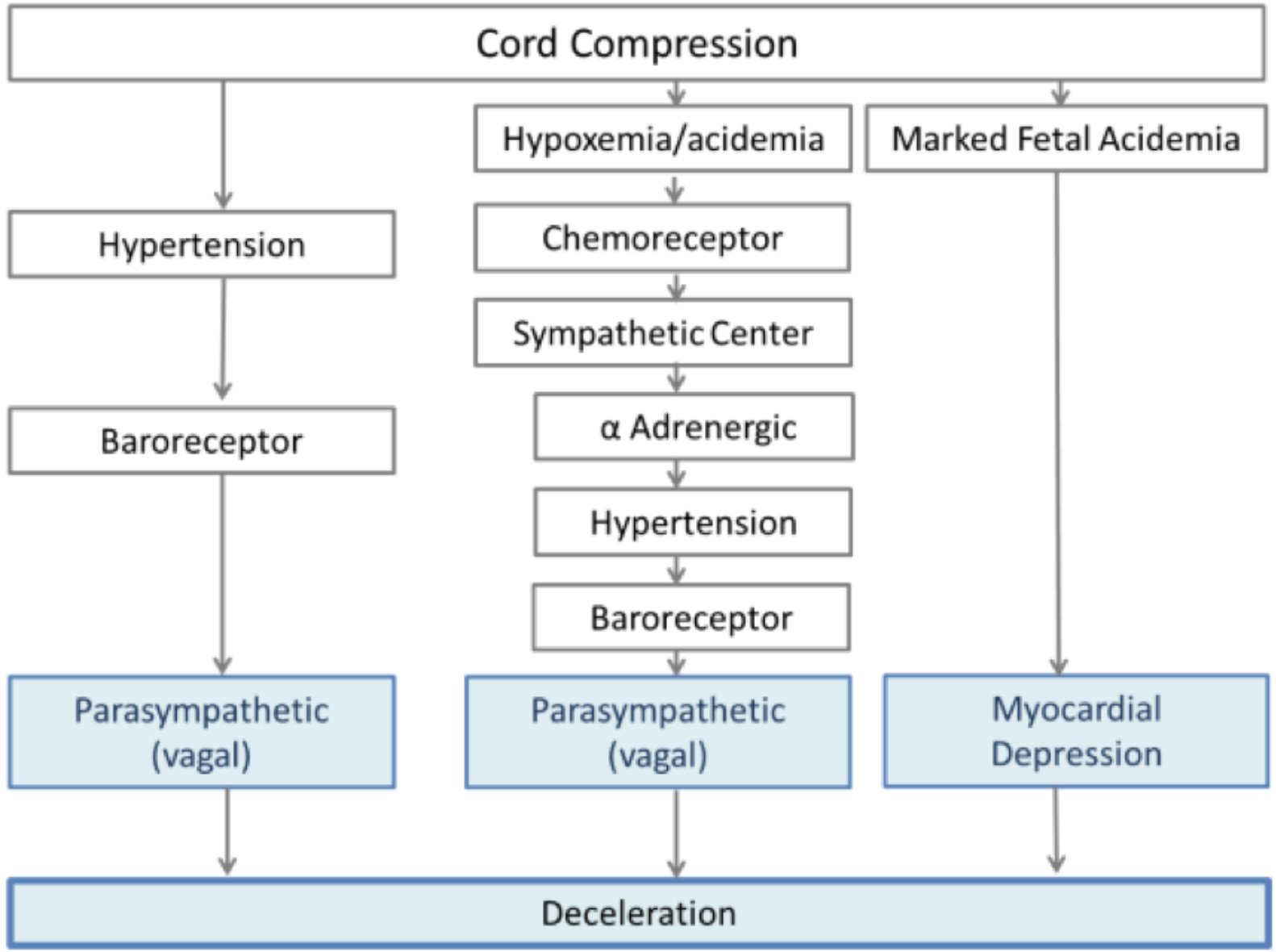
Thank You

PATOPHYSIOLOGY BEHIND ABNORMAL CTG

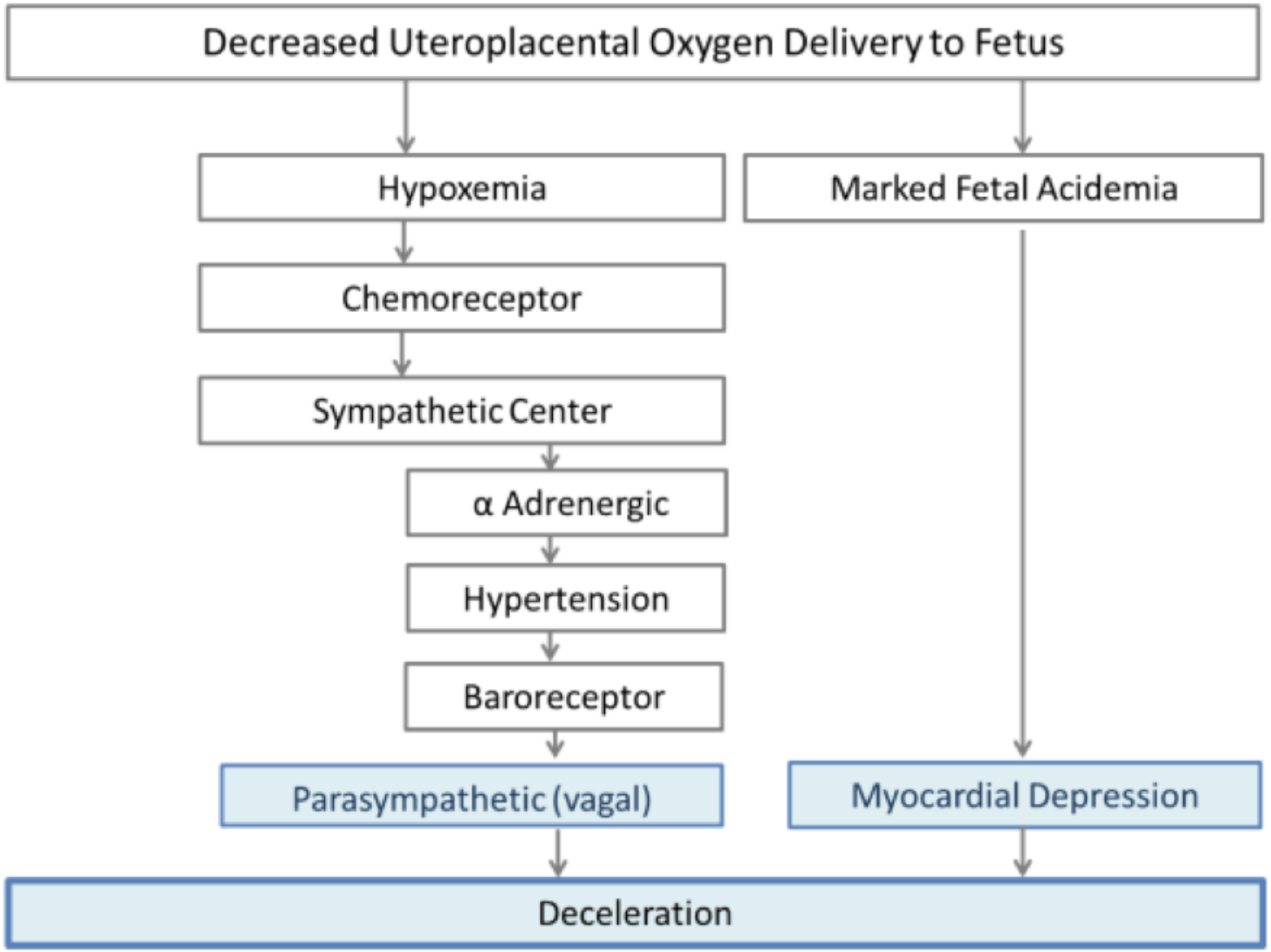


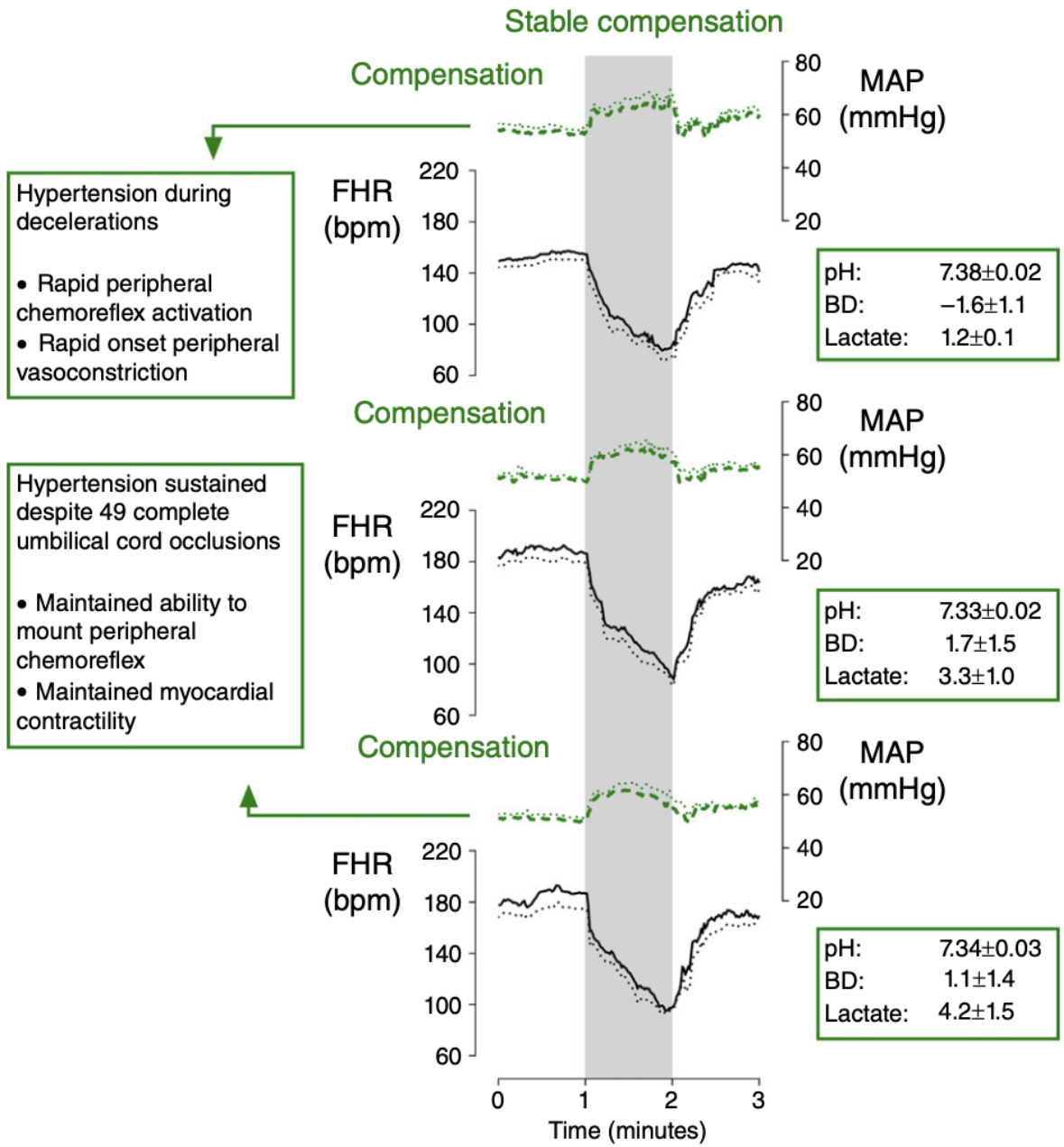
Hamilton and Warrick, 2018

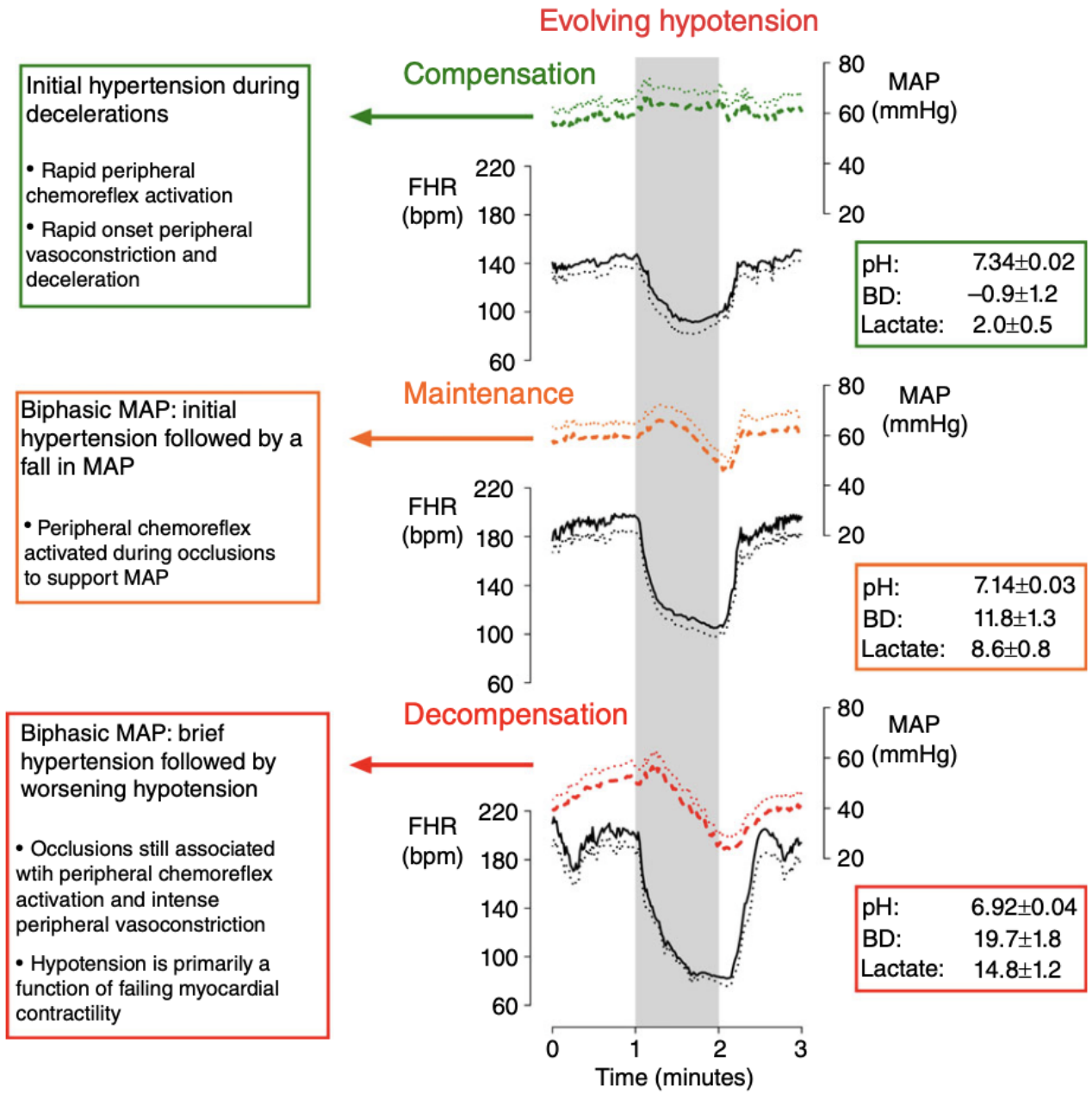
Variable Deceleration



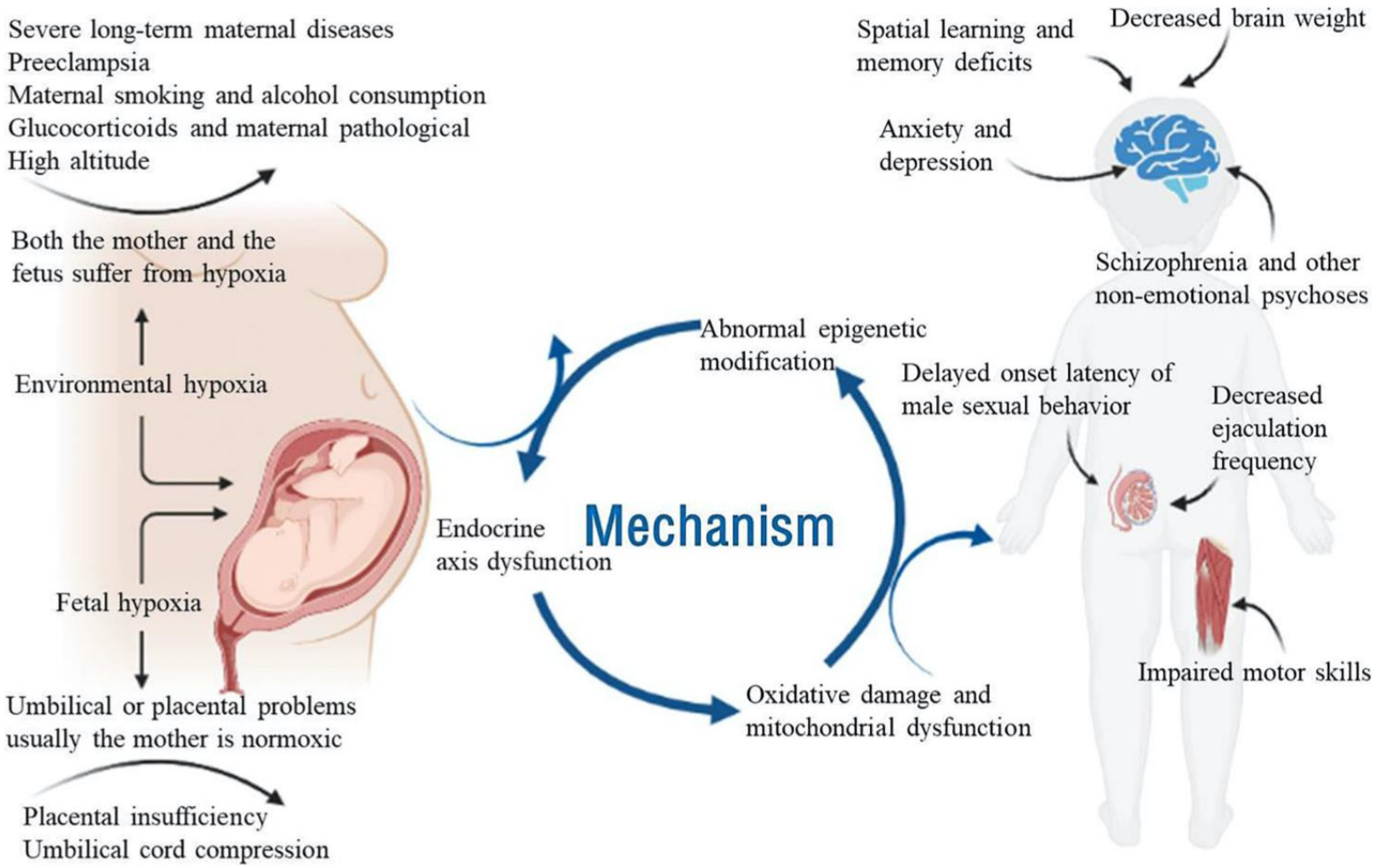
Late Deceleration




Stable compensation
Compensation
Hypertension during decelerations
• Rapid peripheral chemoreflex activation
• Rapid onset peripheral vasoconstriction
FHR (bpm)
220
180
140
100
60
MAP (mmHg)
80
60
40
20
pH: 7.38±0.02
BD: −1.6±1.1
Lactate: 1.2±0.1
Compensation
Hypertension sustained despite 49 complete umbilical cord occlusions
• Maintained ability to mount peripheral chemoreflex
• Maintained myocardial contractility
pH: 7.33±0.02
BD: 1.7±1.5
Lactate: 3.3±1.0
Compensation
pH: 7.34±0.03
BD: 1.1±1.4
Lactate: 4.2±1.5
0
1
2
3
Time (minutes)


Evolving hypotension
Compensation
Initial hypertension during decelerations
• Rapid peripheral chemoreflex activation
• Rapid onset peripheral vasoconstriction and deceleration
FHR (bpm)
220
180
140
100
60
MAP (mmHg)
80
60
40
20
pH: 7.34±0.02
BD: –0.9±1.2
Lactate: 2.0±0.5
Maintenance
Biphasic MAP: initial hypertension followed by a fall in MAP
• Peripheral chemoreflex activated during occlusions to support MAP
