With the advent of point-of-care bedside patient monitors such as the I-Stat and IRMA, blood gas analysis has gone from a seldom-used Ivory Tower monitoring tool to one that is accessible to every practicing veterinarian. Despite this, there is a wide-spread fear of blood gasses and unwillingness to embrace this valuable methodology. This handout will attempt to soothe that fear and make you love blood gasses. Or, at the very least, get you through the night without vomiting if you have to interpret one.

**INDICATIONS – Why bother?:** Evaluation of the respiratory or metabolic/acid-base status of the patient. Any patient with severe respiratory disease (e.g.: pneumonia, suspect PTE) or severe metabolic derangement (e.g: ARF, DKA, status epilepticus) is a candidate for blood gas analysis. Acid-base and respiratory status are commonly altered in many critical illnesses and these alterations can impact cardiovascular function (acidosis decreases cardiac output), neurological function (severe hypocapnia causes cerebral vasosconstriction and ischemia) and a multitude of other body systems. In addition, arterial blood gas analysis is the gold standard for the diagnosis of respiratory failure and the need for mechanical ventilation and can be used to assess the progress of certain therapies.

**Respiratory function:** This evaluates oxygenation and ventilation, and an arterial sample is required. The most accessible arteries in dogs are the dorsal pedal artery and the femoral artery. In cats the femoral artery is most commonly used. Blood from the sublingual vein can be used as a rough approximation of arterial values because the venous and arterial sides of the local circulation are in close approximation. For arterial blood-gas analysis, knowledge of the patient’s temperature and fraction of inspired oxygen (FiO₂) is necessary. Heparinized syringes (fill the syringe with heparin and empty it so only the hub of the syringe contains heparin) and a new needle will facilitate arterial puncture and sample preservation. Samples should be kept on ice until analysis, unless they are to be run immediately. Digital palpation of the artery to localize it and knowledge of its relationship to nearby anatomical structures is helpful.

**Acid-base status:** An arterial or venous sample will suffice.

**INTERPRETATION – What do these numbers mean?:**

The two functions of the respiratory system, oxygenation and ventilation, are assessed by arterial blood gas evaluation. (Metabolic assessment is below)
Oxygenation: The first and most critical evaluation is of PaO$_2$, a measure of oxygenation. It is important to remember that PaO$_2$ reflects oxygen dissolved in plasma, not the amount of oxygen carried by the blood. Normal PaO$_2$ should be approximately five times the inspired oxygen percentage – that is, if a patient is breathing 100% oxygen (intubated) the PaO$_2$ should be in the neighborhood of 500mmHg. For a patient breathing ambient air (at sea level) the normal PaO$_2$ is 100mmHg. PaO$_2$ of less than 80mmHg indicates hypoxia and PaO$_2$ of less than 60mmHg indicates a severe oxygen debt and oxygenation failure – either another method of providing supplemental oxygen is needed or mechanical ventilation is required if at maximal supplementation already.

PaO$_2$ is different from the saturation of hemoglobin (SPO2) but influenced by it – if you have low PaO2 you will most likely have low SPO2. If you calculate this out and they are not five times the inspired oxygen, they have some problem with oxygen uptake (eg: pneumonia, PTE, etc).

For respiratory diseases, we are most concerned about a patient’s ability to oxygenate (get O2 into the blood and thus to the tissues, and measured by PaO2 and SPO2) and ventilate (blow off CO2, and measured by PaCO2 or ETCO2). Realistically, most patients’ respiratory status can be assessed by any good PulseOx, since hypoventilation is relatively rare when compared to hypoxia, and therefore you don’t have to resort to an arterial stick. (This is because CO2 diffuses out of the blood 20X faster than oxygen diffuses in - think of how many patients we have on oxygen compared to how many we have to mechanically ventilate). In order to assess ventilation in a non-intubated patient, however, you need a blood gas.

