Acidosis and Alkalosis
What is an ABG?

• The Components
  – pH / PaCO₂ / PaO₂ / HCO₃ / O₂sat / BE

• Desired Ranges
  – pH - 7.35 - 7.45
  – PaCO₂ - 35-45 mmHg
  – PaO₂ - 80-100 mmHg
  – HCO₃ - 21-27
  – O₂sat - 95-100%
  – Base Excess - +/-2 mEq/L
Why Order an ABG?

- Aids in establishing a diagnosis
- Helps guide treatment plan
- Aids in ventilator management
- Improvement in acid/base management allows for optimal function of medications
- Acid/base status may alter electrolyte levels critical to patient status/care
• PH of arterial blood is 7.35-7.45
This is due to:
• Interacellular chemical buffering
• Lung(CO2)
• Kidney(HCO3)
Henderson-Hasselbalch Equation

\[ \text{pH} = pK_a + \log \frac{[A^-]}{[HA]} \quad \rightarrow \quad \text{pH} = pK_a + \log \frac{[\text{HCO}_3^-]}{[\text{H}_2\text{CO}_3]} \quad \rightarrow \quad \text{pH} = 6.1 + \log \frac{[\text{HCO}_3^-]}{(0.03 \times p\text{CO}_2)} \]

Henderson-Hasselbalch Equation

\[ \text{CO}_2 + \text{H}_2\text{O} \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}^+ + \text{HCO}_3^- \quad (1) \]

\[ \text{CO}_2 + \text{OH}^- \rightleftharpoons \text{HCO}_3^- \quad (2) \]

\[ \text{H}^+ = 24 \frac{P\text{CO}_2}{\text{HCO}_3^-} \quad (4) \]
NORMAL VALUES

Using a normal arterial PCO2 of 40 mm Hg and a normal serum [HCO3⁻] concentration of 24 mEq/L, the normal [H⁺] in arterial blood is

\[ 24 \times \left( \frac{40}{24} \right) = 40 \text{ nEq} / \text{L} \]
When a primary acid-base disturbance alters one component of the PCO2/[HCO3⁻] ratio, the compensatory response alters the other component in the same direction to keep the PCO2/[HCO3⁻] ratio constant.
COMPENSATORY CHANGES

When the primary disorder is metabolic (i.e., a change in \([\text{HCO}_3^-]\), the compensatory response is respiratory (i.e., a change in PCO2), and vice-versa. Compensation is not synonymous with correction.
When a primary acid-base disorder exists, the body attempts to return the pH to normal via the “other half” of acid base metabolism.

Primary metabolic disorder → Respiratory compensation

Primary respiratory disorder → Metabolic compensation
<table>
<thead>
<tr>
<th>Primary Disorder</th>
<th>Compensatory Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>Increased ventilation</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Decreased ventilation</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>Increased renal reabsorption of $\text{HCO}_3^-$ in the proximal tubule, decreased renal excretion of $\text{H}^+$ in the distal tubule</td>
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<tr>
<td>Respiratory alkalosis</td>
<td>Decreased renal reabsorption of $\text{HCO}_3^-$ in the proximal tubule, decreased renal excretion of $\text{H}^+$ in the distal tubule</td>
</tr>
</tbody>
</table>
Role of kidney for regulation of HCO$_3$-

- Reabsorption of filtered HCO$_3$-
- Production of titrable acid
- Excreation of NH$_4$
Kidney excretes 4000 mmol HCO₃⁻, also same amount H⁺.
80-90% absorb in proximal tubule.
Distal tubule excretes 40-60 mmol/day in the form of NH₄⁺ and titrable acid.
Renal Tubule

Filtered

Renal cell

Blood

Filtered

Renal Tubule

HCO₃⁻ + H⁺ + H₂O

H₂CO₃ → CO₂ + H₂O

Blood

Carbonic anhydrase

H⁺ + HCO₃⁻ → CO₂ + H₂O
\[ \text{CO}_2 \text{ from metabolism} \]

\[ \begin{align*}
\text{H}^+ &+ \text{HCO}_3^- \\
\text{PO}_4^{3-} &+ \text{NH}_3 \text{ excreted in urine} \\
\text{NH}_4^+ \text{ in tubule is excreted along with Cl}^- 
\end{align*} \]
Other mechanisms

