Blood Gas Interpretation and Acid Base Balance

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Step-by-step approach to interpreting blood gases

1. Acidosis or alkalosis?
2. Is there a primary respiratory or metabolic problem or is it mixed?
3. Is there any compensation?
4. If there is a metabolic acidosis, assess the anion gap.
5. Assess oxygenation.
6. Put it all together with the clinical situation.
7. Determine the appropriate treatment.
    a. When calling the provider, utilize SBAR format.

Step 1- Acidosis or Alkalosis?

Normal pH= 7.35-7.45

pH< 7.35 = acidosis

pH>7.45 = alkalosis

Acidosis or alkalosis may still be present with a normal pH

Step 2: Is the primary disorder respiratory or metabolic?

2a. Assess pH and CO2

In primary **respiratory** disorders, the pH and PaCO2 change in **opposite** directions

In primary **metabolic** disorders, the pH and PaCO2 change in the **same** direction

Acidosis	pH ↓	PaCO2 ↑	Respiratory
Acidosis	pH ↓	PaCO2 ↓	Metabolic
Alkalosis	pH ↑	PaCO2 ↓	Respiratory
Alkalosis	pH ↑	PaCO2 ↑	Metabolic

Step 2: Is the primary disorder respiratory or metabolic?

2b. Assess the HCO_3

- Normal HCO_3 is 22-26mmol /L
  - $HCO_3 < 22$ indicates metabolic acidosis
  - $HCO_3 > 26$ indicates metabolic alkalosis

2c. Assess the base deficit= how much base is needed to restore the pH to normal; helps assess the severity of acidosis

- Normal base deficit is -2 to +2 mEq/L
  - Mild: -3 to -5mEq/L
  - Moderate: -6 to -9 mEq/L
  - Severe: less than -10 mEq/L
- Treatment: Base required= (BD x -1) x (wt in kg) x 0.4

Step 3: Is there is appropriate compensation for the primary disorder?

Disorder	pH	Primary problem	Compensation
Metabolic acidosis	↓	↓ HCO3	↓ PaCO2
Metabolic alkalosis	↑	↑ HCO3	↑ PaCO2
Respiratory acidosis	↓	↑ PaCO2	↑ HCO3
Respiratory alkalosis	↑	↓ PaCO2	↓ HCO3

Practice Makes Perfect!

1. pH 7.27, PaCO2 56, HCO3 25
    a. Uncompensated respiratory acidosis
2. pH 7.24, PaCO2 45, HCO3 19
    a. Uncompensated metabolic acidosis
3. pH 7.49, PaCO2 42, HCO3 31
    a. Uncompensated metabolic alkalosis
4. pH 7.48, PaCO2 57, HCO3 41
    a. Metabolic alkalosis, partially compensated with respiratory acidosis
5. pH 7.39, PaCO2 24, HCO3 14
    a. Metabolic acidosis, fully compensated with respiratory alkalosis

ABG interpretation practice: https://abg.ninja/abg

Deep Dive into the Degree of Compensation/Chronicity of the Problem

Disorder	Expected Compensation	Correction Factor
Metabolic acidosis	PaCO2 = (1.5 x [HCO3-]) +8	± 2
Metabolic alkalosis	PaCO2 = 40 + 0.6(ΔHCO3-)	
Acute respiratory acidosis	Increase in [HCO3-]= Δ PaCO2/10 **~1mEq/L increase in HCO3** for every 10mmHg PaCO2>40	± 3
Chronic respiratory acidosis	Increase in [HCO3-]= 3.5x (Δ PaCO2/10) **~4mEq/L increase in HCO3** for every 10mmHg PaCO2>40	
Acute respiratory alkalosis	Decrease in [HCO3-]= 2x(Δ PaCO2/10) **~2mEq/L decrease in HCO3** for every 10mmHg PaCO2<40	
Chronic respiratory alkalosis	Decrease in [HCO3-] = 5x(Δ PaCO2/10) to 7x(Δ PaCO2/10) **~5mEq/L decrease in HCO3** for every 10mmHg PaCO2<40	