The only way to assess ventilation is measurement of CO2, either through blood gas analysis or ETCO2. Normal range for CO2 is 35-45mmHg, and low values or high values represent hyper- or hypoventilation, respectively. Therapy for hyperventilation involves treating the primary cause (fear, pain, etc) and therapy for hypoventilation involves treating the primary cause (reverse depressant drugs if possible) or mechanical ventilation.

One of the most important decisions we make when evaluating an arterial blood gas is whether the patient needs to be intubated and artificially ventilated, or if they’re doing just fine on their own, thank you very much. In general, most folks use the “Rule of 60”, which means if the PaO2 is less than 60 mmHg or the PaCO2 is greater than 60mmHg, they just aren’t getting the job done and need ventilation (assuming they’re already on supplemental oxygen). There are a few exceptions to this, including chronic respiratory diseases, in which the patient has lived with low oxygen and or high CO2 for a long time. Also, some patients may have perfectly normal blood gasses, but are working very hard to maintain these numbers and will suffer acute respiratory failure, so work of breathing is another factor to consider (think of a laryngeal paralysis dog).

One method of evaluating respiratory function is calculation of the PaO$_2$/FiO$_2$ ratio (the P/F ratio). This index will allow benchmarking the lungs’ function against various levels of supplemental oxygen. For example – a patient with a PaO$_2$ of 90mmHg may not sound so bad, but if you are told that that patient was on 60% oxygen (where according to the above “five times rule” you would expect a PaO$_2$ of 300mmHg) the patient is quite
far from where they should be. The P/F ratio is calculated by dividing the PaO₂ by the fraction of inspired oxygen as a decimal. Normal P/F ratio is 500. Less than 300 indicates acute lung injury (ALI) or moderate respiratory dysfunction, and less than 200 indicates severe respiratory failure (and is one of the diagnostic criteria for ARDS, the acute respiratory distress syndrome).

**Example:**

1) **Normal Lungs** – dog under anesthesia for an ovariohysterectomy.

   FIO₂ – 100% (Intubated, on 100% oxygen)

   \[
   \text{PaO}_2 = 497 \text{mmHg}
   \]

   P/F Ratio = 497 - normal

2) **Diseased Lungs** – Severe pneumonia, bilateral nasal oxygen at 2 liters each

   FIO₂ – 50%

   \[
   \text{PaO}_2 = 110 \text{mmHg}
   \]

   P/F Ratio = 220, indicating severe hypoxia

In this example, the PaO₂ is technically “normal”, but it still indicates a critical level of pulmonary dysfunction when the supplemental oxygen is considered. This patient should be intubated and given 100% oxygen.

3) **“Warm up the Ventilator” lungs** –

   FIO₂ – 100% (Intubated)

   \[
   \text{PaO}_2 = 55 \text{mmHg}
   \]

   P/F Ratio = 55

Severe hypoxia despite maximal supplemental oxygen indicates the need for mechanical ventilation.

Another useful index that is fun and easy to calculate (OK, so shoot me) is the A-a gradient, or the Alveolar to arterial oxygen gradient. This is a measure of how well oxygen diffuses across into the pulmonary arterioles from the alveoli. It is important to remember that the A-a gradient is accurate only for patients breathing ambient (room) air. A large A-a gradient indicates a barrier to diffusion from the alveoli into the blood such as pulmonary edema or other pulmonary parenchymal disease. Hypoxia due to hypoventilation would be associated with a normal A-a gradient.
Calculation of the A-a gradient involves 2 steps – calculation of the alveolar oxygen content (the big A – \( \text{PAO}_2 \)) and measurement of the arterial oxygen content (the little a – \( \text{PaO}_2 \)). Calculation of the alveolar oxygen content uses the alveolar gas equation:

\[
\text{PAO}_2 = \text{FiO}_2 (\text{Pb}-\text{P}_{\text{H}_2\text{O}}) - \frac{\text{PaCO}_2}{\text{RQ}}
\]

Where \( \text{Pb} \) is barometric pressure (usually 760mmHg) and \( \text{P}_{\text{H}_2\text{O}} \) is the water vapor pressure inside the alveolus (which displaces some of the gasses – usually about 47mmHg). \( \text{PaCO}_2 \) is obtained from a blood gas sample and RQ, or respiratory quotient, is a magical number (like Pi or 42) that you don’t really have to worry about. It is 0.8.