- Liver produces glutamine from amino acids
  - Broken down into bicarb and ammonia in renal cells
- Bowel secretes bicarb-rich fluid by $\text{HCO}_3^- / \text{Cl}^-$ exchange
- RBCs
  
  $\text{H}^+ + \text{Hb} \rightarrow \text{H}^+\text{Hb}$ (buffering)

CO$_2$ → HCO$_3^-$

Chloride shift

Cl$^-$
ROLE OF LUNG

CO2 production and excretion is regulated by neural respiratory factor.

HYPERCAPNEA (hypoventilation)
HYPOCAPNEA (hyperventilation)

Primary changes in CO2 causes respiratory acidosis or respiratory alkalosis.

hyperpnea and hypopnea, refer to the total ventilation.

tachypnea and bradypnea, which indicate the number of breaths per minute.
The relationships among minute volume of ventilation (VE), arterial PCO₂, and the ratio of dead space to tidal volume (VD/VT) are shown. BTPS = volume corrected for body conditions (body temperature, ambient pressure, and saturation with water vapor).
Primary Acid-Base Disorders

As dictated by the Henderson-Hasselbalch equation, disturbances in either the respiratory component (pCO$_2$) or metabolic component (HCO$_3^-$) can lead to alterations in pH.

Metabolic Acidosis  
(Too little HCO$_3^-$)

Metabolic Alkalosis  
(Too much HCO$_3^-$)

Respiratory Acidosis  
(Too much CO$_2$)

Respiratory Alkalosis  
(Too little CO$_2$)
METABOLIC ACIDOSIS

IT HAS THREE MECHANISM:

1. RISE IN ENDOGENOUS ACID
2. LOSS OF HCO3 (diarrhea)
3. ACCUMULATION OF ENDOGENOUS ACID (CRF)
Metabolic Acidosis

• This is a metabolic acidosis as the pH, pCO2, and the HCO3 are all low.

• There are two types of metabolic acidosis: gapped and non-gapped.

• The former is known as an anion gapped acidosis and the latter as a hypercholoremic metabolic acidosis.
Metabolic acidosis

1. The anion gap is the difference between the concentration of sodium cations in the serum and the sum of the serum concentration of chloride anions and bicarbonate anions:

   \[ AG = \text{Na}^+ - (\text{Cl} + \text{HCO}_3^-) \]

2. Because the positive and negative ions must always be equal, under normal circumstances, the gap indicates the presence of unmeasured anions such as sulfates, organic ions, albumin.

3. A normal gap is either 8 to 16 (12)
Calculate the Anion Gap

• 1. Calculate the anion gap as described.
• 2. An anion gap over 25 suggests a severe metabolic acidosis.
• 3. Causes of an high anion gap: ethylene glycol, lactic acid, methanol, paraldehyde, aspirin, renal failure, ketoacidosis (diabetic or ethanol).
Metabolic acidosis

1. If there is a metabolic acidosis present, but there is no gap, check the chloride - which should be elevated. This is a non-gapped acidosis.

2. Non-gapped acidosis is caused by the loss of bicarbonate - either through the GI tract or through the kidney. The most common cause is diarrhea.
Treatment

• the treatment is to improve or repair the underlying cause if possible..
• DKA: insulin
• TOXIN: dialysis
• OR: bicarb
Metabolic alkalosis

• Is due to increase of HCO$_3$ (rare) or increase of paco2 due to hypoventilation or loss of acid (HCL in vomiting)
Treatment

• Acute metabolic alkalosis: pH above 7.55 is considered an emergency. This is treated with normal saline to restore volume and salt deficits.
• Acetazolamide in severe cases.
• HCl is used in extreme cases.
Respiratory Acidosis

• Respiratory acidosis is due to CNS depression, neuromuscular impairment, restricted airway, alveolar involvement such as pneumonia.
Respiratory Acidosis

