ABG INTERPRETATION

Steps to ABG interpretation

Step 1:

Determine the pH –
If pH is < 7.40 – then an acidosis is present
If pH is > 7.40 – then an alkalosis is present

Step 2:

Determine the cause of the Acidosis or Alkalosis by looking at the PCO2 and HCO3-

If pH < 7.40 and the PCO2 is high – then it is a RESPIRATORY ACIDOSIS
If pH < 7.40 and the PCO2 is low – then it is a METABOLIC ACIDOSIS
If pH > 7.40 and the PCO2 is low – then it is a RESPIRATORY ALKALOSIS
If pH > 7.40 and the PCO2 is high – then it is a METABOLIC ALKALOSIS

Therefore, by looking at the pH and the PCO2; you can determine the cause of the acidosis or alkalosis. First look at the pH and then look at the PCO2 to see if it is the cause of the pH change. Since an elevated PCO2 causes acidosis, you can determine whether it is the cause of the pH change that you see.

STEP 3

Determine the degree of Compensation present
The body is continually trying to return its pH back to baseline and will try to compensate to restore itself back to a pH of 7.40. If the cause of the change is pH is a respiratory one; then the body will induce a metabolic compensation – usually through the kidneys. If the change in pH is due to metabolic cause; then the body will compensate through the respiratory system. Full compensation occurs when the pH is close to 7.40. The respiratory compensation can occur over a matter of hours whereas the metabolic compensation takes a few days.

**Key Points**

- **CO₂** - is a weak acid. As it rises, acidosis worsens
- **HCO₃⁻** is a weak base. As is rises, alkalosis worsens
- Compensation is the body’s way of restoring the pH back to a neutral state (pH of 7.40)
- **If the primary problem is an acidosis; then the body will try to shift back to a neutral state via compensatory changes**

**Respiratory Acidosis**

The pH is initially below 7.40 and this is due to an elevated PCO₂. This is due to hypoventilation – Causes can include **COPD, asthma, drug overdose, respiratory fatigue, obstructive sleep apnea and stroke**.

The **pH drops by 0.08** for every increase in **10 of PCO₂**. Therefore if the PCO₂ is 60 (normal is 40) – you would expect the pH to be 7.24 in acute uncompensated respiratory acidosis

If the pH is not 7.24 – then there has been some degree of renal compensation and the HCO₃⁻ will be elevated.

**Acute rise in PCO₂** - The **HCO₃⁻ rises by 1 mmol/L** for every 10 rise in PCO₂ – you would expect the HCO₃⁻ in this case to be 26 (normal being 24)

**Chronic rise in PCO₂** – The **HCO₃⁻ rises by 4 mmol/L** for every 10 rise in PCO₂

Therefore if the patient with a PCO₂ of 60 has a pH of 7.32 – you would expect their baseline PCO₂ to be around 50 (chronic CO₂ retainer) and their HCO₃ to be around 27-28 (chronic renal compensation)
**Metabolic Acidosis**

The pH is initially below 7.40, and this is due to a decrease in the bicarbonate. An exogenous acid is produced or taken in; and it takes up the bicarbonate leading to an acidosis. Causes include:

**Anion Gap Acidosis** \((\text{Na}^+ - (\text{HCO}_3^- + \text{Cl}^-))\) – Anion gap is usually around 12

- Methanol
- Uremia (renal failure)
- Diabetic Ketoacidosis
- Paraldehyde, Phenformin (metformin)
- Iron, Isoniazid
- Lactic acidosis
- Ethanol, Ethylene glycol
- Salicylates

Notice, this spells the pneumonic **MUDPILES**

For severe acidosis – pH < 6.8 – think of DKA or methanol

Lactic acidosis is produced whenever there is anaerobic metabolism. Therefore in can be present is shock states, intestinal ischemia, prolonged seizures, ischemic limbs etc

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**2. Non-anion Gap acidosis**

a. Severe diarrhea
b. Pancreatic Fistulas
c. Renal Tubular acidosis
d. Adrenal insufficiency
e. TPN therapy
f. Carbonic anhydrase inhibitor therapy (diamox)

-The **pH changes by 0.02 for every drop of HCO3- by 1.** Therefore if the HCO3 went down from 24 to 19 – you would expect the pH to drop form 7.40 to 7.30. If the pH is higher, then respiratory compensation is occurring (the patient will be hyperventilating. The maximum hyperventilation that will occur will be to a PCO2 of about 12 – 14. **Kussmaul’s** respirations are present and are seen as rapid deep breaths. The **lowest the HCO3-** can go is **3-4 mmol**

