Umbilical-Cord Blood Gas Analysis in Obstetrical Practice

Webinar - Wednesday, July 1, 2015

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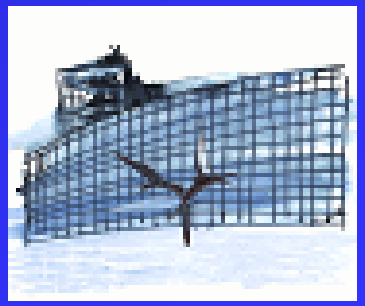
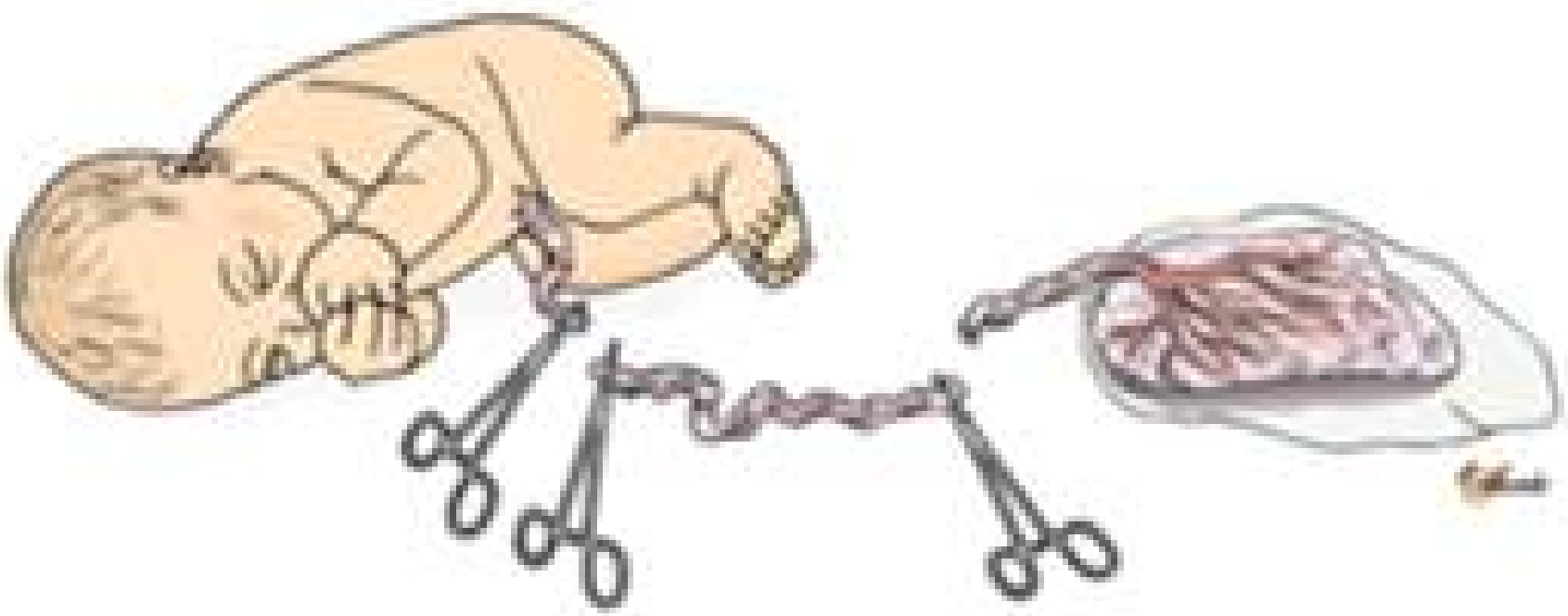


Umbilical-Cord Blood Gas Analysis

- A reliable method to describe fetal oxygenation
- and possible birth asphyxia



Fetal asphyxia

- Asphyxia (from Greek) means no “pulse”
- Usual definition: insufficient oxygen (O_2) supply/ uptake and insufficient carbondixide (CO_2) exchange.
- - This definition is less useful in daily clinical life, as fetal pO_2 is always low in the interuterine life and during labour

Fetal asphyxia

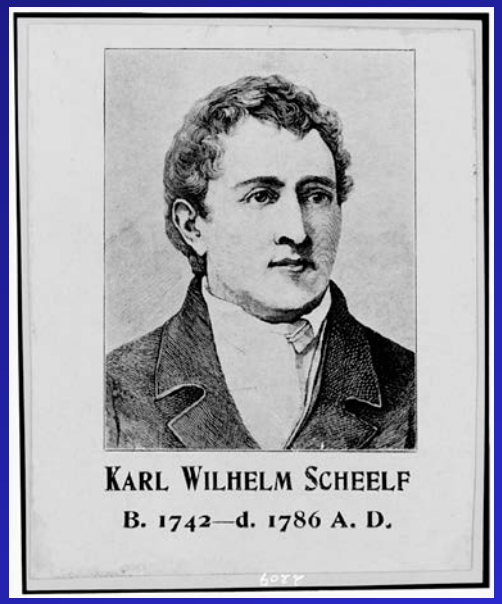
– Accordingly, better described and defined by

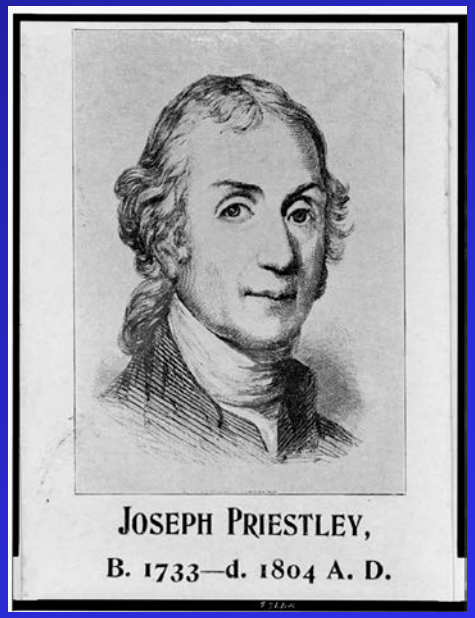
- Apgar scores
- Fetal acid-base status at birth
  - Umbilical-Cord Blood Gas Analysis

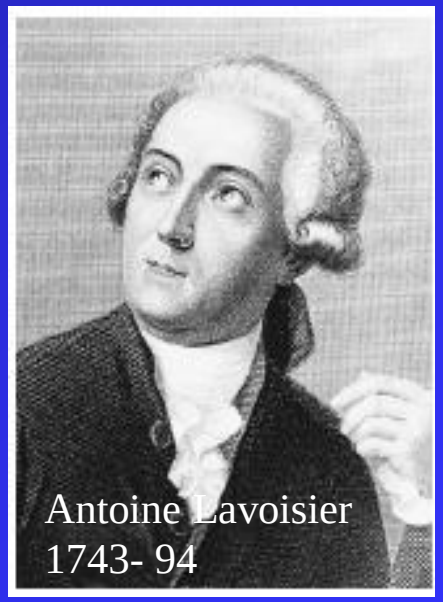
(lack of) Oxygen (O2)

At the end of the day it is all about the presence or abscence of oxygen (O2)

Who discovered oxygen first ?







”Hard-luck Scheele” made a number of chemical discoveries - before others who are generally given the credit for it..

… but here is where fetal surveillance started…

Figure 1.1 Jacques Alexandre de Kergaradec, robed as a Membre de l'Academie de Medicine Paris. (With thanks to Professor J. H. M. Pinkerton, Emeritus Professor of Midwifery and Gynaecology, Queen's University of Belfast)

Intrapartum fetal surveillance

- **1821** First auscultation of FHR
  - *Kergaradec, Geneve*
- **1833** Observations on obstetric auscultation
  - *Kennedy, Dublin*
- **1897** Spasticity might arise in fetal life
  - *Freud, Wien*

Intrapartum fetal surveillance

- **1906** First fetal ECG
  - *Cremer, Germany*

- **1908** First fetal phonocardiogram
  - *Hoffbauer Weiss, Germany*

- **1958** CTG / EFM - *Hon, USA*

- **1958** First Umbilical Cord Blood Gas Analysis
  - *James, USA (N.Z.)*

The Journal of Pediatrics

VOL. 52 APRIL, 1958 No. 4

THE ACID-BASE STATUS OF HUMAN INFANTS IN RELATION TO BIRTH ASPHYXIA AND THE ONSET OF RESPIRATION

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D. A. HOLADAY, M.D., AND V. APGAR, M.D.
NEW YORK, N. Y.

Intrapartum fetal surveillance

- **1961** scalp-pH *Saling, Berlin*
- **1968** scalp-lactate *Monti, Milan*
- **1974** continuous tissue-pH *Stamm, Lausanne*
- **1978** transcutaneous pO_2 and pCO_2 *Huch, Marburg*

Clinical purpose of cord blood gas analysis

- Determine neonatal acid-base status at birth for the detection of birth asphyxia
- Possible assessment tool to document quality of care within obstetrical units
- Documentation of neonatal acid base status at birth in case of litigation towards obstetricians, midwives or obstetrical departments

Facts & figures

Globally, 4 - 9 million neonates suffer from asphyxia each year [1]

1.2 million neonates die from birth asphyxia and about the same number develop severe disabilities [1]

29% of global neonatal deaths are caused by birth asphyxia [1]

1. **Omo-Aghoja L. Maternal and fetal acid-base chemistry:** A major determinant of outcome. Annals of Medical and Health Sciences Research 2014; 4: 8-17

Umbilical-Cord Blood Gas Analysis

- Umbilical-Cord Blood Gas Analysis (UCBGA) provides important information about the past, present and – to some degree – future condition of the newborn infant
- Now recommended in all high-risk deliveries by both ACOG and RCOG
- In many countries, like in Denmark, and in many centres UCBGA is now a routine procedure following all deliveries

Umbilical-Cord Blood Gas Analysis

- UCBGA is of increasing clinical importance,

and in many countries (like in the US and UK) also of medicolegal importance

Clinicians should be familiar with:

- the background to interpret the blood gas values
- the practice to obtain the samples

UCBGA - Clinicians should be familiar with:

- Maternal – fetal gas exchange
- Development of asphyxia
- Normal and pathological values of cord blod gasses
- Factors influencing the blood gasses
- Evaluation and interpretation of fetal acidosis

UCBGA - Clinicians should be familiar with:

- Respiratory acidosis and metabolic acidosis
- Significance of different combinations of acidosis and Apgar scores
- Factors influencing the umbilical cord blood gasses
- Arterio-venous differences and their significance

UCBGA - Clinicians should be familiar with:

- Different prognostic features
- Sampling procedures
- Storage

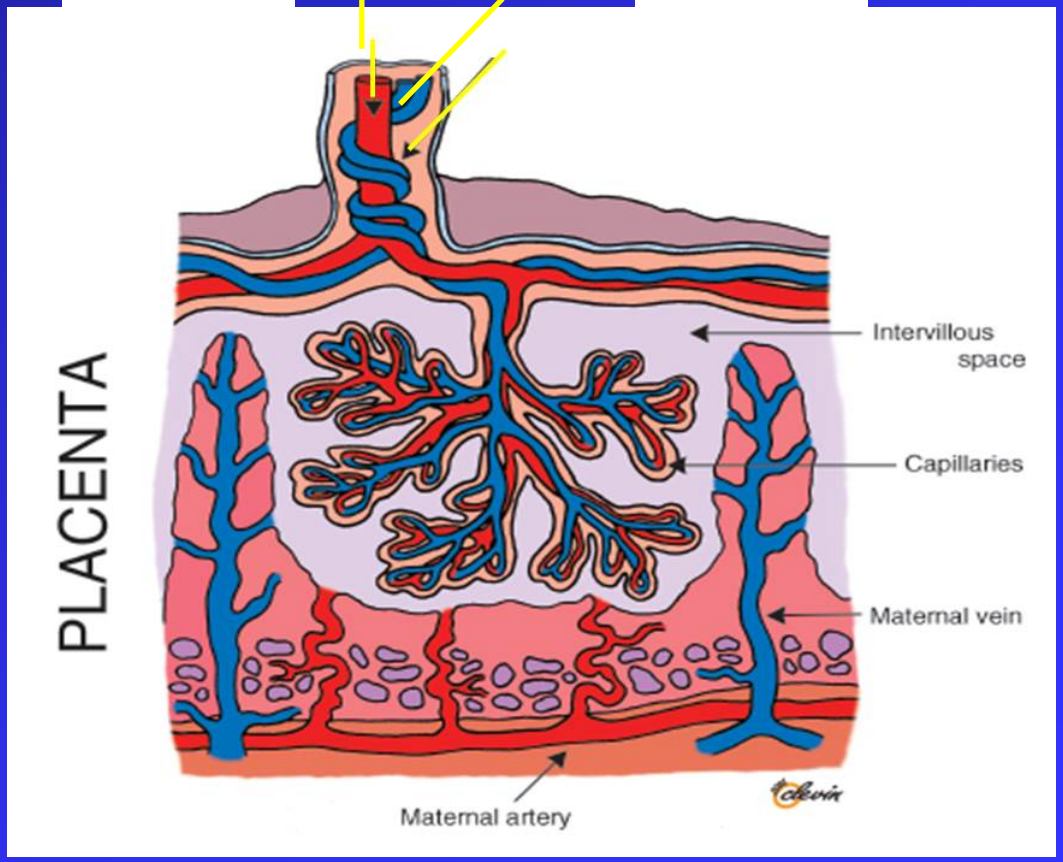
Placental anatomy and physiology

Cord **artery** blood reflects fetal acid-base status whereas the **vein blood** reflects the oxygen (and nutritional) supply form the placenta