The equation can be simplified in patients breathing room air at sea level*, thankfully, to the much more manageable

\[
\text{PAO}_2 = 150 - \left( \frac{\text{PaCO}_2}{0.8} \right).
\]

The formerly unwieldy \( \text{FiO}_2 (\text{Pb}-\text{P}_{\text{H}_2\text{O}}) \), now condensed.

* This equation also works, for some weird reason, in parakeets breathing helium in Ecuador. Who knew? Not me.

Notice how all those crazy water vapor pressures and barometric pressures (c’mon, what are you some sort of weatherman? No, you’re a clinician!) distill down into that nice, round figure of 150. Neat, huh? So by knowing the \( \text{PaCO}_2 \) from your blood gas, you can determine the amount of oxygen in the alveolus. I think of it in simplistic terms as the \( \text{CO}_2 \) “crowding” the oxygen out of the alveolus – more \( \text{CO}_2 \) produced by the body equals less room for oxygen.

To calculate the A-a gradient, you need the second part, the amount of oxygen in the arterial system or \( \text{PaO}_2 \). This is also obtained from your arterial blood gas sample as well. So, the measured arterial \( \text{PaO}_2 \) is subtracted from the calculated \( \text{PAO}_2 \) to give the A-a gradient.

\[
\text{P(A-a)O}_2 = \text{PAO}_2 - \text{PaO}_2
\]

Normal A-a gradient is less than 15mmHg, indicating that a very small gradient exists between the alveolus and pulmonary arterial system, and that oxygen is readily able to diffuse from the alveolus to the artery – nothing stands in the way. An elevated A-a gradient indicates that something is impairing the ability of oxygen to diffuse from the alveolus. As mentioned above, this is generally pulmonary parenchymal disease.

**Example:**

1) Normal Lungs:

- \( \text{PCO}_2 \) (measured) = 40mmHg
- \( \text{PaO}_2 \) (measured) = 95mmHg
- \( \text{PAO}_2 \) (calculated) = \( \text{FiO}_2 (\text{Pb}-\text{P}_{\text{H}_2\text{O}}) - \frac{\text{PaCO}_2}{\text{RQ}} \) = \( (0.21)(760-47) - 40/0.8 \)
Therefore…
\[
P(A-a)O_2 = PAO_2 - PaO_2 = 100 - 95
\]
The A-a gradient is…….5mmHg indicating no diffusion impairment

2) Diseased lungs (pneumonia):
\[
PCO_2 \text{ (measured)} = 40\text{mmHg}
\]
\[
PaO_2 \text{ (measured)} = 60\text{mmHg}
\]
\[
PAO_2 \text{ (calculated)} = \frac{FiO_2 \times (Pb-P_{H_2O}) - PaCO_2/RQ}{(0.21)(760-47) - 40/0.8}
\]
\[
= 150 - 50
\]
\[
= 100
\]
(same as in the normal lung example)
Therefore…
\[
P(A-a)O_2 = PAO_2 - PaO_2 = 100 - 60
\]
The A-a gradient is…….40mmHg indicating severe diffusion impairment

3) Hypoventilation
\[
PCO_2 \text{ (measured)} = 60\text{mmHg}
\]
\[
PaO_2 \text{ (measured)} = 70\text{mmHg}
\]
\[
PAO_2 \text{ (calculated)} = \frac{FiO_2 \times (Pb-P_{H_2O}) - PaCO_2/RQ}{(0.21)(760-47) - 60/0.8}
\]
\[
= 150 - 75
\]
\[
= 75
\]
Therefore…
\[
P(A-a)O_2 = PAO_2 - PaO_2 = 75 - 60
\]
The A-a gradient is…….15mmHg indicating no diffusion impairment – the hypoxia is due to hypoventilation alone.