- The **PCO2 drops by 1.2 – 1.4 for every drop in 1 of the HCO3-**. Therefore if the HCO3- went down to 19 (drop of 5); you would expect to the PCO2 to drop by 7 to compensate – leading to a PCO2 of 33 (drop from 40). The **lowest the PCO2** will go is around **12 mm Hg**
**Winters Rule:** \[ \text{PCO}2 = 1.5 \times (\text{HCO}3) + 8 (+/- 2). \]

If the PCO2 is higher; there is a assoc resp acidosis and if the PCO2 is lower; there is an assoc resp alkalosis

**Respiratory Alkalosis**

The pH is usually > 7.40 and this is due to primary hyperventilation. The PCO2 is low - < 40. May be seen in any hyperventilation state (hypoxia, pain, fear, shock).

The pH rises by 0.08 for every decrease in 10 of the PCO2.

- **Acute hyperventilation** - metabolic compensation - the HCO3 – decreases by **2mmol for every 10 mmHg decrease in the PCO2**
- **Chronic hyperventilation** - metabolic compensation – the HCO3- decreases by **5mmol for every 10 mm Hg decrease in the PCO2**.

**Metabolic Alkalosis**

The pH is > 7.40. The HCO3- will be elevated (above 24) and the PCO2 will be below 40. The most common causes are:
Lost of Gastric Acid – usually from protracted vomiting. (you lose HCL when you vomit)
Excessive Diuresis. This leads to hypokalemia and the cells then shift H+ ions into the cell and move K+ out into the serum to restore the K+ to normal
Exogenous Na+HCO3- administration.

The lungs will try to compensate by hypoventilating leading to a rise in CO2. This is limited by the ensuing hypoxemia that develops.
For every **1 mmol rise in HCO3- the PCO2 should rise by 2 mm Hg for full compensation.** This rarely happens and usually the lungs compensate about 40 – 50% and the patient remains alkalotic until the metabolic abnormality is corrected. Usually Normal Saline administration corrects the alkalosis for the first 2 causes.

**Mixed Acidosis**

When the pH can’t be accounted for by the PCO2 or by the HCO3- a combination of both
may be the cause of the pH. This is seen in **Mixed acidosis** cases. **ASA** is the main one of concern. Can also see this in a patient who is hyperventilating from hypoxemia and has an underlying metabolic Acidosis (sepsis)

**Anion Gap:** $\text{Na}^+ - (\text{HCO}_3^- + \text{Cl}^-) = 10 - 12$ (normal range)

Causes in increased anion gap: - **Mudpiles**
Causes of decreased anion gap: - hypercalcemia, lithium intoxication, bromide intoxication, Hypergammaglobulinemia

**Delta Gap:** The difference between the patient's anion gap and then ormal anion gap. The amount is considered a HCO$_3$- equivalent. The amount the HCO$_3$- decreases should = the anion gap. If the delta gap was added to the HCO$_3$- the result should be a normal HCO$_3$-.
An elevated delta gap indicates a superimposed metabolic acidosis

**Example**

Vomiting ETOH patient (not Betzner)

$\text{Na} - 137\quad \text{K} - 3.8\quad \text{Cl} - 90\quad \text{HCO}_3^- - 22$
$\text{pH} 7.40\quad \text{PCO}_2 - 41\quad \text{PO}_2 - 85$

Anion gap $= 137 - (90 + 22) = 25$ (normal gap is 10)

Delta Gap $= 25 - 10$ (normal gap) $= 15$. Add the 15 to the HCO$_3$ $= 15 + 22 = 37$

The resultant HCO$_3$ (37) is above the normal range (24) and thus indicates a superimposed metabolic alkalosis (from vomiting)

The acidosis would be from AKA

**Oxygenation Calculations**

The PO2 is a function of oxygenation and not ventilation. The oxygen saturation is a function of the arterial PO2
The oxy-hemoglobin saturation curve determines the O2sats for a given alveolar PO2.
In general an arterial **PO2 of 60 = O2 sat of 90%**
The alveolar PO2 is the amount of oxygen in the lung alveoli. This then crosses the alveolar
capillary membrane into the arterial blood. **The difference between the alveolar and arterial PO2 is usually around 10-25 mm Hg (rises with age. 0.4 X age is a reasonable estimate)**

The alveolar PO2 and arterial PO2 can be quite different in cases of V-Q mismatch (Pneumonia, Chf, ARDS, PE)

The alveolar PO2 is determined by the following equation

\[
PO2 = (FiO2 \times (\text{atmospheric pressure} – \text{partial pressure of water}))-\frac{PCO2}{0.8}
\]

\(FiO2 = \text{amount of Oxygen delivered to the patient.}\)

Room air = 21% oxygen.

