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With thanks to Bret Freitag, MD

Cord gases never lie...do they?

Objective:
Understand umbilical cord gases better

Case Study
- 25 year old primip at 41 2/7 weeks
- Induced for post-dates, SROM 10 hours prior to delivery, clear fluid
- At 6 cm developed variable decelerations, then heavy vaginal bleeding with bradycardia when IUPC and FSE were placed
- Emergent C/S under general anesthesia
- 3.4 kg male infant
- Discovery of abruption of ~50%
- Tight nuchal cord x 1

Umbilical Cord Gas Values

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<td>pCO2 (mmHg)</td>
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Interpretation? What next?

BIRTH: poor tone, pale, some grimace, initial HR 60 but rising with stimulation; bloody fluid suctioned from mouth and nose; spontaneous cry shortly after 1 minute

- Tone variable, regular respiratory effort, good HR, still pale color, initial oxygen sats 80’s
- 4-5 MINUTES:
  - Mask CPAP applied with RA then increased to 30-40%
- Apgar scores 3/6/6 at 1, 5, 10 minutes
- 20 MINUTES:
  - Color remained pale
  - Normal respiratory effort and HR
  - PIV placed and glucose obtained

- Nasal CPAP 5 cm H2O room air
  - Oxygen sat mid 90’s
  - HR 130-150, BP mean 42 mmHg
- Glucose 38
  - D5W bolus 2 mL/kg
  - Normal saline 40 mL poor color/perfusion (CFT 5-6 seconds)
- 65 MINUTES:
  - Difficulty drawing labs, UVC placed
  - CBC, blood culture
  - Venous gas: 6.67/75/30/9/-27.6
- 90 MINUTES:
  - UAC placed
  - Arterial gas: 7.06/16/142/5/-23.7
- Hemoglobin/HCT: 7.9/26%
  - 40 mL of emergency O neg blood over 30 minutes
- Admitted to NICU for further evaluation and care
Hospital Course

- Persistent metabolic acidosis
  - Given NaHCO₃, eventual correction at 20 hours of life
- CPAP > intubated, ventilator
- Liver enzymes elevated, coagulopathy
  - Multiple product transfusions without correction
- Essentially anuric, creatinine elevated
- Hypotension > pressor support
- Progressive encephalopathy - very abnormal EEG
- Support withdrawn at 3 days of age

Fetal-placental Circulation

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How could this be with these gases?

Why?

- Debate exists whether blood gas analysis should be universally performed
- Improved gas values and perinatal outcomes independent of obstetric intervention (White et al., 2010)
  - 4 year observational study of universal paired cord blood gas analysis (Australian tertiary care center)
    - 16% reduction in proportion of arterial pH <7.00
    - 25% reduction in BE <5th percentile
    - 60% reduction in lactate >95th percentile
- F/u study demonstrated similar effect in primary and secondary community hospital within 12 months (White et al., 2012)

What?

- Sampling of blood obtained from clamped segment of umbilical vein and artery shortly after delivery
- Values tell us something about status of placenta and baby leading up to and at time of delivery
  - Umbilical vein: reflects uteroplacental status
  - Influenced primarily by maternal conditions
  - Umbilical artery: reflects uteroplacental status plus fetal status

Apgar Scores/Cord Gases Correlation

- 19% of Apgar scores < 7 at 5 minutes had UA pH ≤7.10 BD > -13
- 73% of UA pH <7.10 BD > -13 had Apgar of ≥ 7 at 1 and 86% had Apgar scores of ≥ 7 at 5 minutes
- Gross measure following adaptation to birth
- Equal points for heart rate and cyanosis
- Other things influence Apgars like prematurity, infection
Fetal Heart Rate but Asystolic Newborn
- Doppler detects motion of fetal heart
- Can’t differentiate forceful heartbeat from twitch
- Scalp electrode detects electrical activity
- Maternal-fetal confusion
  > Pulse oximetry during pushing
- Hard to hear/palpate newborn heart beat
- Noise, fear, PPV

