EVALUATION OF A/B STATUS = INTERPRETATION OF BLOOD GASES

When/why do a blood gas? Severe illness with multiple problems, and/or clinical problems identified or suspected on physical examination/laboratory data suggest the presence of an A/B disorder.

How to perform A/B analysis:

- Measure 3 parameters specific to A/B status:
  - pH
  - Respiratory - CO₂
  - Metabolic - HCO₃, TCO₂, and/or BE
- Electrolytes, especially Na and Cl
- Packed cell volume, total protein, +/- albumin
- Venous samples can be used for A/B status
- Arterial samples necessary to evaluate ventilation/gas exchange

- Sampling:
  - Use free-flowing vein/artery
    - Dorsal pedal artery(a.), femoral a. – dogs, cats
    - Transverse facial a., facial a., dorsal metatarsal a., carotid a. – horses
    - Auricular a. – ruminants, pigs
    - Vein Jugular, cephalic – most species; auricular v. – ruminants, pig, dog
  - Heparinized syringes; not too much – over dilution falsely ↓ some results
  - Must cap syringe or needle – no exposure to air!
  - Analyze as soon as possible
  - May keep on ice a few hours, but not ideal
  - Need patient’s current temperature also

- Rules of thumb:
  - Simple Acid/Base disorder – one side is abnormal/one is compensatory
    - Primary component shifts in same direction as pH change
    - Acidemia caused by acidosis, alkalemia caused by alkalosis
    - Compensatory response shifts opposite the change in pH
    - Acidosis balances alkalisos, & vice versa
    - The actual values increase or decrease in parallel because their effects are opposite
    - No overcompensation, in fact, rare to see normal pH
Blood Gas Evaluation

- Rules of thumb: continued

- Mixed acid/Base disorders
  - Normal pH present but pCO₂ or HCO₃ are abnormal
  - Shift in pCO₂ and HCO₃ values are not parallel (both up or down)
  - pH change is opposite to that expected from an abnormal pCO₂ or HCO₃
  - Compensation appears to be > expected
  - What are protein levels?
    - Hypoalbuminemia – metabolic alkalosis - loss of weak acids (A_TOX)
    - Hyperglobulinemia – metabolic acidosis – increased weak acids (A_TOX)

- When metabolic acidosis present
  - Calculate anion gap
  - Look at chloride level

<table>
<thead>
<tr>
<th>Metabolic acidosis</th>
<th>Likely cause</th>
<th>Look at the patient – what disease processes are present that can affect A/B?</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Anion Gap w/ Normal Cl</td>
<td>↑ UA</td>
<td></td>
</tr>
<tr>
<td>↓ Anion Gap w/ High Cl</td>
<td>Loss of HCO₃</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from chart at Cornell Clin Chem Basics website – see refs

<table>
<thead>
<tr>
<th>Normal Blood Gas Values</th>
<th>Dog</th>
<th>Cat</th>
<th>Horse</th>
<th>Cow</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.31-7.42</td>
<td>7.24-7.40</td>
<td>7.32-7.44</td>
<td>7.35-7.50</td>
</tr>
<tr>
<td>paCO₂ mmHg</td>
<td>29-42</td>
<td>29-42</td>
<td>36-46</td>
<td>35-44</td>
</tr>
<tr>
<td>paO₂ mmHg</td>
<td>85-95</td>
<td>85-95</td>
<td>94</td>
<td>92</td>
</tr>
<tr>
<td>HCO₃ mEq/L</td>
<td>17-24</td>
<td>17-24</td>
<td>24-30</td>
<td>20-30</td>
</tr>
<tr>
<td>TCO₂ mEq/L</td>
<td>14-26</td>
<td>13-21</td>
<td>22-33</td>
<td>22-34</td>
</tr>
<tr>
<td>BE mEq/L</td>
<td>-2 to +2</td>
<td>-2 to +2</td>
<td>0 to +5</td>
<td>0 to +5</td>
</tr>
</tbody>
</table>

These values are taken from DiBartola and Latimer references; Be sure to use ranges from lab where tests are performed.

Dogs and cats tend toward lower pH, acidemia
Large animals tend toward higher pH, alkalemia.

Venous samples – more acid present due to > CO₂; pH lower
Blood Gas Evaluation

ACID / BASE CASE EXAMPLES:

Case 1. Healthy dog under anesthesia for ovariohysterectomy

Healthy young dog anesthetized with inhalant anesthetic for a spay; Arterial blood gas taken when the end tidal CO₂ was noted to be quite high (~80) on a capnograph.