- ↓pH, ↑CO$_2$, ↓Ventilation
- Causes
  - CNS depression
  - Pleural disease
  - COPD/ARDS
  - Musculoskeletal disorders
  - Compensation for metabolic alkalosis
Respiratory Acidosis

- Acute Vs Chronic
  - Acute - little kidney involvement
  - Chronic - Renal compensation via synthesis and retention of $\text{HCO}_3^-$
Clinical feature

- Acute rise in Paco2 cause anxiety, dyspnea, confusion, psychosis, hallucination and coma.
- Chronic rise cause sleep and memory disturbances, somnolence, astrixis and in advance cases headache, papiledema
TREATMENT

- Acute: intubation
- Chronic: gradual correction
Respiratory Alkalosis

• \( \text{pH} \uparrow, \text{CO}_2 \downarrow, \text{Ventilation} \uparrow \)
• \( \text{CO}_2 \downarrow \Downarrow \text{HCO}_3 \)
• Causes
  – Intracerebral hemorrhage
  – Salicylate and Progesterone drug usage
  – Anxiety \( \Downarrow \) lung compliance
  – Cirrhosis of the liver
  – Sepsis
  – Exercise
  – Hypoxia
• Decrease in brain perfusion, confusion, convulsion, numbness and lightheadedness
TREATMENT

• Underlying cause
• Rebreathing in bag
Interpretation of Arterial Blood Gases

\[ [\text{H}^+] = \frac{24(\text{PaCO}_2)}{[\text{HCO}_3^-]} \]
<table>
<thead>
<tr>
<th>&quot;Clue&quot;</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>High anion gap</td>
<td><em>Always</em> strongly suggests a metabolic acidosis.</td>
</tr>
<tr>
<td>Hyperglycaemia</td>
<td>diabetic ketoacidosis</td>
</tr>
<tr>
<td>Hypokalemia and/or hypochloremia</td>
<td>Suggests metabolic alkalosis</td>
</tr>
<tr>
<td>Hyperchloremia</td>
<td>Common with normal anion gap acidosis</td>
</tr>
<tr>
<td>Elevated creatinine and urea</td>
<td>Suggests uremic acidosis or hypovolemia (prerenal renal failure)</td>
</tr>
<tr>
<td>Urine dipstick tests for glucose and ketones</td>
<td>Glucose detected if hyperglycaemia; ketones detected if ketoacidosis</td>
</tr>
</tbody>
</table>
# Interpretation of Arterial Blood Gases

<table>
<thead>
<tr>
<th>pH</th>
<th>Approximate [H+] (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.00</td>
<td>100</td>
</tr>
<tr>
<td>7.05</td>
<td>89</td>
</tr>
<tr>
<td>7.10</td>
<td>79</td>
</tr>
<tr>
<td>7.15</td>
<td>71</td>
</tr>
<tr>
<td>7.20</td>
<td>63</td>
</tr>
<tr>
<td>7.25</td>
<td>56</td>
</tr>
<tr>
<td>7.30</td>
<td>50</td>
</tr>
<tr>
<td>7.35</td>
<td>45</td>
</tr>
<tr>
<td>7.40</td>
<td>40</td>
</tr>
<tr>
<td>7.45</td>
<td>35</td>
</tr>
<tr>
<td>7.50</td>
<td>32</td>
</tr>
<tr>
<td>7.55</td>
<td>28</td>
</tr>
<tr>
<td>7.60</td>
<td>25</td>
</tr>
<tr>
<td>7.65</td>
<td>22</td>
</tr>
</tbody>
</table>
Interpretation of Arterial Blood Gases

Is there alkalemia or acidemia present?

\[ \text{pH} < 7.35 \quad \text{acidemia} \]
\[ \text{pH} > 7.45 \quad \text{alkalemia} \]
## Interpretation of Arterial Blood Gases

Is the disturbance respiratory or metabolic?

<table>
<thead>
<tr>
<th></th>
<th>Type</th>
<th>pH</th>
<th>PaCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidosis</td>
<td>Respiratory</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Acidosis</td>
<td>Metabolic</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Alkalosis</td>
<td>Respiratory</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Alkalosis</td>
<td>Metabolic</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>
## Interpretation of Arterial Blood Gases

Is there appropriate compensation for the primary disturbance?