pH: 7.14±0.03
BD: 11.8±1.3
Lactate: 8.6±0.8
Decompensation
Biphasic MAP: brief hypertension followed by worsening hypotension
• Occlusions still associated wtih peripheral chemoreflex activation and intense peripheral vasoconstriction
• Hypotension is primarily a function of failing myocardial contractility
pH: 6.92±0.04
BD: 19.7±1.8
Lactate: 14.8±1.2
0
1
2
3
Time (minutes)

Schematic diagram of the effects of prenatal hypoxia on neurological development and related diseases [Wang et al., 2027]



Species	Method	Offspring influence	References
Rat	Exposure to acute hypoxia on day 14 of pregnancy	The number of synaptopodin-positive dendritic spines was reduced, learning and memory deficits	Vasilev et al., 2016; Zhuravin et al., 2019
Rat	Uterine artery ligation on the 16th day of pregnancy	Pax6 immunoreactivity showed diverse patterns in the neurogenic zone	So et al., 2017
Rat	Pregnant female rats were injected with 5, 25, and 50 mg/kg sodium nitrite	Impaired spatial memory	Sosedova et al., 2019
Rat	Unilateral ligation of uterine artery at E17	White and gray matter damage, myelin loss, motor, sensorimotor and short-term memory deficits	Delcour et al., 2012b
Rat	Clamping of the uterine vascular system of pregnant rats at 17 days gestation for 30 min	Learning deficit	Cai et al., 1999