<table>
<thead>
<tr>
<th>Blood gas results: Case 1- Dog under anesthesia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>pH</strong></td>
</tr>
<tr>
<td><strong>paCO₂</strong></td>
</tr>
<tr>
<td><strong>paO₂</strong></td>
</tr>
<tr>
<td><strong>HCO₃</strong></td>
</tr>
<tr>
<td><strong>TCO₂</strong></td>
</tr>
<tr>
<td><strong>BE</strong></td>
</tr>
</tbody>
</table>

- **Interpretation:** Simple disorder
  - **Respiratory acidosis** is present
    - pH & pCO₂ both shifted towards acid
    - pCO₂ increased from hypoventilation
  - Physiochemical compensation is present
  - Respiratory compensation is inhibited under anesthesia.

Remember this equation: \( \text{HCO}_3^- + \text{H}^+ \rightleftharpoons \text{H}_2\text{CO}_3 \rightleftharpoons \text{H}_2\text{O} + \text{CO}_2 \)

Hypoventilation is also seen in awake patients w/ sedation, depression, and muscle weakness as well.

Note the pO₂ is high because the dog is breathing close to 100% oxygen; oxygenation is very good in spite of significant hypoventilation. If she were breathing room air, 21% O₂, she would be hypoxemic.

- **Treatment:** Mechanical ventilation; CO₂ and pH corrected quickly.

Hypoventilation with resultant respiratory acidosis is VERY COMMON under anesthesia

The respiratory center is affected by anesthetic drugs and the respiratory threshold for CO₂ is "reset".

The respiratory threshold for CO₂ is increased above normal and breathing is not stimulated until the pCO₂ exceeds the reset value.
Case 2. Alpaca under anesthesia for orthopedic surgery

Adult alpaca under inhalant anesthesia for fracture repair; ventilated at 10 breaths/minute and 10 ml/kg/breath

**Blood gas results: Case 2- Alpaca under anesthesia**

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>pH</strong></td>
<td>7.609</td>
</tr>
<tr>
<td><strong>paCO₂</strong></td>
<td>19.7</td>
</tr>
<tr>
<td><strong>paO₂</strong></td>
<td>293</td>
</tr>
<tr>
<td><strong>HCO₃⁻</strong></td>
<td>19.5</td>
</tr>
<tr>
<td><strong>TCO₂</strong></td>
<td>20.2</td>
</tr>
<tr>
<td><strong>BE</strong></td>
<td>+2.0</td>
</tr>
</tbody>
</table>

- **Interpretation:** Simple disorder
  - **Respiratory alkalosis** - pH very alkalotic, pCO₂ quite low, alkalotic
    - Hyperventilation; incorrect ventilator settings (although “usual” guidelines were followed)
  - Some **physiochemical compensation** - HCO₃⁻, and TCO₂ low.
  - **Remember that respiratory compensation is inhibited under anesthesia** - respiratory center is affected by anesthetic drugs - threshold for CO₂ is elevated
  - Actual metabolic compensation by the kidney takes too long to be seen in this case

  *Remember this equation* - HCO₃⁻ + H⁺ ⇌ H₂CO₃ ⇌ H₂O + CO₂

- **Treatment:** Decrease ventilator settings
Case 3. Sick cow

A 4 yr old Holstein/mixed breed cow is presented with weakness and depression, continuously drooling. She lives in a large pasture with 30 other cows and calves.

Her rumen sounds are present but very infrequent; rumen fill appears decreased. Her eyes are sunken and the skin on her neck remains tented for 2-3 seconds.

On examination, an apple is found lodged in her esophagus (‘Choke’). Temp=103 °F, heart rate is 100 beats/min, respiratory rate is 60 breaths/min.

Blood work reveals a PCV of 55, TS of 8.0, increased albumin, creatinine, and BUN. The blood gas sample was obtained from an auricular artery. The cow is estimated to be 8% dehydrated.

