ABG Interpretation: 
A Respirologist’s approach
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Outline
• A quick review of acid-base physiology
• The 8 steps to ABG interpretation
• Discuss the causes of hypoxemia and hypercapnea

What use is an ABG?
• Assess acid-base balance
• Assess adequacy of ventilation
• Assess oxygenation

Acid-Base Disturbances
• Acidosis = process that makes the blood acidic
• Alkalosis = process that makes the blood alkaline
  • This is a diagnosis
  • Multiple disorders can exist simultaneously
• Acidemia = blood pH below 7.35
• Alkalemia = blood pH above 7.45
  • This is a sign
  • Net result of all concurrent disorders

Two kinds of pH disorders
1. “Respiratory” = 1º abnormality in ventilation (CO2)
2. “Metabolic” = 1º abnormality in any other acid or base

Acid-Base Disturbances
• When acidosis or alkalosis occurs, the body tries to normalize pH by “compensating” using buffers
  • If the primary process is metabolic,
    • We use lungs to increase or decrease ventilation to alter \( p_{\text{CO}_2} \)
    • This “respiratory compensation” takes minutes
  • If the primary process is respiratory,
    • We use kidneys to excrete either acid (NH4) or base (NaHCO3)
    • This “metabolic compensation” takes hours or days
• Compensation is always in the same direction as the primary problem
  • If \( p_{\text{CO}_2} \) rises, appropriate compensation increases HCO3-
  • If \( p_{\text{CO}_2} \) falls, appropriate compensation decreases HCO3-
  • If HCO3- rises, appropriate compensation increases \( p_{\text{CO}_2} \)
  • If HCO3- falls, appropriate compensation decreases \( p_{\text{CO}_2} \)

Alveolar Ventilation
• CO2 is normally tightly regulated
  • Small changes to CO2 alter ventilation
• Carotid body is essential to this regulation
  • This is a cluster of chemoreceptors in the carotid artery
  • Detects levels of [O2], [CO2] and [H+] 
  • Sends signals to the brain
  • Alters ventilation in response to [CO2] and [H+]
• Carotid body response:
  • When patient has acidemia (low pH)
    • Carotid body makes you more sensitive to \([\text{CO}_2]\) ventilation
  • When patient has alkalemia (high pH)
    • Carotid body makes you less sensitive to \([\text{CO}_2]\) ventilation
Respiratory Acidosis

\[ \uparrow \text{CO}_2 + \text{H}_2\text{O} \rightarrow \uparrow \text{H}^+ + \uparrow \text{HCO}_3^- \]

- Hypoventilation causes rise in \( p_{\text{CO}_2} \), shifts equilibrium to the right.
  - Acutely (10:1)
    - For each 10 mm Hg rise in \( p_{\text{CO}_2} \), \( \text{HCO}_3^- \) should increase by 1 mEq/L
    - This is due to equilibrium shift (buffering)
  - Chronically (10:3)
    - For each 10 mm Hg rise in \( p_{\text{CO}_2} \), \( \text{HCO}_3^- \) should increase by 3 mEq/L
    - This is due to renal compensation (excretion of \( \text{H}^+ \))
- Common causes
  - Lung disease
  - Neuromuscular disease
  - Sedative drugs
  - Adaptation to extreme obesity and sleep apnea

\[ [\text{H}^+] = 24 \times p_{\text{CO}_2} \]

\[ [\text{HCO}_3^-] \]

Respiratory Alkalosis

\[ \downarrow \text{CO}_2 + \text{H}_2\text{O} \leftarrow \downarrow \text{H}^+ + \downarrow \text{HCO}_3^- \]

- Hyperventilation causes \( p_{\text{CO}_2} \) to fall, shifts equilibrium to the left.
  - Acutely (10:2)
    - For each 10 mm Hg fall in \( p_{\text{CO}_2} \), \( \text{HCO}_3^- \) should decrease by 2 mEq/L
    - This is due to equilibrium shift (buffering)
  - Chronically (10:4)
    - For each 10 mm Hg fall in \( p_{\text{CO}_2} \), \( \text{HCO}_3^- \) should decrease by 4 mEq/L
    - This is due to renal compensation (excretion of \( \text{HCO}_3^- \))
- Common causes
  - Anxiety / panic (including panic attacks)
  - Pregnancy
  - Early sepsis
  - Drugs (one component of ASA toxicity)
  - Mechanical ventilation at excessive rate or volumes

\[ [\text{H}^+] = 24 \times p_{\text{CO}_2} \]

\[ [\text{HCO}_3^-] \]