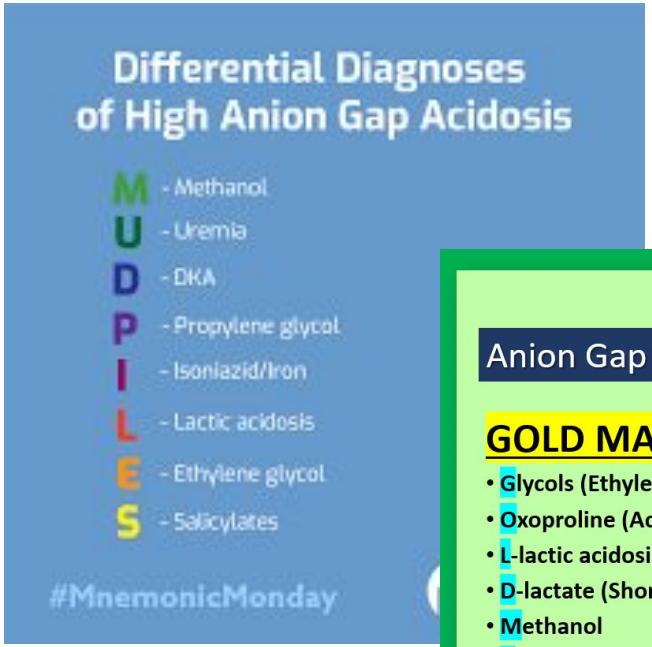
Deep Dive into the Degree of Compensation/Chronicity of the Problem

Predicted change in pH

Acute Respiratory Acidosis	0.08 x [PaCO2-40÷10]
Chronic Respiratory Acidosis	0.03 x [PaCO2- 40÷10]
Acute Respiratory Alkalosis	0.08 x [40 - PaCO2÷10]
Chronic Respiratory Alkalosis	0.03 x [40-PaCO2÷10]

Step 4: If there is a metabolic acidosis, assess the anion gap

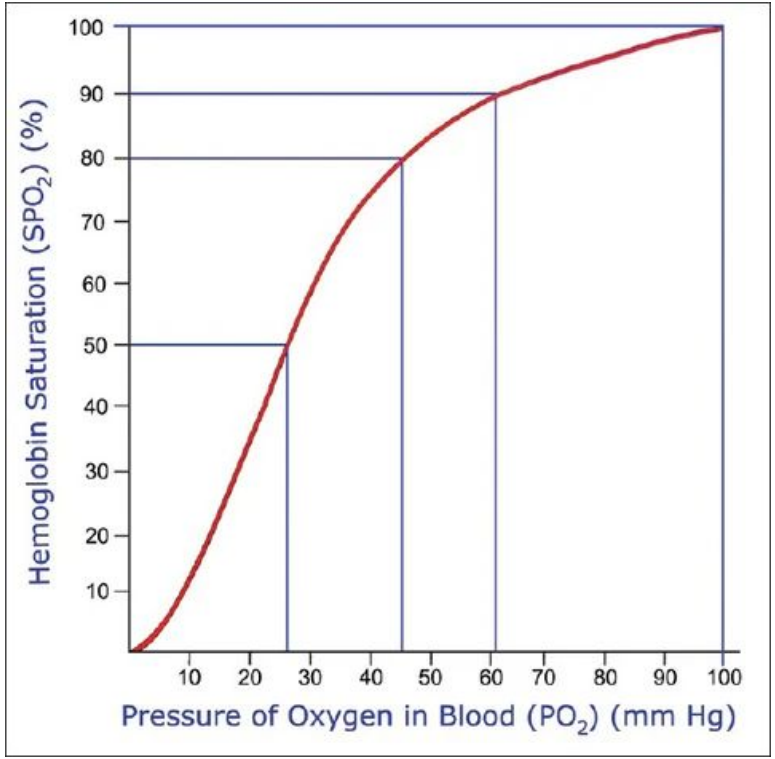
1. Anion gap = [Na+]-([Cl-] + [HCO3-])-12 ± 2
2. Normal anion gap ~12
   a. Non-anion gap acidosis:
      i. GI losses of bicarbonate (diarrhea)
      ii. Failure of the kidneys to eliminate acid or produce bicarbonate
      iii. Hyperchloremia
   b. Elevated anion gap:
      i. MUDPILES vs GOLDMARK





