Arterial Blood Gases

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Arterial Blood Gases

- **Normal values:**

<table>
<thead>
<tr>
<th>ABG Value</th>
<th>Normal</th>
<th>Arterial Range</th>
<th>Venous Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.4</td>
<td>7.35-7.45</td>
<td>7.32-7.42</td>
</tr>
<tr>
<td>HCO3</td>
<td>25</td>
<td>22-26</td>
<td>19-25</td>
</tr>
<tr>
<td>PaCO2</td>
<td>40</td>
<td>35-45</td>
<td>38-52</td>
</tr>
<tr>
<td>PaO2</td>
<td>80</td>
<td>80-100</td>
<td>28-48</td>
</tr>
</tbody>
</table>

(Values from globalrph.com)

**Approach**

1. pH acidemic (pH < 7.35), alkalemic (pH > 7.45) or normal (pH 7.35-7.45)
2. What is the primary disturbance?
   - metabolic: change in HCO3 and pH in same direction
   - respiratory: change in HCO3 and pH in opposite directions
3. Is compensation appropriate?
   - Metabolic compensation: usually takes 2-3d (renal HCO3 production/excretion change)
   - Respiratory compensation: occurs immediately (PaCO2 change)
   - Inadequate compensation: usually means second acid-base disorder
     - Trick: can look at pH and see if pH is well compensated or not
   - Expected compensations:

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>$P_aCO_2$ (mmHg)</th>
<th>$HCO_3^-$ (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory Acidosis</td>
<td>↑10</td>
<td>↑1</td>
</tr>
<tr>
<td>Acute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic</td>
<td>↑10</td>
<td>↑3</td>
</tr>
<tr>
<td>Respiratory Alkalosis</td>
<td>↓10</td>
<td>↓2</td>
</tr>
<tr>
<td>Acute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chronic</td>
<td>↓10</td>
<td>↓5</td>
</tr>
<tr>
<td>Metabolic Acidosis</td>
<td>↓1</td>
<td>↓1</td>
</tr>
<tr>
<td>Metabolic Alkalosis</td>
<td>↑5-7</td>
<td>↑10</td>
</tr>
</tbody>
</table>

- Compensation
  - Resp Acidosis Acute --> Main Mechanism is Cellular buffering (1:10)
  - Resp Acidosis Chronic --> Renal excretion of H+ (3:10)
  - Resp Alkalosis Acute --> Cellular Buffering (2:10)
  - Resp Alkalosis Chronic --> Decreases renal excretion of H+ (4:10)
- Other ways:
  - Winter’s Formula: Expected $pCO2 = 1.5 \times [HCO3] + 8$ (based on real patients)
  - (Even easier formula) : $pCO2$ similar to last two digits of pH
    - I.e. pH = 7.21 (then $pCO2$ should be about 21)
    - Obviously won’t work if pH < 7
  - Metabolic Alkalosis
    - Formulas are not reliable, generally do not stop breathing for resp compensation
4. If there is metabolic acidosis --> calculate anion and osmolar gap
   - **anion gap** = $[Na^+] - ([Cl^-] + [HCO3^-])$; Normal ≤10-15 mmol/L
   - Do not include K+ because it’s a small player and always ~4.
   - Normal AG = 10... if > 10, then suspect additional acid.
   - Low Albumin and unmeasured cations (such as serum light chains) decrease anion gap
   - Drop normal AG by 2.5 for every 10 decrease in albumin. Normal AG = 10-2.5*(40-Albumin)/10
Osmolar gap = measured osmolarity - calculated osmolarity =
= measured - (2[Na+] + glucose + urea); Normal ≤ 10

Causes of elevated anion gap:
- KARMEL (ketoacidosis, ASA, Renal failure, methanol, ethylene glycol, lactic acid.), MUDPILES also used.
- Acute EtOH does NOT cause anion gap metabolic acidosis!! (if anion gap then another reason such as another alcohol)

Causes of elevation of osmolar gap:
- Alcohols (including EtOH)
- ... others

5. If metabolic acidosis with anion gap: Calculate Delta-Delta to see if there is another acid/base abnormality. Is extra anion gap same as change in bicarbonate?

\[
\text{Delta/Delta} = \frac{\text{delta AG}}{\text{delta HCO}_3^-} \]

When anion gap goes up by 1 point, bicarb does not go down by 1 (have other buffers).