Wang et al., 2021

Species	Method	Offspring influence	References
Sheep	The fetus was intubated through the umbilical cord vasculature to a pumpless extracorporeal oxygenator for 22 ± 2 days in mid-gestation.	Increase of white matter vessels, the decrease of neuronal density and the damage of myelination	Lawrence et al., 2019
Sheep	Fetal lambs (111 ± 3 days) were oxygen delivery was limited by the umbilical circuit oxygenator maintained in the artificial womb for a mean of 22 ± 6 days	Altered cerebrovascular resistances and loss of brain mass	McGovern et al., 2020
Sheep	Pregnant ewes undergo a sterile procedure between 88 and 92 days of gestational age to control blood flow to the brain through the common carotid artery and vertebral artery.	Destroy the dendration and activity of neurons in the subplate of preterm fetal sheep	McClendon et al., 2017

Wang et al., 2021

Species	Method	Offspring influence	References
Human	Autopsy of the human neonate to perinatal hypoxia/ischemia	Neurological and/or cognitive deficits, dopaminergic neurotransmitter dysfunction	Giannopoulou et al., 2018
Human	Not mentioned	Cognitive disorders, Alzheimer's disease	Nalivaeva et al., 2018
Human	Not mentioned	Attention-deficit/hyperactivity disorder	Getahun et al., 2013; Owens and Hinshaw, 2013

