ABG TUTORIAL

The following is a systematic way to interpret SIMPLE acid/base disorders involving either primary respiratory or metabolic dysfunction, with or without compensation. Mixed disorders (combined respiratory and metabolic dysfunction) will be covered in the renal course.

**STEP 1. Classify the pH.** Acidemia is pH < 7.35 and alkalemia is pH > 7.45

**STEP 2. Is the 1° disturbance respiratory (look at PaCO2)?**
If acidemia is present (or arterial pH is on the acid side of normal) and PaCO2 > 45 mmHg, then hypoventilation is partly, or entirely, responsible for the acidosis.

If alkalemia is present (or arterial pH is on the alkaline side of normal) and PaCO2 < 35 mmHg, then hyperventilation is partly, or entirely, responsible for the alkalosis.

**EXPLANATION:** Normally, ventilation is matched to CO2 production and PaCO2 is tightly regulated at ~ 40 mmHg. When breathing normal air (not CO2-enriched air), the ONLY way to change PaCO2 is by affecting alveolar ventilation. A high PaCO2 is due to alveolar hypoventilation (ventilation that is inadequate for the rate of CO2 production) and causes respiratory acidosis. The formation of H+ is evident from Eq 1 which moves to the right by mass action when CO2 increases:

\[
CO_2 + H_2O \leftrightarrow H^+ + HCO_3^-
\]

A low PaCO2 (alveolar hyperventilation) causes respiratory alkalosis because a decrease in PCO2 decreases [H+] by moving Eq 1 to the left.

**STEP 3. Is the 1° disturbance metabolic (look at HCO3-)?**
Plasma HCO3- is a good (but not definitive) indicator of the metabolic involvement. The normal range is 22-26 mM (or mEq/L). If acidemia is present and [HCO3-] is < normal, then metabolic acidosis may be partly or entirely responsible for the acidemia. If alkalemia is present and [HCO3-] is > normal, then metabolic alkalosis may be entirely or partly responsible for the alkalemia.

**EXPLANATION:**
In METABOLIC ACIDOSIS, both pH and HCO3- (a base) decrease. The fall in HCO3- can be due either to direct loss of HCO3- (e.g. diarrhea) or gain of nonvolatile acid (e.g., lactic acid). A decrease in HCO3- increases [H+] by moving Eq 1 to the right. Metabolic increases in [H+] decrease HCO3- by moving Eq 1 to the left.

In METABOLIC ALKALOSIS both pH and HCO3- increase. A gain of HCO3- (e.g., ingestion) decreases [H+] by moving Eq 1 to the left and loss of nonvolatile acid (e.g., vomiting) increases HCO3- by moving Eq 1 to the right.

**CAVEAT:** Bicarbonate is also affected by the respiratory component (i.e., the mass action effect of CO2 in Eq 1), but the effect is relatively small. The predicted changes are +1 mM Δ in [HCO3-] per 10 mmHg increase in PaCO2 above the normal value of 40 mmHg or -2 mM Δ in [HCO3-] per 10 mmHg decrease in PaCO2 below normal. Any changes beyond what is predicted from PaCO2 are of metabolic origin.
NOTE: A more definitive measure of metabolic involvement is the base excess, which is not influenced by CO2.

**Step 4. Assess for compensation.**

If the 1° abnormality is respiratory, the kidneys compensate (correct the pH abnormality towards normal) by retaining or removing bicarbonate from plasma. Renal compensation takes time (3-5 days for completion), thus when the respiratory disturbance is uncompensated, this implies an acute rather than chronic respiratory defect. You can evaluate the compensation by looking at the magnitude of the change in pH (as per handout) or in [HCO3-]. Compensation is classified as **uncompensated**, **partial** (compensation has begun but pH is still abnormal), or **complete** (restoration to the acid or basic side of normal).

If the 1° disturbance is metabolic, the respiratory system compensates by adjusting PaCO2. Respiratory compensation is rapid (begins within minutes and complete within 12-24 hours) and usually more effective in the acute stages of metabolic dysfunction.

<table>
<thead>
<tr>
<th>PRIMARY DISORDER</th>
<th>Plasma [HCO3-]</th>
<th>PaCO2</th>
<th>Arterial pH</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Chronic</td>
<td>↑↑</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acute</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Chronic</td>
<td>↓↓</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
</tbody>
</table>

**Base excess and Anion gap analysis** will be covered in the Renal course.
PRACTICE CASES

We will guide you through three cases, then provide two cases for practice interpretation.

CASE #1. An 18 year male is hit by a car. He has a collapsed trachea, but appears to be in good health otherwise.