Preferably parameters derived from **both** cord **artery and vein blood** are used to assess neonatal condition at delivery

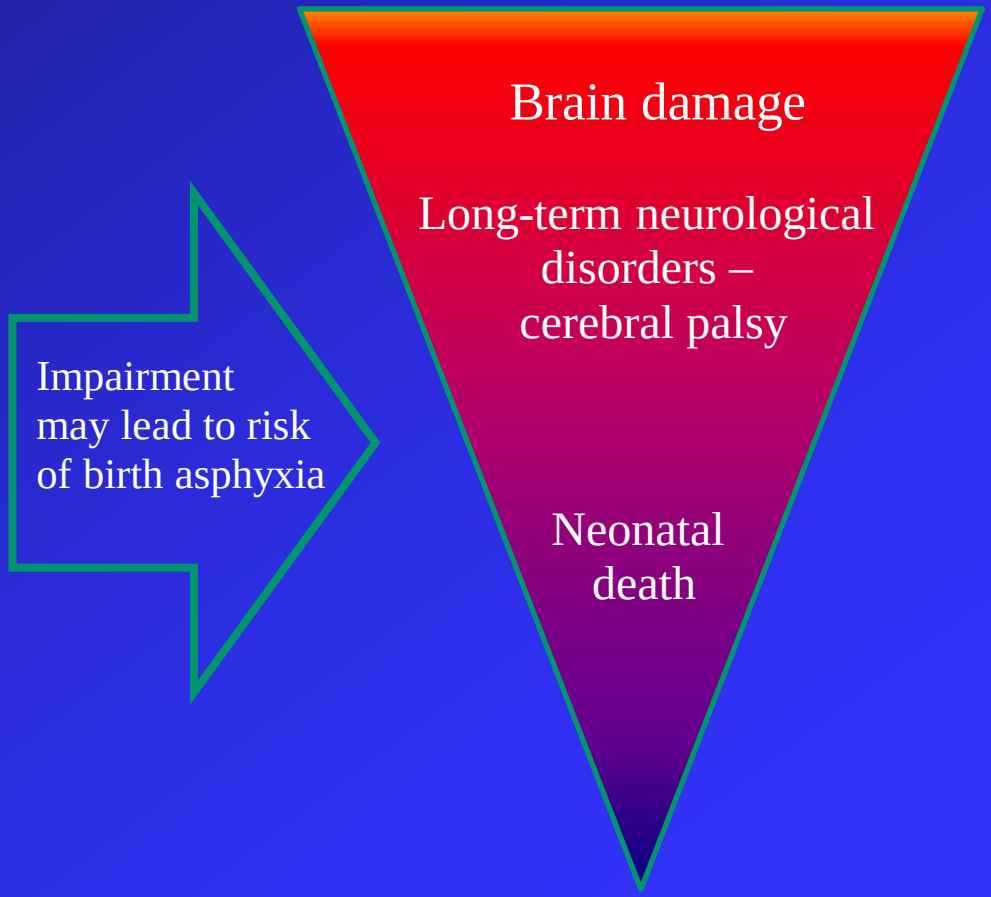
One large cord **vein** carries oxygenated blood and nutrient to the fetus

Two small cord **arteries** carry deoxygenated blood and waste products (CO2) from the fetus



Understanding gas exchange during labour

- Adequate supply of oxygenated maternal blood reaching placenta
- Gas exchange across placenta
- Supply of oxygenated blood to fetus through open umbilical vein
- Sufficient metabolic reserve in fetus to withstand “hypoxic effect” of uterine contractions



What can cause foetal hypoxia/asphyxia:

Cause:	**Effect:**
Maternal hypotension - suppine position, anaesthesia, vasodilation (epidural)	Utero-placental flow ↓
Maternal hypoventilation - apnoe /eclampsia	Maternal pO_2 / SO_2 ↓
Maternal cathecolamines ↑ (adrenalin) ↑ fear, pain, stress	Utero-placental flow ↓ (from animal experiments)

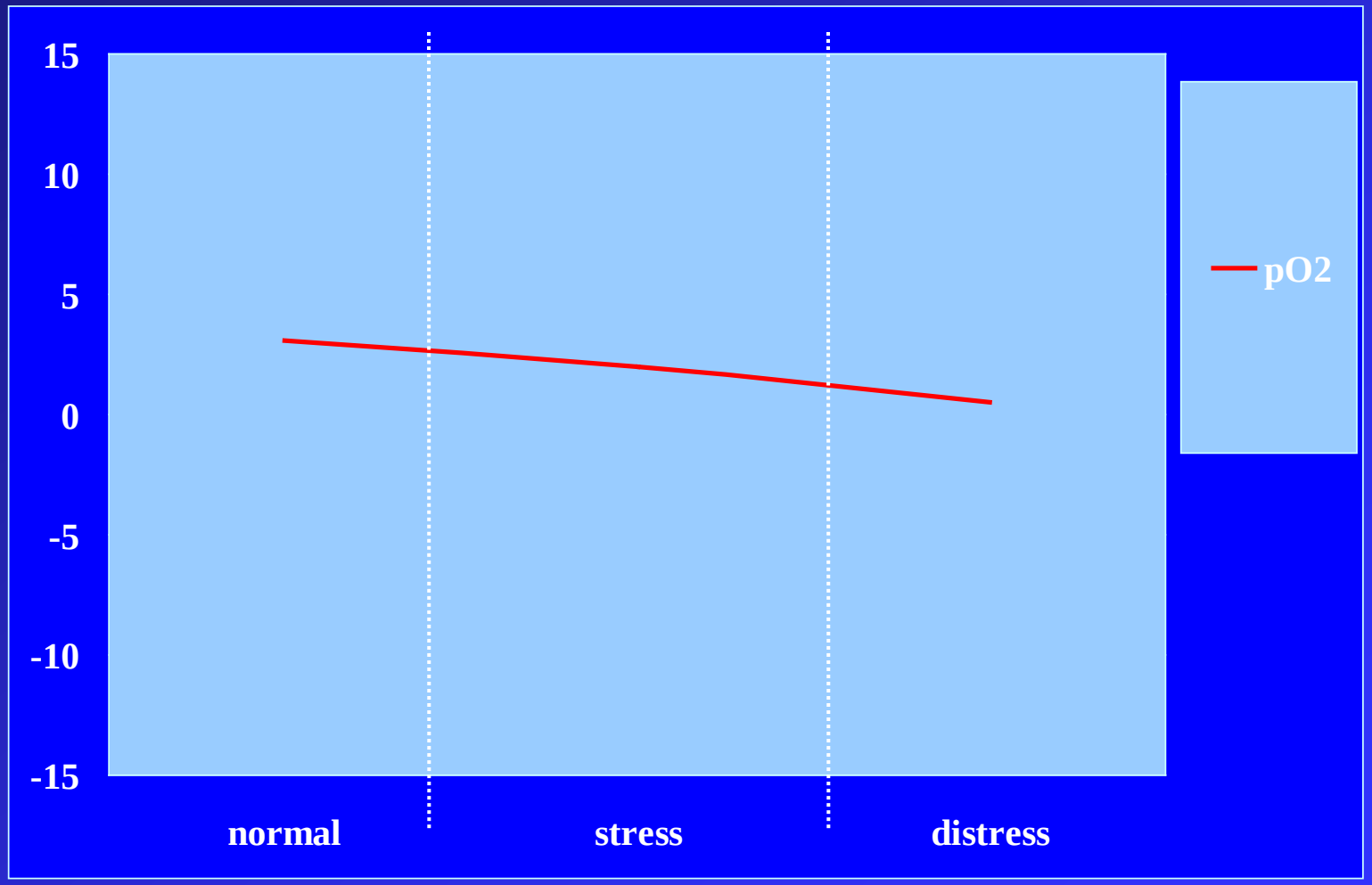
What can cause foetal hypoxia/asphyxia:

Cause:	**Effect:**
Uterine hypertonia hyperstimulation overefficient uterine activity	Utero-placental flow ⇓
Cord compression - oligohydramnios, (maternal) position, breech, cord entanglement, nuchal cord prolapse	Foeto-placental flow ⇓ decreased/blocked O_2/CO_2 - exchange
Placental abruption / insufficiency	Foeto-placental flow ⇓ decreased/blocked O_2/CO_2 exchange

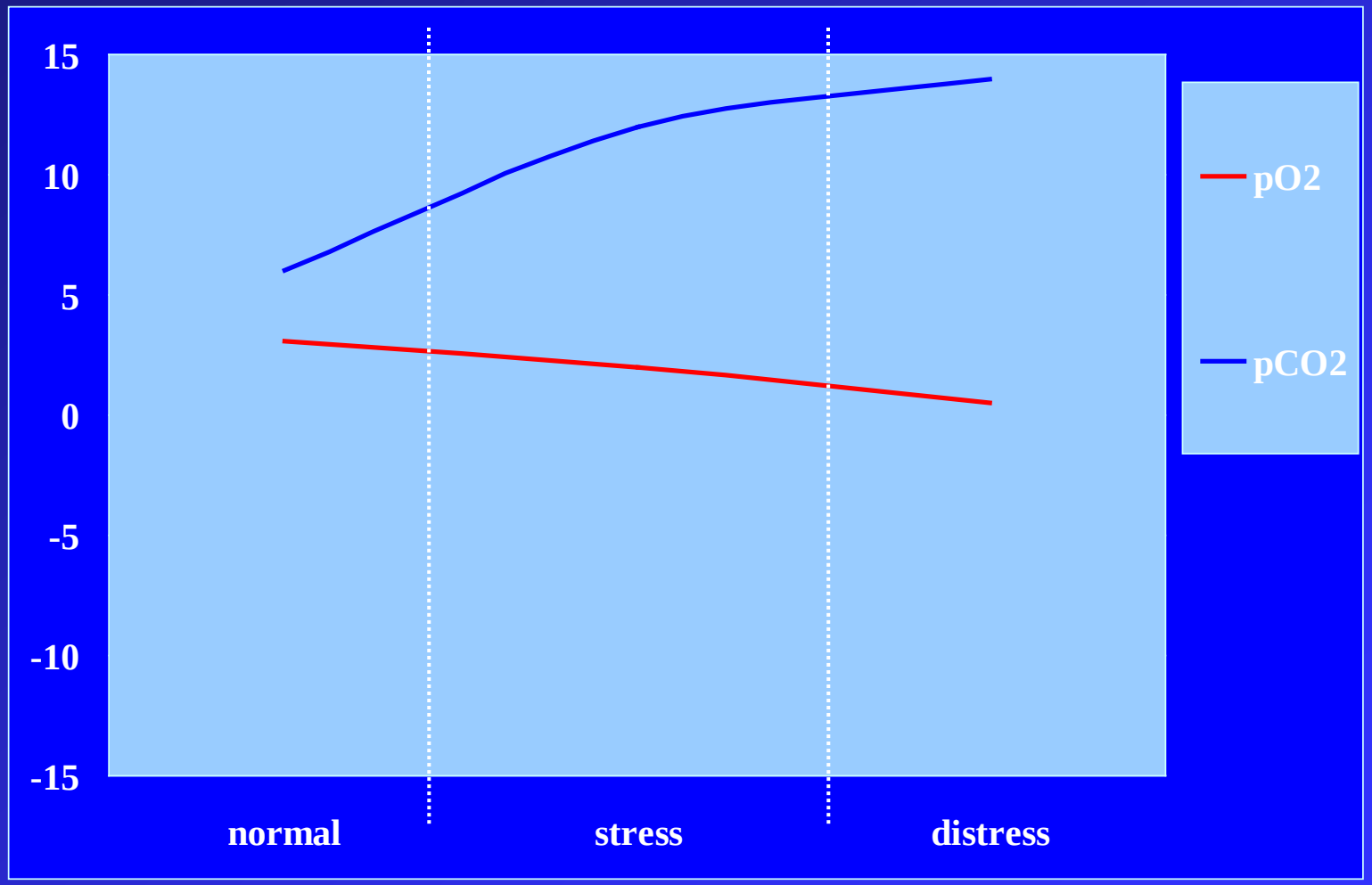
Cord entanglement, a knot – or rather ”a tie”