**Blood gas results: Case 3- Sick cow**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.101</td>
<td>Low</td>
</tr>
<tr>
<td>paCO₂</td>
<td>26.8</td>
<td>Low</td>
</tr>
<tr>
<td>paO₂</td>
<td>64.8</td>
<td>Low</td>
</tr>
<tr>
<td>HCO₃</td>
<td>8.2</td>
<td>Low</td>
</tr>
<tr>
<td>TCO₂</td>
<td>9.0</td>
<td>Low</td>
</tr>
<tr>
<td>BE</td>
<td>-19.0</td>
<td>*HIGH</td>
</tr>
</tbody>
</table>

* a negative BE = a base deficit = excess acid

**Interpretation – a simple disorder with 2 causes of metabolic acidosis**

- pH low – acidemia
- pCO₂ low – alkalemia
- HCO₃, TCO₂, are low and BE is very negative - acidemic
- **Metabolic acidosis** is the primary component
  - pH & all metabolic parameters have shifted towards acid side
  - Loss of HCO₃ in saliva and dehydration from lack of intake
    - Loss of base and increased acid - additive effects
- **Respiratory alkalosis** is the compensatory response; a low CO₂ is alkalemia
  - Response is inadequate in this case – in spite of tachypnea - cannot correct this pH
    - The acidosis is severe; hypoventilation is limited by oxygenation
    - This cow’s is already hypoxemic, pO₂ is low, < 80 mmHg

**Treatment:**

- Fluid therapy to replace losses
  - Large volume of lactated ringers or other balanced electrolyte solution
    - Oral (once choke is resolved) and IV if possible (expense).
    - Enteral fluid therapy works quite well in cows.
  - Specific replacement of HCO₃ is probably not necessary
    - With rehydration, the liver will process the ↑ lactate & regenerate HCO₃

↑ PCV, TS, albumin, Creatinine, & BUN result from dehydration;

In fact, the ONLY cause of ↑ albumin is fluid loss.
Case 4: Fresh cow, on and off problems with milk production and appetite

4 yr old Holstein dairy cow, 4 wks fresh, presents with decreased appetite and milk production on and off since calving. Physical exam is normal except the cow is a bit thin.

A large "ping" developed on the left side of the abdomen during the night. Recheck of blood gases/electrolytes shown.

**Interpretation** – simple disorder (second sample)
- Admission – normal blood gases/electrolytes
- **Next morning - Severe metabolic alkalosis**
  - All metabolic parameters shifted towards alkalosis
- **Some respiratory compensation**
  - $pCO_2$ is increased - produced by hypoventilation
  - pH still high - limited by oxygenation
  - $pO_2$ is close to the low end of normal (80-100 mmHg)

  Note – we can evaluate $pO_2$ since its an arterial sample

**Cause** – Partial upper GI obstruction with sequestration of gastric acid – a "swinging" displaced abomasum (DA).

It corrected somewhat on the trailer ride to the hospital - obstruction was relieved, blood gases and electrolytes corrected.

Overnight, the cow ate grain and hay, displacement reoccurred – partial obstruction with the resultant pH and electrolyte abnormalities.

$\downarrow$ $Cl^-$ is due to sequestration in the abomasum, preventing absorption in the small intestine.

In kidney - need a (-) ion to resorb with Na, usually Cl. When Cl is not available, it saves $HCO_3$ instead, causing alkalosis.

The sequestration of $H^+$ and $Cl^-$ and the actions of the kidney combine to produce the severe metabolic alkalosis.

$\downarrow$ $K^+$ develops from gastric loss, decreased intake, and as a response to the high pH – $K^+$ shifts intracellularly, $H^+$ moves extracellularly.

**Treatment**: Rolling is sometimes attempted, but surgical intervention is most often necessary.

Try this link to UPenn’s excellent CAL site - [Bovine physical exam](#) – great photos of ping locations –

### Blood gas results: Case 5- Cow with a “ping” on L

<table>
<thead>
<tr>
<th></th>
<th>Day one</th>
<th>Next AM</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.408</td>
<td>N 7.554</td>
</tr>
<tr>
<td>$pCO_2$</td>
<td>43.3</td>
<td>N 50.9</td>
</tr>
<tr>
<td>$pO_2$</td>
<td>29.6</td>
<td>N 81.3</td>
</tr>
<tr>
<td>$HCO_3$</td>
<td>26.8</td>
<td>N 44.6</td>
</tr>
<tr>
<td>TCO$_2$</td>
<td>28.0</td>
<td>N 46.1</td>
</tr>
<tr>
<td>BE</td>
<td>+2.4</td>
<td>N +22.6</td>
</tr>
<tr>
<td>Na</td>
<td>134</td>
<td>N 142</td>
</tr>
<tr>
<td>K+</td>
<td>4.3</td>
<td>N 2.3</td>
</tr>
<tr>
<td>Cl</td>
<td>101</td>
<td>N 91</td>
</tr>
</tbody>
</table>

Note that this compensatory response is towards acidemia, the **opposite** direction to the pH. The VALUES actually move in parallel (all go up) but the A/B effect is opposite.
Blood Gas Evaluation

Case 5. 18 year old cat in renal failure

Depressed, lethargic, 18 yr old cat in renal failure. This is a venous blood gas.

