

The Physiology of Electronic Fetal Monitoring

Emily Hamilton, MD CM
SVP Clinical Research

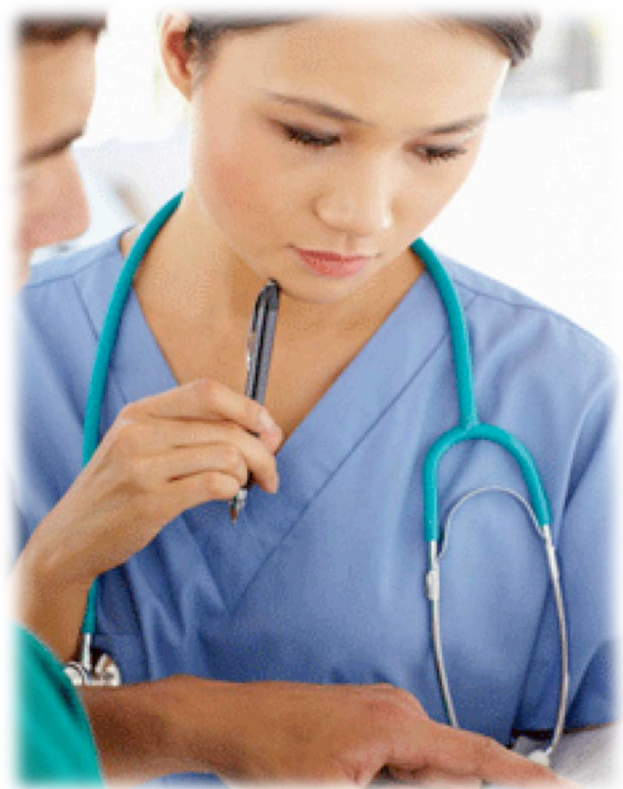


Featuring Emily Hamilton, MDCM Senior Vice President of Clinical Research

An experienced obstetrician, Dr. Hamilton is currently an Adjunct Professor of Obstetrics and Gynecology at McGill University, as well as leading PeriGen's clinical research team.

Dr. Hamilton is the inventor of the PeriCALM advanced fetal monitoring system, holding 32 US and international patents for her research work. She is an internationally-known clinical thought leader on the use of technology to improve obstetric outcomes. She presents her research regularly at obstetric conferences and in peer-reviewed journals.



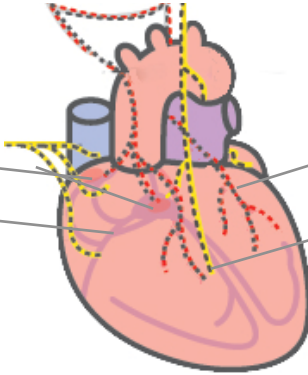


1. Physiology of heart rate regulation
2. How scientists simulated labor stresses and determined deceleration mechanisms
3. Relevance to EFM usage in humans



Intrinsic

- Myocardium
- Pacemakers
- Conducting system
- Adrenergic Receptors α , β
- Cholinergic Receptors



Extrinsic

- Neuronal connections to
- Sympathetic
- Parasympathetic systems
- Circulating catecholamines



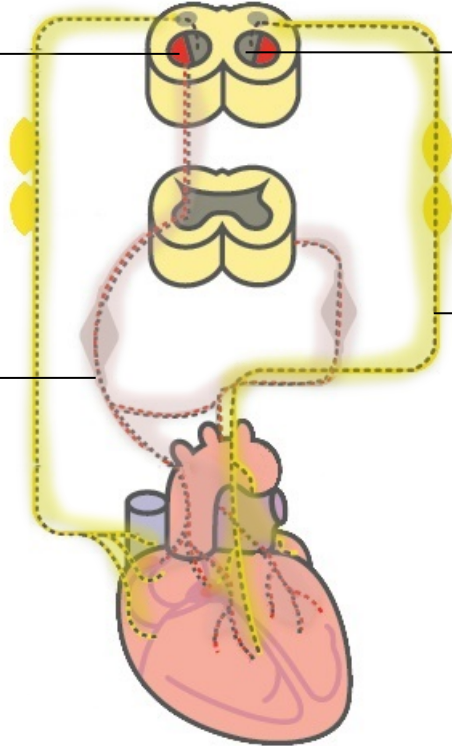
Cardioregulatory Center

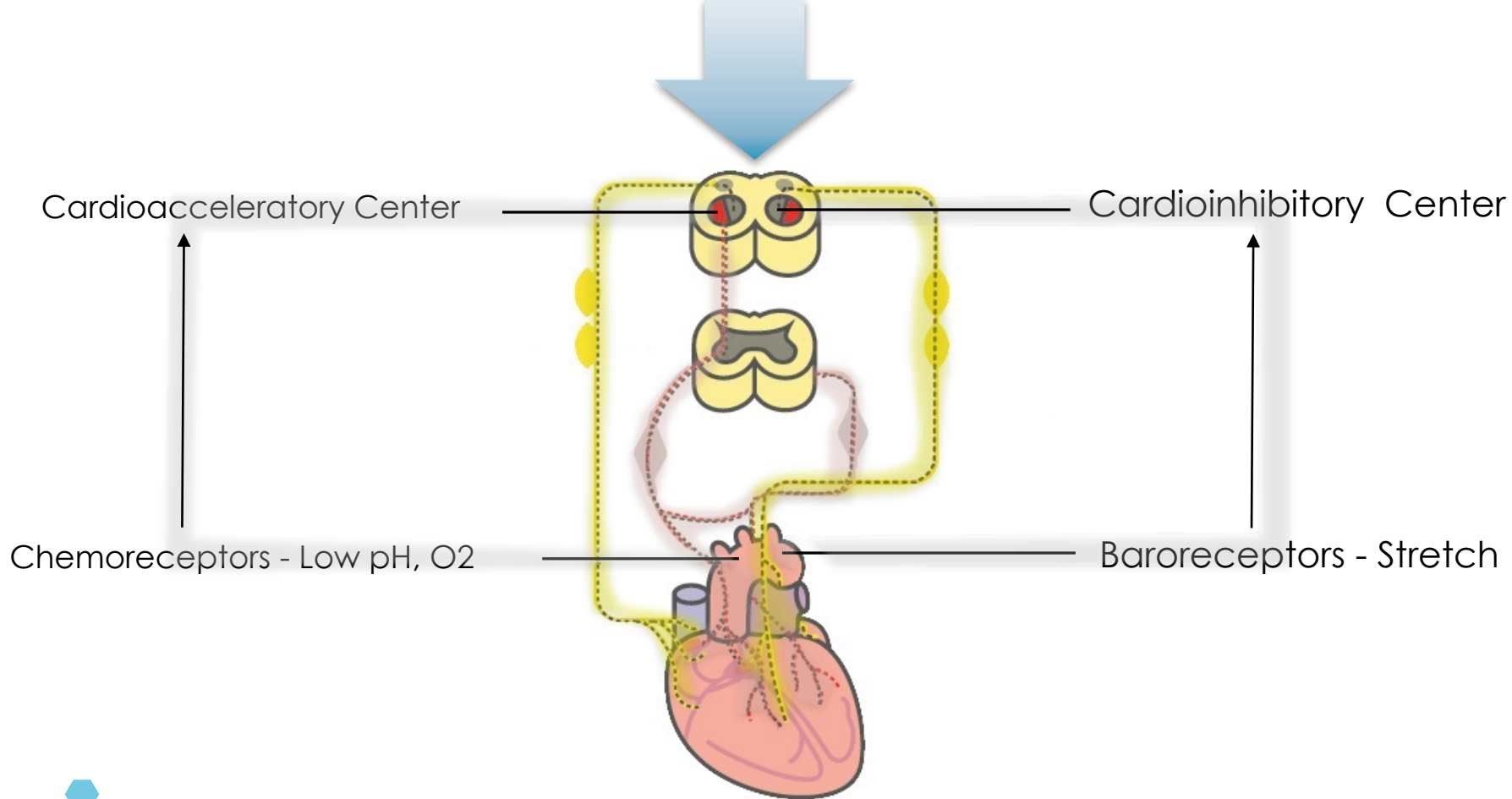
Cardioacceleratory Center

Cardioinhibitory Center

Sympathetic

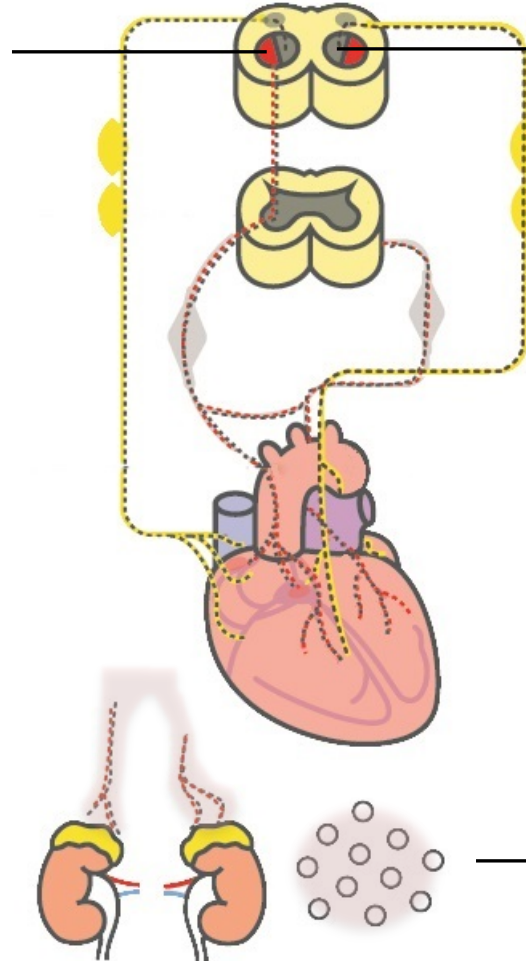
Parasympathetic (vagus)





