ABG Interpretation:  
A Respirologist’s approach

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September 2020
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" = 1° abnormality in ventilation (CO2)
2. "Metabolic" = 1° 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 $p_a$CO2
    • 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 $p_a$CO2 rises, appropriate compensation increases HCO3-
  – If $p_a$CO2 falls, appropriate compensation decreases HCO3-
  – If HCO3- rises, appropriate compensation increases $p_a$CO2
  – If HCO3- falls, appropriate compensation decreases $p_a$CO2
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 chemoceptors 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 [CO2] = ↑ ventilation
  – When patient has alkalemia (high pH)
    • Carotid body makes you less sensitive to [CO2] = ↓ ventilation
Respiratory Acidosis

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

- Hypoventilation causes rise in \( p_a \text{CO}_2 \), shifts equilibrium to the right.

  - **Acutely (10:1)**
    - For each 10 mm Hg rise in \( p_a \text{CO}_2 \), HCO3 should increase by 1 mEq/L
    - This is due to equilibrium shift (buffering)

  - **Chronically (10:3)**
    - For each 10 mm Hg rise in \( p_a \text{CO}_2 \), HCO3 should increase by 3 mEq/L
    - This is due to renal compensation (excretion of H+)

- **Common causes**
  - Lung disease
  - Neuromuscular disease
  - Sedative drugs
  - Adaptation to extreme obesity and sleep apnea

\[
[\text{H}^+] = \frac{24 \times p_a \text{CO}_2}{[\text{HCO}_3^-]} 
\]
Respiratory Alkalosis

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

- Hyperventilation causes \( p_a \text{CO}_2 \) to fall, shifts equilibrium to the left.
  
  - Acutely (10:2)
    - For each 10 mm Hg fall in \( p_a \text{CO}_2 \), \( \text{HCO}_3^- \) should decrease by 2 mEq/L
    - This is due to equilibrium shift (buffering)
  
  - Chronically (10:4)
    - For each 10 mm Hg fall in \( p_a \text{CO}_2 \), \( \text{HCO}_3^- \) 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

\[
[\text{H}^+] = \frac{24 \times p_a \text{CO}_2}{[\text{HCO}_3^-]} \]
Metabolic Alkalosis

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

• Increase in HCO3- shifts equilibrium to the left
  – \( p_a \text{CO}_2 \) increases
  – Alkalemia makes the carotid body less sensitive to [CO2]
  – We “allow” the \( p_a \text{CO}_2 \) to stay elevated (maximum ~ 50 mmHg)

• Compensation (1:0.7)
  – For every 1 mEq rise in HCO3-, \( p_a \text{CO}_2 \) increases by 0.7 mmHg

• Causes
  – Volume depletion ("contraction alkalosis")
  – Nasogastric suction
  – Diuretics
  – Hyperaldosteronism

\[ [\text{H}^+] = \frac{24 \times p_a \text{CO}_2}{[\text{HCO}_3^-]} \]
Metabolic Acidosis

• Two possible mechanisms of onset
  – Loss of HCO3-
  – Gain of H+

• Mechanisms of compensation are a bit more complex
Metabolic Acidosis – Bicarb Loss

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

- Loss of bicarbonate (ie. diarrhea) creates an acidemia (a relative increase in [H+])
  - Acidemia makes the carotid body more sensitive to [CO2] leading to increased ventilation

- Compensation (1:1)
  - For every drop of 1 mEq of HCO3-, \( p_a \text{CO}_2 \) falls by 1 mmHg

\[
\text{[H+] = } \frac{24 \times p_a \text{CO}_2}{[\text{HCO}_3^-]}\]
Metabolic Acidosis – Acid Gain

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

• Some acid gets added to the body (ie. excercising muscle makes lactate), the equilibrium gets shifted to the left (which lowers \( \text{HCO}_3^- \) and \( \text{H}^+ \))
  – This causes a transient rise in \([\text{CO}_2]\)
  – Excess \text{CO}_2 is quickly exhaled
  – \([\text{CO}_2]\) 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 \( \text{HCO}_3^- \), \( p_a\text{CO}_2 \) 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 (acetic acid)
  – ASA (acetylsalicylic acid)

\[ [\text{H}^+] = \frac{24 \times p_a\text{CO}_2}{[\text{HCO}_3^-]} \]
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

  140 | 3.9 / 100 | 35 \ 85

Please interpret this ABG
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

• 7.30 / 80 / 45 / 38
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: Determine the primary process:**
  - Is it an acidosis or an alkalosis?
  - Is the primary problem respiratory or metabolic?

7.30 / 80 / 45 / 38
Step 2: What is the primary process?