- Interpretation: Mixed acid base disorder – more than one problem is present
  - Both CO₂ and metabolic parameters have shifted towards the acidic side. One should shift to the opposite side if this were a simple disorder
  - CO₂ has not shifted as expected with this pH
  - Main problem is a metabolic acidosis - metabolic parameters are most abnormal.
  - A mild respiratory acidosis also present, likely due to hypoventilation in a depressed patient.
  - What kind of metabolic acidosis is present? Calculate the anion gap:

    \[ \text{Anion Gap} = (Na + K) - (HCO₃ + Cl^-) = (153 + 4.8) - (12 + 126) = 157.8 - 138 = 19.8 \text{ High} \]

    - ↑ AG and N chloride - increased organic acids or a ‘titration’ acidosis
    - Common with renal failure and uremia
  - Treatment: Diuresis via fluid therapy. If in end stage renal failure, consider euthanasia

Blood gas & blood chemistry results:
Case 5- Renal failure cat

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>pCO₂</th>
<th>pO₂</th>
<th>HCO₃</th>
<th>TCO₂</th>
<th>BE</th>
<th>Na</th>
<th>K</th>
<th>Cl</th>
<th>Cr</th>
<th>BUN</th>
<th>AG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Value</td>
<td>7.024</td>
<td>44.8</td>
<td>40</td>
<td>12</td>
<td>13</td>
<td>-19.0</td>
<td>153</td>
<td>4.8</td>
<td>126</td>
<td>12.6</td>
<td>181</td>
<td>20</td>
</tr>
<tr>
<td>Result</td>
<td>Very low</td>
<td>Mild</td>
<td>Low normal</td>
<td>Low</td>
<td>Acidosis</td>
<td>High</td>
<td>Acidosis</td>
<td>High normal</td>
<td>normal</td>
<td>High normal</td>
<td>Very high</td>
<td>Very high</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>acidemia</td>
<td>↑ acidosis</td>
<td></td>
<td>Acidosis</td>
<td></td>
<td>Acidosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Anion gap interpretation:

<table>
<thead>
<tr>
<th>Metabolic acidosis</th>
<th>Likely cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Anion Gap w/ Normal Cl</td>
<td>↑ UA</td>
</tr>
<tr>
<td>Normal Anion Gap w/ High Cl</td>
<td>Loss of HCO₃</td>
</tr>
</tbody>
</table>

Adapted from chart at Cornell Clin Chem Basics website
2 week old Holstein calf with history of diarrhea for 1 week. Also has pneumonia, Se deficiency; HR=90, RR=60; hypothermic 35°C (95°F) at presentation.

- Interpretation: Simple disorder
  - **SEVERE** metabolic acidosis
    - loss of base and ↑ acid (lactic)
      - Neonatal diarrhea in calves - Opposing effects on anion gap
        - Loss of HCO₃⁻ very often present – AG usually N, Cl ↑
        - Severe fluid loss as well – lactic acidosis – AG usually ↑, Cl N
      - K ↑ with acidosis, see bradycardia, arrhythmias
  - **Respiratory** alkalosis - attempt to compensate
  - Se deficiency may complicate this case
    - With cardiac effects – hypotension – lactic acidosis
    - Hypothermia – shock – poor perfusion - lactic acidosis

- Treatment:
  - Fluid therapy to replace losses and match deficits IV in this calf
    - 0.9% Saline
    - +/- hypertonic saline
    - HCO₃⁻ is given to replace loss and help A/B status
    - Enteral therapy as soon as possible
  - Warming procedures – fluid therapy will help the most – Rx shock
  - Selenium injection, antibiotics, glucose if needed, occasionally Ca²⁺
  - Treat blood pressure with inotropes, vasopressors, if needed
  - **Many of these calves do very well in spite of such severe acid/base changes**
    Will be up and bawling for milk in a few hours!