Cardioacceleratory Center

Cardioinhibitory Center



Vasomotor Center

- Autonomic control of dilation and constriction of peripheral vessels

Fetal Heart Regulation

Mechanisms of late decelerations in the fetal heart rate. A study with autonomic blocking agents in fetal lambs

C.B. Martin, Jr., J. de Haan *, B. van der Wildt, H.W. Jongsma, A. Dieleman and T.H.M. Arts
Department of Obstetrics and Gynecology, Catholic University and Sint Radboudziekenhuis, Nijmegen, The Netherlands

CLINICAL OPINION

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OBSTETRICS

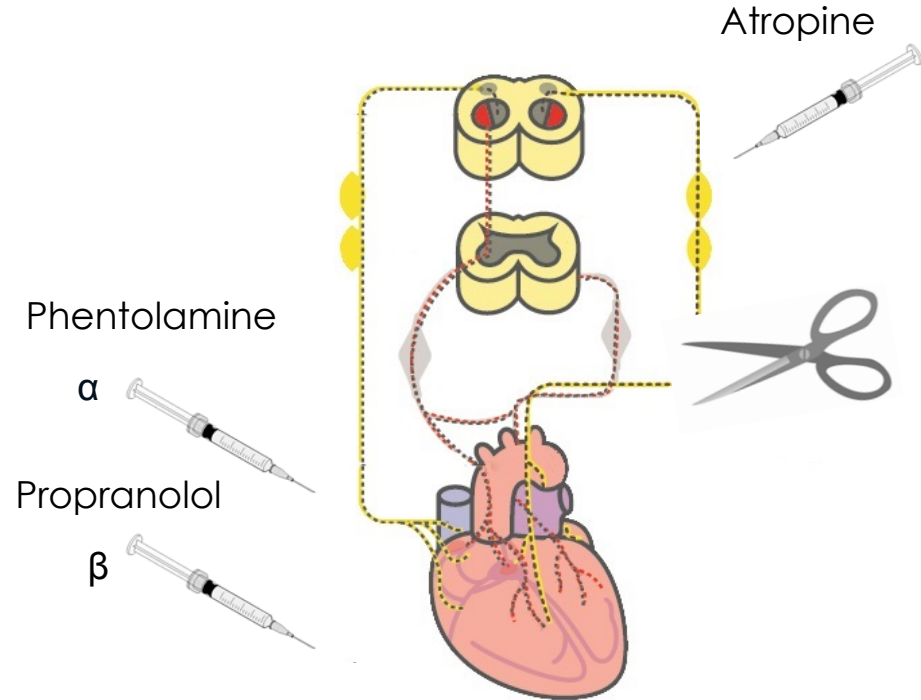
The intrapartum deceleration in center stage: a physiologic approach to the interpretation of fetal heart rate changes in labor

Jenny A. Westgate, MBChB, MD; Bert Wibbens, MD; Laura Bennet, PhD; Guido Wassink, MSc; Julian T. Parer, MD, PhD; Alistair J. Gunn, MBChB, PhD



Animal Experimentation

1. Umbilical cord compression
Mechanical devices to occlude the cord
2. Uteroplacental insufficiency
Compression of arteries outside the uterus
3. Implanted devices for BP, acid base status



Decelerations from Uteroplacental Dysfunction

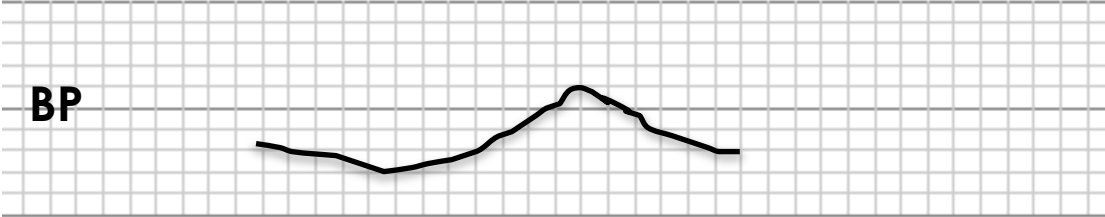
**Mechanisms of late decelerations
in the fetal heart rate.
A study with autonomic blocking agents in fetal lambs**

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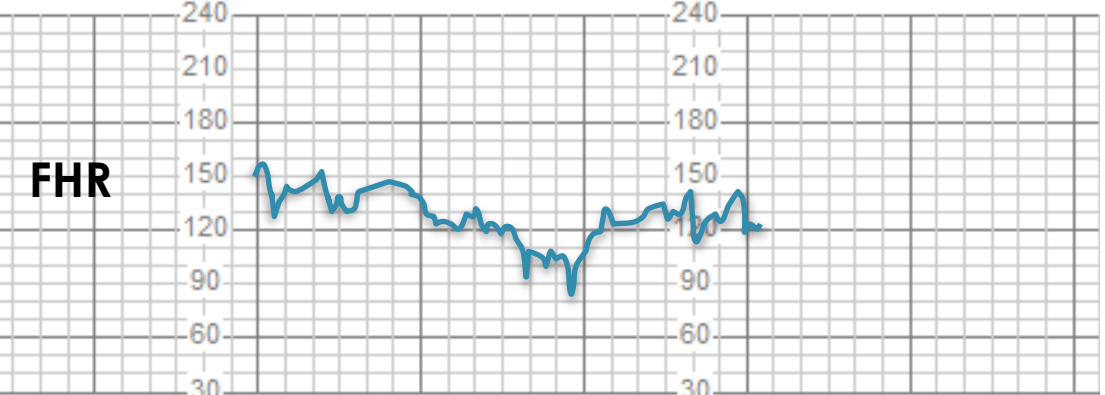
Department of Obstetrics and Gynecology, Catholic University and Sint Raadboudziekenhuis, Nijmegen, The Netherlands

pH 7.40 BE +1.3

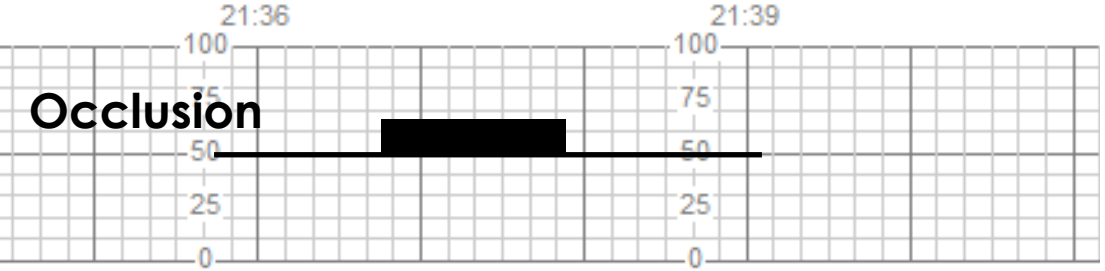
BP



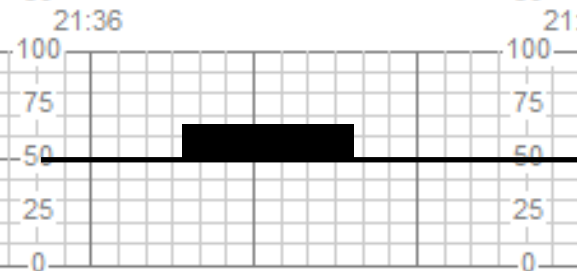
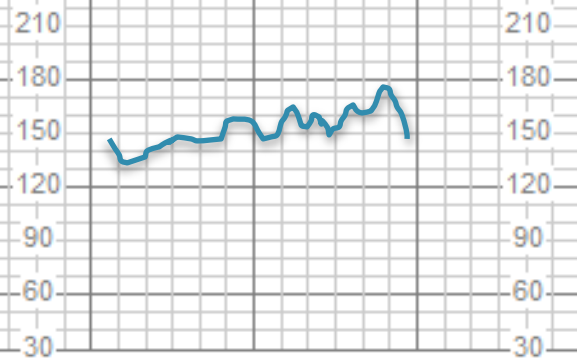
FHR