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Expected compensation</th>
<th>Correction factor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>( \text{PaCO}_2 = (1.5 \times [\text{HCO}_3^-]) + 8 )</td>
<td>± 2</td>
</tr>
<tr>
<td>Acute respiratory acidosis</td>
<td>Increase in ([\text{HCO}_3^-] = \Delta \text{PaCO}_2/10)</td>
<td>± 3</td>
</tr>
<tr>
<td>Chronic respiratory acidosis (3-5 days)</td>
<td>Increase in ([\text{HCO}_3^-] = 3.5(\Delta \text{PaCO}_2/10))</td>
<td></td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Increase in (\text{PaCO}_2 = 40 + 0.6(\Delta \text{HCO}_3^-))</td>
<td></td>
</tr>
<tr>
<td>Acute respiratory alkalosis</td>
<td>Decrease in ([\text{HCO}_3^-] = 2(\Delta \text{PaCO}_2/10))</td>
<td></td>
</tr>
<tr>
<td>Chronic respiratory alkalosis</td>
<td>Decrease in ([\text{HCO}_3^-] = 5(\Delta \text{PaCO}_2/10)) to 7((\Delta \text{PaCO}_2/10))</td>
<td></td>
</tr>
</tbody>
</table>
Interpretation of Arterial Blood Gases

Calculate the anion gap
Interpretation of Arterial Blood Gases

If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in $[\text{HCO}_3^-]$.

$\frac{\Delta \text{AG}}{\Delta [\text{HCO}_3^-]}$
# Interpretation of Arterial Blood Gases

## Selected mixed and complex acid-base disturbances

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Characteristics</th>
<th>Selected situations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis with metabolic acidosis</td>
<td>↓ in pH&lt;br&gt;↓ in HCO&lt;sub&gt;3&lt;/sub&gt;-&lt;br&gt;↑ in PaCO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>• Cardiac arrest&lt;br&gt;• Intoxications&lt;br&gt;• Multi-organ failure</td>
</tr>
<tr>
<td>Respiratory alkalosis with metabolic alkalosis</td>
<td>↑ in pH&lt;br&gt;↑ in HCO&lt;sub&gt;3&lt;/sub&gt;-&lt;br&gt;↓ in PaCO&lt;sub&gt;2&lt;/sub&gt;</td>
<td>• Cirrhosis with diuretics&lt;br&gt;• Pregnancy with vomiting&lt;br&gt;• Over ventilation of COPD</td>
</tr>
<tr>
<td>Respiratory acidosis with metabolic alkalosis</td>
<td>pH in normal range&lt;br&gt;↑ in PaCO&lt;sub&gt;2&lt;/sub&gt;,&lt;br&gt;↑ in HCO&lt;sub&gt;3&lt;/sub&gt;-</td>
<td>• COPD with diuretics, vomiting, NG suction&lt;br&gt;• Severe hypokalemia</td>
</tr>
</tbody>
</table>
## Interpretation of Arterial Blood Gases

### Selected mixed and complex acid-base disturbances

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Characteristics</th>
<th>Selected situations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory alkalosis with metabolic acidosis</td>
<td>pH in normal range ↓ in PaCO₂ ↓ in HCO₃</td>
<td>• Sepsis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Salicylate toxicity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Renal failure with CHF or pneumonia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Advanced liver disease</td>
</tr>
<tr>
<td>Metabolic acidosis with metabolic alkalosis</td>
<td>pH in normal range HCO₃- normal</td>
<td>• Uremia or ketoacidosis with vomiting, NG suction, diuretics, etc.</td>
</tr>
</tbody>
</table>
Base excess

Base excess beyond the reference range indicates

• **metabolic alkalosis** if too high (more than +2 mEq/L)
• **metabolic acidosis** if too low (less than −2 mEq/L)