- Relationship: for every 1-2mEq of anion gap, HCO3- falls by 1.
  - Lactic acidosis is ~1.6:1, in DKA it is more like 1:1 (ketones lost in urine)
  - If HCO3- falls too much, then overlying metabolic acidosis that is Non-anion gap.
  - If HCO3- falls too little, then something bringing bicarb up (i.e. metabolic alkalosis)

<table>
<thead>
<tr>
<th>Delta/Delta =</th>
<th>No other acid-base abnormality (HCO3 went down as expected)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1-2</td>
<td>Overlying metabolic alkalosis (HCO3 decr. less than expected)</td>
</tr>
<tr>
<td>&gt; 2</td>
<td>Overlying non-anion gap metabolic acidosis (HCO3 much lower than expected)</td>
</tr>
</tbody>
</table>

Differential Diagnosis

- Anion Gap Metabolic Acidosis
  - KARMEL
    - Ketoacidosis
    - ASA
    - Renal Failure (uremia)
    - Methanol
    - Ethylene glycol
    - Lactic Acidosis
  - MUDPILES
    - If don't like mnemonics, use Major Categories:
      1. Renal Failure (Uremia, with buildup of phosphates, etc.)
      2. Ketoacidosis (alcohol, survation, diabetic)
      3. Ingestion (Those that metabolize to acids)
        - Salicilates, Methanol, Ethylene Glycol
      4. Lactic Acidosis

<table>
<thead>
<tr>
<th>DDx of Respiratory Acidosis</th>
<th>DDx of Respiratory Alkalosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased PaCO2 due to hypoventilation</td>
<td>Decreased PaCO2 due to hyperventilation</td>
</tr>
</tbody>
</table>

Respiratory Center Depression (decreased RR)

- Drugs (anesthesia, sedation, narcotics)
- Trauma, Increased ICP, Encephalitis, stroke, central apnea, supplemental O2 in chronic CO2 retainers (i.e. COPD)

Neuromuscular Disorders (Decreased vital capacity)

- Myasthenia gravis
- Guillain-Barre syndrome
- Poliomyelitis

Hypoxemia

- Pulmonary Disease (pneumonia, edema, PE, interstitial fibrosis)
- Severe anemia
- Heart Failure
- High altitude

Respiratory Center Stimulation

- CNS disorders
- Hepatic failure
Approach to Oxygenation

- A-a gradient is the alveolar-arterial oxygen tension difference
  - Oxygen gradient between alveolus and pulmonary capillaries.
  1. What is PaO2? (normal 95-100%)
  2. What is the A-a gradient?
    - A-aDO2 = PAO2 (alveolar) - PaO2 (arterial) = [FiO2 (Patm- PH2O) - PaCO2/RQ] - PaO2
    - ... 
    - At sea level: A-a gradient = [150-1.25(PaCO2)] - PaO2
    - Normal A-a gradient increases with age. (roughly 15-20)
  3. Find the cause:

<Diagram>

Notes

- Methanol and Ethylene glycol intoxication
  - Consider "fomepizole", which is a competitive alcohol dehydrogenase inhibitor.
  - Consider dialysis
- Ethylene glycol intoxication causes calcium oxalate crystals in urine in later stages (usually >24hrs)
  - Direct toxicity, cortical edema, and inhibition of mitochondrial activity, as evidenced by decreased succinate dehydrogenase activity, are possible mechanisms of crystal damage. Since calcium oxalate monohydrate
crystals are transported intracellularly by kidney cells, the renal toxicity of ethylene glycol may result from inhibition of mitochondrial respiratory function in proximal tubular cells by calcium oxalate monohydrate crystals.

- **PaO2/FiO2 ratio**
  - Normal 550
  - <300 respiratory distress
  - <200 respiratory failure

- **NAGMA**
  - Diarrhea, Medications, Aldosterone
  - Urine anion gap, urine osmolar gap, and urine pH

**Respiratory Acidosis**

- Inadequate ventilation
- **Symptoms:**
  - If Severe: Impairs mentation, decr cerebral blood flow, cerebral edema, asterixis.
  - Cardiac arrhythmias/contractility