Occlusion

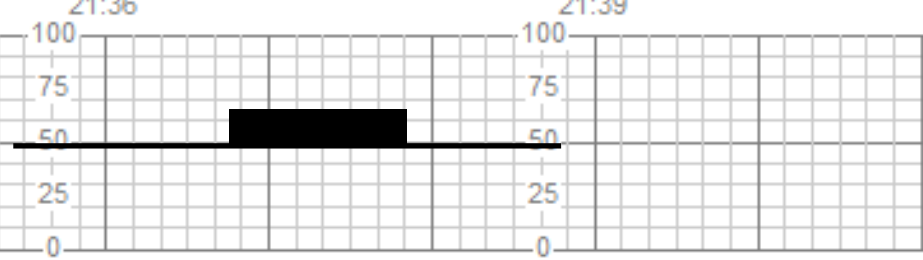
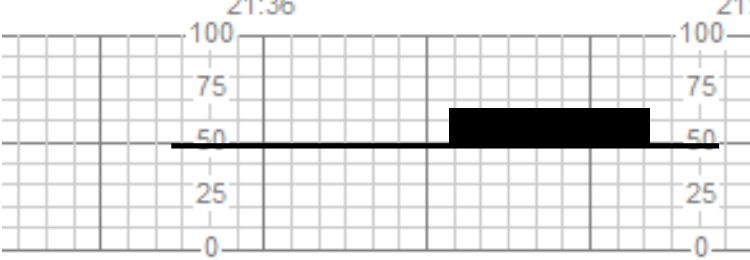
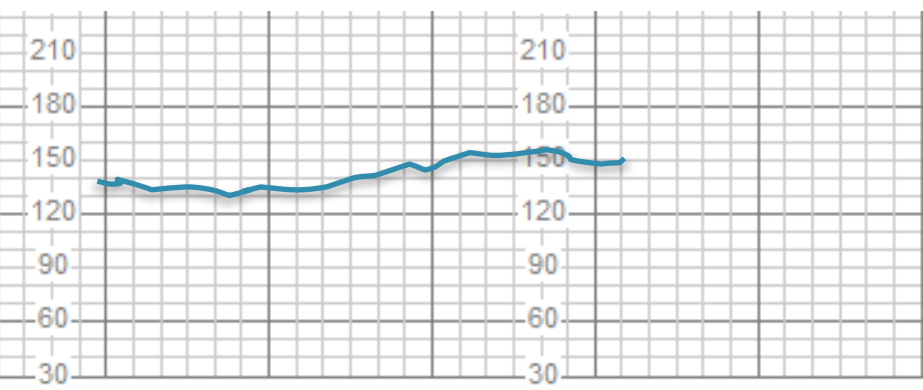
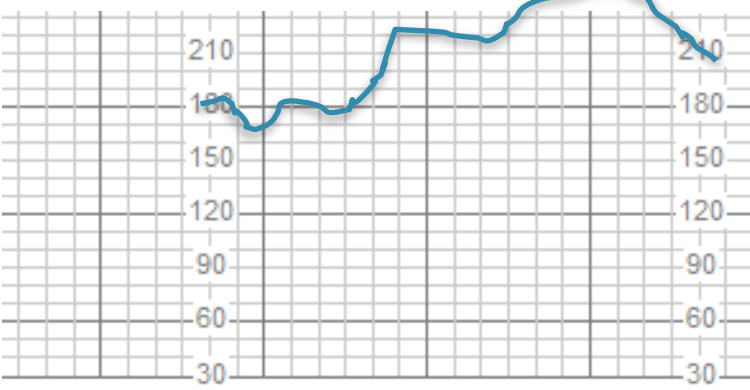
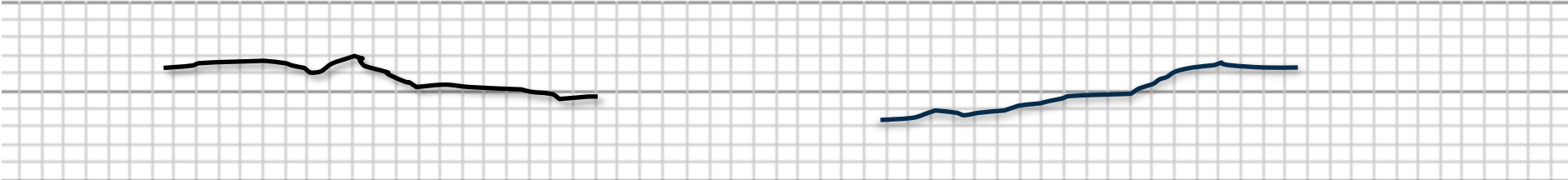


Phentolamine pH7.35 BE -0.7

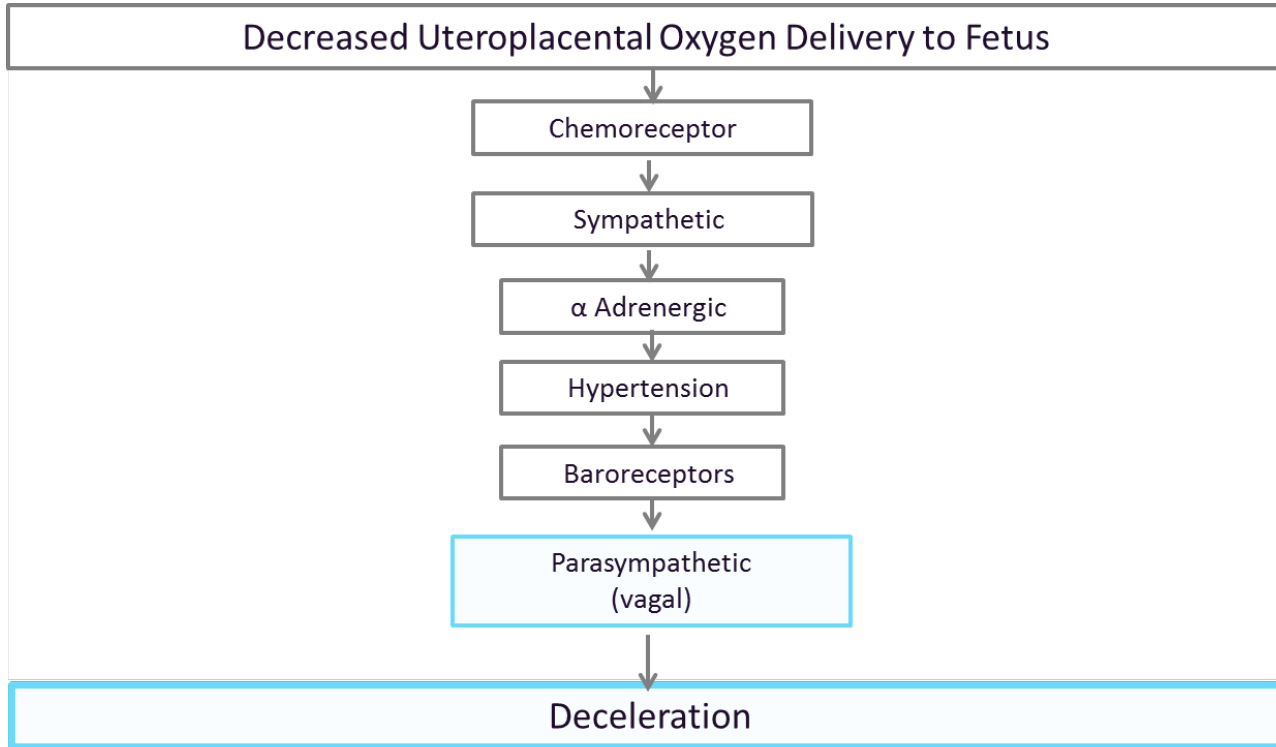


Pentolamine atropine
pH7.33 BE -4.8

Pentolamine atropine propranolol
pH7.33 BE -5.5



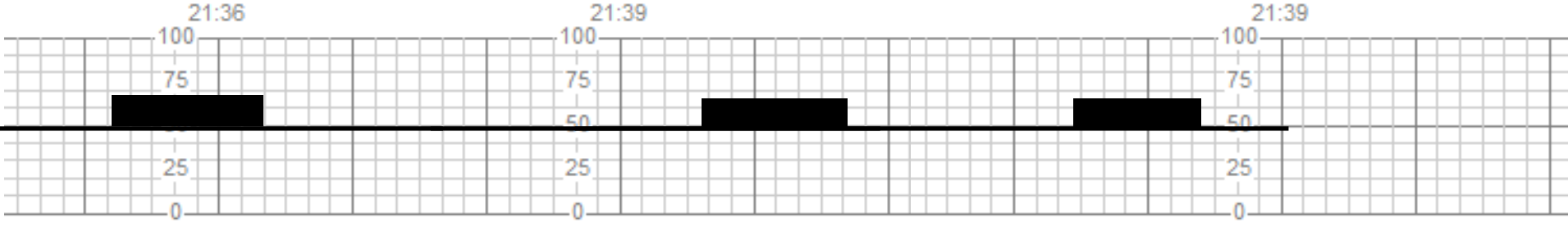
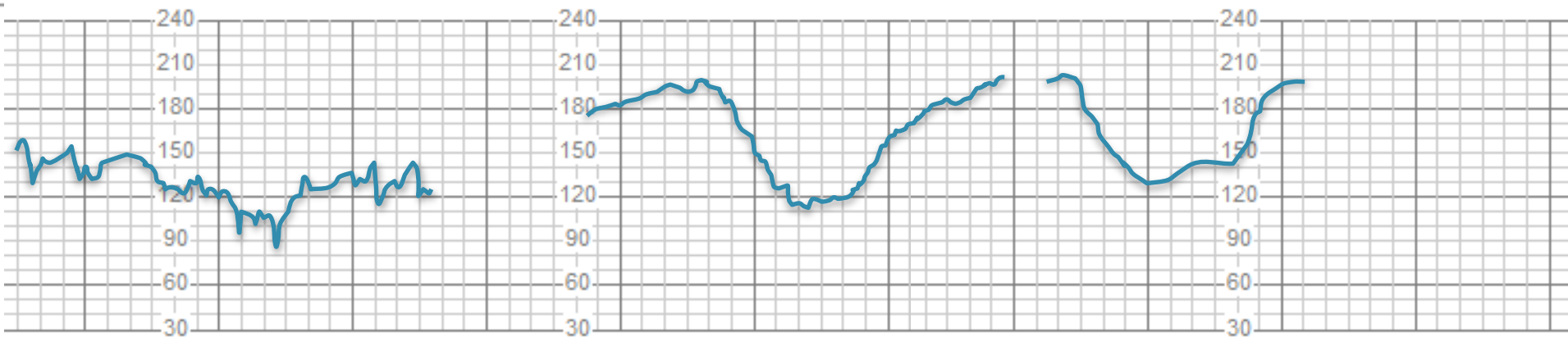
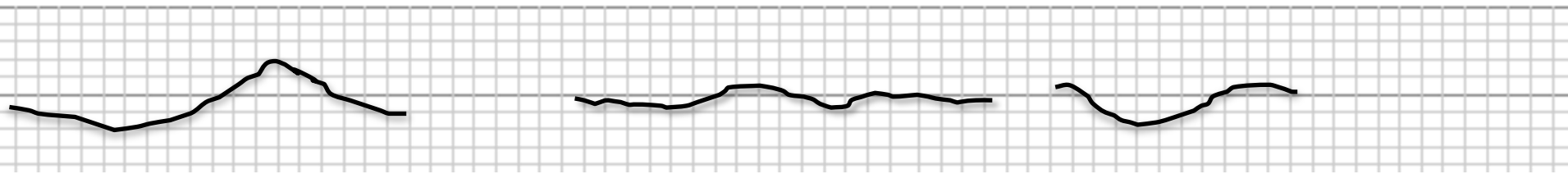
Physiological Mechanisms