CASE BASED DISCUSSION SESSION

Ny A, 27 tahun

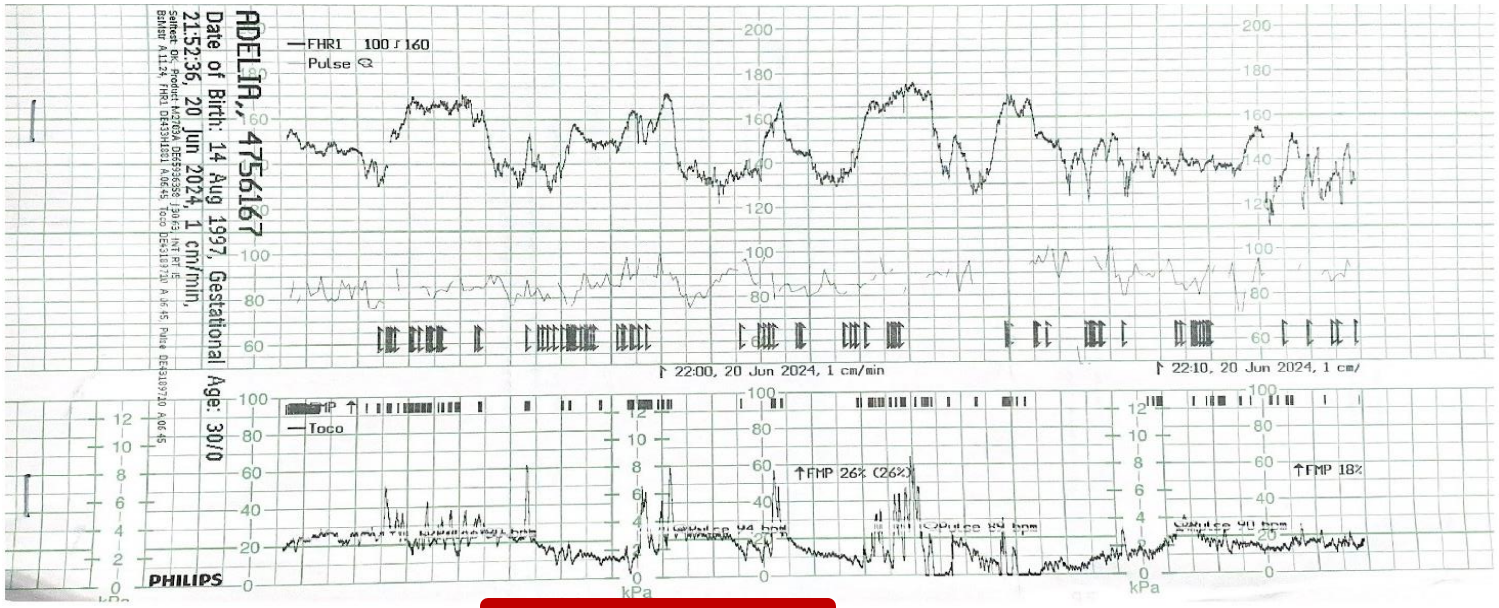
S: Pasien datang dengan keluhan mulas-mulas sejak 12 jam SMRS, Keluhan mulas-mulas dirasakan semakin sering sejak 12 jam SMRS. Keluar lendir darah atau air-air disangkal. Gerak janin dirasakan aktif.

O: BP 115/77 mmHg, HR 84 bpm, RR 20 x/min, T 36.5 C;
FH 31 cm, FHR 140 bpm, contraction irregular;
Io: portio licin, OUE tertutup, fluxus (-), fluor (-);
VT: portio posterior, kenyal, OUE tertutup

Ultrasound
BPD/HC/AC/FL 93/344/327/71
TBJ 3117 g, ICA 7
SDAU 2.25, CPR>1
PIMCA 0.9, PIAU 0.7
Kesimpulan :
Hamil sesuai 39 minggu (serial). Aktivitas dan pertumbuhan janin baik. Air ketuban berkurang. Tidak tampak tanda hipoperfusi janin.

G1 40 weeks of gestational age, twin pregnancy (fetal A head presentation, fetal B vanished twin), DCDA, diminished amniotic fluid without ROM (AFI 7), not in labor

Laboratorium
DPL 12.7/37/7450/267000//90/30/34
PT/aPTT 0.83x/0.88x
Ur/Cr/eGFR 15/0.50/132
SGOT/PT 30/24
SI/TIBC/Sat transferin 117/399/29
Na/K/Cl 135/3,7/105,4
GDS 61 TSH/FT4 0.736/1.08
USG Akut (20/06/24)
JPKTH, plasenta di corpus anterior, DJJ 152 dpm
Prolaktin 200

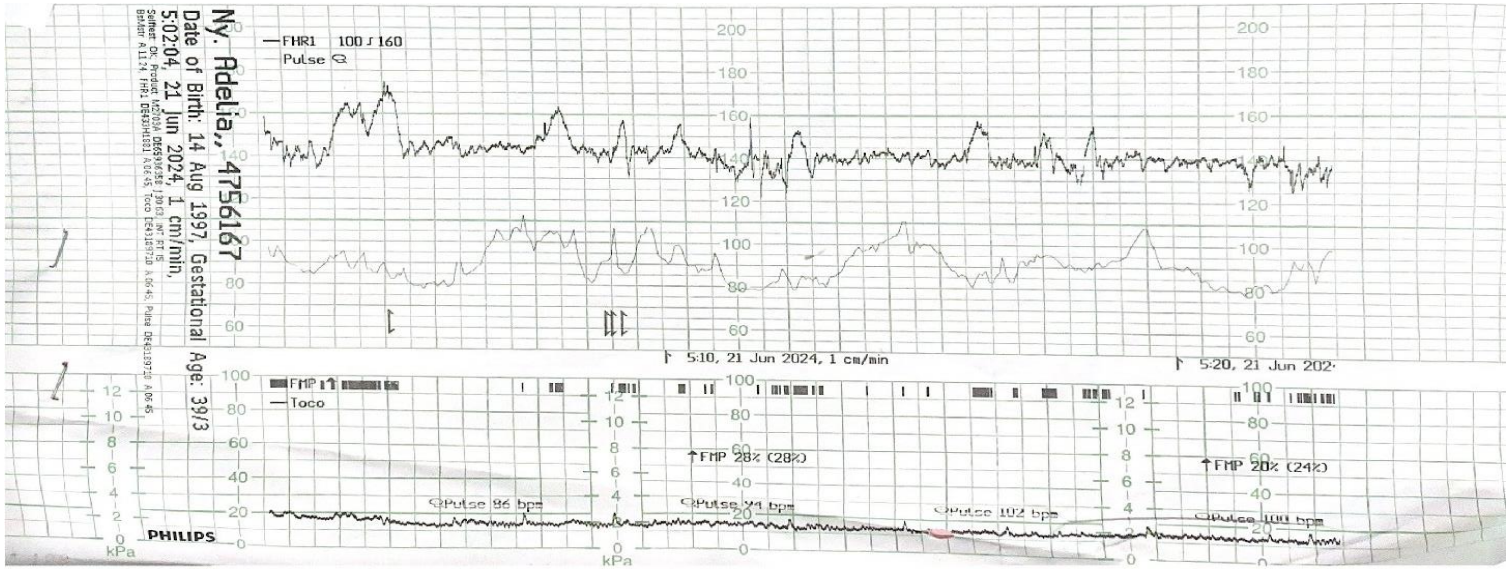


20 Juni 21:52

Induction of labor with Misoprostol 25 mcg/4 jam (1st Dose)

VT: Portio kenyal, posterior, OUE tertutup, tebal 3 cm (PS 0)

Baseline: 140 dpm
Variabilitas: Moderate
Akselerasi; 5x/10 menit
Deselerasi: Variabel 1x
Kontraksi: Ireguler
Gerak Janin: aktif
Kesan: CTG kategori I