Every litre via prongs adds 3% upto about 15% total (5 litres/min)

NRB is about 80% FiO2

Intubated patients have 100% FiO2

Atmospheric pressure in - Calgary 660 mm Hg

sea level 760 mm Hg

partial pressure of water = 47 mm Hg

0.8 is a constant that the CO2 is divided by

Therefore in Calgary the equation would be:

\[
(\text{FiO2} \times (660 – 47)) – \frac{\text{PCO2}}{0.8} = \text{alveolar PO2}
\]

eg: if the patient was on a NRB in Calgary and had a normal PCO2 (40)

then the equation would be

\[
0.80 \times (660-47) = 490
\]

\[
490 – 40/0.8 = 440 – \text{Final alveolar PO2. The arterial PO2 should be around 420 mm Hg.}
\]

**Case 2**

A patient is on NRB and has a sat of 93%. What is the arterial PO2? What is the expected arterial PO2.

The expected arterial PO2 is what was calculated earlier – around 420 mm Hg (may vary by 10-20 mm Hg)
The patient has a sat of 93%. We know that a sat of 90% correlates with a PO2 of 60. Therefore a sat of 93% correlates with a PO2 of 65 – 70 mm Hg.
This patient has a A-A gradient (alveolar – arterial gradient) of 420-70 = 350. This is massive gradient indicative of significant V-Q mismatch.

**The oxygen saturation that is measured must be taken in context of the FiO2 that is being administered.**

Venous Blood Gases

The Venous blood Gas has a close approximation to the arterial blood gases. They will tend to differ more when the patient is in shock. The differences are as follows:

1. **pH** – will be about 0.04-0.07 lower with venous gases. Therefore, add this amount to the measured level and that will be a close approximation of the arterial blood gas
2. **PCO2** - the PCO2 will be about 6-7 mm HG greater than with an ABG. Therefore, subtract this amount to get a close approximation of the arterial PCO2.
3. **HCO3-** the HCO3 – will be about 1-2 mm higher on the venous blood gas.

Electrolytes and Hemoglobin will be accurate

Capillary Blood Gases

The Capillary blood Gases will also have an approximation to Arterial Blood Gases
It will differ slightly in shock states

1. **pH** – will be about 0.04 lower than arterial gases

PCO2 – will be very similar to arterial gases. This can help determine the ventilation rate

HCO3- will be about 1-2 mm higher that an arterial gas
PO2 – will not be accurate.
Electrolytes and Hemoglobin will be accurate

CASES

Case 1

A 25 year old female who has a history of IDDM presents with nausea and vomiting. Pt is confused and tachypneic.

Vitals – 108/80  P 130   RR  36

ABG done – pH -6.82.
PCO2 - 23 mm Hg
pO2 - 48 mm Hg
HCO3- 4 mmol

Question - What is the primary pH disorder?
What is the main cause of the disorder?
What degree of compensation is present?

Answer

The patient has a metabolic acidosis. The pH is < 7.4 therefore and acidosis is present. The PCO2 is low and therefore is not the cause of the acidosis. The HCO3- is low and is the cause of the acidosis.
The pH drops by 0.02 for every drop in 1 of the HCO3-.
The HCO3 is at 4 mmol (the lowest it can go)
The PCO2 compensation is 1.25 X the drop in HCO3

Calculation – pH decrease = 7.40 – 6.82 = 0.58 mm Hg
The HCO3 – should decrease by 0.58/2 = 29 mmol, however the maximum decrease that is possible is 20 mmol and that is what has occurred (24 mmol – 20 mmol = 4 mmol
PCO2 compensation = 1.25 X 20 mmol = 25 mmHG. Therefore the expected PCO2 should be 40 mm Hg – 25 mm Hg = 15 mm HG

In this case the PCO2 is 23 mmHg. Therefore full compensation is not present. This may be due to altered LOC or inability to hyperventilate adequately

**Cause** - The cause of the acidosis is DKA – the excess ketones cause a metabolic acidosis. The patient is also markedly hypoxic. The expected PO2 should be

\[ 0.21 \times (660 - 47) - 23/0.8 = 103 \text{ mm Hg} \]

It is 48 mm Hg

The patient may have pneumonia as the cause of her DKA or she may have V-Q mismatch from her acidosis.