Protective amniotic (sac) fluid

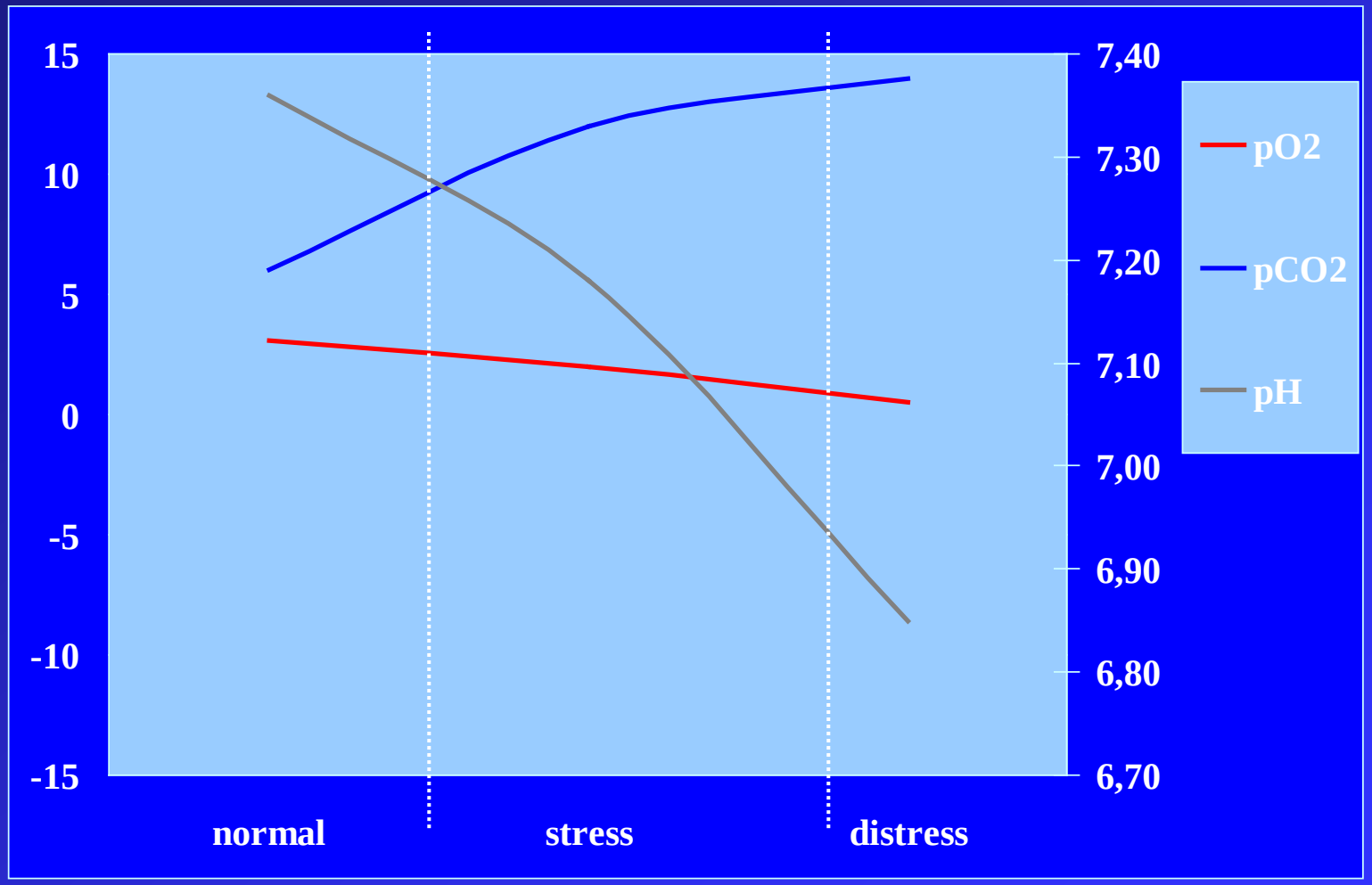
Asphyxia during labour pO_2



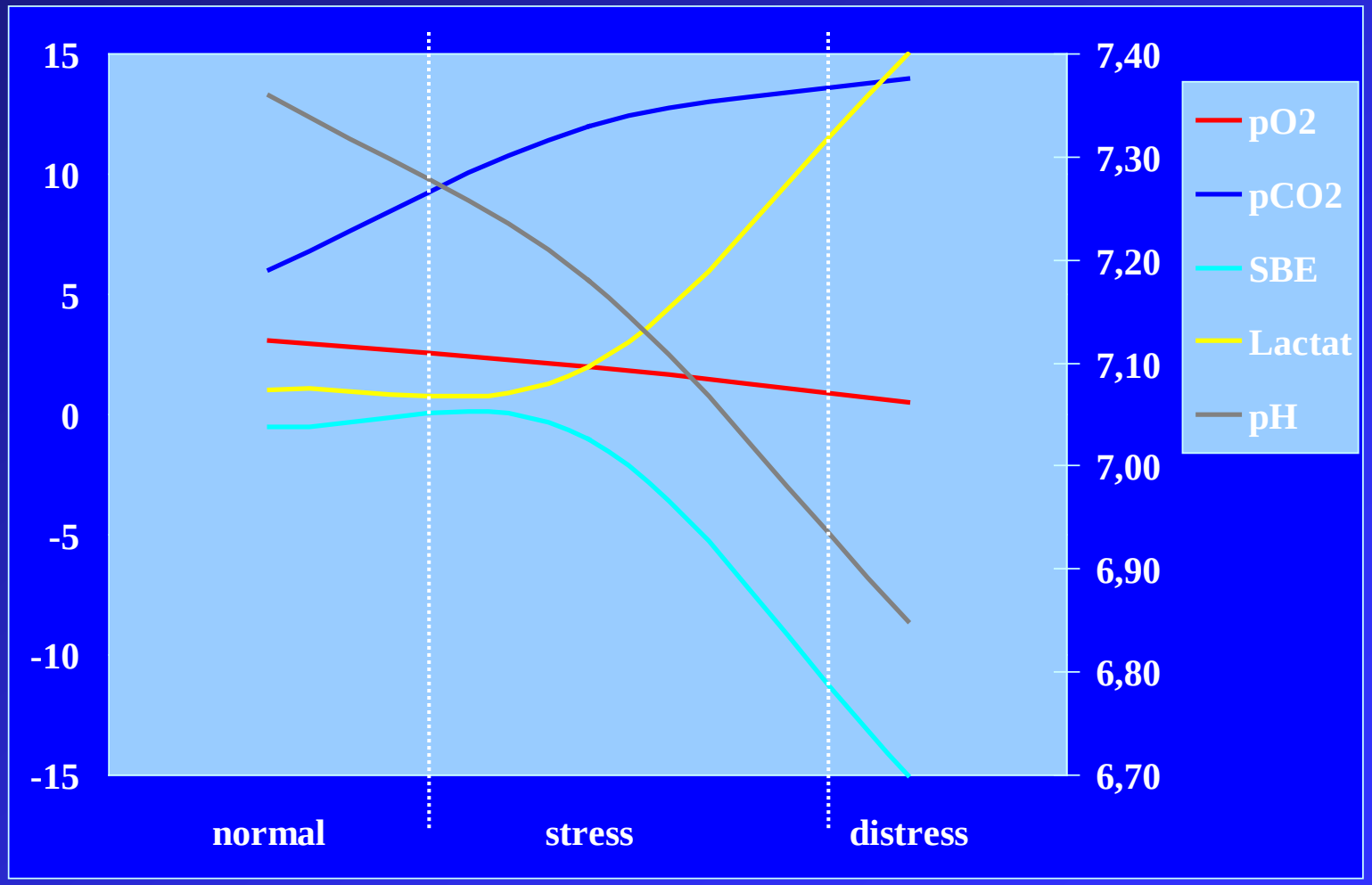
Asphyxia during labour pCO_2



Asphyxia during labour pH

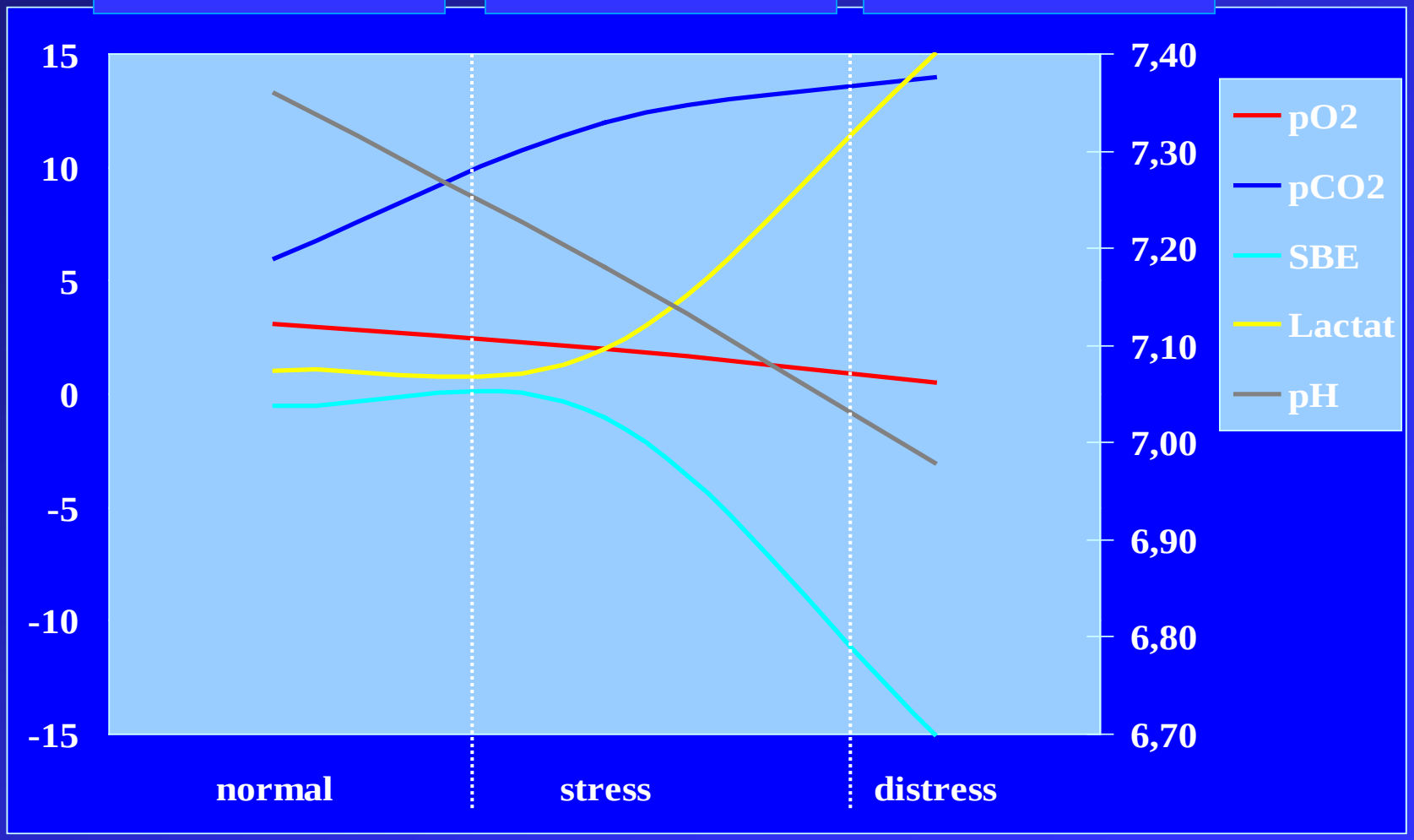


Asphyxia during labour SBE, lactate

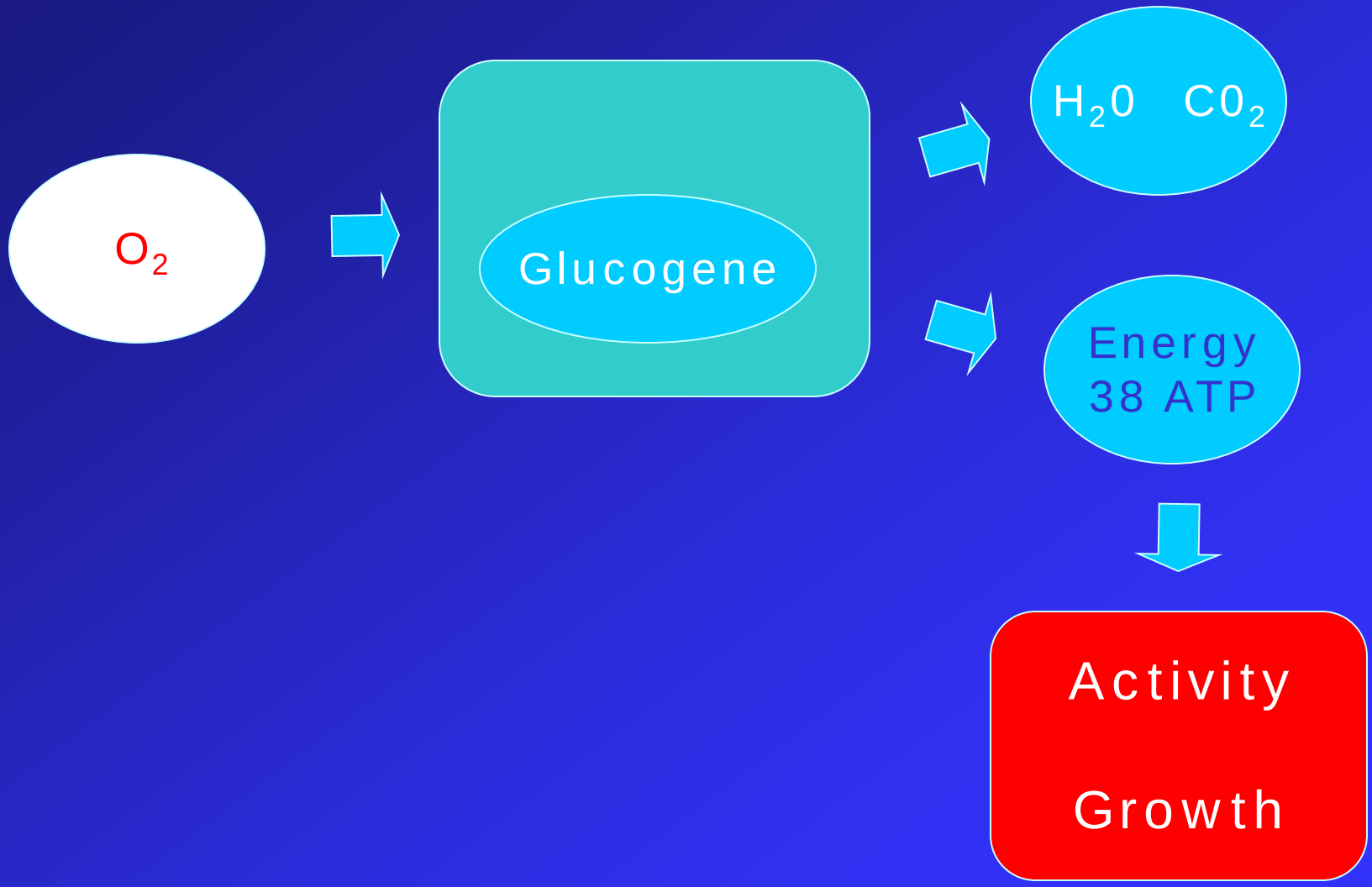


Asphyxia during labour

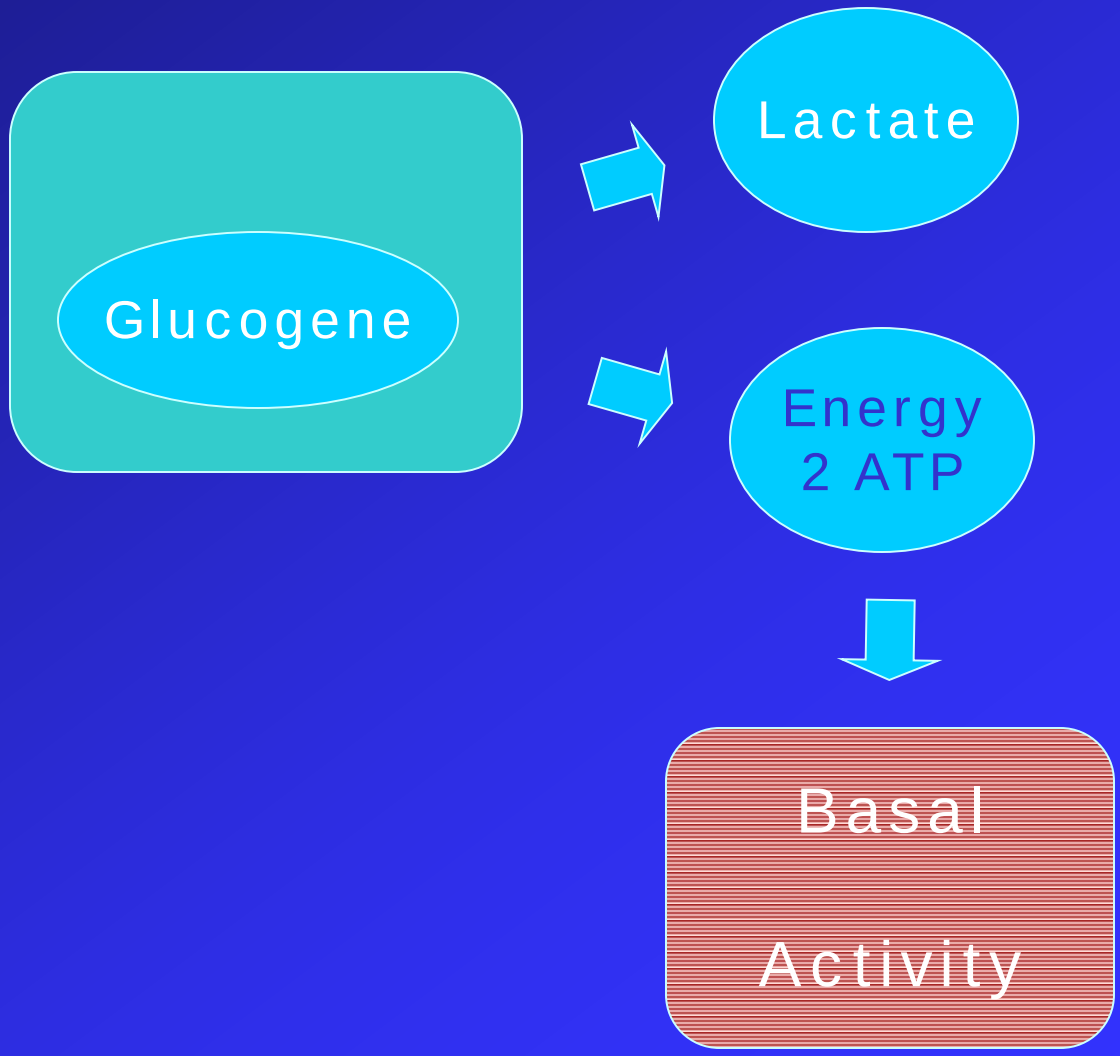
Pre-acidotic | Respiratory acidosis | Metabolic acidosis



Aerobic metabolism



Anaerobic metabolism



Fetal physiology during labour

Pre-acidotic period

- Increasing oxygen utilisation (Bohr effect)
- Decreasing activity

Fetal physiology during labour – preacidotic period

Blodgas-værdier		
pH	7,282	
pCO_2	5,85	kPa
pO_2	3,97	kPa
Syre-Base-status		
$cHCO_3^-(P)_c$	20,0	mmol/L
$ctCO_2(P)_c$	21,4	mmol/L
ABE_c	-6,1	mmol/L
SBE_c	-5,5	mmol/L
$cHCO_3^-(P,st)_c$	18,7	mmol/L
Oximetri-værdier		
ctHb	8,6	mmol/L
FO_2Hb	0,598	
FMetHb	0,008	
FCOHb	0,008	
sO_2	0,608	
Elektrolyt-værdier		
cK^+	4,4	mmol/L
cNa^+	134	mmol/L
cCa^{2+}	1,47	mmol/L
Beregnede værdier		