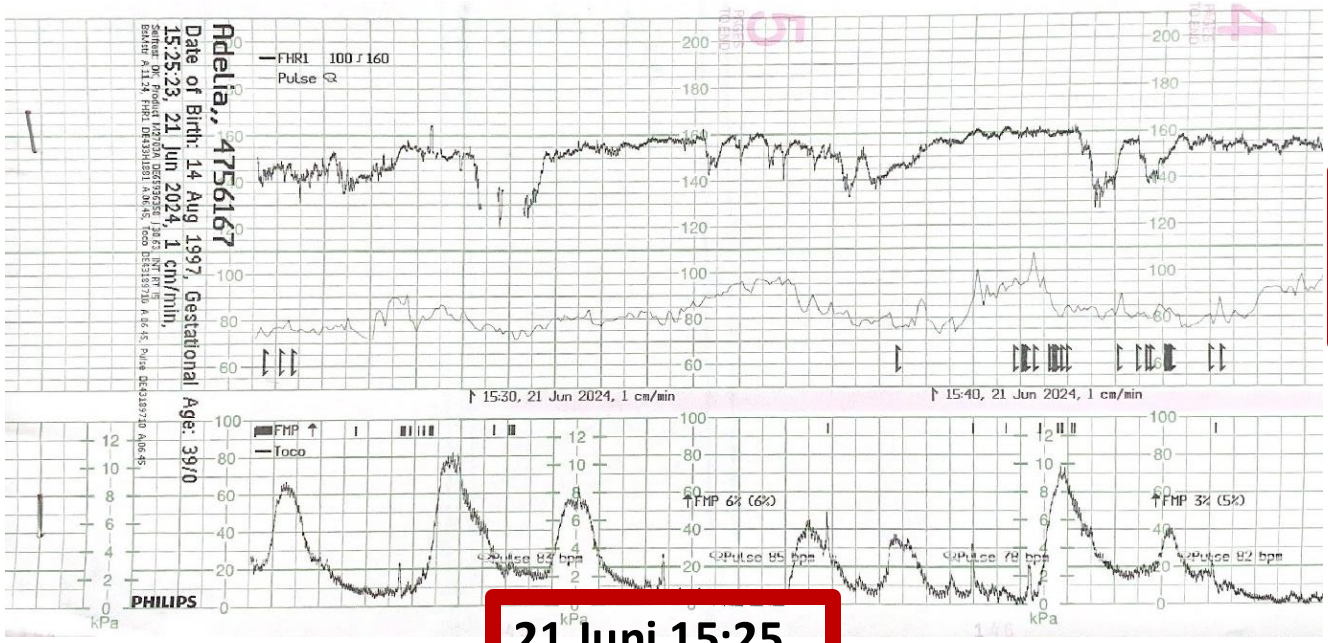


21 Juni 05:24

Induction of labor with Misoprostol 25 mcg/4 jam (3rd Dose)

VT: Portio kenyal, posterior, OUE tertutup, tebal 3 cm (PS 0)

Baseline: 140 dpm
Variabilitas: Moderate
Akselerasi; 3x/10 menit
Deselerasi: Variabel 1x
Kontraksi: Tidak ada
Gerak Janin: aktif
Kesan: CTG kategori I

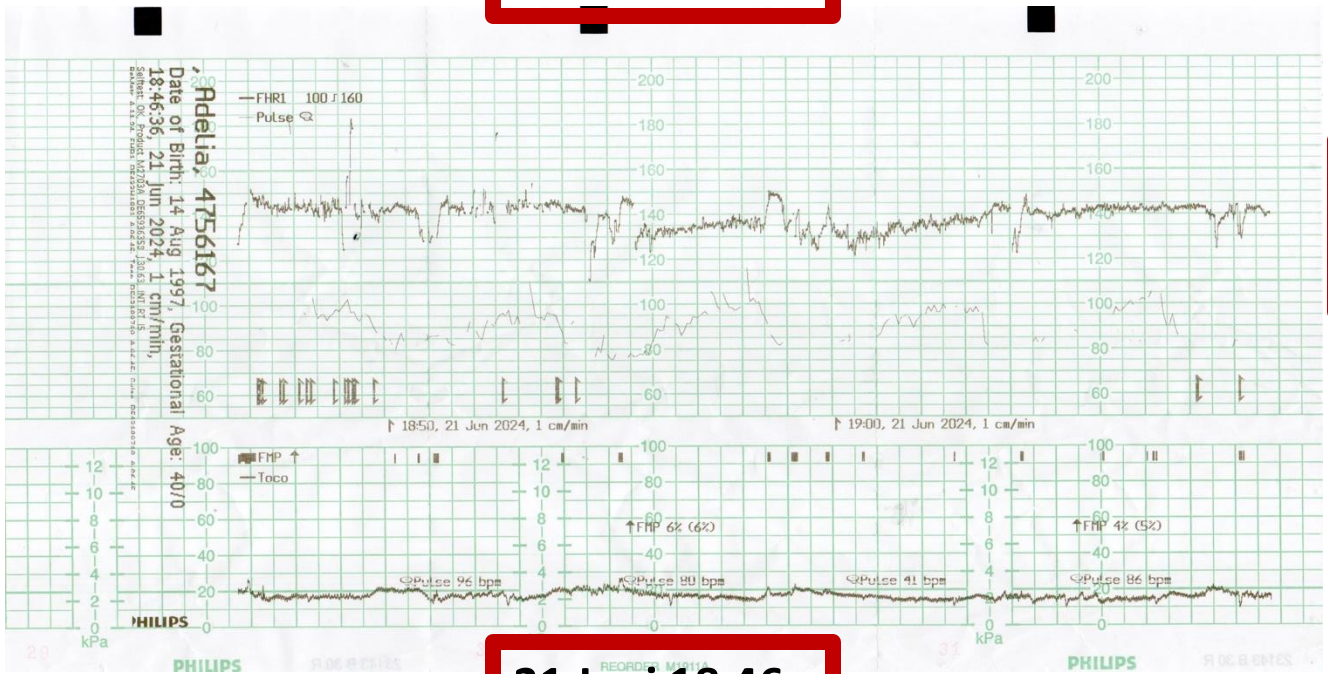


21 Juni 15:25

Induction of labor with Misoprostol 25 mcg/4 jam (5th Dose)

VT: Portio lunak, posterior, OUE tertutup, tebal 3 cm (PS 1)

Baseline: 160 dpm
Variabilitas: Moderate
Akselerasi; 0x/10 menit
Deselerasi: 3x Deselerasi Lambat
Kontraksi: Ireguler
Gerak Janin: aktif
Kesan: CTG kategori II