pH 7.40 BE 1.3 I

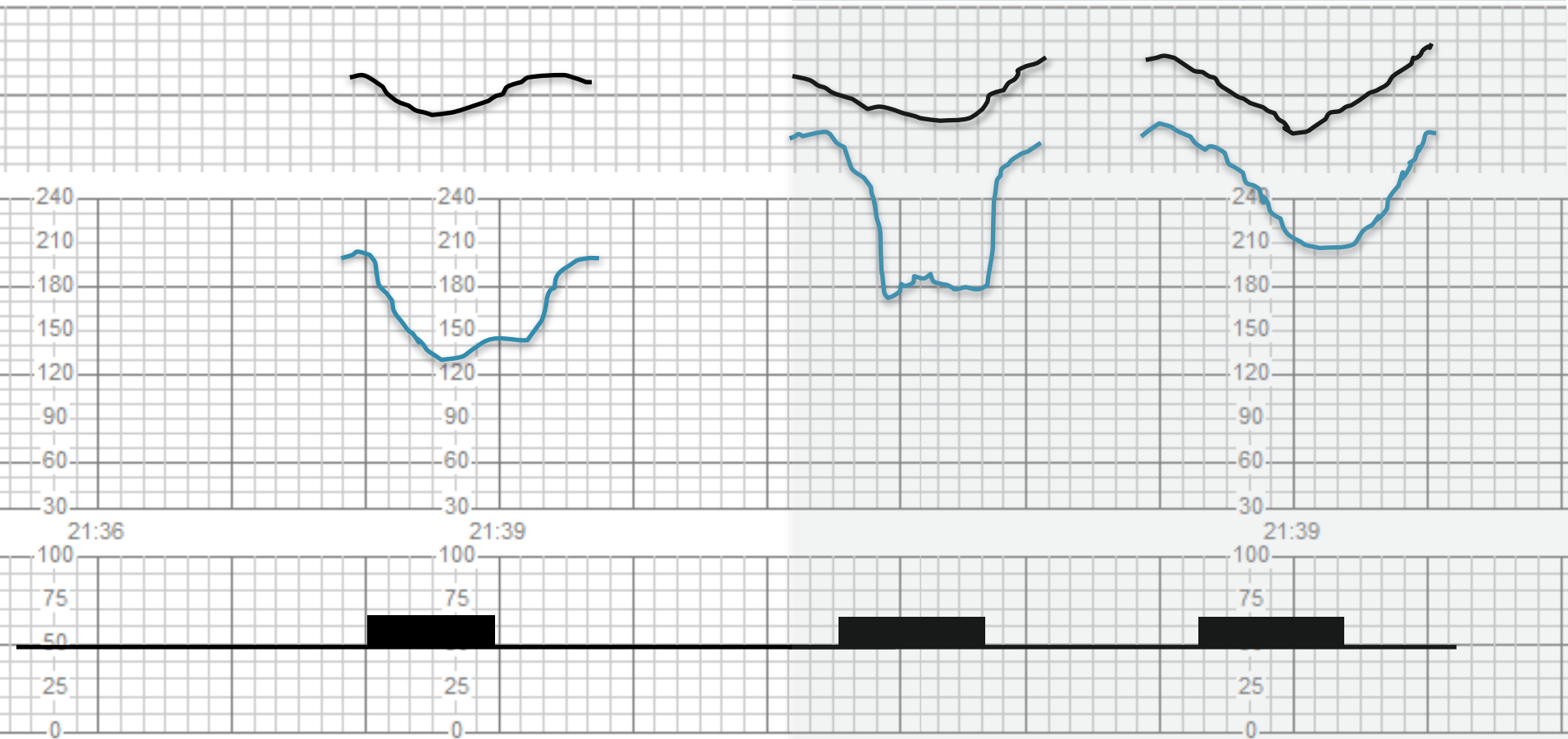
pH 7.14 BE -13.1

pH 6.96 BE -19.7

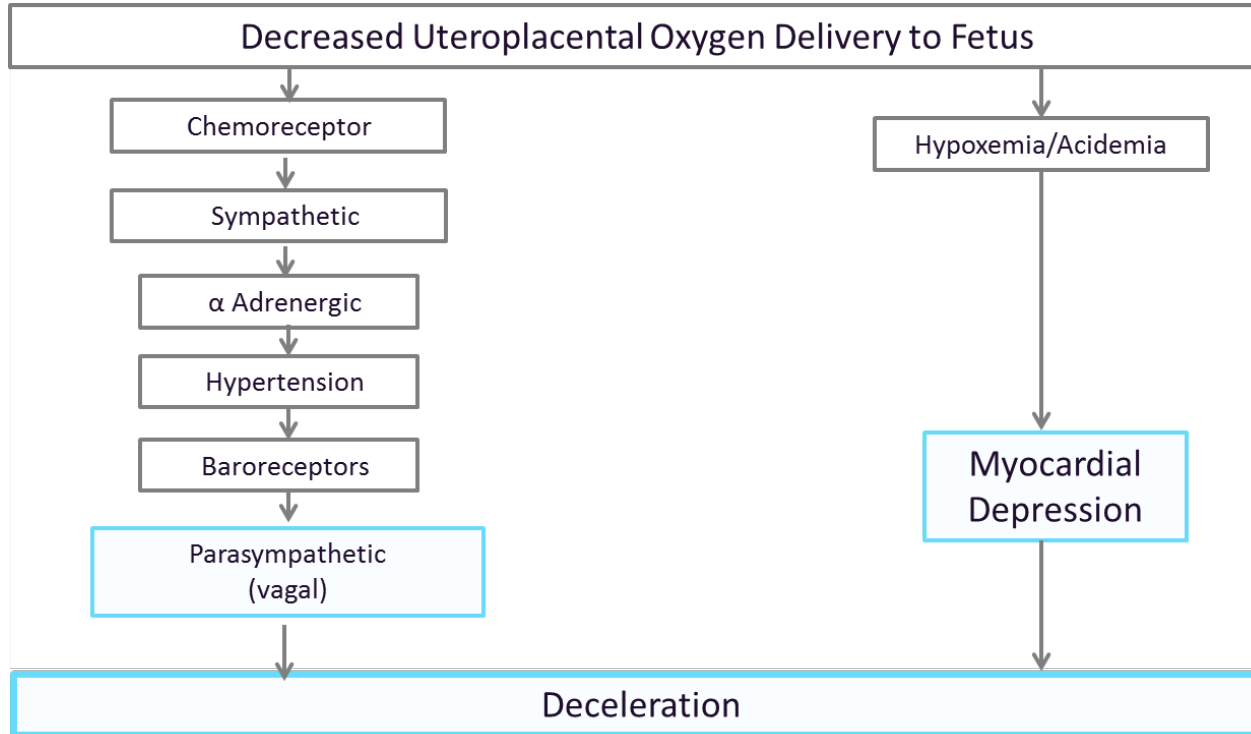


pH 6.96 BE -19.7

pH 6.96 BE -19.7
with Atropine



Physiological Mechanisms



Recurrent Lates Incidence and Outcome

Low risk women **normal** tracing on admission

- Incidence of **sporadic** Lates 5.5% 1% pH<7.1
- With normal variability 5.4%
- Incidence of **recurrent** Lates 1.8%
- With normal variability 1.4% 10% → pH< 7.1
- With minimal/absent variability 0.36% 20% → pH< 7.1

With **low variability and recurrent Lates/PD** on admission

- Incidence 0.43% **42%** → pH < 7.0
- Died or developed CP **33%**

Conclusions: Decelerations from Uteroplacental dysfunction “Late” deceleration

1. Uncommon pattern
2. Multiple pathways and shapes
3. Not all late decelerations indicate myocardial hypoxia or acidemia
4. Presence of very low variability with recurrent Lates on admission VERY concerning



Case Presentation # 1

- 25 year old P0 with an uncomplicated antenatal course, admitted in spontaneous labor at term.
- Delivered spontaneously 12 hrs later, a 3100 gram baby
No oxytocin, no meconium
Labor progression normal
- Would you deliver at point **1 or 2 or 3**