Metabolic Alkalosis

\[ \uparrow \text{CO}_2 + \text{H}_2\text{O} \rightarrow \uparrow \text{H}^+ + \uparrow \text{HCO}_3^- \]

- Increase in \( \text{HCO}_3^- \) shifts equilibrium to the left
  - \( p_{\text{CO}_2} \) increases
  - Alkalemia makes the carotid body less sensitive to \([\text{CO}_2]\)
  - We “allow” the \( p_{\text{CO}_2} \) to stay elevated (maximum ~ 50 mmHg)
- Compensation (1:0.7)
  - For every 1 mEq rise in \( \text{HCO}_3^- \), \( p_{\text{CO}_2} \) increases by 0.7 mmHg
- Causes
  - Volume depletion (“contraction alkalosis”)
  - Nasogastric suction
  - Diuretics
  - Hyperaldosteronism

\[ [\text{H}^+] = 24 \times p_{\text{CO}_2} \]

\[ [\text{HCO}_3^-] \]

Metabolic Acidosis

\[ \downarrow \text{CO}_2 + \text{H}_2\text{O} \leftarrow \downarrow \text{H}^+ + \downarrow \text{HCO}_3^- \]

- Two possible mechanisms of onset
  - Loss of \( \text{HCO}_3^- \)
  - Gain of \( \text{H}^+ \)
- Mechanisms of compensation are a bit more complex

\[ [\text{H}^+] = 24 \times p_{\text{CO}_2} \]

\[ [\text{HCO}_3^-] \]

Metabolic Acidosis – Bicarb Loss

\[ \downarrow \text{CO}_2 + \text{H}_2\text{O} \leftarrow \downarrow \text{H}^+ + \downarrow \text{HCO}_3^- \]

- Loss of bicarbonate (ie. diarrhea) creates an acidemia (a relative increase in \([\text{H}^+]\))
  - Acidemia makes the carotid body more sensitive to \([\text{CO}_2]\) leading to increased ventilation
- Compensation (1:1)
  - For every drop of 1 mEq of \( \text{HCO}_3^- \), \( p_{\text{CO}_2} \) falls by 1 mmHg

\[ [\text{H}^+] = 24 \times p_{\text{CO}_2} \]

\[ [\text{HCO}_3^-] \]

Metabolic Acidosis – Acid Gain

\[ \downarrow \text{CO}_2 + \text{H}_2\text{O} \leftarrow \uparrow \text{H}^+ + \downarrow \text{HCO}_3^- \]

- Some acid gets added to the body (ie. exercising muscle makes lactate), the equilibrium gets shifted to the left (which lowers \( \text{HCO}_3^- \) and \( \text{H}^+ \))
  - This causes a transient rise in \([\text{CO}_2]\)
  - \( \text{CO}_2 \) falls even lower than baseline, because acidemia makes the carotid body more sensitive to \([\text{CO}_2]\) leading to increased ventilation
- Compensation
  - For every drop of 1 mEq of \( \text{HCO}_3^- \), \( p_{\text{CO}_2} \) falls by 1 mmHg
- Examples of metabolic acidosis
  - Diarrhea/GI losses (loss of \( \text{HCO}_3^- \))
  - Lactic acidosis (lactic acid)
  - Renal failure (metabolic acids and loss of \( \text{HCO}_3^- \))
  - Diabetic ketoacidosis (acetate acid)
  - ASA (acetylsalicylic acid)
ABG Interpretation

“ABG’s in 8 steps”

A Case

- You get a call from your clinical clerk...
  - “I need your input on Ms. K. She is a 65 year old woman who is here for a small bowel obstruction. Med consults is following her for long standing back pain and they are working her up for possible cancer.”
- ABG (pH / PaCO2 / PaO2 / HCO3-)
  - 7.30 / 80 / 45 / 38
  - 3.6
  - 140 / 3.9 /
  - 100 / 35 \ 
  - 85