When?
A segment of the umbilical cord will be obtained immediately after delivery by the OB provider and saved in the delivery room until the case is reviewed for inclusion criteria to send cord gases as listed below:
- Category II or III tracings
- Cesarean deliveries: emergent or for fetal indication
- Maternal complications
- Intrapartum fever >38°C
- Intrapartum bleeding
- Multi-fetal gestation
- Shoulder dystocia
- Vaginal breech delivery
- Gestation <37 or >41 weeks
- 5 minute Apgar <7
- Newborn resuscitation requiring PPV
- Suspected IUGR
- At the request of the obstetric or pediatric provider

ACOG 2006 (reaffirmed 2012)
- “Umbilical cord arterial blood acid-base and gas assessment remains the most objective determination of the fetal metabolic condition at the moment of birth.”
- Moderate or severe neonatal complications occurred in:
  > 10% of newborns with BE of -12 to -16
  > 40% of newborns with BE less than -16
  > No significant increase when BE is better than -12; no increase in complications at 4-8 years of age

Vein can be easier to access, so we get both to be sure we have the arterial gas

How?
- 10-20 cm segment clamped immediately
- Syringe coated with powdered heparin
- Analyze as soon as possible (White, 2012)
  – Least change in syringe at room temperature for up to 60 minutes
  – Statistically significant changes over first 60 minutes cord vs syringe & ice vs room temperature (next slide)
- What about delayed cord clamping? Does it affect the values?
  – We’ll come back to that…

When?
ACOG:
- Physicians should attempt to obtain venous and arterial cord blood samples in the following situations:
  > C/S for fetal compromise
  > 5-minute Apgar <6
  > Severe growth restriction;
  > Abnormal FHT
  > Maternal thyroid disease
  > Intrapartum fever
  > Multiple gestation
**Can cord gases ever be misleading?**

“**You should never accept an umbilical cord blood gas as correctly reflecting the condition of an infant when your common sense tells you it does not**” (Pomerance, 2012)

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**Reasons for non-correlation**

- Terminal fetal bradycardia secondary to either cord occlusion or fetal heart failure
- Acute fetal hemorrhage
- Any condition when fetal circulation is poor or non-existent at birth

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**Rules**

#1: umbilical vein *always* has higher pH
#2: umbilical vein *always* has lower pCO2
#3: umbilical vein *always* has higher pO2
#4: base deficit and bicarbonate are *usually* similar in both
   - If one is significantly worse, it must be arterial sample

**BASE DEFICIT IS MORE RELIABLE THAN BICARBONATE AS A MEASURE OF METABOLIC ACIDOSIS**

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**The Reason BD is a Better Indicator…**

<table>
<thead>
<tr>
<th>UV</th>
<th>UA</th>
</tr>
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<tbody>
<tr>
<td>pH</td>
<td>6.83</td>
</tr>
<tr>
<td>pCO2</td>
<td>100</td>
</tr>
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<td>10</td>
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- Base deficit takes into account pCO₂ value
- Allows for appreciation of metabolic acidosis before CO₂ is normalized
What if the results don’t follow the rules?

- If 1-3 of the 4 relationships are opposite to rules, maybe samples are from the same vessel
  - Technically easier to sample vein than artery
- If all four are opposite, samples may be mislabeled
- A pO₂ >60mm is reportedly physiologically impossible, and likely represents air bubble contamination (CO₂ should also be low)
  - Today’s blood gas analyzers are smart—will generally not report out a value if there is evidence of air contamination

What about delayed cord clamping?

