University Health Network
Toronto General Hospital Toronto Western Hospital Princess Margaret Hospital

# ABG Interpretation: A Respirologist's approach

Dr. Shane Shapera
Division of Respirology
University Health Network
October 2014

## 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
  - Mutliple 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 $p_aCO2$
    - 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 $p_aCO2$ rises, appropriate compensation increases HCO3-
  - If $p_aCO2$ falls, appropriate compensation decreases HCO3-
  - If HCO3- rises, appropriate compensation increases $p_aCO2$
  - If HCO3- falls, apppropriate compensation decreases $p_aCO2$

## 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 chemoerceptors 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

$\uparrow CO_2 + H_2O \rightarrow \uparrow H^+ + \uparrow HCO_3^-$

- Hypoventilation causes rise in $p_aCO2$, shifts equilibrium to the right.
  - Acutely (10:1)
    - For each 10 mm Hg rise in $p_aCO2$, HCO3 should increase by 1 mEq/L
    - This is due to equilibrium shift (buffering)
  - Chronically (10:3)
    - For each 10 mm Hg rise in $p_aCO2$, 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

$[H+] = \frac{24 \times p_aCO2}{[HCO3]}$

## Respiratory Alkalosis

$\downarrow CO_2 + H_2O \leftarrow \downarrow H^+ + \downarrow HCO_3^-$

- Hyperventilation causes $p_aCO2$ to fall, shifts equilibrium to the left.
  - Acutely (10:2)
    - For each 10 mm Hg fall in $p_aCO2$, HCO3 should decrease by 2 mEq/L
    - This is due to equilibrium shift (buffering)
  - Chronically (10:4)
    - For each 10 mm Hg fall in $p_aCO2$, HCO3 should decrease by 4 mEq/L
    - This is due to renal compensation (excretion of HCO3-)
  - Common causes
    - Anxiety / panic (including panic attacks)
    - Pregnancy
    - Early sepsis
    - Drugs (one component of ASA toxicity)
    - Mechanical ventilation at excessive rate or volumes

$[H+] = \frac{24 \times p_aCO2}{[HCO3]}$

## Metabolic Alkalosis

$\uparrow CO_2 + H_2O \leftarrow H^+ + \uparrow HCO_3^-$

- Increase in HCO3- shifts equilillbrium to the left
  - $p_aCO2$ increases
  - Alkalemia makes the carotid body less sensitive to [CO2]
  - We "allow" the $p_aCO2$ to stay elevated (maximum ~ 50 mmHg)
- Compensation (1:0.7)
  - For every 1 mEq rise in HCO3-, $p_aCO2$ increases by 0.7 mmHg
- Causes
  - Volume depletion ("contraction alkalosis")
  - Nasogastric suction
  - Diuretics
  - Hyperaldosteronism

$[H+] = \frac{24 \times p_aCO2}{[HCO3]}$

## 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

$\downarrow CO_2 + H_2O \Leftrightarrow H^+ + \downarrow HCO_3^-$

- Loss of bicarbinate (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-, $p_aCO2$ falls by 1 mmHg

$[H+] = \frac{24 \times p_aCO2}{[HCO3]}$

## Metabolic Acidosis – Acid Gain

$\downarrow CO_2 + H_2O \leftarrow \uparrow H^+ + \downarrow HCO_3^-$

- Some acid gets added to the body (ie. excercising muscle makes lactate), the equilibrium gets shifted to the left (which lowers HCO3- and H+)
  - This causes a transient rise in [CO2]
  - Excess CO2 is quickly exhaled
  - [CO2] falls even lower than baseline, because acidemia makes the carotid body more sensitive to [CO2] leading to increased ventilation
- Compensation
  - For every drop of 1 mEq of HCO3-, $p_aCO2$ falls by 1 mmHg
- Examples of metabolic acidosis
  - Diarrhea/GI losses (loss of HCO3)
  - lactic acidosis (lactic acid)
  - renal failure (metabolic acids and loss of HCO3)
  - diabetic ketoacidosis (acetic acid)
  - ASA (acetylsalicylic acid)

$[H+] = \frac{24 \times p_aCO2}{[HCO3]}$

# ABG Interpretation

## "ABG's in 8 steps"

## A Case

- You get a call from a 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 **Please interpret this ABG**

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: Determine the primary process:**
  - Is it an acidosis or an alkalosis?
  - Is the primary problem respiratory or metabolic?