Step 1

- Step 1: Obtain ABG and electrolytes
  - If you don’t perform the test, you’ll never know what is going on with the patient
  - An ABG and a lactate are the 2 best tests to help you get a sick patient to the ICU

Step 2

- Step 1: Obtain ABG and electrolytes
  - If you don’t perform the test, you’ll never know what is going on with the patient
  - An ABG and a lactate are the 2 best tests to help you get a sick patient to the ICU

Step 2: What is the primary process?

A. Look at the pH.
   - Is it normal, acidemic or alkalemic?

B. Look at pCO2 – is it “concordant” with pH change?
   - Is CO2 an acid... So, is ΔCO2 in the direction that would cause pH change?
   - If concordant, the primary problem is respiratory
     - Low pH and high pCO2 indicates respiratory acidosis
     - High pH and low pCO2 indicates respiratory alkalosis
   - If not concordant, the primary problem is metabolic
     - Low pH and low pCO2 indicates metabolic acidosis
     - High pH and high pCO2 indicates metabolic alkalosis

Step 3: What is the compensation?

- Then look at HCO3-
  - Has it changed by the expected amount?
    - It doesn’t have to be “perfect”
  - Change in HCO3 can tell you
    - if the disorder is acute or chronic
    - Whether multiple disorders are present

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<td>↓ 4</td>
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<tr>
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</tr>
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- 7.30 / 80 / 45 / 38
Step 3: What is the compensation?

- If compensation is "right", there is one process
- If compensation doesn’t "fit", there may be more than one process going on

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Step 4: Determine the Anion Gap

- 3.6
- 140 | / |
- 100 | 35 \ |
- 85

Anion Gap = Na⁺ - Cl⁻ - HCO3⁻
= 140 – 100 – 35
= 5 (normal is < 12)

Anion Gap – DDx

- Medical student answer: MUDPILES
- Real life answer...
  - Lactic acidosis
  - Ketosis (DKA, starvation, alcohol)
  - Renal failure
  - Poison (alcohols, ASA, cyanide)

Step 5: If an Anion Gap is present, is it the only process?

- Each molecule of unmeasured anion (ie. Lactate) donates a H⁺ which binds to HCO3⁻.
- H⁺ + HCO3⁻ → H₂O and CO₂
- Therefore, if there is only one process,
  - Amount of added acid = the increase in H⁺ = the fall in HCO3⁻
  - The amount of added acid is measured using the anion gap
  - So, the change in Anion Gap should equal the change in HCO3⁻.

Step 5: If an Anion Gap is present, is it the only process?

- Calculate ΔAG/ΔHCO3⁻ ratio
  - ΔAG = measured AG – 12
  - Δ HCO3⁻ = 24 – measured HCO3⁻
**Step 5: If an Anion Gap is present, is it the only process?**

- **Calculate ΔAG/ΔHCO3- ratio**
- If ΔAG/ΔHCO3- ratio = 1 → no other process
  - Ratio > 1, HCO3 is too low → concomitant non-AG acidosis
  - Ratio < 1, HCO3 is too high → concomitant alkalosis

**Step 6: Determine the Osmolar (OSM) Gap**

- OSM gap = measured OSM - calculated OSM
  - Measured OSM: given by the lab
  - Calculated OSM = (2 x Na*) + BG + BUN
    - *“Two salts and a sugar bun.”*
  - Normal Osmolar gap < 10

**Step 6: Determine the Osmolar (OSM) Gap**

- DDx of a high osmolar gap
  - Methanol*
  - Ethylene glycol*
  - Ethanol
  - Mannitol
  - Acetone
  - Isopropyl alcohol
  - Others…

* Anion gap AND osmolar gap

**Step 7: Calculate the A-a gradient**

- A-a gradient = PAO2 – PaO2
    = [(713) x FiO2] – [PaCO2/RQ]
  - PaO2 = measured with ABG

**Step 7: Calculate the A-a gradient**

- Normal A-a gradient
  - A-a gradient < 10 is normal
  - A-a gradient is higher in elderly (up to 20)
Step 8: Causes of hypoxemia

- List the 5 major causes of hypoxemia
- Which have a normal A-a gradient?
- Which have a high A-a gradient?