### Blood gas & blood chemistry results:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value (mmol/L)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>6.989</td>
<td>very low acidemia</td>
</tr>
<tr>
<td>pCO₂</td>
<td>27</td>
<td>low alkalosis</td>
</tr>
<tr>
<td>pO₂</td>
<td>26</td>
<td>very low acidosis</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>6.7</td>
<td>very low acidosis</td>
</tr>
<tr>
<td>TCO₂</td>
<td>7.7</td>
<td>very low acidosis</td>
</tr>
<tr>
<td>BE (ECF)</td>
<td>-25.3</td>
<td>very high acidosis</td>
</tr>
<tr>
<td>Na⁺</td>
<td>132</td>
<td>mild ↓</td>
</tr>
<tr>
<td>K⁺</td>
<td>6.4</td>
<td>high</td>
</tr>
<tr>
<td>Cl⁻</td>
<td>106</td>
<td>normal</td>
</tr>
<tr>
<td>nCa²⁺</td>
<td>0.78 mmol/L</td>
<td>low</td>
</tr>
<tr>
<td>Hct</td>
<td>26</td>
<td>low</td>
</tr>
</tbody>
</table>

Anion Gap = (132 + 6.4) - (6.7 + 06) = 138.4 - 112.7 = **25.7**

Bloodwork courtesy Dr. L George

Will be up and bawling for milk in a few hours!
Case 7. Down foal, dribbling urine

4 day old male quarterhorse foal was presented recumbent with a distended abdomen and a history of dribbling urine, straining to urinate, and then quickly becoming anorexic, weak, and depressed.

Diagnosed with pneumonia, sepsis, and uroperitoneum from a ruptured bladder.

- **Interpretation: Mixed disorder**
  - Respiratory & metabolic parameters shifted towards the acidic side
  - **Severe Respiratory acidosis**
    - Hypoventilation from atelectasis and pneumonia
  - **Moderate Metabolic acidosis**
    - Dehydration – lactic acidosis
    - Shock - tissue hypoxia from hypotension, vasodilation – poor perfusion – lactic acidosis
    - Uremic acids
  - Metabolic parameters should $\downarrow$ to balance pH but can’t.
    - High pCO$_2$ shifts equilibrium equation snf metabolic acids present

This foal is very sick; this pH is *incompatible* with life for very long. The CO$_2$ is high enough to cause sedation/narcosis.

The history of urinary difficulty, his age, and the electrolyte changes - low sodium and high potassium - are characteristic of uroperitoneum.

Ruptured bladder often confirmed via ultrasound of the abdomen.
Urine - high in potassium, low in sodium. Plasma - high in sodium, low in potassium.

Electrolytes (& other substances, Creatinine, BUN, etc.) equalize across the peritoneal membrane, resulting in the changes seen and a “titration” metabolic acidosis in plasma.

- **Treatment:**
  - Drain abdomen of urine, +/- indwelling abdominal drain.
  - Stabilize foal via fluid replacement and diuresis, and perform surgical repair of bladder.
  - Ventilation is *required* in this foal to decrease the CO$_2$.

At surgery, the CO$_2$ normalized with mechanical ventilation. The foal improved post surgery but later succumbed to sepsis.

---

**Blood gas & blood chemistry results:**

<table>
<thead>
<tr>
<th>Case 7 - Down foal</th>
<th>pH 6.938</th>
<th>pcO$_2$ 104.9</th>
<th>pvO$_2$ 43.7</th>
<th>HCO$_3$ 22.4</th>
<th>TCO$_2$ 25.6</th>
<th>BE -9.2</th>
<th>Na 116</th>
<th>K 5.65</th>
<th>Ca$^{2+}$ 5.4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acidemic</strong></td>
<td></td>
<td>very high</td>
<td>normal</td>
<td>mild ↓</td>
<td>normal</td>
<td>high</td>
<td>Low</td>
<td>high</td>
<td>normal</td>
</tr>
</tbody>
</table>

**Mixed disorder - respiratory & metabolic sides shift in the same direction rather than opposite directions as seen with a compensatory response**
**Images and Links worth a look:**

**Cornell University**  [Clinical Chemistry basics](#), [Acid/base](#), [Mixed acid base](#), [Bicarbonate and Anion Gap](#)

**University of Pennsylvania**  [Clin Path case studies](#)

**Tufts University**  [“Strong Ion” theory of Acid Base](#)

**References:** Unless otherwise noted, images are courtesy of Dr. JG Adams