Case 2

66 year old male who has a hx of COPD. Pt presents with increasing respiratory distress.

Vitals - P 130    RR 36    BP 140/80    T 37.6

**ABG Done**

- pH - 7.13
- pCO2 - 85 mm Hg
- pO2 - 88 mm Hg
- HCO3 – 28 mmol

**Question** - What is the primary pH disorder?
What is the cause of the disorder?
What is the degree of compensation present?

The primary disorder is an acidosis (the pH is < 7.40). The cause is the PCO2 as it is elevated. The HCO3- is elevated; which means there is some degree of renal compensation. This means that some degree of CO2 elevation has been present > 48 hrs (length of time for renal compensation)

The pH decreases by 0.08 for every rise of 10 in PCO2

Chronic renal compensation. The HCO3 rises by 4 mmol for every 10 mm Hg rise in PCO2
Calculation
The pH decrease is 0.27 (7.40-7.13). The expected PCO2 rise would be 0.27/0.08 X 10 = 34 mm Hg. Given that it was 85 mmHg (one would expect the baseline to be around 51 mmHg).
The HCO3 has gone up by 4 mmol. This would occur if the PCO2 has gone up by 10 mm Hg chronically (chronic CO2 retainer). The PCO2 would be expected to rise from 40 mm Hg to 50 mmHg. This correlates with the expected baseline of 51 that was determined by the pH and rise in PCO2.

The final explanation for this patient would be that he is a chronic CO2 retainer with a baseline CO2 of 50 mm Hg. The patient has had a respiratory deterioration leading to an acute rise in PCO2 to 85 and this has lead to a pH drop to 7.13.

Case 3

A 30 year old female who has an eating disorder and hx of bulimia; presents with a history of protracted vomiting

<table>
<thead>
<tr>
<th>ABG done</th>
<th>pH</th>
<th>-7.50</th>
</tr>
</thead>
<tbody>
<tr>
<td>pCO2</td>
<td>- 45 mm Hg</td>
<td></td>
</tr>
<tr>
<td>HCO3-</td>
<td>- 34 mmol</td>
<td></td>
</tr>
</tbody>
</table>

What is the pH disorder?
What is the cause of the disorder?
What degree of compensation is present?

The primary disorder is an alkalosis. The CO2 is elevated and is therefore not the cause. The HCO3- is elevated. This is a Metabolic Alkalosis. The HCO3- is elevated by 10 mmol. The PCO2 should rise by 0.02/1mmol rise in HCO3- for full compensation. The expected PCO2 would be 60 if full compensation was present (the pH would also be close to 7.40 if full compensation was present). Given that the PCO2 is 45, we would say the patient has a Metabolic alkalosis with partial respiratory compensation.

Case 4
A 41 year old male presents with dyspnea. Pt states that he has felt SOB for about 1 day. The patient has a left leg cast on.

Vitals – P 120     RR  32     T 37.3     BP 130/85       Sat 91%

**ABG Done**
- pH – 7.50
- pCO2 - 28
- PO2 – 61
- HCO3- 22

What is the pH disorder?
What is the cause of the disorder?
What degree of compensation is present?

The patient has an alkalosis present (pH > 7.40). The PCO2 is low and is therefore the cause of the alkalosis. The HCO3- is low and not the cause of the alkalosis. The patient has a **Respiratory alkalosis**. The HCO3- decreases by 2 mmol for every 10 decrease in the PCO2. In this case, the PCO2 is down by 12 and therefore we would expect the HCO3- to drop by 2 mmol – which it has in this case.

There is also V-Q mismatch present. We can calculate the expected PO2 by the following equation

\[
(FiO2 \times (660 – 47)) – PCO2/0.8 \\
0.21 \times (613) – 28/0.8 = 94
\]

The expected PO2 is 94, but the patient’s PO2 is 61 on the ABG. This tells you that the patient has significant V-Q mismatch.

Most likely the patient has a **Pulmonary Embolus** causing his hypoxemia and leading to hyperventilation to compensate. This leads to a respiratory alkalosis.

**Caveats**
Always look at the PO2 in context of the FiO2 the patient is being administered

If the patient has a significant metabolic acidosis and has respiratory compensation; caution must be taken when intubating the patient. Unless the same minute ventilation is maintained; the acidosis may worsen. The end-tidal PCO2 or cap gas PCO2 should be the same pre/post intubation in these cases or the acidosis will worsen.

Cap gases are useful for lytes, hemoglobin, and PCO2. The pH should be adjusted, but the trend is useful.