$cCa^{2+}(7.4)_c$	1,38	mmol/L
Metabolit-værdier		
cLac	3,5	mmol/L

Noter

Advarsel: Der er detekteret og korrigeret for HbF

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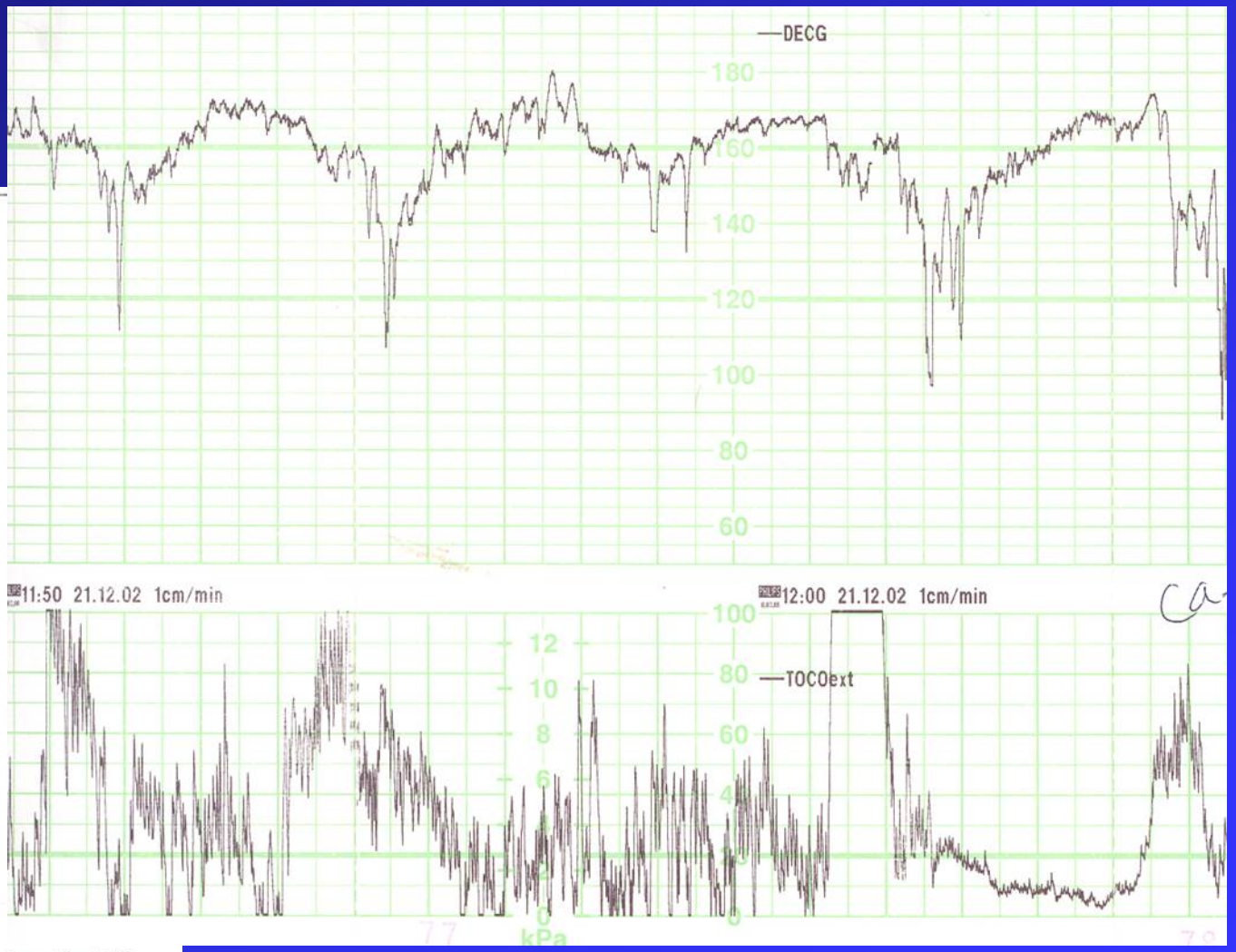

Fetal physiology during labour

Respiratory (hypercapnic) acidosis

- release of stress hormones
- redistribution of foetal blood flow
- anaerobic metabolism in peripheral tissue

Fetal physiology during labour - Respiratory acidosis

Blodgas-værdier		
pH	7,116	
pCO_2	9,20	kPa
pO_2	0,14	kPa
Syre-Base-status		
$cHCO_3^-(P)_c$	21,3	mmol/L
$ctCO_2(P)_c$	23,4	mmol/L
ABE_c	-10,5	mmol/L
SBE_c	-6,8	mmol/L
$cHCO_3^-(P,st)_c$	14,4	mmol/L
Oximetri-værdier		
ctHb	11,1	mmol/L
FO_2Hb	0,035	
FMetHb	0,012	
FCOHb	0,003	
sO_2	0,036	
Elektrolyt-værdier		
cK^+	4,3	mmol/L
cNa^+	134	mmol/L
cCa^{2+}	1,55	mmol/L
Beregnede værdier		
? $cCa^{2+}(7.4)_c$	1,31	mmol/L
Metabolit-værdier		
cLac	11,0	mmol/L

Noter

Ca (7,4) kan ikke benyttes

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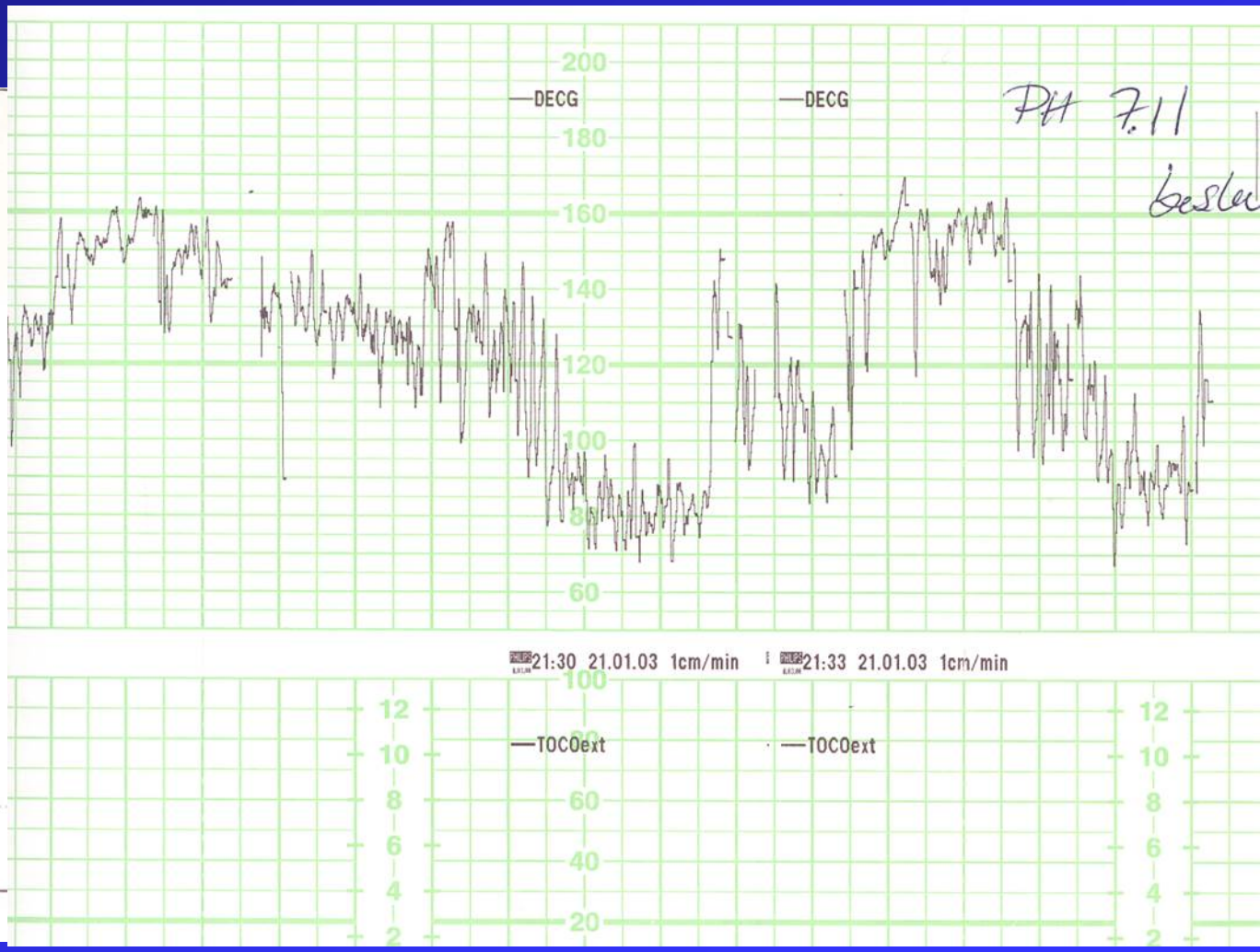