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>PaO2 (mmHg)</th>
<th>PaCO2 (mmHg)</th>
<th>Bicarb (mM)</th>
<th>Tot CO2 (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>(7.35-7.45)</td>
<td>80-100</td>
<td>(35-45)</td>
<td>(22-26)</td>
<td>(23-27)</td>
</tr>
<tr>
<td>Patient</td>
<td>7.30</td>
<td>78</td>
<td>54</td>
<td>25.9</td>
<td>27.5</td>
</tr>
</tbody>
</table>

**Step 1. Classify the pH.** The patient has acidemia.

**Step 2. Analyze the respiratory involvement (PaCO2).** PaCO2 is high indicating respiratory acidosis (hypoventilation) as a cause of (or a contributor to) the acidemia.

**Step 3. Analyze the metabolic component (HCO3-):** If metabolic acidosis is contributing to the acidemia, [HCO3-] would be low. It is on the high side of normal, therefore it cannot be the cause of the acidemia.

**Step 4. Is compensation present?** If the kidneys are compensating for the respiratory acidosis by retaining HCO3-, the [HCO3-] will be higher than the small amount expected from CO2. The predicted increase from CO2 is ~1.4 mM (about + 1 mM for every 10 mmHg increase in PCO2 above 40 mmHg). The observed increase in HCO3- is very small (25.9 mM minus the normal value of 24 mM = 1.9 mM) and accounted for by the 14 mmHg increase in PaCO2. Therefore, renal compensation is absent.

The present case is uncompensated (acute) respiratory acidosis, consistent with acute alveolar hypoventilation from a collapsed trachea.

CASE #2. A 45-year-old man has had recurrent episodes of vomiting for 1 week.

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>PaO2 (mmHg)</th>
<th>PaCO2 (mmHg)</th>
<th>Bicarb (mM)</th>
<th>Tot CO2 (mM)</th>
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<tbody>
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<td>(35-45)</td>
<td>(22-26)</td>
<td>(23-27)</td>
</tr>
<tr>
<td>Patient</td>
<td>7.49</td>
<td>72</td>
<td>55</td>
<td>40</td>
<td>41.6</td>
</tr>
</tbody>
</table>

**Step 1. Classify the pH.** The patient has alkalemia.

**Step 2. Analyze the respiratory component (PCO2):** The PCO2 is on the high side of normal, therefore, it cannot be a cause of alkalemia (a high PCO2 would cause acidosis).

**Step 3. Analyze the metabolic component (HCO3-):** The [HCO3-] is much higher than predicted from the increase in PaCO2, indicating 1° metabolic alkalosis.

**Step 4. Is compensation present?** Some of you may think that the high PaCO2 is caused by the metabolic increase in HCO3- that drives Eq 1 to the left; but this is wrong. Additional CO2 is indeed formed, but any increased production of CO2 is immediately removed from the system by the lung (i.e., assuming the lung is not dysfunctional). Rather, when alkalemia is present it is sensed by the chemoreceptors and results in diminished drive for ventilation. The resulting hypoventilation increases PaCO2 and causes a respiratory acidosis that helps to offset the metabolic alkalosis. This is called respiratory compensation.
CASE #3. A markedly obese 24 yo male complains of dyspnea on exertion. No other significant pathophysiological abnormalities evident.

<table>
<thead>
<tr>
<th></th>
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<th>PaCO2 (mmHg)</th>
<th>Bicarb (mM)</th>
<th>Tot CO2 (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7.35-7.45</td>
<td>80-100</td>
<td>(35-45)</td>
<td>(22-26)</td>
<td>(23-27)</td>
</tr>
<tr>
<td>Patient</td>
<td>7.33</td>
<td>59</td>
<td>65</td>
<td>33.3</td>
<td>35.3</td>
</tr>
</tbody>
</table>

**Step 1. Classify the pH.** Acidemia is present.

**Step 2. Analyze the respiratory component (PCO2):** The high PCO2 indicates presence of respiratory acidosis as one cause of the acidemia.

**Step 3. Analyze the metabolic component (HCO3-):** The HCO3- is high, therefore, metabolic acidosis cannot be the cause of the acidemia.

**Step 4. Assess for compensation.** The increase in HCO3- (+9.3 mM) is higher than would be expected (+2.5 mM) from the elevation in CO2 (+1 mM per 10 mmHg increase in CO2); therefore, the kidney is adding base to the blood to counter the addition of H+ from the lung. The kidney is compensating in a normal way to make up for the dysfunction of the respiratory system.

This patient has respiratory acidosis with renal compensation. The presence of renal compensation (which takes time) indicates a **chronic** respiratory acidosis.

**How do you know that this is not a metabolic alkalosis (high HCO3) with respiratory compensation (high PCO2)?** Compensatory mechanisms tend to restore pH towards normal and do not overshoot. If the primary disturbance was metabolic alkalosis, we would have expected pH to be alkaline or on the alkaline side of normal. The case history is also important.
Cases for practice interpretation

**CASE #4.** This ABG was taken from a 40-year-old female diagnosed with diffuse pulmonary embolism. After 2 days of oxygen therapy, the PaO2 increased but the other values were about the same.