- Wiberg et al (2008): 70 infants 36-42 weeks not needing resuscitation were evaluated with three serial samples (0 sec, 45 sec, and 90 sec)
  - Mean arterial values changed more & between 0-90 seconds in 1st 45 seconds
    - pH decreased 7.24 to 7.21, BD increased 4.85 to 6.14 mmol/L, and CO₂ increased 57.3 to 80.5 mm Hg
  - Mean venous changes were less & mostly between 45 and 90 seconds
    - pH decreased 7.32 to 7.31, BD increased 4.93 to 5.19 mmol/L, and CO₂ increased 42.7 to 43.6 mm Hg
- DePaco et al (2011): 158 healthy term infants randomized to early (<10 sec) or late (2 minute) clamping
  - No significant differences in acid-base and gas analysis results, except for higher pO₂ values in delayed group

Other General Rules

- Arterial pH should generally be 0.04 – 0.10 pH units lower and pCO₂ 4-18 mm Hg higher than venous under normal circumstances
- Venous sample is generally a good proxy for arterial values if:
  - Normal, non-asphyxiated newborn, or
  - Newborn depressed secondary to uteroplacental insufficiency
- Newborns depressed secondary to cord occlusion or fetal heart failure with terminal bradycardia—arterial values may be much worse than venous
  - Umbilical cord values only reflect fetal tissue status up to time flow in cord ceases

Mokarami et al, 2013

- Umbilical gases from 124 newborns (36-42 weeks)
  - 66 vaginal deliveries (VD), 58 planned c-sections
    - 19% of VD had category 2, 4.7% had category 3 (1 baby required resuscitation)
    - Cord not clamped, placed on abdomen/ warm towel between legs
    - Blood gases at birth (T0) and 45 seconds later (T45), and compared between time points and vaginal vs C/S
- Findings
  - Small but significant decrease in arterial pH, and increase in pO₂, pCO₂, lactate, and hemoglobin between T0 and T45 in both groups
  - Small but significant difference in pH, lactate, and hemoglobin in VD vs C/S

Other Perspectives: “Hidden Acidosis”?

1. During labor fetal blood flow is limited; fetus preserves central perfusion at expense of non-vital tissues (diving reflex)
   - Increase in lactic acid
   - Increase in pH
2. Normal transitional circulation improves; “hidden” acid load is washed out, measured in neonatal blood, particularly umbilical artery
Uteroplacental Insufficiency: Causes

- Maternal anemia or \( \text{O}_2 \) desaturation
- Hypotension
- Excessive uterine activity
- Medications and drugs
- Abnormal uteroplacental vasculature
- Abruption
- Uterine rupture without expulsion

Uteroplacental Insufficiency: Pathophysiology

As umbilical arterial blood moves through the intervillous space, uteroplacental insufficiency leads to poor exchange of \( \text{CO}_2 \) and \( \text{O}_2 \), resulting in umbilical venous blood with:

- Higher \( \text{pCO}_2 \)
- Lower \( \text{O}_2 \)
- Little or no correction of any underlying metabolic acidosis

- Fetus becomes progressively acidotic (respiratory and metabolic), with decelerations (lates), and elevated baseline HR, and eventual bradycardia (very late)

Cord Occlusion with Terminal Fetal Bradycardia

- Ultimately more devastating: in animal models ischemia is a much more potent cause of brain injury than hypoxia alone
- Umbilical cord is at risk for occlusion due to both stretch and compression
  - Compression is easier when stretch is already present
- Umbilical vein is more susceptible to occlusion due to thin walled nature
- Risk increased with thin/short cord, oligohydramnios

Hallmarks of Uteroplacental Insufficiency

ELEVATED \( \text{PCO}_2 \) & DEPRESSED VENOUS \( \text{PO}_2 \)
TOGETHER WITH APPROXIMATELY EQUAL
DERANGEMENTS OF BOTH UMBILICAL VENOUS
AND ARTERIAL BASE DEFICITS

Mechanism of Impaired Umbilical Blood Flow

**Stretch**
- Short cord
- Cord around neck or other structures
- True knot in cord
- Descent of fetus
- Shoulder dystocia (possible)
- Breech delivery with trapped head (stretch and compression)

**Compression**
- Kinked cord
- True knot in cord
- Torsion of cord
- Entwined cords (mono-mono twins)
- Hematoma of cord
- Cysts of cord
- Prolapsed cord
- Breech delivery with trapped head (stretch and compression)
- Stricture of cord
- Shoulder dystocia (probable)

What if...?