Fetal physiology during labour

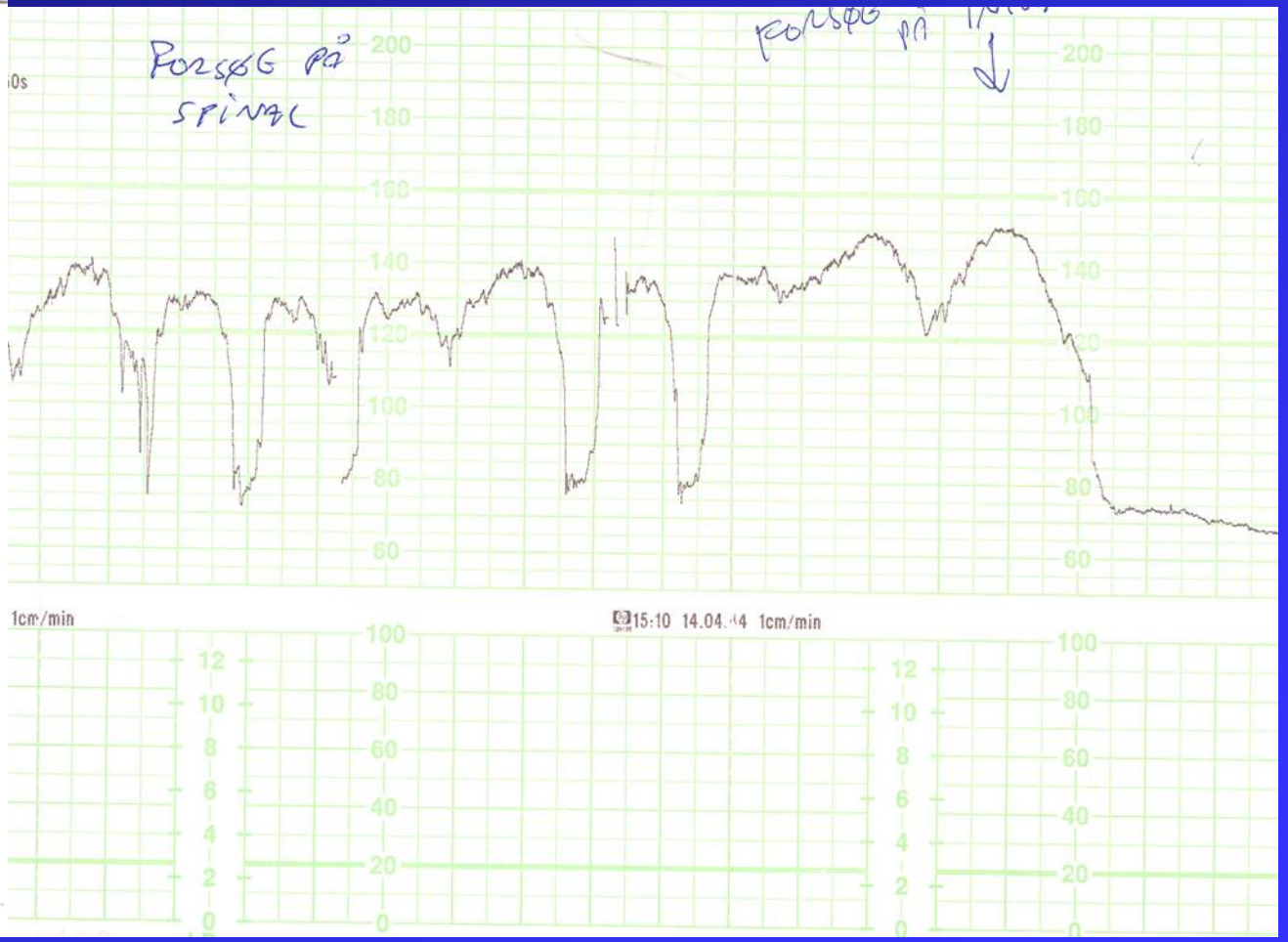
Metabolic acidosis

- anaerobic metabolism in vital organs
- risk of heart and brain failure

Fetal physiology during labour - metabolic acidosis

Blodgas-værdier		
pH	6,904	
pCO_2	13,5	kPa
pO_2	0,01	kPa
Syre-Base-status		
$cHCO_3^-(P)_c$	19,0	mmol/L
$ctCO_2(P)_c$	22,1	mmol/L
ABE_c	-19,8	mmol/L
SBE_c	-12,4	mmol/L
$cHCO_3^-(P,st)_c$	9,5	mmol/L
Oximetri-værdier		
ctHb	10,9	mmol/L
FO_2Hb	0,018	
FMetHb	0,013	
FCOHb	0,011	
sO_2	0,018	
Elektrolyt-værdier		
↑ cK^+	6,1	mmol/L
cNa^+	135	mmol/L
cCa^{2+}	1,66	mmol/L
Beregnede værdier		
? $cCa^{2+}(7.4)_c$	1,22	mmol/L
Metabolit-værdier		
cLac	15	mmol/L

Ioter



Normal and pathological values of cord blood gasses

Table 1 Studies reporting umbilical cord values for term and preterm infants

	Umbilical artery				Umbilical vein					
Author	pH	Base excess (mmol/l)	Pco_2 (kPa)	Po_2 (kPa)	pH	Base excess (mmol/l)	Pco_2 (kPa)	Po_2 (kPa)	Number	Population studied
Victory *et al*[21] 2004	7.24 (0.07)	−5.6 (3.0)			7.33 (0.06)	−4.5 (2.4)			20 456	Term non-anomalous singletons
Helwig *et al*[22] 1996	7.26 (0.07)	−4.0 (3.0)	7.05 (1.33)	2.26 (0.8)	7.34 (0.06)	−3.0 (3.0)	5.45 (0.93)	3.86 (0.93)	15 073	All gestations, all delivery types, Apgar >7[5]
Thorp *et al*[18] 1989	7.24 (0.07)	−3.6 (2.7)	7.49 (1.14)	2.38 (0.92)	7.32 (0.06)	−2.9 (2.4)	5.83 (0.89)	3.82 (0.97)	1694a 1820v	Term, nulliparous, SOL, all delivery types
Riley and Johnson[20] 1993	7.27 (0.07)	−2.7 (2.8)	6.69 (1.48)	2.45 (1.09)	7.34 (0.06)	−2.4 (2.0)	5.41 (1.05)	3.79 (1.02)	3522	Term singleton infants, vaginal delivery
Dickinson *et al*[23] 1992	7.26 (0.08)	−3.2 (2.9)	7.05 (1.33)	2.53 (1.05)	7.33 (0.07)	−2.6 (2.5)	5.77 (1.1)	3.88 (1.29)	1393a 1526v	Preterm (24–36 weeks), normal CTG

Data are presented as mean (SD). Arterial (a) and venous (v) sample numbers are given separately where available.
CTG, cardiotocogram; SOL, spontaneous onset of labour; SVD, spontaneous vertex delivery.

	Umbilical artery	Umbilical vein
pH	7.24-7.27	7.32-7.34
BE (mmol/l)	-2.7 - -5.6	-2.4 - -4.5
pCO2 (kPa)	6.69-7.49	5.54 – 5.83
pO2	2.26-2.45	3.79 – 3.88

… values in mm Hg, Lactate - and human adult values for comparison

Table I. Median ranges for umbilical cord blood gas, base excess and lactate values [8].

	Umbilical Artery (n =12,345)	Umbilical Vein (n =12,345)	Adult artery (non-cord) blood values (for comparison only)
pH median	7.27	7.35	7.40
pO_2 median (kPa)	2.2	3.7	12.0
pO_2 median (mm Hg)	16.3	27.9	90
pCO_2 median (kPa)	7.3	5.4	5.3
pCO_2 median (mmHg)	55.1	40.4	40
Base excess (mmol/L)	-3.00	-3.00	0
Lactate (mmol/L)	3.7		1.0

8. White C *et al*. Benefits of introducing universal cord blood gas and lactate analysis into an obstetric unit. Australia and New Zealand J of Obstetrics and Gynaecology 2010; 50: 318-28.

Factors influencing the UC blood gasses

- Mode of delivery
- Gestational age
- Parity
- Fetal presentation (Breech)
- Cord entanglement
- Oligohydramnios
- Multiple pregnancies
- Regional anesthesia

- (Fever – chorionamnitis)

Arterio-venous differences and their significance

British Journal of Obstetrics and Gynaecology
December 1994, Vol. 101, pp. 1054–1063

OBSTETRICS

Umbilical cord blood gas analysis at delivery: a time for quality data

JENNIFER WESTGATE *Lecturer/Honorary Senior Registrar*, JONATHAN M. GARIBALDI *Research Assistant*,
KEITH R. GREENE *Consultant/Honorary Senior Lecturer*

Perinatal Research Group, Postgraduate Medical School, Department of Obstetrics, Derriford Hospital, Plymouth

Conclusions Both artery and vein cord samples must be taken and the results screened to ensure separate vessels have been sampled. Interpretation of the results requires the examination of pCO_2 and base deficit of the extracellular fluid from each vessel as well as the pH. Confusion about the value of cord gas measurements may be due to the use of erroneous data and inadequate definitions of acidosis which do not differentiate between respiratory and metabolic components.

Verifying that both cord artery - and vein sample was obtained

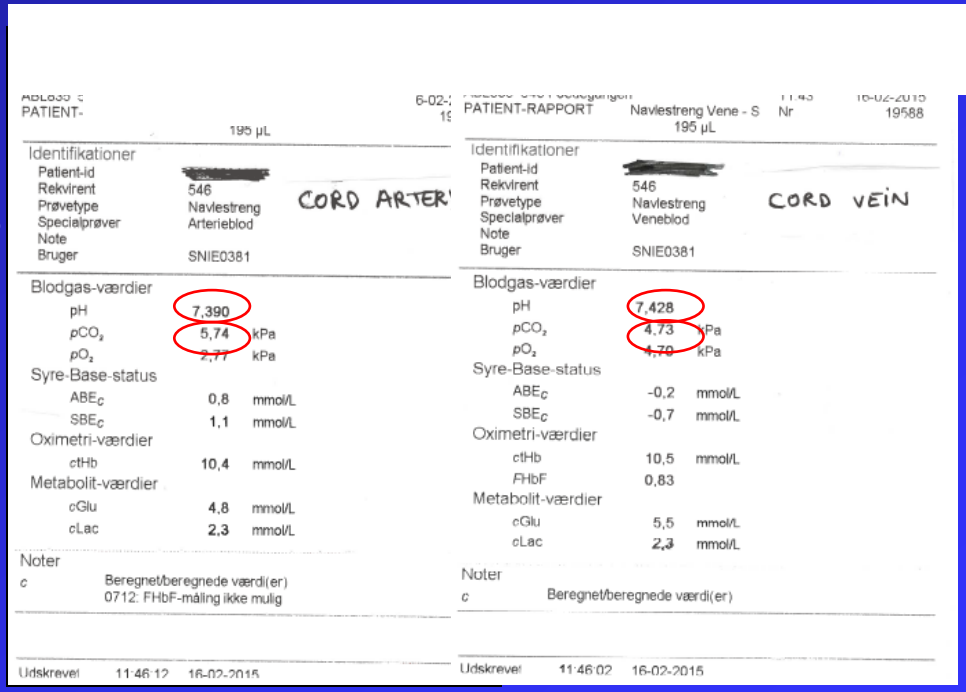
Blood from both cord artery and cord vein should preferably be collected and analyzed

To validate that a sample form cord artery has truly been obtained:

- Arterio-venous (A-V) differences for:

  pH > 0.02

  pCO_2 > 0.5 kPa/3.75 mmHg



PATIENT-

195 µL

Identifikationer

Patient-id	[redacted]
Rekvirent	546
Prøvetype	Navlestreng
Specialprøver	Arterieblod
Note	CORD ARTER
Bruger	SNIE0381

Blodgas-værdier

pH	7,390	
pCO_2	5,74	kPa
pO_2	2,77	kPa

Syre-Base-status

ABE_c	0,8	mmol/L
SBE_c	1,1	mmol/L

Oximetri-værdier

ctHb	10,4	mmol/L

Metabolit-værdier

cGlu	4,8	mmol/L
cLac	2,3	mmol/L

Noter

c Beregnet/beregnede værdi(er)
0712: FHbF-måling ikke mulig

Udskrevet 11:46:12 16-02-2015

PATIENT-RAPPORT Navlestreng Vene - S Nr 19588

195 µL

Identifikationer

Patient-id	[redacted]
Rekvirent	546
Prøvetype	Navlestreng
Specialprøver	Veneblod
Note	CORD VEIN
Bruger	SNIE0381

Blodgas-værdier

pH	7,428	
pCO_2	4,73	kPa
pO_2	4,79	kPa

Syre-Base-status

ABE_c	-0,2	mmol/L
SBE_c	-0,7	mmol/L

Oximetri-værdier

ctHb	10,5	mmol/L
FHbF	0,83	

Metabolit-værdier

cGlu	5,5	mmol/L
cLac	2,3	mmol/L

Noter

c Beregnet/beregnede værdi(er)

Udskrevet 11:46:02 16-02-2015

Insight into cause of acid-base disturbance

1. American College of Obstetricians and Gynecologists Committee on Obstetric Practice. Umbilical cord blood gas and acid-base analysis. Obstet Gynecol 2006; 108: 1319-22.
2. Westgate J *et al*. Umbilical cord blood gas analysis at delivery: a time for quality data. Br J of Obstetrics and Gynaecology 1994; 101: 1054-63.

Interpretation of low and high A-V differences – in relation to acidosis and aphyxia

- A wide difference between umbilical artery and vein blood gas values is often due to an obstructed cord as for instance ”nuchal cord” (*Martin*)
- A small difference is most likely caused by impairment of maternal perfusion of the placenta as in case of placental abruption *(Johnson)*
- When UcA-pH < 7.0 : The magnitude of A-V difference in pCO2 is directly correlated to the risk of developing HIE (*Belai*)

Interpretation of low and high A-V differences – in relation to acidosis and aphyxia

For prognostic value -

- It is of outmost importance to sample both arterial and venous blood for bloodgasses – when the newborn is depressed, as…

- normal UcV blood gasses in the case of an obstructed umbilical cord

- could ”hide” a severe acidosis with a high risk of an adverse outcome

Normal Cord Blood pH (both artery and vein) at birth does not entirely exclude acute intrapartum asphyxia:

- Sudden an total obstruction of cord vessels
- Sudden fetal cardiac arrest
- .. in these cases blood gasses taken post partum would reveal severe acidosis

• What is severe fetal acidosis?

- Most authors agree on ph < 7.0 as severe acidosis
- Prevalence: 0.4 – 1 %
- Low pH in combination with other abnormal clinical patterns (e.g. cardio-pulmonary) is associated with high risk of poor long-term outcome
- This also counts for pathological intrapartum findings

25-25 term newborns	seizures	no seizures	p-value
pH	6.84 ± 0.12	6.89 ± 0.11	NS
BD	-18.1 ± 9.1	-16.6 ± 6.1	NS
Baseline FHR	143 ± 11	146 ± 16	NS
Bradycardia	56%	84%	‘0.06’
Decelerations	36, 32 %	50, 52 %	NS
Accelerations	24%	48%	NS
Min/absent variab.	64%	36%	‘0.08’
Duration abnormal	72 ± 12 min	36 ± 18 min	0.001

Williams and Galerneau. J Perinat Med 2004; 32: 422-5

- Signifance of different combinations of acidosis and Apgar scores

Low pH - but normal Apgar scores:

- Short period of acidosis (most likely respiratory)
- Fair prognosis

• Signifance of different combinations of acidosis and Apgar scores

Normal pH - but low Apgar scores:

- Chronically sick child
  - no hypoxia during the last part of the delivery
- Earlier condition of e.g. hypoxia, infection, malformation or prematurity
- Prognosis - depending on the cause

• Signifance of different combinations of acidosis and Apgar scores

Low Apgar scores - and low pH:

- Severe asphyxia - of a certain duration
  - during labour
  (most likely metabolic acidosis)

- Prognosis: pH – but also BE (lactate)
  is of prognostic importance

pH is no ideal measure for cumultative exposure to acidosis due to anaerobic metabolism

- pH is logarithmic (nor linear) -
  directly correlated to pCO2 accumulation
- Base excess provides a more linear measure of the accumulation of metabolic acid

  - adjusted for pCO2

- Sampling procedures
- Storage

The cord blood sampling should be performed by *either* method 1 *or* method 2:

Method 1:

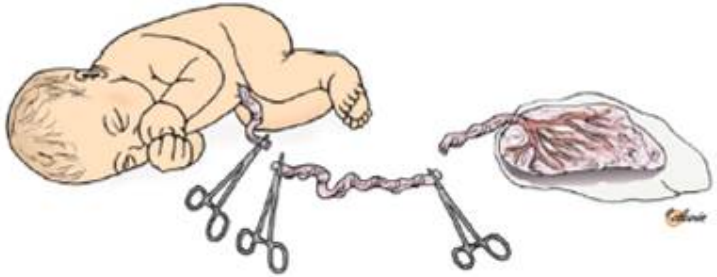
The cord blood sample must be collected immediately and within one minute after delivery of the neonate.

So, the blood is collected before the placenta is delivered and before the cord is clamped and separated from the neonate [27]

Method 2:

A segment of the cord must be isolated immediately and within one minute after delivery of the neonate.

When the cord and placenta are separated from the neonate, the cord segment is placed on the delivery table.



The cord blood sample must be collected within 60 minutes after delivery [27-29]

For method 1 and 2 the collection should be performed as follows:

For method 1 and 2 the collection should be performed as follows:

Collect the cord *artery* sample first. Push the plunger down as far as it can go. Insert the cannula parallel to the artery; pull the plunger for collection of the cord artery blood sample.

Then collect the cord *vein* sample. Push the plunger down as far as it can go. Insert the cannula parallel to the vein; pull the plunger for collection of the cord vein blood sample.

Delayed cord clamping

In recent years there has been increasing acceptance of delaying the cord clamping procedure by 2-3 minutes after delivery for the benefit of placental blood transfusion (extra blood volume) to the neonate [39].

A recent Cochrane review of studies in this area concluded that the benefit to the neonate associated with delayed cord clamping (higher birth weight, increased hemoglobin concentration and iron reserves) outweighs the increased risk of jaundice. It states that a more liberal approach to delayed cord clamping is warranted [39]. The policy of delayed cord clamping clearly poses a potential problem for accurate assessment of neonatal acid-base status at the moment of delivery, because of the "hidden acidosis" phenomenon (See section

A solution to this problem has been validated by the results of two recent clinical studies [30, 40]. The solution, which is standard practice in some units, is to sample blood directly from the still pulsating unclamped umbilical cord, at the moment of delivery, rather than from a separated clamped cord segment. This way there is no risk of "hidden acidosis" and the neonate can take advantage of the delayed clamping.

Storage

- Double-clamped (10 cm) piece of cord
  - or in syringe
- On ice – for up to 60 minutes…..

Asphyxia - prognosis

- Apgar score - by it self -
  has a poor prognostic value

- Both the Apgar score - as well as pH / BE -
  should be used to more precisely
  predict the prognosis at birth

Asphyxia – prognosis
Does pH correlate to longterm outcome?

BMJ

RESEARCH

Strength of association between umbilical cord pH and perinatal and long term outcomes: systematic review and meta-analysis

Gemma L Malin, clinical research fellow,[1] Rachel K Morris, clinical research fellow,[1] Khalid S Khan, professor of obstetrics, gynaecology, and clinical epidemiology[1,2]

Cite this as: *BMJ* 2010;340:c1471

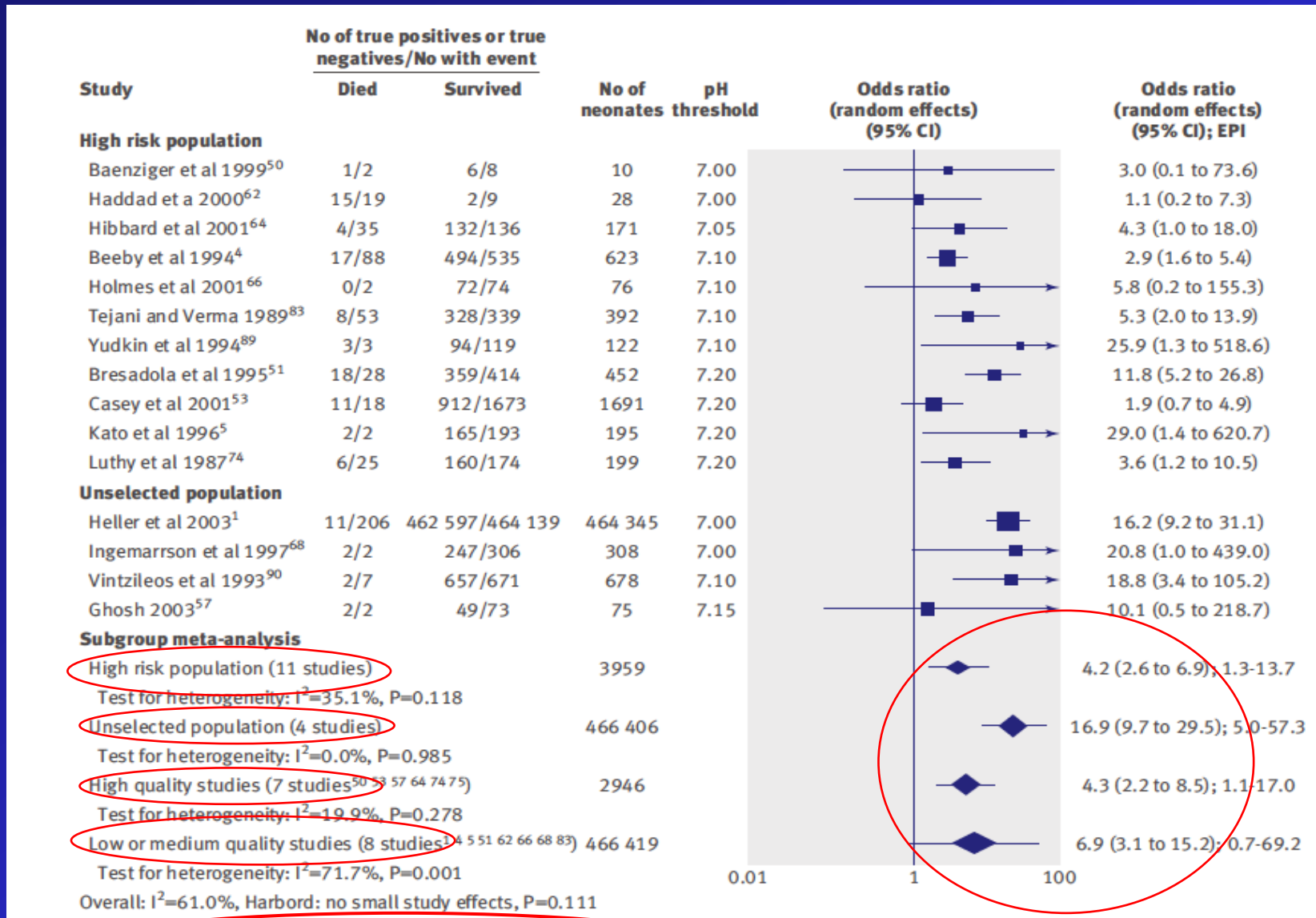
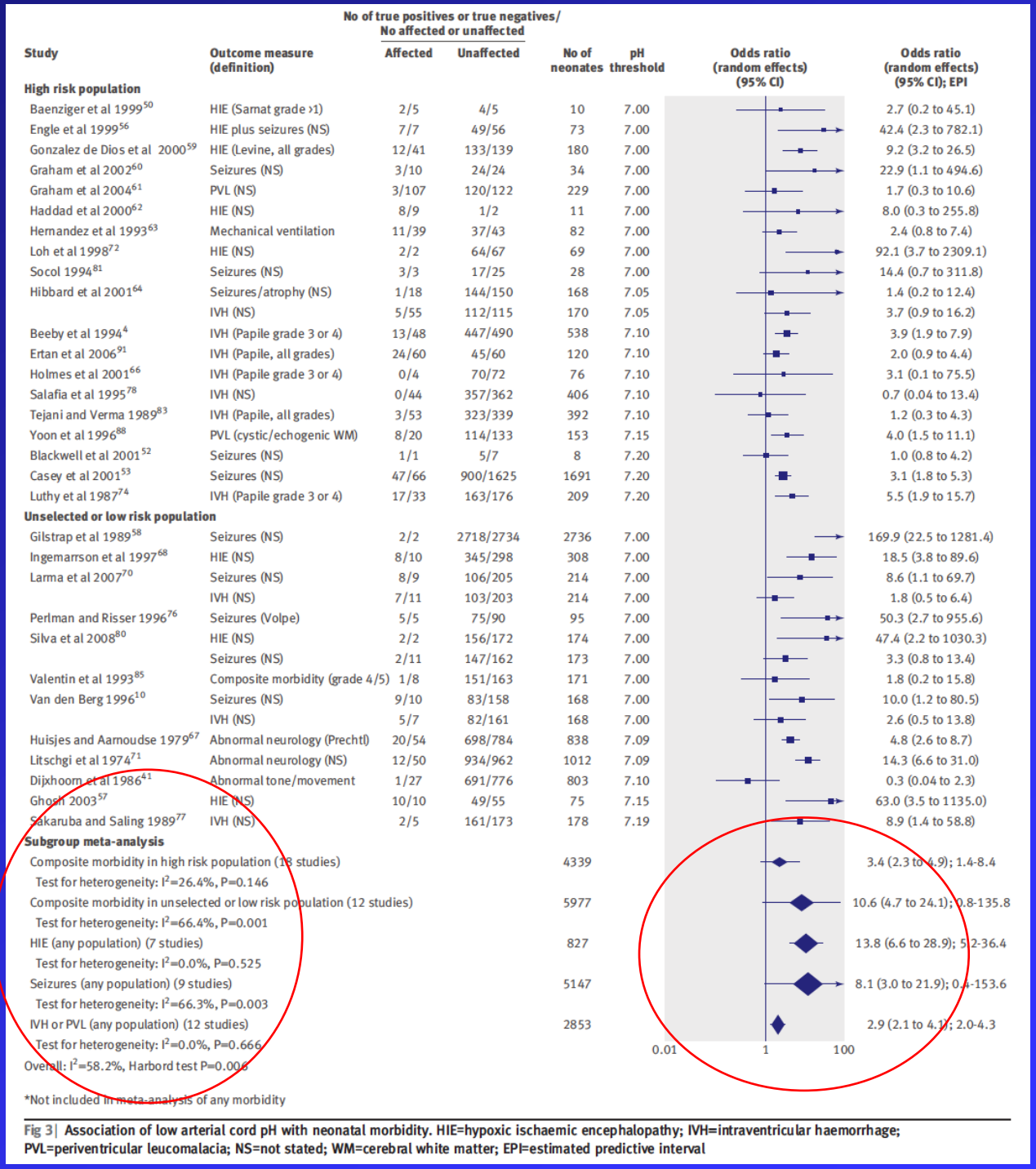