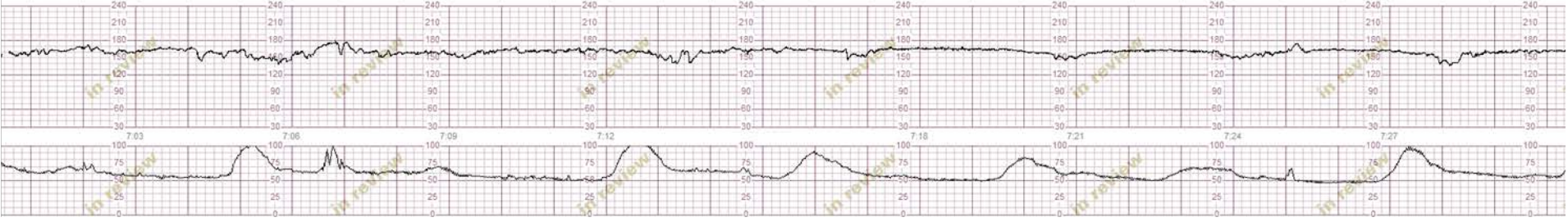
Admission 03:30 2 cm /50%/ -3



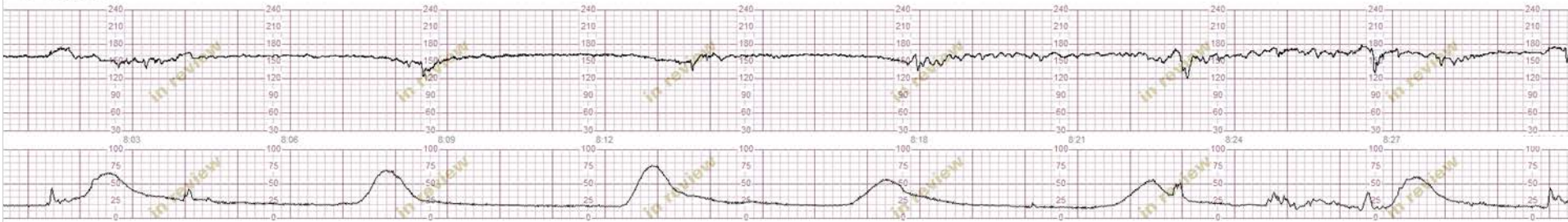
05:00



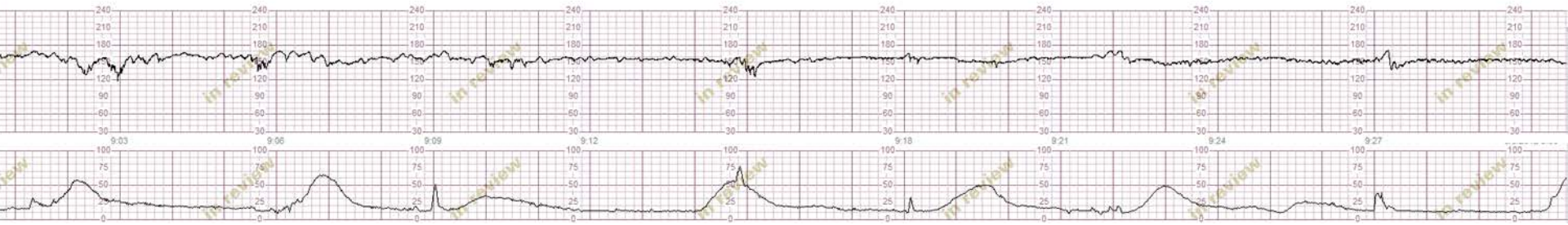
07:00 3 cm/80%/-2



08:00

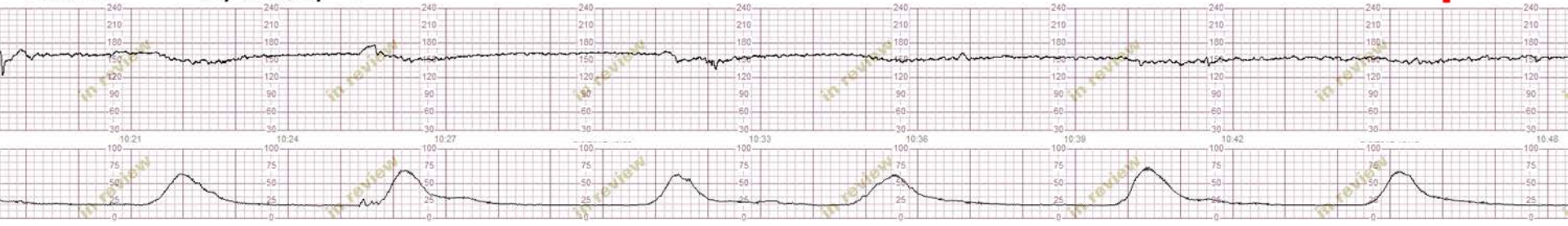


09:00

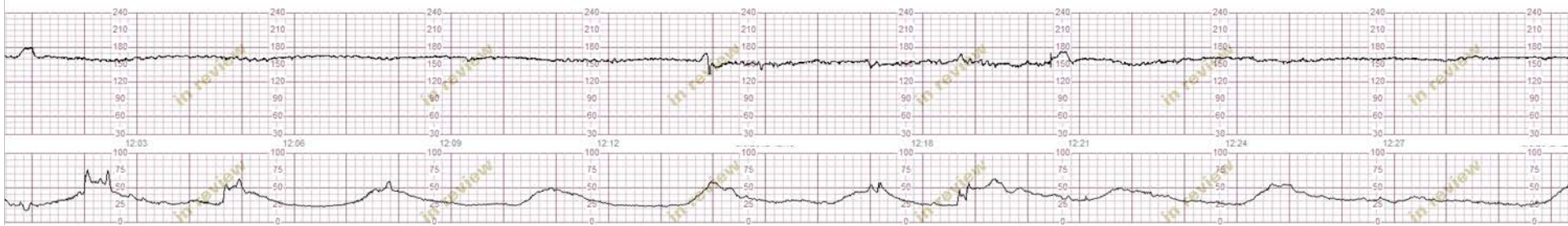


10:20 5/90%/-1

1



12:00

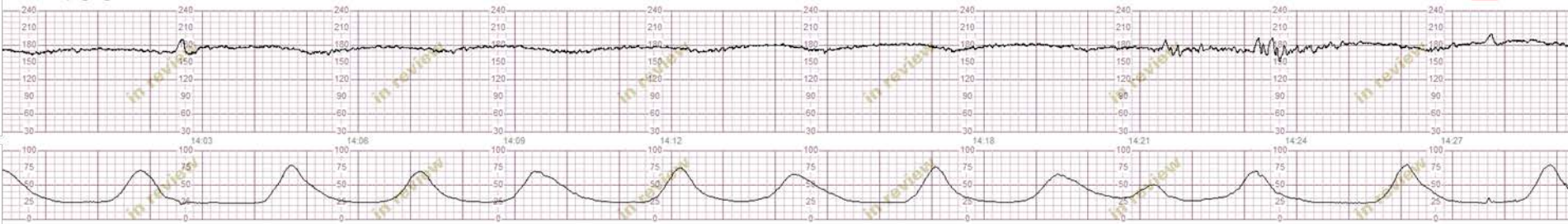


13:00 6/100/+1



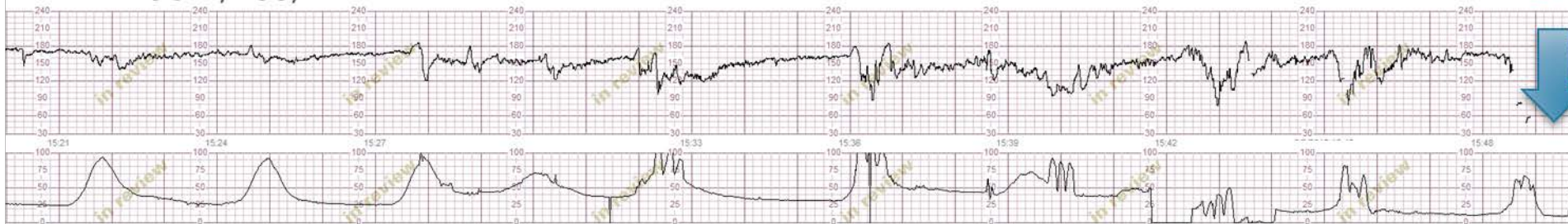
14:00

2



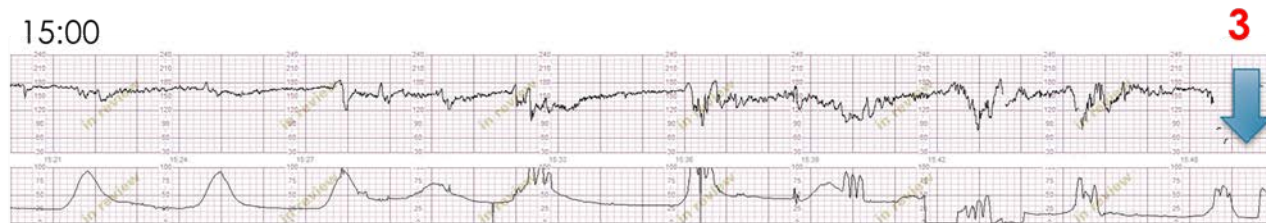
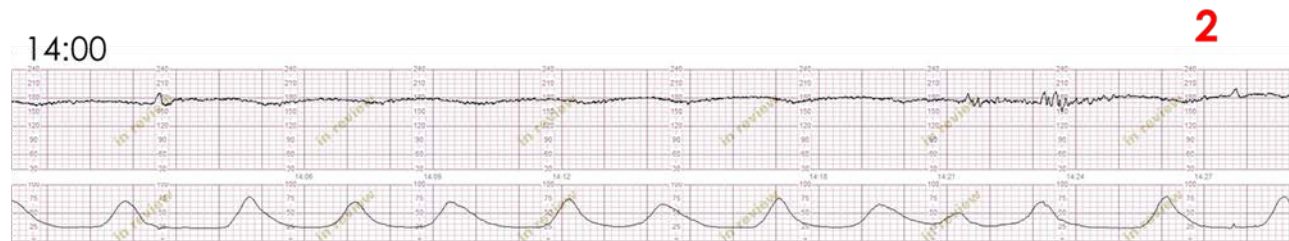
15:00 10cm/100/+1