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

B. Look at $p_aCO_2$ – is it “concordant” with pH change? (i.e. CO2 is an acid… So, is $\Delta CO_2$ in the direction that would cause pH change?)
   - If concordant, the primary problem is respiratory
     - Low pH and high $p_aCO_2$ indicates respiratory acidosis
     - High pH and low $p_aCO_2$ indicates respiratory alkalosis
   - If not concordant, the primary problem is metabolic
     - Low pH and low $p_aCO_2$ indicates metabolic acidosis
     - High pH and high $p_aCO_2$ indicates metabolic alkalosis
Step 3: What is the compensation?

- Then look at $\text{HCO}_3^-$
  - Has it changed by the expected amount?
    - It doesn’t have to be “perfect”
  - Change in $\text{HCO}_3^-$ can tell you
    - if the disorder is acute or chronic
    - Whether multiple disorders are present

<table>
<thead>
<tr>
<th></th>
<th>$\Delta p_a\text{CO}_2$</th>
<th>$\Delta \text{HCO}_3^-$</th>
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<tbody>
<tr>
<td>Acute Respiratory Acidosis</td>
<td>$\uparrow 10$</td>
<td>$\uparrow 1$</td>
</tr>
<tr>
<td>Acute Respiratory Alkalosis</td>
<td>$\downarrow 10$</td>
<td>$\downarrow 2$</td>
</tr>
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<td>$\downarrow 10$</td>
<td>$\downarrow 4$</td>
</tr>
<tr>
<td>Metabolic Alkalosis</td>
<td>$\uparrow 0.7$</td>
<td>$\uparrow 1$</td>
</tr>
<tr>
<td>Metabolic Acidosis</td>
<td>$\downarrow 1$</td>
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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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$7.30 / 80 / 45 / 38$
Step 4: Determine the Anion Gap

\[
\text{Anion Gap} = \text{Na}^+ - \text{Cl}^- - \text{HCO}_3^-
\]

\[
= 140 - 100 - 35
\]

\[
= 5 \text{ (normal is < 12)}
\]

\[7.30 / 80 / 45 / 38\]
Anion Gap

- What causes an increased anion gap?

An extra unmeasured anion

All the cations in the body

(Na+)

Phosphate, Pyruvate, Sulfate, Lactate

Albumin

HCO₃⁻

Cl⁻
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-

- \( H^+ + HCO3^- \rightarrow H2O \text{ and } CO2 \)

- 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 $\Delta AG/\Delta HCO_3^-$ ratio

  $\Delta AG = \text{measured AG} - 12$

  $\Delta HCO_3^- = 24 - \text{measured HCO}_3^-$

- $7.30 / 80 / 45 / 38$
Step 5: If an Anion Gap is present, is it the only process?

• Calculate $\Delta AG/\Delta HCO_3$- ratio

• If $\Delta AG/\Delta HCO_3$- ratio = 1 $\rightarrow$ no other process
  
  – Ratio > 1, HCO$_3$ is too low $\rightarrow$ concomitant non-AG acidosis
  
  – Ratio < 1, HCO$_3$ is too high $\rightarrow$ concomitant alkalosis

• 7.30 / 80 / 45 / 38
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

- 7.30 / 80 / 45 / 38
Step 7: Calculate the A-a gradient

- \( PAO2 = [(Pbar - PH20) \times FiO2] - [PaCO2/RQ] \)

- \( A-a = PAO2 - PaO2 \)
- \( A-a = [(Pbar - PH20) \times FiO2] - [PaCO2/RQ] - PaO2 \)
- \( A-a = [(760 - 47) \times 0.21] - [PaCO2 / 0.8] - PaO2 \)
- \( A-a = [(760 - 47) \times 0.21] - [1.25 \times PaCO2] - PaO2 \)
- \( A-a = [(713) \times 0.21] - [1.25 \times PaCO2] - PaO2 \)
- \( A-a = [150] - [1.25 \times PaCO2] - PaO2 \) - Simplified version for pt on R/A

- 7.30 / 80 / 45 / 38
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

7.30 / 80 / 45 / 38

140 / 3.6
100 / 35 \ 85
Summarize this ABG

- Step 1: done
- Step 2: chronic respiratory acidosis
- Step 3: compensated appropriately (10:3.5)
- Step 4: anion gap = 5 (normal)
- Step 5: no anion gap present
- Step 6: osmolar gap (can’t do)
- Step 7: A-a gradient = 5 (normal)
- Step 8: hypoxemia due to hypoV

7.30 / 80 / 45 / 38
140 | ___ / 3.6
100 | 35 \ 85
Causes of Hypercapnia

• What are the determinants of PaCO2?

• \( \text{PaCO2} = \frac{(\text{VCO2})}{\text{RR} \times (\text{Vt-Vd})} \times K \)
  
  – 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 × PaCO2)] – PaO2
  – A-a = [150 – (1.25 × 80)] – 30
  – A-a = [150 – 100] – 30
  – A-a = 20

• 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
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   - Intra-pulmonary
     • 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 pneumomitis
  – 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