Step 5: Look at the oxygenation status

- Normal PaO2 = 80-100mmHg
- Normal SpO2 = 92-100%
- Expected PaO2 = 5x FiO2



Oxyhemoglobin Dissociation Curve

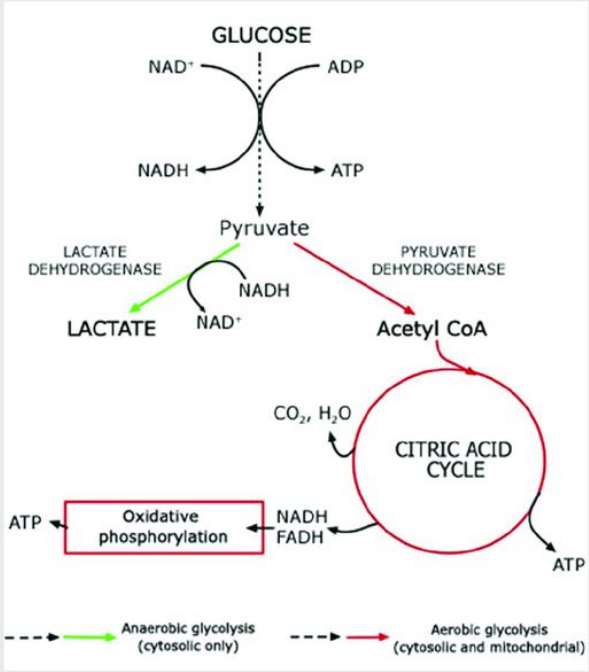
Normal Values

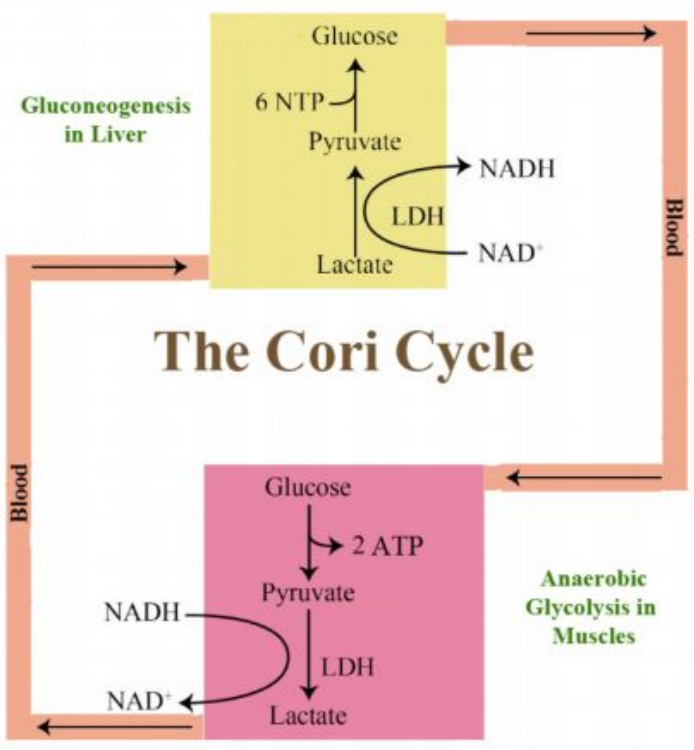
- Arterial samples: pH 7.35-7.45, HCO_3 22-26, PCO_2 35-45
- Venous: Arterial pH ~0.05 units lower, CO_2 ~5 higher
  - VBG: pH 7.3 and CO_2 50 → ABG: pH 7.35 and CO_2 45
  - No indication of oxygenation
  - Venous saturation can give an indication of oxygen consumption
- Capillary: similar to arterial (assuming no prolonged tourniquet use, ischemia, etc)
  - Mix of oxygenated arterial blood and deoxygenated venous blood