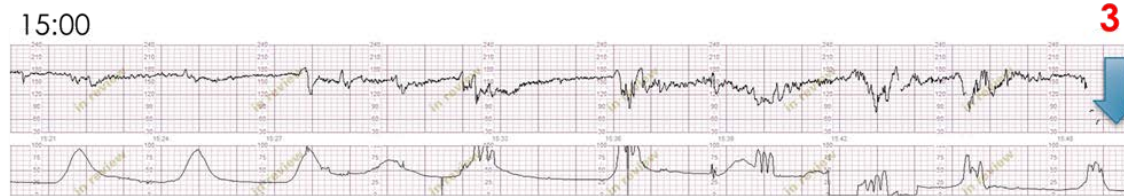
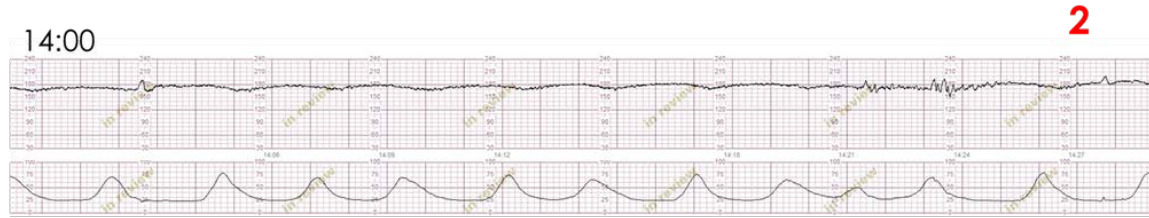
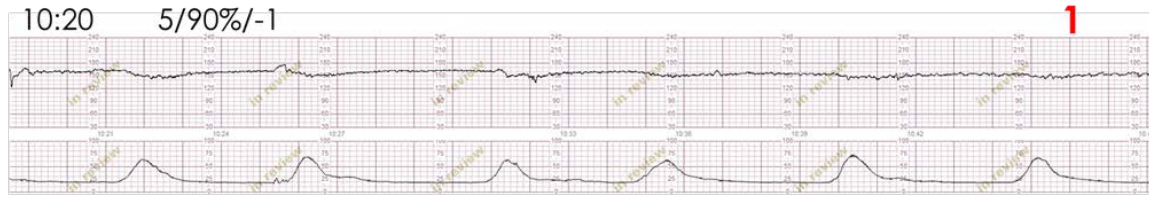
3





Delivery intervention recommended at point





SVD, Apgar 8,9 pH 7.25 Base deficit 4 mmol/L



Case Presentation #2

G1P0

41+ weeks

Admitted in spontaneous labor

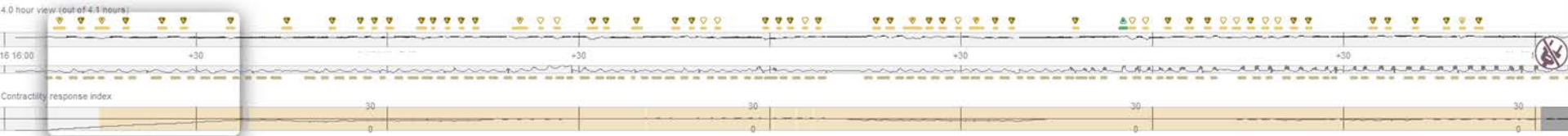
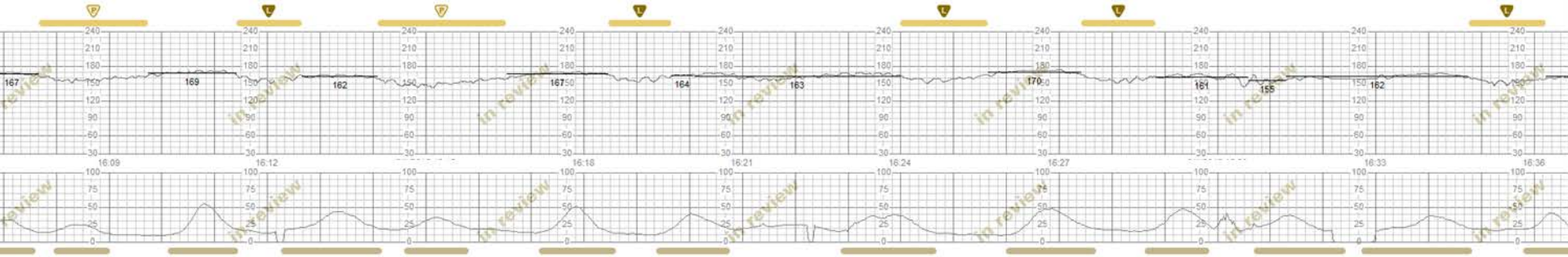
Spontaneous Delivery 4 hrs later, 3750 g

pH 6.91 BD 20.7

Apgars 3/6

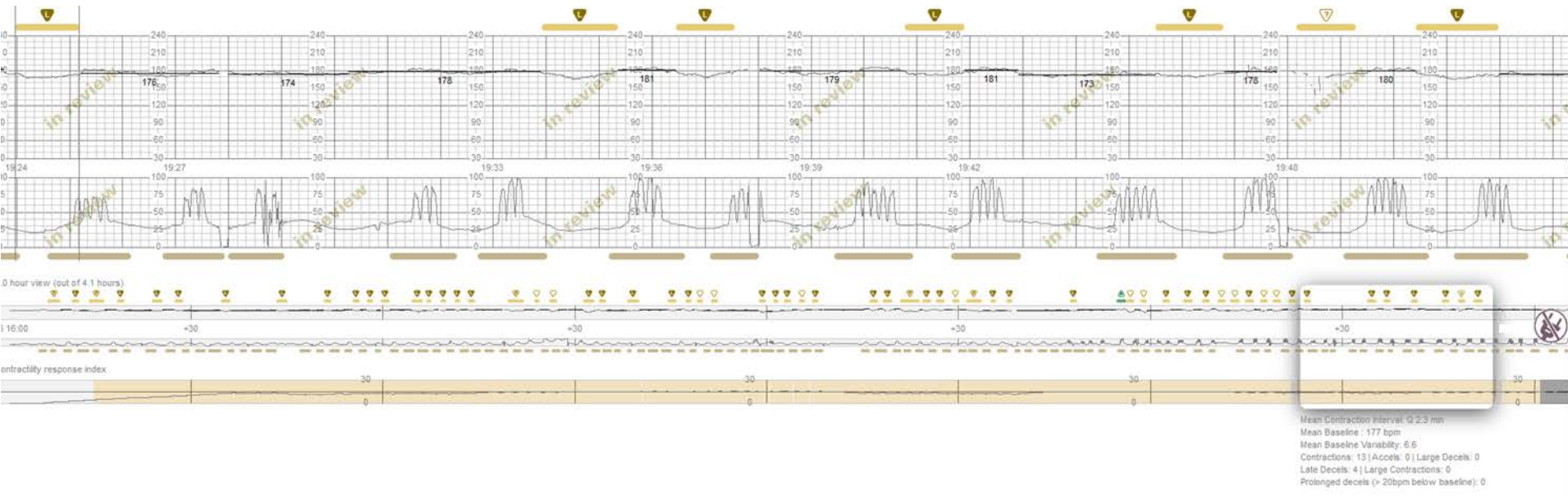


Tracing on Admission



Mean Contraction Interval: 2.3 min
Mean Baseline: 164 bpm
Mean Baseline Variability: 7.1
Contractions: 13 | Accels: 0 | Large Decels: 0
Late Decels: 5 | Large Contractions: 1
Prolonged decels (> 20bpm below baseline): 0

Tracing minutes before vaginal delivery





This tracing is highly concerning because of

1. Abnormal EFM patterns on admission
2. Decelerations with almost every contraction
3. High and rising baseline with low baseline variability
4. Abnormalities persist for >4hrs
5. All of the above



PeriGen®

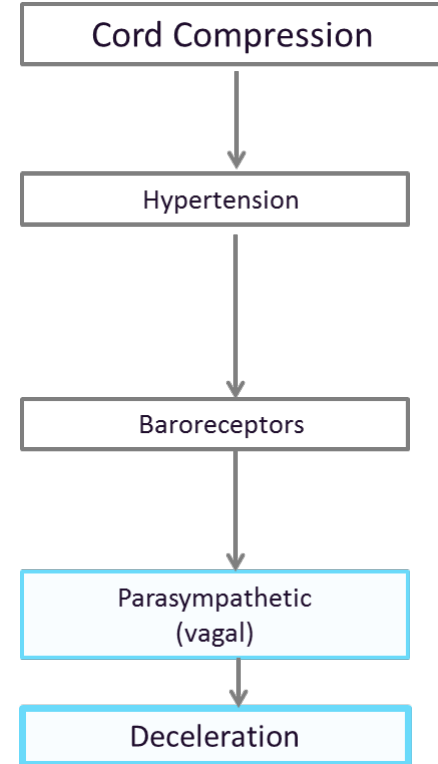
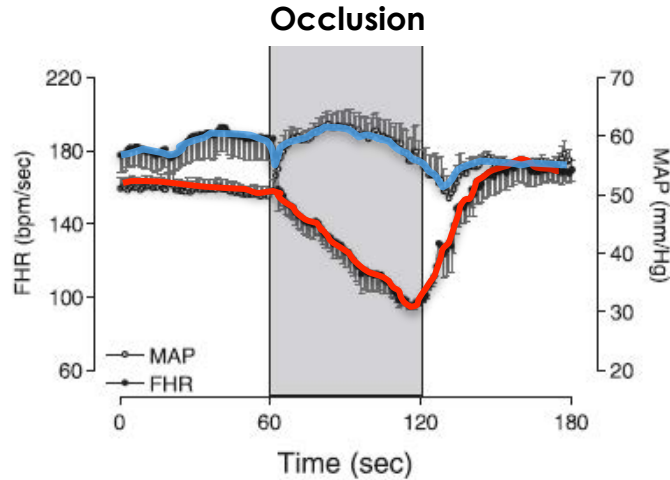
Empowering Clinicians. Advancing Perinatal Systems.