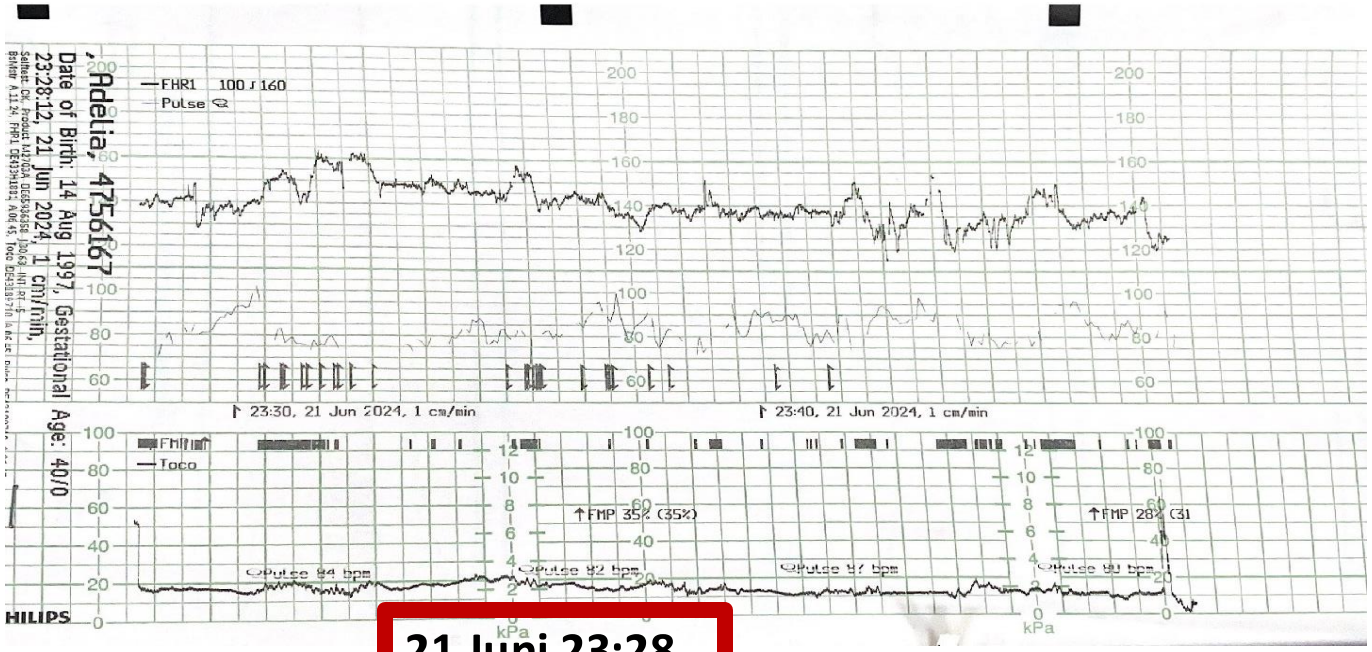


21 Juni 18:46

Induction of labor with Misoprostol 25 mcg/4 jam (6th Dose)

VT: Portio lunak, posterior, OUE tertutup, tebal 3 cm (PS 1)

Baseline: 160 dpm
Variabilitas: Moderate
Akselerasi; 2x/10 menit
Deselerasi: Tidak ada
Kontraksi: Ireguler
Gerak Janin: aktif
Kesan: CTG kategori I

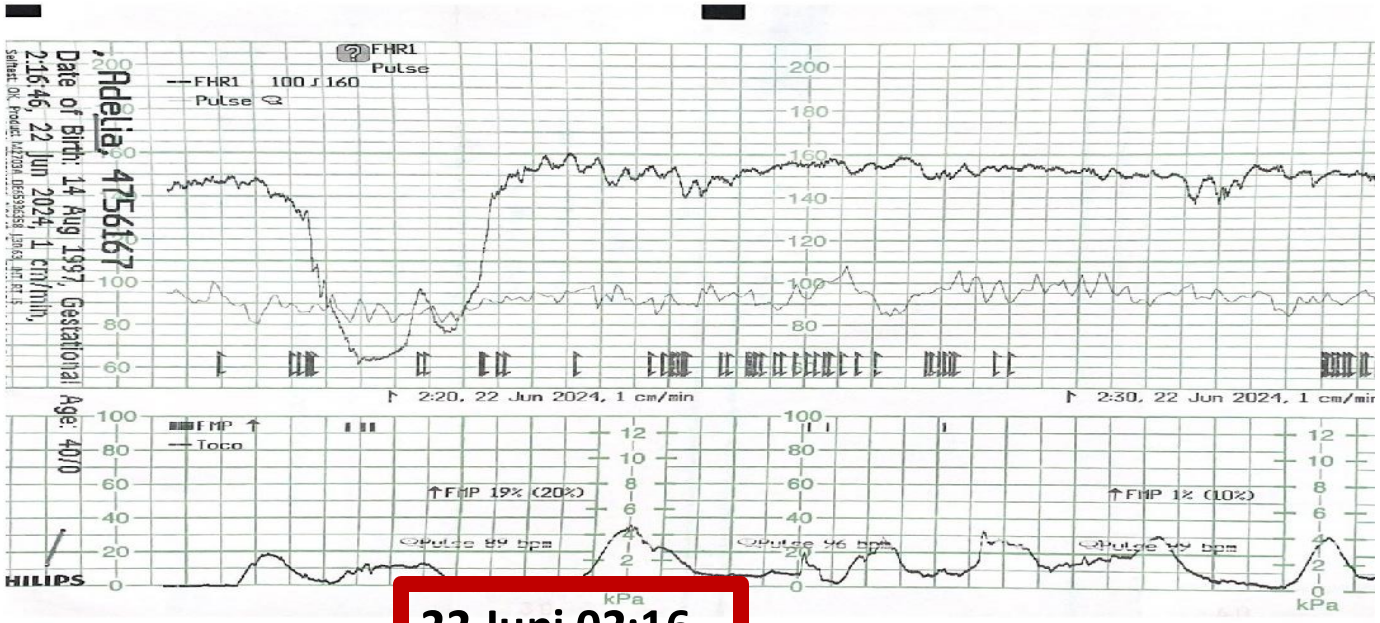


21 Juni 23:28

Induction of labor with Misoprostol 50mcg/4 jam (7th Dose)

VT: Portio lunak, posterior, OUE tertutup, tebal 3 cm (PS 1)

Baseline: 140 dpm
Variabilitas: Moderate
Akselerasi; 5x/10 menit
Deselerasi: Tidak ada
Kontraksi: Tidak ada
Gerak Janin: aktif
Kesan: CTG kategori I



22 Juni 02:16

CTG Interpretation :
Kategori III (Prolonged deseleration dengan riwayat late deseleration)

Baseline: 160 dpm
Variabilitas: Moderate
Akselerasi; 0x/10 menit
Deselerasi: Prolonged deseleration Kontraksi: Ireguler
Gerak Janin: aktif
Kesan: CTG kategori III

Clinical Decision?

Pertanyaan

1. Sampai pada tahap manakah dalam kasus ini proses induksi masih dapat dilakukan?
2. Apakah jika dilakukannya resusitasi intrauterine akan menunda rencana C-Section?

Ny Y, 29 tahun

Fetal distress on G1 37 weeks of gestational age, singleton live head presentation, oligohydramnios without rupture of membrane (AFI 5), mother with Sjogren syndrome ESSDAI 2, cervicitis on therapy, iron depletion (ferritin 16)

S: Pasien mengeluhkan mulas sejak 12 jam yang lalu disertai nyeri perut hilang timbul. Keluhan lain seperti keluar air-air disangkal. Gerakan janin dirasakan aktif