<table>
<thead>
<tr>
<th></th>
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<td>(7.35-7.45)</td>
<td>80-100</td>
<td>(35-45)</td>
<td>(22-26)</td>
<td>(23-27)</td>
</tr>
<tr>
<td>Patient</td>
<td>7.47</td>
<td>85</td>
<td>22</td>
<td>15.4</td>
<td>-</td>
</tr>
</tbody>
</table>

a. **Interpret the ABG.**
   - [ ] Normal
   - [ ] Respiratory acidosis with complete renal compensation
   - [ ] Respiratory alkalosis, uncompensated
   - [ ] Respiratory alkalosis with partial renal compensation
   - [ ] Metabolic alkalosis with respiratory compensation
   - [ ] Metabolic acidosis with respiratory compensation

b. **Calculate the Tot CO2.**

**CASE #5.** A one year old with a history of profuse diarrhea of 4 days duration is listless and unresponsive. Skin turgor is poor and eyes are sunken. Temp 98 °F, HR = 130/min, respirations 30 and deep.

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>PaO2 (mmHg)</th>
<th>PaCO2 (mmHg)</th>
<th>Bicarb (mM)</th>
<th>Tot CO2 (mM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>(7.35-7.45)</td>
<td>80-100</td>
<td>(36-42)</td>
<td>(22-26)</td>
<td>(23-27)</td>
</tr>
<tr>
<td>Patient</td>
<td>7.2</td>
<td>ND</td>
<td>16</td>
<td>6.1</td>
<td>6.6</td>
</tr>
</tbody>
</table>

a. **Evaluate the ABG.**
   - [ ] Normal
   - [ ] Respiratory acidosis, uncompensated
   - [ ] Respiratory acidosis with partial metabolic compensation
   - [ ] Respiratory alkalosis with metabolic compensation
   - [ ] Metabolic alkalosis
   - [ ] Metabolic acidosis
   - [ ] Metabolic acidosis with partial respiratory compensation
   - [ ] Metabolic acidosis with complete respiratory compensation

b. **Compare cases #4 and #5.**

Annotated answers to cases 4 and 5.

**Answers to Case #4.**
a. This is an alkalemia caused by alveolar hyperventilation (low PaCO2). The low HCO3- indicates that a metabolic dysfunction cannot be causing the alkalemia. Renal compensation is present because the decrease in bicarbonate (8.6 mM) is much larger than the small reduction
expected (-3.6 mM HCO3-) from the fall in PCO2 (~2 mM per 10 mmHg fall in PCO2). This is a chronic, partially compensated, respiratory alkalosis.

b. TCO2 is the sum of the bicarbonate and the dissolved CO2. Most clinical path reports will calculate TCO2 for you, but we thought you might want to know where the value comes from. Multiply PCO2 by its solubility coefficient (0.03 mM/mmHg) to determine how many mM of CO2 are dissolved in the plasma at the given PCO2 then add it to the [HCO3-].

\[
TCO2 = [HCO3-] + (0.03 \times PCO2) = 15.4 \text{ mM} + (0.03 \text{ mM/mmHg} \times 22 \text{ mmHg}) = 16.1 \text{ mM}
\]

Notice that a PCO2 as high as 60 mmHg would still only contribute 1.8 mEq to TCO2. Thus, TCO2 is largely [HCO3-].

FYI. Pulmonary embolism is often associated with hyperventilation that is not corrected by O2 therapy, indicating that the hyperventilation was not initiated by arterial hypoxia. It is presumed that the hyperventilation is due to intrapulmonary receptors (juxtapulmonary receptors in the interstitium may be activated by edema or pulmonary vascular congestion) that stimulate the central respiratory controller.

**Answers to Case #5.**

a. Respiratory acidosis cannot be the cause of the acidemia because PaCO2 is low. The decrease in [HCO3-] of 17.9 mM (the normal of 24 mM minus 6.1 mM) is much larger than the 4.6 mM decrease predicted from the low PCO2 (-2 mM/L per 10 mmHg in PCO2) and consistent with 1° metabolic acidosis. The acidemia has stimulated chemoreceptors that increase ventilation and, therefore, the rate of CO2 elimination. The hyperventilation causes a respiratory alkalosis that helps compensate for the metabolic acidosis.

b. Cases 4 and 5 both have low HCO3- and low PaCO2, but the 1° disorders are different. Case 5 (diarrhea) is a metabolic acidosis with partial respiratory compensation. Because the PCO2 is low, it may be tempting to interpret case #5 as a respiratory alkalosis with metabolic compensation. Suppose that respiratory alkalosis is the primary dysfunction. The low PaCO2 will force Eq 1 to the left and [H+] and HCO3 will decrease. The kidney compensates by excreting bicarbonate and conserving H+. Exactly what we want. But the pH in this patient is 7.2. Do you think the kidney would take the pH down to 7.4 then go past normal and end up at 7.2? Not likely. That is why we begin our analysis by evaluating whether the pH is on the acidic or alkaline side of normal.