Decelerations From Umbilical Cord Compression

Cord Compression

Bennet L, Westgate JA, Liu YC, Wassink G, Gunn AJ. J Appl Physiol (1985). 2005



Repeated 1 min Cord Occlusion

Bennet L, Westgate JA, Liu YC, Wassink G, Gunn AJ. J Appl Physiol (1985). 2005

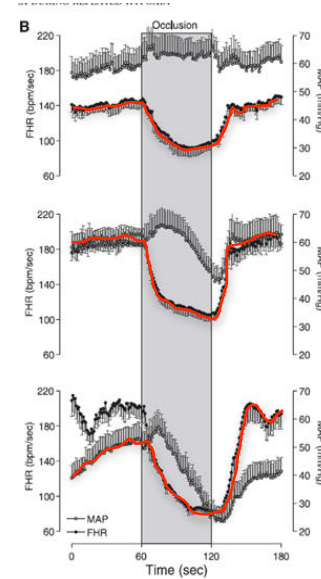
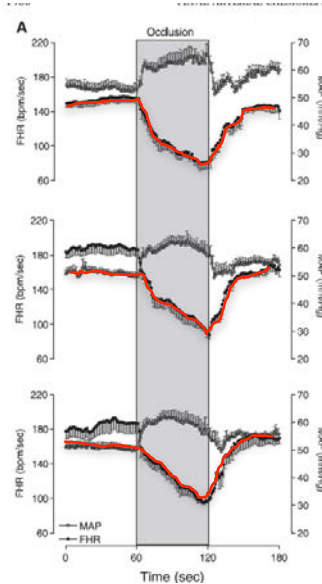
5 mins

2.5 min

First 30 min

Mid 30 min

Last 30 min



pH 7.34 stable...
BD 1.3 stable ...

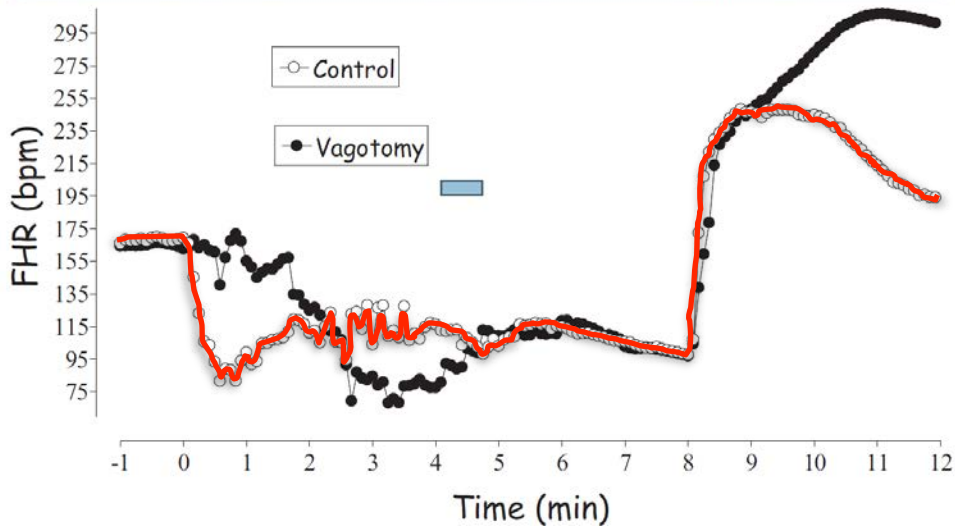
pH 7.25 7.14 **6.92**
BD 3.3 13.6 **19.2**

Non Vagal Component

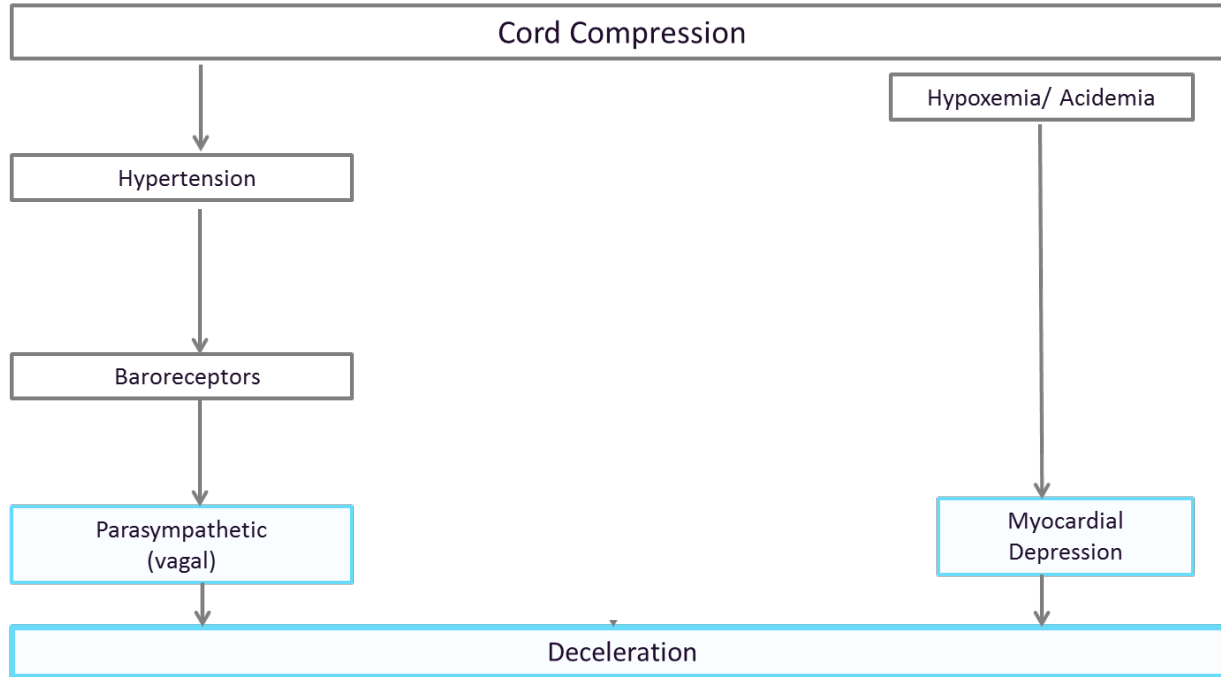
Westgate JA, Wibbens B, Bennet L, Wassink G, Parer JT, Gunn AJ. Am J Obstet Gynecol. 2007

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



Physiological Mechanisms



Baseline Variability

1. Heart

Myocardium and its pacemakers contribute to variability

2. Central Nervous System

“Push Pull” effect of sympathetic and parasympathetic system

Drugs, malformations, sleep

3. Circulating catecholamines



NICHD 2008 EFM Update Category I,II,III Defined

“Moderate FHR variability reliably predicts the absence of fetal acidemia at the time that it is observed.”

How reliable ?

How much acidemia?