Step 8: Causes of hypoxemia

1. Low inspired O2 content (low FiO2 or low PiO2)
2. Hypoventilation

3. V/Q mismatch
   - Asthma, COPD, Alveolar filling (fluid, blood, pus), pHTN
4. Shunt
   - Physiologic shunt
   - Intra-cardiac (ASD, PFO or VSD)
   - Intra-pulmonary
     - With normal capillaries: atelectasis or consolidation
     - With abnormal capillaries: pAVM’s or HPS
5. Diffusion abnormality
   - Severe ILD, severe COPD, etc…

Summarize this ABG

- Step 1: Get the ABG
- Step 2: Determine primary abnormality
- Step 3: What is the compensation
- Step 4: Assess the anion gap
- Step 5: Is the anion gap the only process
- Step 6: Calculate the osmolar gap
- Step 7: Calculate the A-a gradient
- Step 8: Cause of hypoxemia

Causes of Hypercapnia

- What are the determinants of PaCO2?
- PaCO2 = (VCO2) / RR (Vt-Vd) x K
  - CO2 production
  - Respiratory rate
  - Tidal volume
  - Dead space volume

Causes of Hypercapnia

PaCO2 = (VCO2) / RR (Vt-Vd) x K

- High VCO2
  - fever, sepsis, seizures
- Low RR
  - drugs, brainstem lesions, hypothyroid
- Low Vt
  - muscle weakness (rapid shallow breathing pattern), neuromuscular disease, low chest wall compliance
- High Vd
  - ARDS, PE, COPD
Back to the case

- You get a call from your clinical clerk...
  - “I need your input on Ms. K. She is a 65 year old woman who is here for a small bowel obstruction. Med consults is following her for long standing back pain and they are working her up for possible cancer.”
- ABG (pH/PaCO2/PaO2/HCO3-)
  7.30 / 80 / 45 / 38
- You diagnose a chronic respiratory acidosis with a normal A-a gradient due to hypoventilation
  - You remove the fentanyl patch from her arm
  - You transfer her to the ICU

Back to the case

- 15 minutes later
  - Patient arrives in ICU
  - RT feels patient is worse
- ABG: 7.30 / 80 / 30 / 38
  - What happened?

Baseline ABG:
7.30 / 80 / 45 / 38

Back to the case

- ABG: 7.30 / 80 / 30 / 38
  - Acid base status unchanged
  - PaO2 fell from 45 → 30
- A-a gradient has increased
  - $A-a = PAO2 – PaO2$
  - $A-a = [150 – (1.25 x PaCO2)] – PaO2$
  - $A-a = [150 – (1.25 x 80)] – 30$
  - $A-a = 100 – 30$
  - $A-a = 70$
- DDx?

Baseline ABG:
7.30 / 80 / 45 / 38

Back to the case:
Causes of hypoxemia

1. Low inspired O2 content (low FiO2 or low PIO2)
2. Hypoventilation

3. V/Q mismatch
   - Asthma, COPD, Alveolar filling (fluid, blood, pus), pHTN
4. Shunt
   - Physiologic shunt
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     - With normal capillaries: atelectasis or consolidation
     - With abnormal capillaries: pAVM’s or HPS
5. Diffusion abnormality
   - Severe ILD, severe COPD, etc...

Back to the case:
DDx of acute rise in A-a gradient

- V/Q mismatch
  - Aspiration pneumonitis
  - Flash pulmonary edema
  - Mucous plug
  - Pneumothorax
  - PE
  - (ARDS)

Review:
ABG interpretation in 8 steps

- Step 1: Get the ABG
- Step 2: Determine primary abnormality
- Step 3: What is the compensation
- Step 4: Assess the anion gap
- Step 5: Is the anion gap the only process
- Step 6: Calculate the osmolar gap
- Step 7: Calculate the A-a gradient
- Step 8: Causes of hypoxemia