## Step 2: What is the primary process?

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

B. Look at $p_aCO2$– is it "concordant" with pH change?
(i.e. CO2 is an acid… So, is ΔCO2 in the direction that would cause pH change?)

- If concordant, the primary problem is respiratory
  - Low pH and high $p_aCO2$ indicates respiratory acidosis
  - High pH and low $p_aCO2$ indicates respiratory alkalosis
- If not concordant, the primary problem is metabolic
  - Low pH and low $p_aCO2$ indicates metabolic acidosis
  - High pH and high $p_aCO2$ indicates metabolic alkalosis

## Step 3: What is the compensation?

- Then look at $HCO_3^-$
  - 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

| | Δ $p_aCO2$ | Δ HCO3- |
|---|---|---|
| Acute Respiratory Acidosis | ↑ 10 | ↑ 1 |
| Acute Respiratory Alkalosis | ↓ 10 | ↓ 2 |
| Chronic Respiratory Acidosis | ↑ 10 | ↑ 3 |
| Chronic Respiratory Alkalosis | ↓ 10 | ↓ 4 |
| Metabolic Alkalosis | ↑ 0.7 | ↑ 1 |
| Metabolic Acidosis | ↓ 1 | ↓ 1 |

## 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

| | Δ $p_aCO2$ | Δ HCO3- |
|---|---|---|
| Acute Respiratory Acidosis | ↑ 10 | ↑ 1 |
| Acute Respiratory Alkalosis | ↓ 10 | ↓ 2 |
| Chronic Respiratory Acidosis | ↑ 10 | ↑ 3 |
| Chronic Respiratory Alkalosis | ↓ 10 | ↓ 4 |
| Metabolic Alkalosis | ↑ 0.7 | ↑ 1 |
| Metabolic Acidosis | ↓ 1 | ↓ 1 |

•7.30 / 80 / 45 / 38

## Step 4: Determine the Anion Gap

3.6

140 | /

100 | 35 \

85

Anion Gap = $Na^+ - Cl^- - HCO3^-$

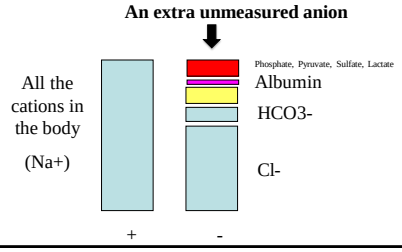
= 140 – 100 – 35

= 5 (normal is < 12)

•7.30 / 80 / 45 / 38

## Anion Gap

- What causes an increased anion gap?




## 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-
- $H^+ + HCO3^- \rightarrow$ H20 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-

•7.30 / 80 / 45 / 38

## 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

•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
- PAO2 = [(Pbar – PH20) x FiO2] – [PaCO2/RQ]
  = [(760 - 47) x FiO2] – [PaCO2/RQ]
  = [(713) x FiO2] – [PaCO2/RQ]
- PaO2 = measured with ABG

•7.30 / 80 / 45 / 38

## Step 7: Calculate the A-a gradient

- PAO2 = [(Pbar – PH20) x FiO2] – [PaCO2/RQ]
- A-a = PAO2 – PaO2
- A-a = [(Pbar – PH20) x FiO2] – [PaCO2/RQ] – PaO2
- A-a = [(760 – 47) x 0.21] – [PaCO2 / 0.8] – PaO2
- A-a = [(760 – 47) x 0.21] – [1.25 x PaCO2] – PaO2
- A-a = [(713) x 0.21] – [1.25 x PaCO2] – PaO2
- **A-a = [150] – [1.25 x 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

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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?
- 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 a 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 = [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

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**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…**

## 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