Fig 2 | Association of low arterial cord pH with neonatal mortality. EPI=estimated predictive interval

Association of : low arterial cord pH - with neonatal morbidity



Study	Outcome measure (definition)	No of true positives or true negatives/ No affected or unaffected: Affected	Unaffected	No of neonates	pH threshold	Odds ratio (random effects) (95% CI)	Odds ratio (random effects) (95% CI); EPI
High risk population							
Baenziger et al 1999[50]	HIE (Sarnat grade >1)	2/5	4/5	10	7.00		2.7 (0.2 to 45.1)
Engle et al 1999[56]	HIE plus seizures (NS)	7/7	49/56	73	7.00		42.4 (2.3 to 782.1)
Gonzalez de Dios et al 2000[59]	HIE (Levine, all grades)	12/41	133/139	180	7.00		9.2 (3.2 to 26.5)
Graham et al 2002[60]	Seizures (NS)	3/10	24/24	34	7.00		22.9 (1.1 to 494.6)
Graham et al 2004[61]	PVL (NS)	3/107	120/122	229	7.00		1.7 (0.3 to 10.6)
Haddad et al 2000[62]	HIE (NS)	8/9	1/2	11	7.00		8.0 (0.3 to 255.8)
Hernandez et al 1993[63]	Mechanical ventilation	11/39	37/43	82	7.00		2.4 (0.8 to 7.4)
Loh et al 1998[72]	HIE (NS)	2/2	64/67	69	7.00		92.1 (3.7 to 2309.1)
Socol 1994[81]	Seizures (NS)	3/3	17/25	28	7.00		14.4 (0.7 to 311.8)
Hibbard et al 2001[64]	Seizures/atrophy (NS)	1/18	144/150	168	7.05		1.4 (0.2 to 12.4)
	IVH (NS)	5/55	112/115	170	7.05		3.7 (0.9 to 16.2)
Beeby et al 1994[4]	IVH (Papile grade 3 or 4)	13/48	447/490	538	7.10		3.9 (1.9 to 7.9)
Ertan et al 2006[91]	IVH (Papile, all grades)	24/60	45/60	120	7.10		2.0 (0.9 to 4.4)
Holmes et al 2001[66]	IVH (Papile grade 3 or 4)	0/4	70/72	76	7.10		3.1 (0.1 to 75.5)
Salafia et al 1995[78]	IVH (NS)	0/44	357/362	406	7.10		0.7 (0.04 to 13.4)
Tejani and Verma 1989[83]	IVH (Papile, all grades)	3/53	323/339	392	7.10		1.2 (0.3 to 4.3)
Yoon et al 1996[88]	PVL (cystic/echogenic WM)	8/20	114/133	153	7.15		4.0 (1.5 to 11.1)
Blackwell et al 2001[52]	Seizures (NS)	1/1	5/7	8	7.20		1.0 (0.8 to 4.2)
Casey et al 2001[53]	Seizures (NS)	47/66	900/1625	1691	7.20		3.1 (1.8 to 5.3)
Luthy et al 1987[74]	IVH (Papile grade 3 or 4)	17/33	163/176	209	7.20		5.5 (1.9 to 15.7)
Unselected or low risk population							
Gilstrap et al 1989[58]	Seizures (NS)	2/2	2718/2734	2736	7.00		169.9 (22.5 to 1281.4)
Ingemarrson et al 1997[68]	HIE (NS)	8/10	345/298	308	7.00		18.5 (3.8 to 89.6)
Larma et al 2007[70]	Seizures (NS)	8/9	106/205	214	7.00		8.6 (1.1 to 69.7)
	IVH (NS)	7/11	103/203	214	7.00		1.8 (0.5 to 6.4)
Perlman and Risser 1996[76]	Seizures (Volpe)	5/5	75/90	95	7.00		50.3 (2.7 to 955.6)
Silva et al 2008[80]	HIE (NS)	2/2	156/172	174	7.00		47.4 (2.2 to 1030.3)
	Seizures (NS)	2/11	147/162	173	7.00		3.3 (0.8 to 13.4)
Valentin et al 1993[85]	Composite morbidity (grade 4/5)	1/8	151/163	171	7.00		1.8 (0.2 to 15.8)
Van den Berg 1996[10]	Seizures (NS)	9/10	83/158	168	7.00		10.0 (1.2 to 80.5)
	IVH (NS)	5/7	82/161	168	7.00		2.6 (0.5 to 13.8)
Huisjes and Aarnoudse 1979[67]	Abnormal neurology (Prechtl)	20/54	698/784	838	7.09		4.8 (2.6 to 8.7)
Litschgi et al 1974[71]	Abnormal neurology (NS)	12/50	934/962	1012	7.09		14.3 (6.6 to 31.0)
Dijxhoorn et al 1986[41]	Abnormal tone/movement	1/27	691/776	803	7.10		0.3 (0.04 to 2.3)
Ghosh 2003[57]	HIE (NS)	10/10	49/55	75	7.15		63.0 (3.5 to 1135.0)
Sakaruba and Saling 1989[77]	IVH (NS)	2/5	161/173	178	7.19		8.9 (1.4 to 58.8)
Subgroup meta-analysis							
Composite morbidity in high risk population (18 studies)				4339			3.4 (2.3 to 4.9); 1.4-8.4
Test for heterogeneity: I^2=26.4%, P=0.146							
Composite morbidity in unselected or low risk population (12 studies)				5977			10.6 (4.7 to 24.1); 0.8-135.8
Test for heterogeneity: I^2=66.4%, P=0.001							
HIE (any population) (7 studies)				827			13.8 (6.6 to 28.9); 5.2-36.4
Test for heterogeneity: I^2=0.0%, P=0.525							
Seizures (any population) (9 studies)				5147			8.1 (3.0 to 21.9); 0.4-153.6
Test for heterogeneity: I^2=66.3%, P=0.003							
IVH or PVL (any population) (12 studies)				2853			2.9 (2.1 to 4.1); 2.0-4.3
Test for heterogeneity: I^2=0.0%, P=0.666							
Overall: I^2=58.2%, Harbord test P=0.006							

0.01 1 100

*Not included in meta-analysis of any morbidity

Fig 3| Association of low arterial cord pH with neonatal morbidity. HIE=hypoxic ischaemic encephalopathy; IVH=intraventricular haemorrhage; PVL=periventricular leucomalacia; NS=not stated; WM=cerebral white matter; EPI=estimated predictive interval

Association of low arterial cord pH - with cerebral palsy

Study	Definition	No of true positives or true negatives/ No with or without cerebral palsy: Cerebral palsy	No cerebral palsy	No of children	pH threshold	Odds ratio (random effects) (95% CI)	Odds ratio (random effects) (95% CI); EPI
Ingemarrson et al 1997[68]	NS	0/2	139/200	202	7.00		0.5 (0.02 to 9.6)
Socol 1994[81]	NS	5/6	3/6	12	7.00		5.0 (0.3 to 72.8)
Gaudier et al 1994[49]	Abnormal movements/posture	3/30	182/189	219	7.05		2.9 (0.7 to 11.9)
Beeby et al 1994[4]	NS	1/20	219/235	255	7.10		0.7 (0.1 to 5.7)
Murphy et al 1995[75]	Permanent movement disorder	6/24	112/128	152	7.10		2.3 (0.8 to 6.8)
Kato et al 1996[5]	NS	3/8	98/113	121	7.20		3.9 (0.9 to 18.1)
Luthy et al 1987[74]	NS	3/19	126/137	156	7.20		2.2 (0.5 to 8.5)
Overall (7 studies)				1117			2.3 (1.3 to 4.2); 1.1-5.0

Test for heterogeneity: I^2=0.0%, P=0.777

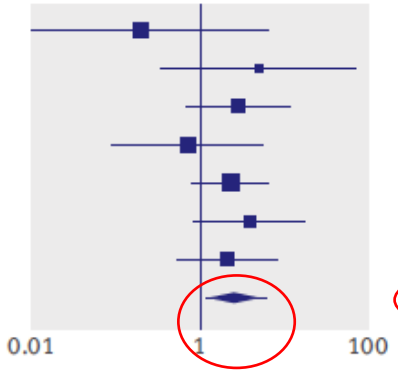


Fig 4 | Association of low arterial cord pH with cerebral palsy. NS=not stated; EPI=estimated predictive interval

WHAT IS ALREADY KNOWN ON THIS TOPIC

Umbilical cord pH at birth is frequently used to measure perinatal asphyxia

Neonatal and childhood mortality and morbidity, including cerebral palsy, are often attributed to fetal acidosis, as defined by a low cord pH at birth

Existing reports of the association between cord pH and adverse outcome are conflicting

WHAT THIS STUDY ADDS

Low cord pH is substantially associated with neonatal mortality and morbidity and cerebral palsy in childhood

These outcomes justify the increased surveillance of infants born with a low cord pH

Further research is, however, needed to explore the cost effectiveness of doing this test in all neonates

Conclusions and practice implications

Cord pH is currently assessed in infants believed to be at high risk for neonatal asphyxia. Our results suggest, however, that the strength of association with cord pH and outcome is not limited to this high risk population. Therefore future research should assess the use of cord pH across neonatal populations, particularly exploring the cost effectiveness of testing all neonates.

Conclusions and practice implications

Cord pH is currently assessed in infants believed to be at high risk for neonatal asphyxia. Our results suggest, however, that the strength of association with cord pH and outcome is not limited to this high risk population. Therefore future research should assess the use of cord pH across neonatal populations, particularly exploring the cost effectiveness of testing all neonates.