Lactic Acidosis

- Serum lactate serves as a surrogate measure of cardiac output and tissue oxygen delivery
- Type A: occurs in the context of cellular (mitochondrial) hypoxia, as a result of lactate overproduction and is usually associated with diminished oxygen delivery
  - Elevated lactate:pyruvate ratio
- Type B: associated with normal cardiac output and oxygen delivery, likely re: systemic inflammatory reaction from CPB and endogenous catecholamine surge
  - Normal lactate:pyruvate ratio, indicating no cellular hypoxia
  - Well described after adult cardiac surgery and is associated with an excellent postoperative diagnosis

Glucose Metabolism





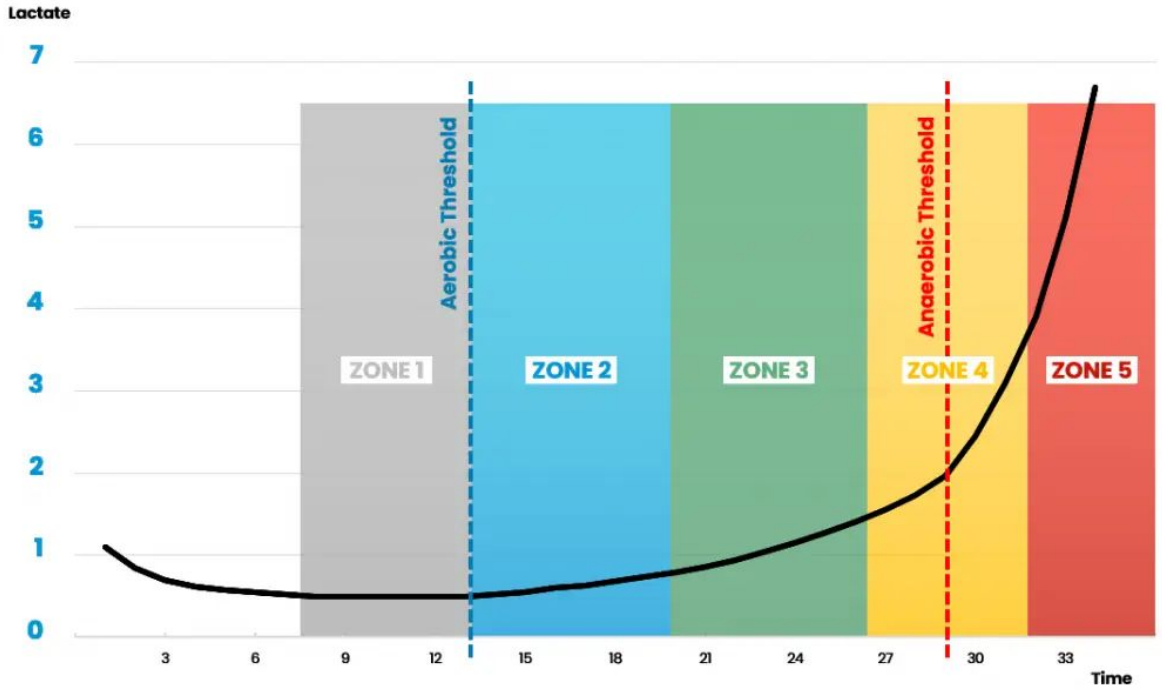
What does that mean in real life?

Zone 1= recovery, body is using oxygen as energy source

Zones 2-3 = aerobic endurance, body is using oxygen and stored energy

Zone 4 = anaerobic threshold, increased lactate production

Zone 5= VO2 max, maximal oxygen consumption, lactate continues to rise



“Benign” lactic acidosis is common in adolescents and children following congenital heart surgery

- Observation: Children and adolescents frequently have elevated lactate levels postoperatively without clinical signs of low CO
- Objective: evaluate children with postoperative lactic acidosis and describe a distinct group of patients with lactic acidosis and low inotrope scores called “benign” lactic acidosis
- Design: retrospective chart review of 105 patients, ages 5-21 years old
  - 3 groups:
    - Classic lactic acidosis: elevated lactate and inotrope scores
    - Benign lactic acidosis: elevated lactate and low inotrope score
    - Control: low lactate and low inotrope score