A base excess (positive value) indicates an excess of base in the body and so mirrors a raised HCO₃⁻ level (metabolic alkalosis). A base deficit (negative value) indicates a lack of base in the body and so mirrors a reduced HCO₃⁻ level (metabolic acidosis).
• Very sick 56 year old woman being evaluated for a possible double lung transplant
• Dyspnea on minimal exertion
• On home oxygen therapy (nasal prongs, 2 lpm)
• Numerous pulmonary medications
While she is being assessed an arterial blood gas sample is taken, revealing the following:

- pH: 7.30
- PCO2: 65 mm Hg
- [HCO3⁻]: 31.1 mEq/L
CHRONIC RESPIRATORY ACIDOSIS
An obese 70 year old man has diabetes of 25 years duration complicated by coronary artery disease (CABG x 3 vessels 10 years ago), cerebrovascular disease (carotid artery endarterectomy 3 years ago)
Patient with Ischemic Bowel

ABGs obtained in the ICU

pH 7.18
PCO2 20 mmHg
HCO3 7 mEq/L

Expected PCO2 in metabolic acidosis

\[ \text{PCO2} = 1.5 \times \text{HCO3} + 8 \text{ (range: +/- 2)} \]

\[ = 1.5 \times 7 + 8 = 18.5 \]
Expected PCO2 in metabolic acidosis

\[ = 1.5 \times \text{HCO}_3^- + 8 \text{ (range: +/- 2)} \]

\[ = 1.5 \times 7 + 8 = 18.5 \]
<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum sodium</td>
<td>135 mEq/L</td>
</tr>
<tr>
<td>Serum bicarbonate</td>
<td>7 mEq/L</td>
</tr>
<tr>
<td>Serum chloride</td>
<td>98 mEq/L</td>
</tr>
</tbody>
</table>
Anion Gap =
Serum Sodium –
Serum Chloride –
Serum Bicarbonate

Anion Gap =
= 135 - 98 - 7 mEq/L
= 30 mEq/L
(ELEVATED)

SERUM ELECTROLYTE DATA

<p>| | |</p>
<table>
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<tr>
<td>Serum chloride</td>
<td>98 mEq/L</td>
</tr>
</tbody>
</table>
For an increased anion gap metabolic acidosis, are there other derangements?

\[ \frac{\Delta AG}{\Delta [\text{HCO}_3^-]} = \frac{18}{17} \]

Lactic Acidosis
Because of the extreme pain, the patient is given morphine

<table>
<thead>
<tr>
<th>pH</th>
<th>7.00 (was 7.18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCO2</td>
<td>25 mmHg (was 20)</td>
</tr>
<tr>
<td>HCO3</td>
<td>7 mEq/L</td>
</tr>
</tbody>
</table>

REMEMBER THAT MORPHINE IS A RESPIRATORY DEPRESSANT AND WILL ELEVATE PCO2
The expected degree of respiratory compensation is not present.

\[
\text{Expected PCO}_2 \text{ in metabolic acidosis} \]
\[
= 1.5 \times \text{HCO}_3 + 8 \quad \text{(range: +/- 2)}
\]
\[
= 1.5 \times 7 + 8 = 18.5
\]

BUT … we got a PCO2 of 25 mm Hg (as a result of respiratory depression from morphine administration) so the expected degree of respiratory compensation is not present.
A 23-year-old woman is 12 weeks pregnant. For the last with 10 days she has had worsening nausea and vomiting. When seen by her physician, she is dehydrated and has shallow respirations. Arterial blood gas data is as follows:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.56</td>
</tr>
<tr>
<td>PCO2</td>
<td>54 mm Hg</td>
</tr>
<tr>
<td>Hco3</td>
<td>45</td>
</tr>
</tbody>
</table>
Increase in PaCO$_2$ = $40 + 0.6(\Delta$HCO$_3^-$)

= $40 + 0.6(45-24) = 52$

Metabolic Alkalosis from Persistent Vomiting
The atmospheric pressure at Mount Everest is about a third that at sea level. When an ascent is made without oxygen, extreme hyperventilation is needed.

\[
\begin{align*}
\text{pH} &= 7.7 \\
\text{PCO}_2 &= 7.5 \\
\text{HCO}_3 &= 9
\end{align*}
\]