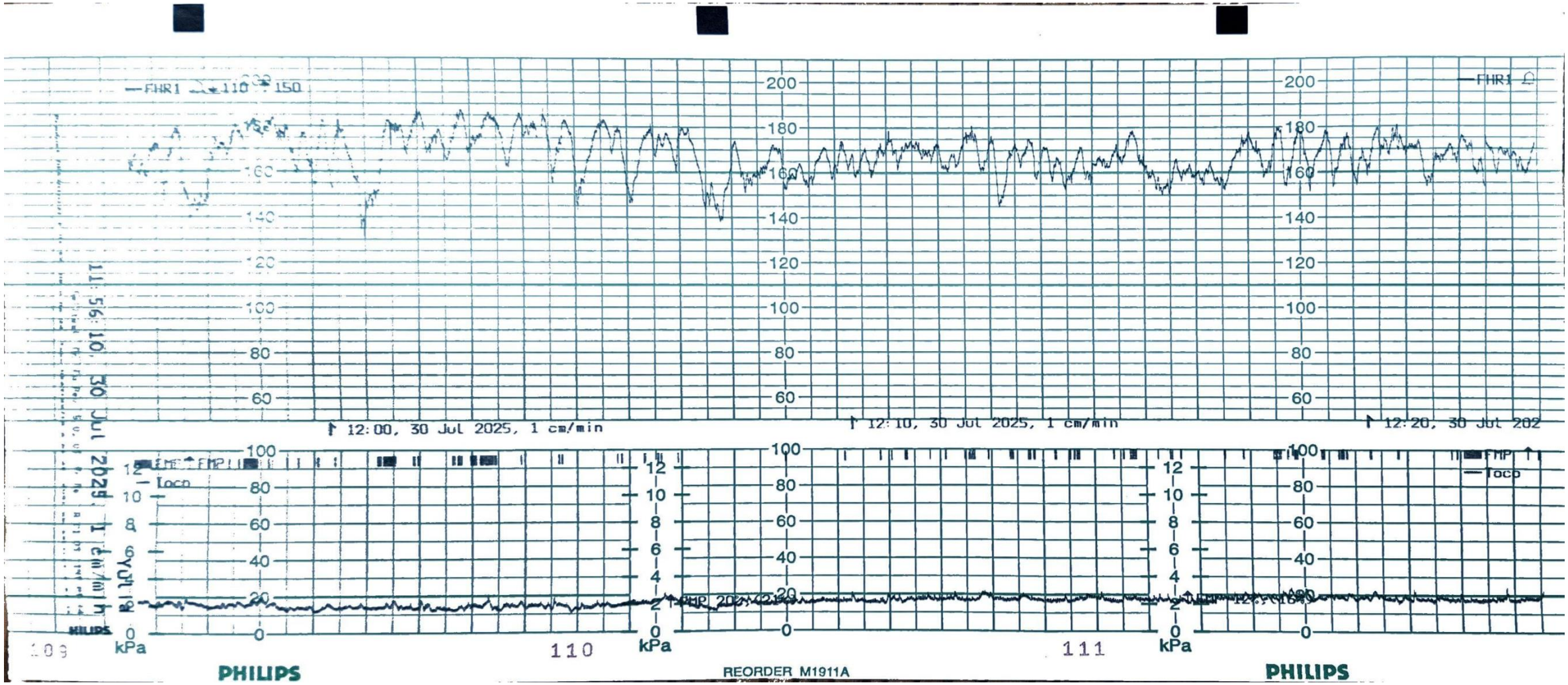
O: BP 115/62 mmHg, HR 98 x/min, RR 20 x/min, T 36.5C; FH 33 cm, head presentation, no contraction, FHR 170 bpm; Io: smooth portio, closed OUE, fluor positive, fluxus negative, pooling (-), valsava (-), nitrazine test (-); US exam: EFW 3150 gram, AFI 5 cm

Laboratorium

DPL 14.3/42.1/13970/392000//81.2/27.4/34
NLR 5.4
Ferritin 16
SI/TIBC/TSAT 53/667/8
SGOT/SGPT 27/22.3
RBG 64
Ur/Cr/eGFR -/0.38/143

Ultrasound

Singleton live head presentation, anterior placenta, FHR 168 bpm
BPD/HC/AC/FL 94/337/332/70
EFW 3150 gram, AFI 5 cm
PIMCA 1.4, PIAU 0.9, CPR >1, SDAU 2.5



30 Juli 2025 11:56

CTG:
Zig zag pattern, increase fetal heart rate
baseline : 165-170 dpm

Clinical Decision ?

Pertanyaan

1. Apakah hasil CTG tersebut dapat langsung ditarik kesimpulan atau masih perlu dipanjangkan?
2. Bila CTG dipanjangkan, bagaimana prognosis dan apakah dapat mempengaruhi keputusan klinis?

Ny F, 29 tahun

Fetal distress on G2P0A1 36 weeks of gestational age, singleton live head presentation, fetus IUGR (p 1.9), diminished amniotic fluid without rupture of membrane (AFI 6.5), mother with SLE hematology, musculoskeletal, mucocutaneus involvement (SLEDAI 2K 2) on therapy, nephritis lupus WHO clinical class II

S: Pasien sebelumnya dikonsulkan dari poli alergi imunologi karena hamil dengan SLE sejak 30 Januari 2025. Pasien mengaku didiagnosis SLE dengan gangguan hematolog riwayat anemia Hb 4 g/dL, trombosit 3000 dengan gejala hematoma di lengan kanan kiri dan bibir sejak September 2014

O: BP 118/80 mmHg, HR 90 x/m, T 36.5 C; RR 20 x/min; TFU 25 cm, DJJ 142 bpm, presentasi kepala, tidak ada kontraksi; Io: portio licin, OUE tertutup, fluor (-), fluksus (-), VT: portio posterior, kenyal, tebal 3 cm, OUE tertutup

Laboratorium

Lab 7/8/25
CBC
11.3/31.8/9610/303000//
79/28/35
Ur/Cr 25.3/0.44

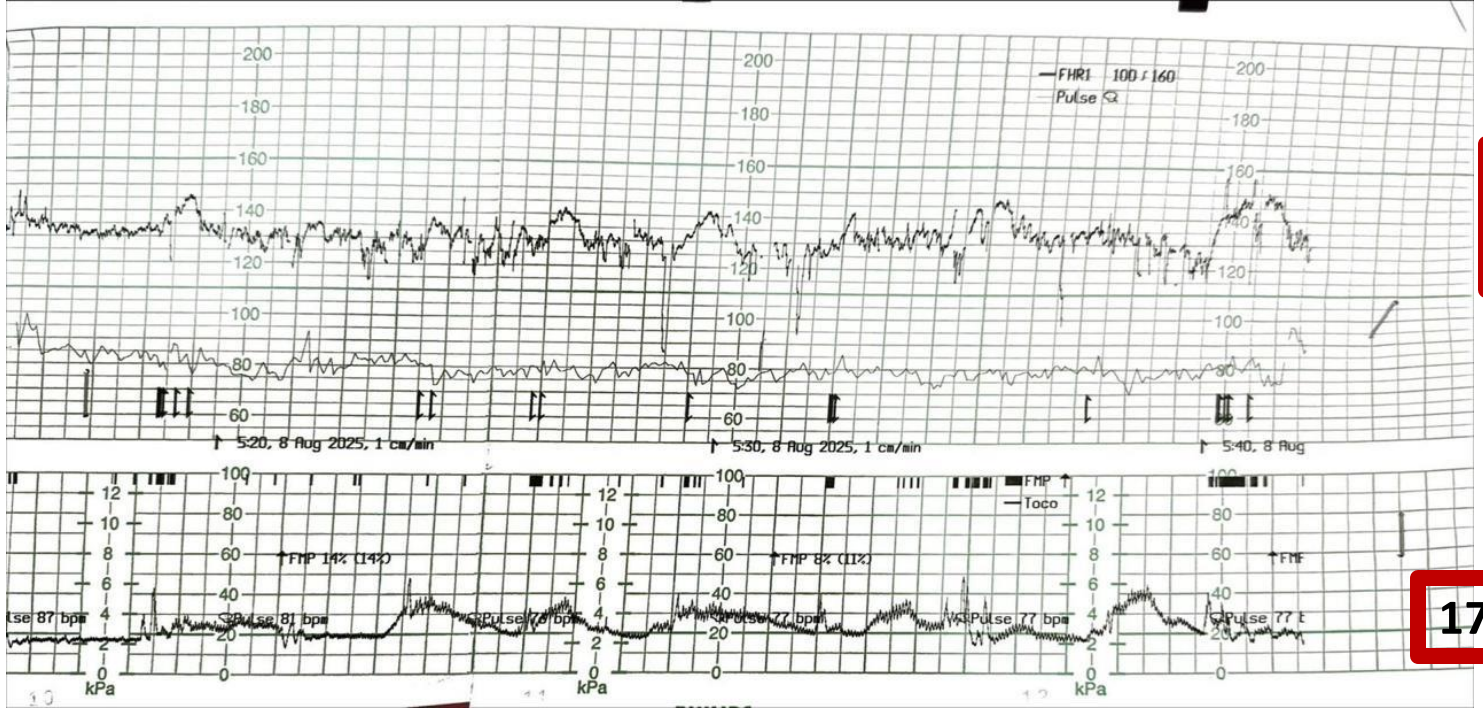
Lab 15/6/2025
PUK 142.8

Lab 18/7/2025
Anti DsDNA 807
C3/C4 113/19

Lab 7/1/25
Anti ds-DNA 1100.5
C3/C4 87/12
Lab 10/9/24
Anti dsDNA 831.7

Ultrasound

Singleton live head presentation, posterior placenta, FHR 148 bpm
BPD/HC/AC/FL 74/283/247/55
EFW 1300 gram, AFI 1 cm
PIMCA 1.1 PIAU 1.4 CPR < 1, SDAU 5.1
Increased umbilical artery resistance