“Benign” lactic acidosis is common in adolescents and children following congenital heart surgery

- Findings:
  - Lactic acidosis in the benign group was less pronounced and resolved earlier than in the classic group.
  - The classic and benign groups had significantly elevated serum glucose.
  - The classic group had significantly longer CPB time, time to extubation, and hospital length of stay compared to the benign and control groups.
  - The classic group had significantly lower systolic blood pressure, higher heart rate and CVP, and lower UOP than the benign and control groups.
- The Bottom Line: Benign lactic acidosis is clinically distinct from classic lactic acidosis. It has faster resolution and patients exhibit stable postoperative hemodynamics with similar clinical outcomes as patients in the control group

Patient Scenario 1

8 year old female with single ventricle physiology admitted from the OR after MPA band revision, intubated and on epinephrine infusion at 0.04mcg/kg/min. Intraoperative course was uneventful. CPB: 61 minutes. CC: 30 minutes. Plan to extubate when awake and alert.

VS: HR 113, BP 103/60 (76), SpO2 82% on 0.3 FiO2, RR 22, T 37.2, NIRS 76/86

ABG: 7.23/58/59/24/-4/83%

VBG: 7.24/62/51/26/-1/76%

Lactate: 7.72

Glucose: 209

Patient Scenario 1

Patient starts to wake up and is quite agitated, receives morphine x1. Epinephrine infusion is turned off. She is switched to a spontaneous breathing mode on the ventilator.

VS: HR 113, BP 108/57 (76), SpO2 79% on 0.21 FiO2, RR 14, T 37.2, NIRS 68/79

1 hour ABG: 7.33/50/41/26/0/73%

Lactate: 8.6

Glucose: 162

Patient Scenario 1

Patient is extubated to 4L LFNC. She appears comfortable. VS and exam are stable.

ABG (4 hours after arrival to ICU, 1 hour post-extubation):

7.41/46/49/30/4.1/83%

Lactate: 3.5

Glucose: 162

Patient Scenario 2

- FT baby girl with HLHS with prenatal concern for severe TR and a restrictive atrial septum
- Plan: Proceed to cath lab for BAS and cannulate onto ECMO if needed.
- Patient did not have a restrictive atrial septum and had appropriate saturations on 0.21 FiO2; therefore did not proceed with BAS and was admitted to the CICU around 1300.

Patient Scenario 2

- Initial VS:
  - Temp: 37.1 °C (98.7 °F)
  - Pulse: 157
  - Resp: 30
  - BP: 55/41
  - SpO2: 83%
  - SIMV PRVC: PS 12, PEEP 6, 21%, TV 20, R30
- Initial Exam (focused):
  - Orally intubated female
  - Warm, 2+ pulses, mottled, +murmur
  - Lungs CTAB
  - Abdomen soft, NTND, normoactive BS
  - Moves all extremities, anterior fontanelle is soft and flat

Patient Scenario 2

- Echo confirms HLHS (AA, moderative MV hypoplasia), slightly restrictive atrial septum (5mmHg gradient) with LTR shunt, 2 smaller ASDs, dysplastic TV with severe TR, lg PDA (bid), nml RV fxn
- 7.38/35/36/21/-5/72
- Lactate 1.84
- SpO2 83%
- NIRS 49/50

Patient Scenario 2

- At 1800, patient is noted to be cooler and now has 1+ pulses
  - Temp: 35.6C
  - Pulse: 107
  - Resp: 36
  - BP: 60/39
  - SpO2: 79%
  - NIRS: 48/48
  - 7.36/35/41/20/-6/78, lactate 1.62

Patient Scenario 2

- At 2200, exam is stable. Patient has had volatile BP with no changes to her exam or NIRs. She is not on any sedation.
- Patient has also remained cool and had an episode of hypoglycemia, prompting neonatal septic workup and initiation of antibiotics and steroids.
- 7.37/30/37/17/-8/72, lactate 2.4