- 29 yo G1P0
- Uncomplicated pregnancy at 39 2/7 weeks
- Normal FHT, membranes intact

Magically double clamp the umbilical cord in utero without otherwise disturbing the fetus, immediately causing cessation of all umbilical flow, then deliver the baby 40 minutes later

1) Describe the condition of the newborn
2) Describe the cord blood gases
**Answers**

1) Dead

2) Normal

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**Explanation**

- Animal data of complete cord occlusion shows that over 1st 12-15 minutes, fetal BP and cardiac output progressively fail.
- Fetal aorta blood gas changes:
  - \( \text{pH} 0\text{.038 units/minute} \)
  - \( \text{CO}_2 \text{ of 6.4 mmHg/minute} \)
  - \( \text{BD of 1\text{.1 mmol/L/minute}} \)
- WHEN UMBILICAL VESSELS ARE OCCLUDED VALUES WILL ONLY REFLECT BLOOD GAS STATUS PRIOR TO OCCLUSION.

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**How much difference is significant?**

- \( \text{BD: generally >4 mmol/L} \)
- \( \text{pH: generally > 0\text{.10 units}} \)
- Wider the difference, the longer standing the venous occlusion with partial umbilical arterial flow.
- If/when umbilical arterial flow ceases, umbilical arterial value will not change further and will only reflect status of infant up until the time that flow ceased.
- May underrepresent acidosis at fetal level, that is not appreciated until circulation is restored.

INITIAL POSTNATAL BLOOD GAS MAY HAVE MUCH MORE SEVERE METABOLIC ACIDOSIS THAN CORD GASES IN THIS SITUATION

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**But in real life...**

- Umbilical cord venous compression may be brief, intermittent, or continuous.
  - Period of “slowed” umbilical venous flow may be associated with elevated \( \text{pO}_2 \), assuming placental function is adequate.
- Umbilical arterial occlusion is frequently overcome, at least temporarily or intermittently, by augmented fetal blood pressure.
- Leads to net blood volume loss to fetus, worsening hypoxia, ischemia, and metabolic status.
  - Net result is arterial values that are worse than venous values.

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**Back to the original case...**

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(Possible) Interpretation

- Venous: mild respiratory acidosis, moderately elevated \( pO_2 \)
- Arterial: mild mixed acidosis, elevated \( pO_2 \)?
  - Could this be venous?
  - Acidosis and increased \( CO_2 \) shift the oxygen dissociation curve to the right, so increased free \( O_2 \)?
  - Fetal anemia may increase the amount of free \( O_2 \)
  - Maternal oxygen administration?
- Venoaerterial difference is mildly significant
  - This would fit with variable deceleration pattern on FHT

How did initial UAC/UVC gases get so bad?

- Most likely reperfusion acidosis
  - Cord arterial gas "underrepresented" fetal state
- Likely some contribution from anemia as well
  - Baby persistently pale at birth despite fairly normal VS
  - Initial HCT measured was 25%, although this was after receiving 47 mL of fluid (40 mL of saline + 7 mL of D10), which would be about 16% of his blood volume (3.5 kg x 85 mL/kg = ~300mL)
  - So true HCT at birth was closer to 25 (25 x 1.16), still quite abnormal
  - Abruption very likely slowly acute – enough for infant to begin to equilibrate hematocrit
  - REMEMBER: Kleihauer-Betke testing of mom

Opportunities for Improvement

- Assess HCT sooner given clinical situation
  - Can HCT be done on cord gas analyzer?
- 50% abruption associated with high mortality
  - 30-50% reported in the literature
- Central access = high priority
- Emergency release blood available at delivery
  - Better choice than saline for 1st volume expander
- Candidate for cooling
  - Send baby’s blood gas before 60 minutes of life
- Consider ventilation support any time there is severe metabolic acidosis

References


Test yourself!

https://secure1.csmc.edu/nicu/cbg/
THANK YOU
for taking such good care
of our littlest patients