Experimental Hypoxemia Increases FHR Variability initially

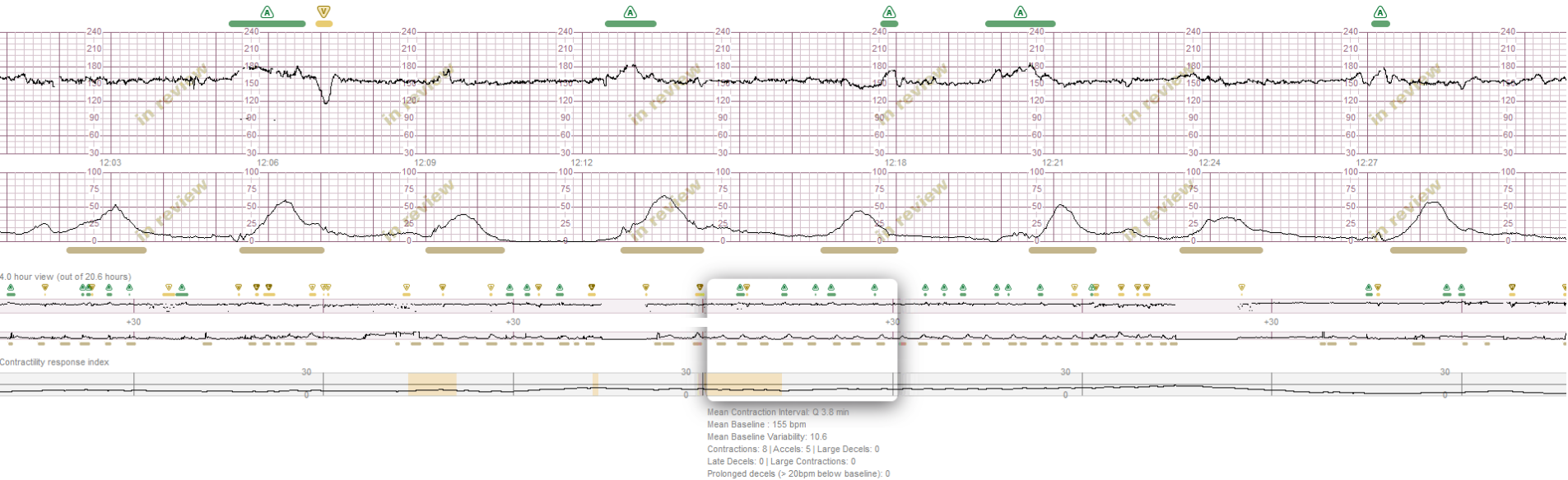
	Animal	Intervention	Variability change
Ikenoue et al 1981	monkeys	hypoxemia	Increased
Dalton, Dawes et al 1977	sheep	hypoxemia	Increased
Murotsuki, Bocking et al 1997	IUGR sheep	hypoxemia	Initial increase in variability After 21 days of hypoxemia variability decreased by 20%
Martin 1979	Sheep	Hypogastric occlusion pH 6.96	Increased variability
Kozuma et al 1997	sheep	severe acidosis (mean pH 6.92)	Decreased in 2/3 Increased in 1/3

Low Variability in Humans

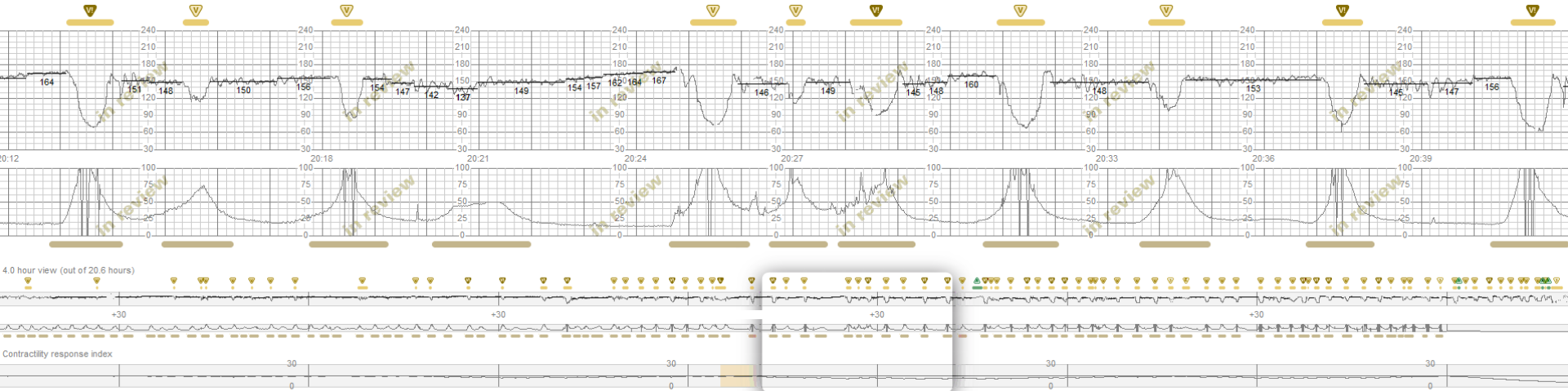
	Outcome	Number	With Low variability close to birth	Normal Variability
Samueloff 1994	pH <7.2	303	26%	74%
Cahill 2012	pH <7.1	57	9%	91%
Williams and Galarneau 2003	BD >12	36	42%	58%
Low 1999	BD >16	71	68%	32%
Williams and Galarneau 2003	BD >16	13	85%	15%

G1 40+wks labor X 12.5Hrs 3600g baby

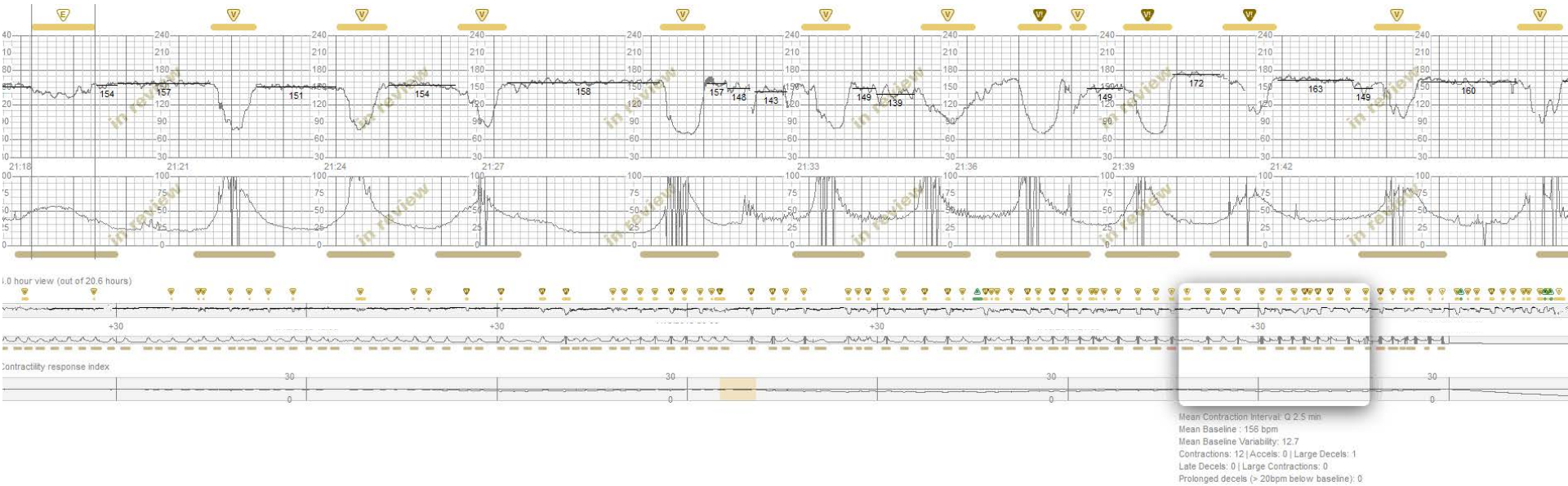
pH 6.89 BD 21.4 Apgar 3/5



2.5 hours before birth

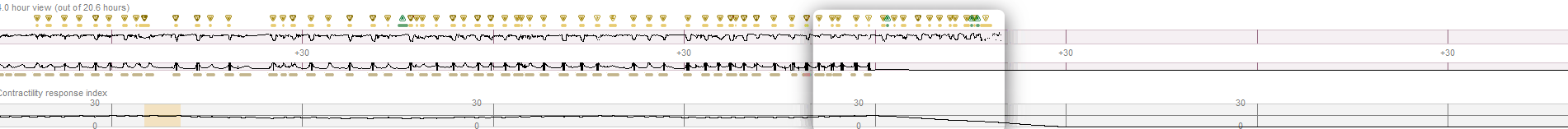
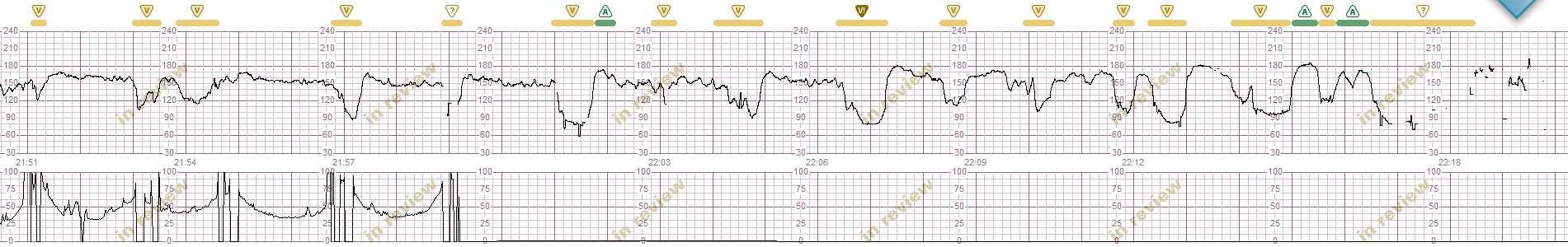


1 hour before birth



Last 30 minutes

pH 6.89 BD 21.4 Apgar 3/5



Mean Contraction Interval: 6.0 min
Mean Baseline : 157 bpm
Mean Baseline Variability: 12.9
Contractions: 5 | Accels: 3 | Large Decels: 0
Late Decels: 0 | Large Contractions: 0
Prolonged decels (> 20bpm below baseline): 1

Baseline Variability

- Initial response to hypoxemia in experimental conditions is an increase in variability
- Low variability appears with advanced acidemia
- And /or with other “depressing” or “chronic ”central nervous system factors



SUMMARY

- EFM patterns are the result of many interacting physiological pathways that overlap and change in the presence of severe acidemia
- Important considerations for EFM interpretation
 1. Decelerations from uteroplacental insufficiency and cord compression share common physiological pathways
 2. Size, frequency of decelerations
 3. Duration of EFM problem and its progression
 4. Loss of baseline variability is a late change
 5. Underlying fetal tolerance and cause



The Physiology of Electronic Fetal Monitoring

Thank you for joining us