Patient Scenario

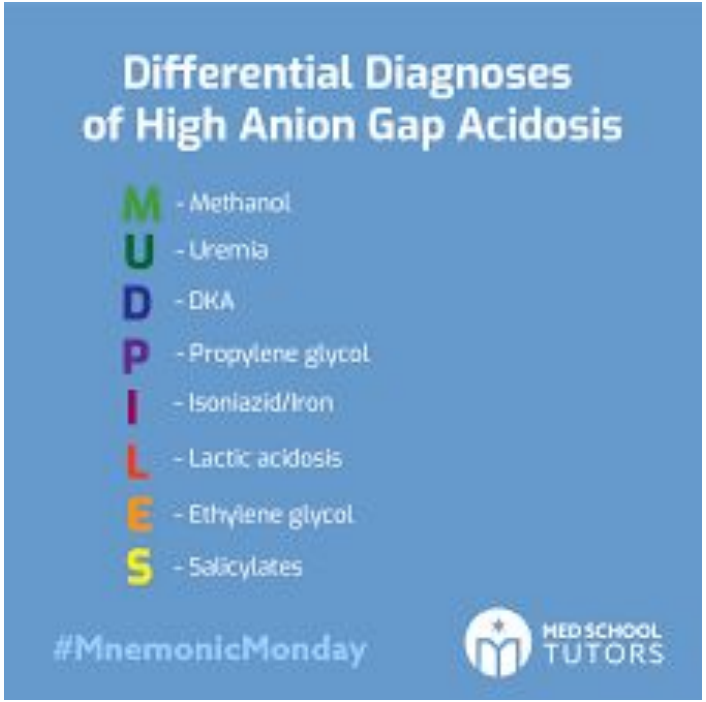
- 0200: 7.33/37/43/19/-6/83%, lactate 2.8
  - VS trend: HR 110s-140s, BP 40s-60s/30s (MAP~40), T 36C, SpO2 88%
  - Bedside Qp:Qs 2:1
  - We continued to wean the ventilator rate down to allow her CO2 to rise **(why?)**
- 1100: 7.36/25/41/14/-11/79%, lactate 10.5
  - PEEP increased from 5 to 7 during rounds (why?)
  - Started milrinone during rounds
  - After this gas, the patient was paralyzed, but lactate never less than 8, so ultimately cannulated onto ECMO
- Diagnosis: Type A Lactic Acidosis 2/2 poor CO

Treatment

- Address the underlying cause to treat the derangement
- Respiratory acidosis: low pH, high CO2
  - Causes:
    - Airway obstruction, CNS depression, ventilatory restriction, incorrect ventilator settings
  - Treatment:
    - Increase minute ventilation by treating the airway obstruction, decreasing sedation, aid ventilation with non-invasive or invasive measures, increase rate and/or TV
- Respiratory alkalosis: high pH, low CO2
  - Causes:
    - Hyperventilation 2/2 fever, pain, fear, anxiety; incorrect ventilator settings
  - Treatment:
    - Antipyretics, pain medication, sedation, decrease rate and/or TV

Treatment

- Metabolic Acidosis: low pH, low HCO3
  - Causes: MUDPILES, GOLD MARK
  - Treatment:
    - Uremia:
      - Consider dialysis if kidney failure
    - Lactic acidosis: increase CO and/or oxygen delivery
      - DO2= COxCaO2
      - CaO2= (Hgb x SpO2 x1.34) + (PaO2 x 0.003)



Treatment:

- Metabolic Alkalosis: high pH, high HCO3
  - Cause: Hypovolemia with Cl depletion
    - GI loss of H+ from vomiting, high gastric output
    - Renal loss of H+ from diuretics
  - Treatment:
    - Control vomiting, wean diuretics
    - Consider acidifying agents, such as acetazolamide or arginine chloride

Summary

1. Acidosis or alkalosis?
2. Is there a primary respiratory or metabolic problem or is it mixed?
3. Is there any compensation?
4. If there is a metabolic acidosis, assess the anion gap.
5. Assess oxygenation.
6. Put it all together within the clinical situation.
7. Determine the appropriate treatment.
    a. When calling the provider, utilize SBAR format.

References

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