Intrapartum fetal surveillance

- *CTG/EFM:*
  - Introduced world-wide after 1970 without proper evidence
    - Intention and expectation was to get rid of CP due to intrapartum asphyxia
    - Low specificity causing high CS-rate
    - FBS was introduced meanwhile, and was found to increase the specificity


SCALP SERVICE STATION
HORSE SHOW
Sunday 9 July
club
Orange
HB
ICE CREAM
HB
ICE CREAM

History of Biochemical Monitoring of the Fetus During Labor

Archiv für Gynäkologie 197, 108—122 (1962)

Aus der Städtischen Frauenklinik und Hebammenlehranstalt Berlin-Neukölln
(Ärztlicher Direktor: Dr. E. JUNG)

Neues Vorgehen zur Untersuchung des Kindes unter der Geburt*

Einführung, Technik und Grundlagen

Von

ERICH SALING

Mit 7 Textabbildungen

(Eingegangen am 10. April 1961)

Fetal Scalp Sampling (FBS)

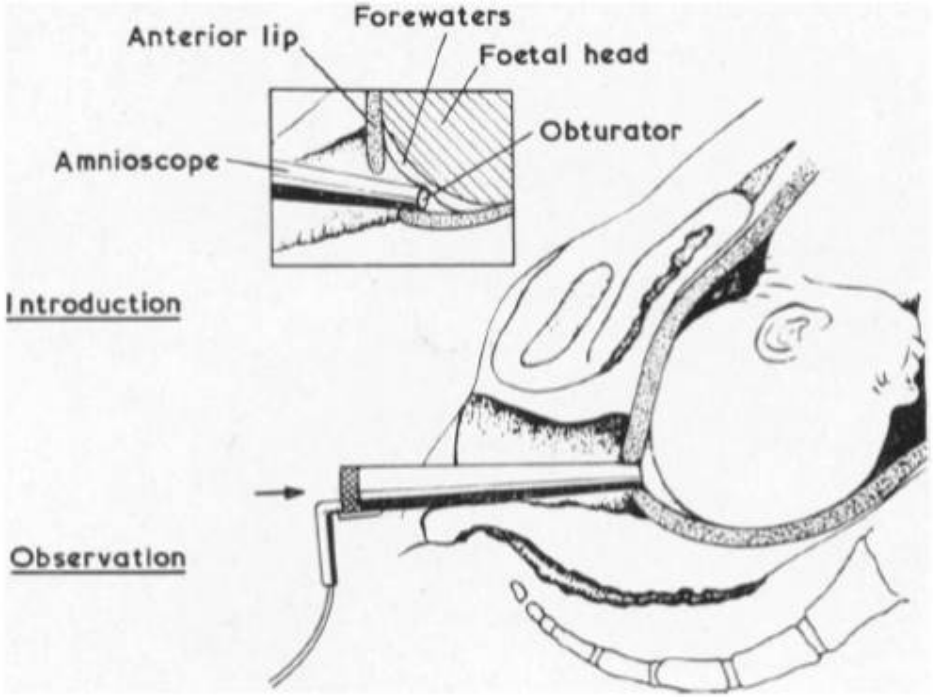


FIG. 1.—*Amnioscopy. Inset shows introduction of the instrument. Below, the instrument in place with the obturator withdrawn.*

FBS

NB: suction by mouth

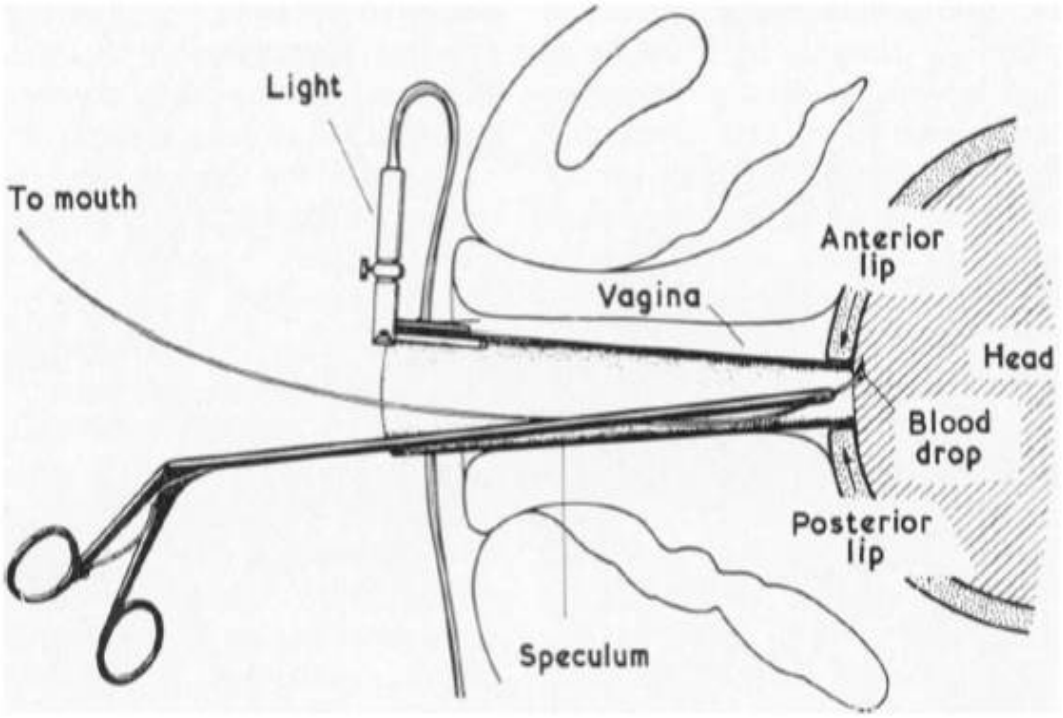


FIG. 2.—*Foetal blood sampling. The presenting part has been pricked, and a drop of blood is about to be sucked into a heparinized tube, held in special forceps.*

Scalp-blood sampling
FBS (pH)

Normal values:

Scalp-pH is slowly decreasing during normal labour, with values between 7.45 og 7.25
(Weber 79)

No upper limits of normal scalp-pH have been described

Scalp-blood sampling
FBS (pH)

pH decrease during normal labour:
(Weber 79)

- I. stage: 0.016 pH unit per hour
- II. stage: 0.11 pH unit per hour

Scalp-blood sampling
FBS (pH)

- By anoxia (no oxygen supply at all)
  - e.g. total umbilical cord compression

- pH drops by 0.04 pH unit per min !

  – e.g. from 7.20 $\Rightarrow$ 6.80 in 10 minutes

  (Myers 72)

Scalp-pH – Intrauterine rescuscitation

- pH < 7.20
  - Incipient acidosis
    Risk of developing asphyxia
  - Consider intrauterine rescuscitation (tocolysis)
  - Continue CTG in theatre, if improvement after IUR – avoid general anaesthesia
  - Deliver the baby

Scalp-pH – Acidosis - Hypoxia

- Hypoxia $\Rightarrow$ Acidosis
  - CO_2 accumulation
  - Anaerobic metabolism, accumulation of lactate
- Low scalp-pH $\Rightarrow$ low cord-pH
- Hence, scalp-pH can predict fetal acidosis

Scalp-blood sampling
FBS (pH)

- Low pH is connected with fetal hypoxia

but

- So far, no single study has proven better neonatal outcome, nor decreased incidence of cerebral palsy

  - by the use of scalp-pH

"…..the pan-galactical trial"

Scalp-blood sampling
FBS (pH)

- Special conditions to consider :
  - Prematurity (< 34 weeks)
  - Chorionamnitis

Scalp-blood sampling
FBS (pH)

- Conclusion:
  - scalp-pH in comb. with CTG is the mainstay
  - at present no other (and for sure - no better) supplement with CTG

British Journal of Obstetrics and Gynaecology
April 1992, Vol. 99, pp. 307–309

FETAL AND NEONATAL MEDICINE

Fetal scalp and umbilical artery blood lactate measured with a new test strip method

L. NORDSTRÖM
B. PERSSON
Associate Professor

St. Görans Pediatric Hospital
Karolinska Institutet, Sweden

ABSTRACT

Objective To compare the measurement of lactate in fetal scalp and umbilical artery blood by a new dry reagent strip method with a commercially available enzymatic method using plasma (Monotest).

Design Comparative study.

ejog
European Journal of Obstetrics & Gynecology and Reproductive Biology
www.elsevier.com/locate/ejogrb

Available online at www.sciencedirect.com
ScienceDirect
European Journal of Obstetrics & Gynecology and Reproductive Biology 139 (2008) 16–20

Umbilical cord blood lactate: A valuable tool in the assessment of fetal metabolic acidosis

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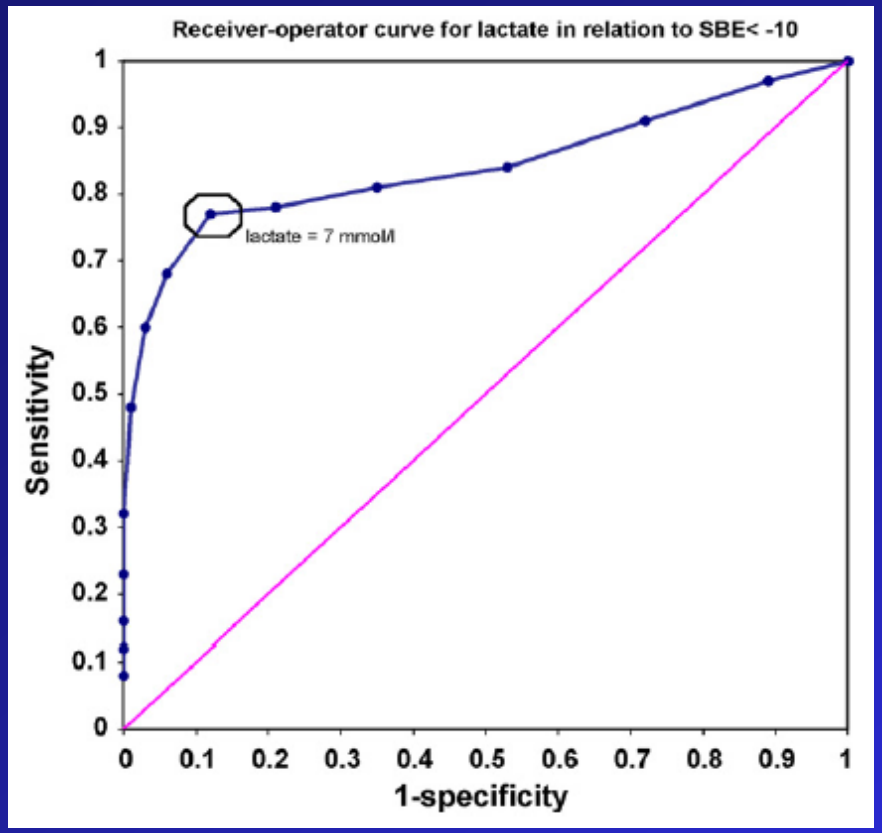
[b] *Department of Obstetrics and Gynaecology, Odense University Hospital, Odense, Denmark*

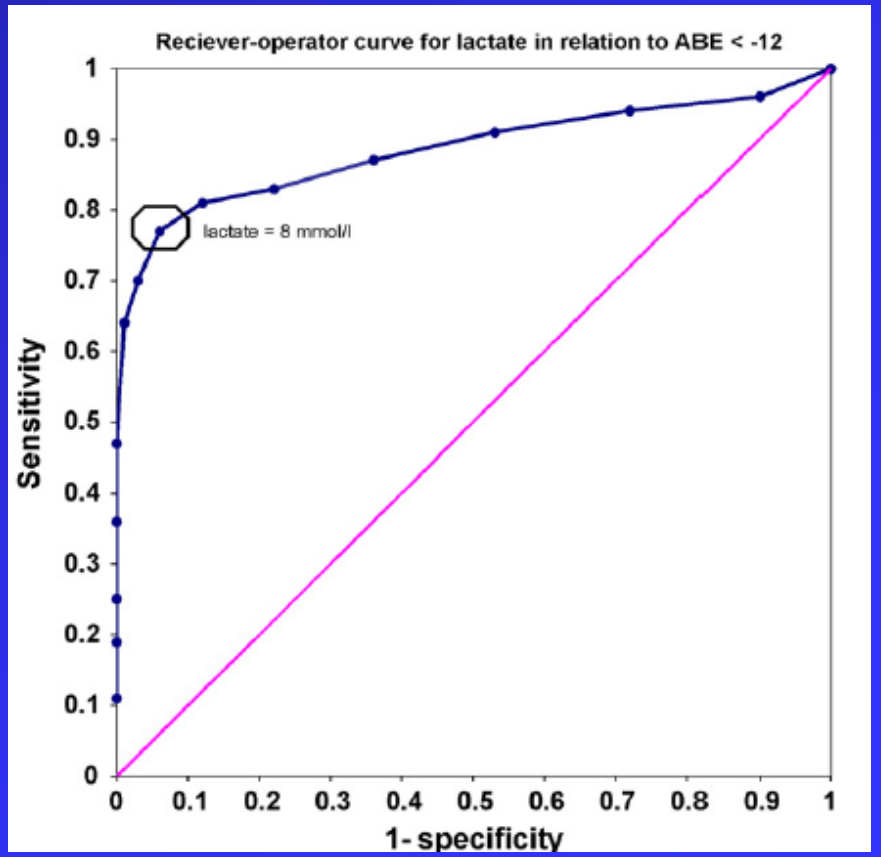
umbilical cord arterial blood samples from 2554 singleton deliveries

Conclusion: Lactate in arterial umbilical cord blood might be a more direct and accordingly more correct indicator of fetal asphyxia at delivery than pH and SBE (or ABE). Its potential as a predictor of neonatal outcome needs to be evaluated in future studies.



SBE< - 10

Lactate 7 mmol/l



ABE< - 12

Lactate 8 mmol/l

• Most important take home messages

- UCBGA is recommended in high-risk deliveries, but ought to be after **ALL** deliveries – since early intervention can be considered (e.g. cooling)
- Optimal interpretation only when both art. and ven. samples are obtained - after immediate double clamping of segment of umbilical cord.
- Low pH in vigourous newborns has a fair prognosis,
  - whereas non-vigourous newborns with pH<7.0 are at high risk of HIE
- SR+MA: Even in low risk populations, low pH is substantially associated with neonatal morbidity and mortality - and later cerebral palsy
- Scalp-pH (FBS) is gold standard in conjunction with CTG as monitor of fetal wellbeing during labour
- Lactate in both FBS and in UCBGA may be the future