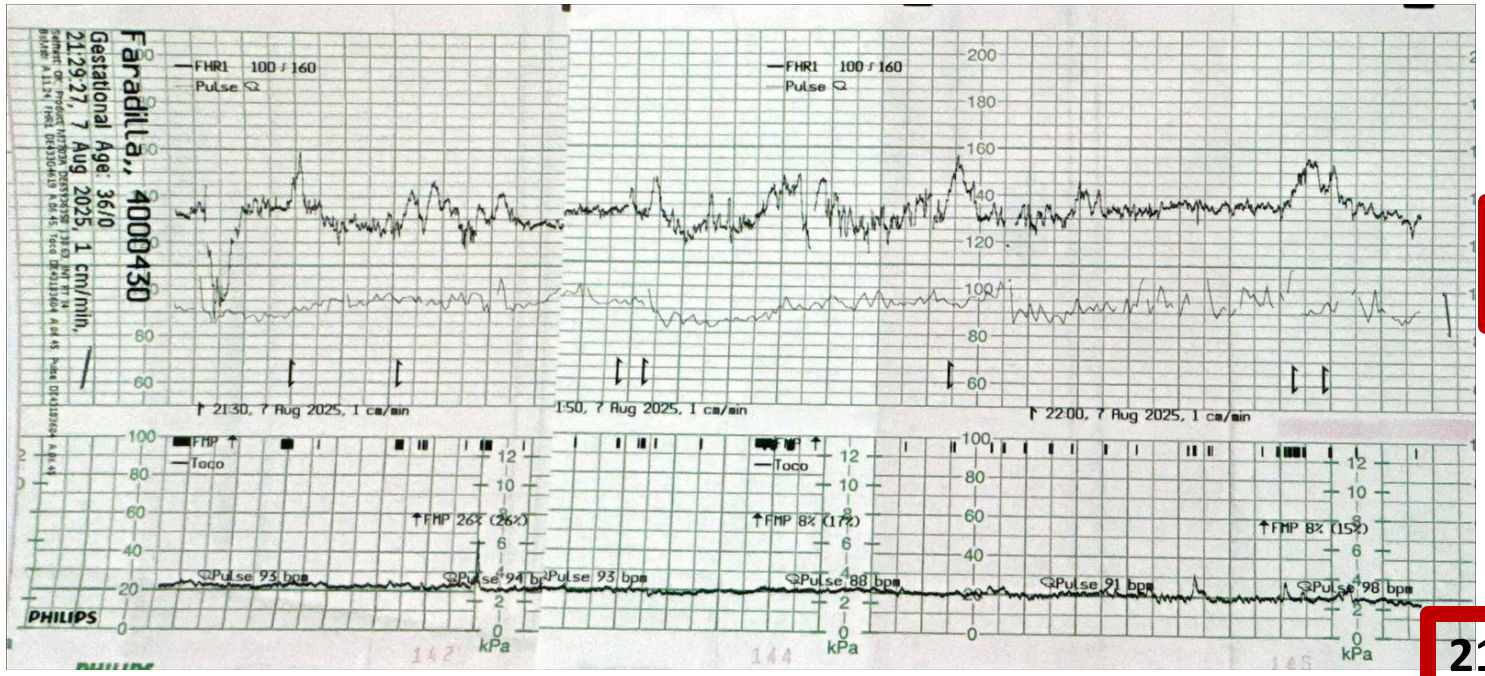
Induction of labor with Misoprostol 50 mcg/4 jam (6th Dose)

VT: Portio aksial, lunak, tebal 2 cm, pembukaan 2 cm, kepala Hodge I

Baseline: 130 dpm
Variabilitas: Moderate
Akselerasi; 3/5x/10 menit
Deselerasi: Tidak ada
Kontraksi: Reguler
Gerak Janin: aktif
Kesan: CTG kategori I

17:26

Clinical Decision ?

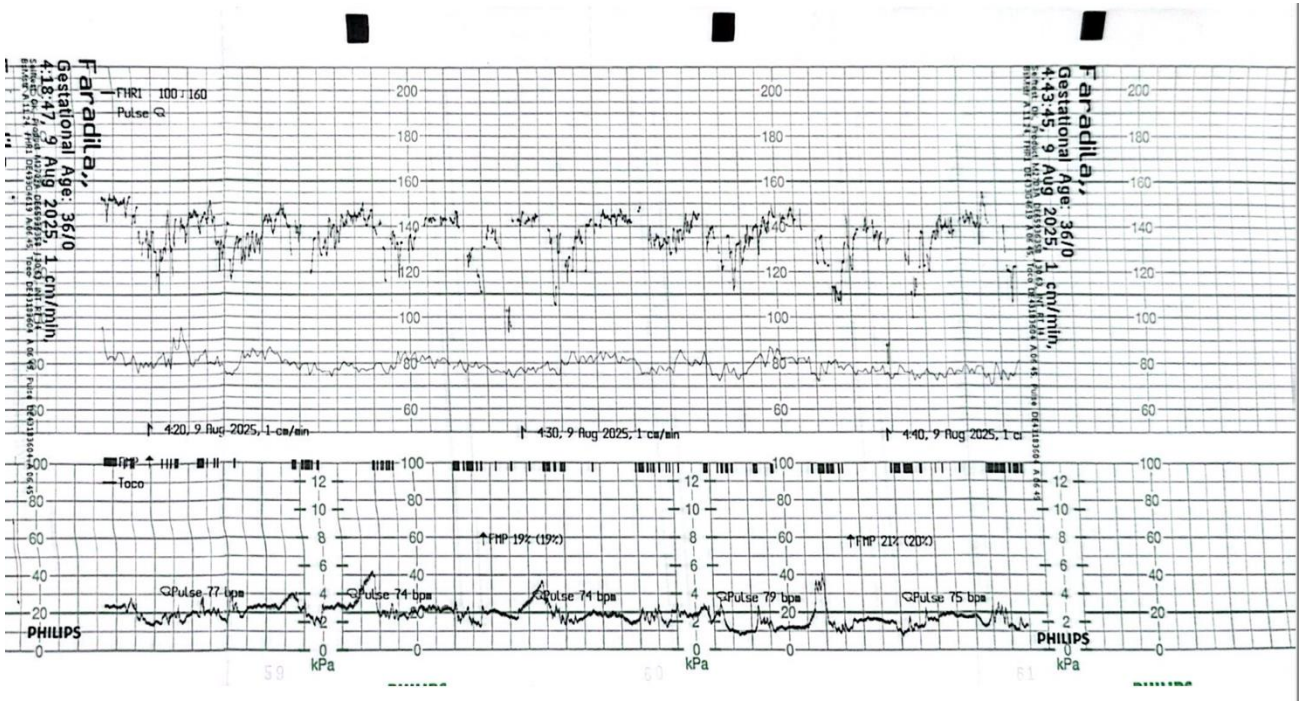


Induction of labor with Misoprostol 50 mcg/4 jam (8th Dose)

VT: Portio aksial, lunak, tebal 2 cm, pembukaan 2 cm, kepala Hodge I

Baseline: 130 dpm
Variabilitas: Moderate
Akselerasi; 3-5x/10 menit
Deselerasi: Deseleration(+) 1x Variable
Kontraksi: Tidak ada
Gerak Janin: aktif
Kesan: CTG kategori I

21:29



04:18

Induction of labor with Misoprostol 50 mcg/4 jam (8th Dose)

VT: Portio aksial, lunak, tebal 2 cm, pembukaan 2 cm, kepala Hodge I

Baseline: 130 dpm
Variabilitas: Moderate
Akselerasi; 3-5x/10 menit
Deselerasi: Deselerasi Lambat
Kontraksi: Reguler
Gerak Janin: aktif
Kesan: CTG kategori I

Clinical Decision ?