\textbf{Metabolic/Acid-Base:} First, a few boring definitions. An acid is any molecule that donates a proton, while a base accepts one. A buffer can be a weak acid or base, and helps to protect against wide swings in pH. The primary extracellular buffer is bicarbonate. Intracellular buffers are phosphate, proteins and hemoglobin. pH is the measure of acidity/alkalinity, and is equal to negative the log of the H+ concentration. Acidemia is a blood pH less than 7.35, while alkalemia is a blood pH of greater than 7.45. Acidosis and alkalosis refer to the process that is causing the pH disturbance. Normal ranges for pH, PaCO₂, PaO₂, HCO₃⁻ and base excess are listed below.
Normal values for blood gases

<table>
<thead>
<tr>
<th>Canine</th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.35-7.45</td>
<td>7.35-7.45</td>
</tr>
<tr>
<td>PO2 (mm Hg)</td>
<td>90-100</td>
<td>30-42</td>
</tr>
<tr>
<td>PCO2 (mm Hg)</td>
<td>35-45</td>
<td>40-50</td>
</tr>
<tr>
<td>HCO3- (mmol/L)</td>
<td>20-24</td>
<td>20-24</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>-4-+4</td>
<td>-4-+4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Feline</th>
<th>Arterial</th>
<th>Venous</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.34 ± 0.1</td>
<td>7.30 ± 0.08</td>
</tr>
<tr>
<td>PO2 (mm Hg)</td>
<td>102.9 ± 15</td>
<td>38.6 ± 11</td>
</tr>
<tr>
<td>PCO2 (mm Hg)</td>
<td>33.6 ± 7</td>
<td>41.8 ± 9</td>
</tr>
<tr>
<td>HCO3- (mmol/L)</td>
<td>17.5 ± 3</td>
<td>19.4 ± 4</td>
</tr>
<tr>
<td>BE (mmol/L)</td>
<td>-6.4 ± 5</td>
<td>-5.7 ± 5</td>
</tr>
</tbody>
</table>

Metabolic (acid-base) disturbances can change the patient’s pH away from the normal range (7.3-7.45) in either the acidic direction (pH<7.3) or alkalotic direction (pH>7.45). As an example, one of the most common conditions requiring measurement of acid-base status is diabetic ketoacidosis. The buildup of ketones, which are acidic byproducts of fatty acid metabolism, cause the blood to become acidic, and result in a decrease in the body’s main buffer, bicarbonate (HCO3) to balance the acid (like adding Tums to your stomach to reduce acidity). The way the body compensates for the decreased pH is to increase respiration and “blow off” CO2 which acts like and acid in the body. For an alkalosis, which is less common, the body tries to retain acid (CO2, by breathing slower) or decrease the amount of buffer (less HCO3 produced by the kidneys).

As a general rule, the body compensates for an abnormality in one body system (for example metabolic, mostly controlled by the kidneys) by changing something in the other system. This compensation is the body’s way of maintaining the pH in the very narrow range that the body prefers. Outside of this range, enzyme and other systems do not function properly and the patient is referred to as a “Gomer” and the owner is instructed not to buy large bags of dog food.

It is possible to calculate the amount of compensation that the body should be aiming for and decide if compensation is occurring or if there is a mixed disorder (for example, a DKA with pneumonia that has both a metabolic acidosis and respiratory acidosis) but that is beyond the scope of this talk and I have to pull out a bunch of textbooks to make it make any sense at all.

Treatment of metabolic abnormalities always involves therapy for the inciting disease (for example giving insulin to a DKA so the body can metabolize the acidic ketones), as well as other steps like giving bicarbonate or increasing/decreasing the respiratory rate to return the pH to normal. For many metabolic acidoses, giving IV fluids to increase
tissue perfusion and decrease lactate production will be enough to solve the problem without having to resort to giving bicarbonate.

There are 4 main acid-base derangements:

Primary disturbance: ↓HCO₃ ‒ pH
Compensation: ↑PaCO₂
Example: DKA

Primary Disturbance: ↑pH, ↑HCO₃
Compensation: ↑CO₂
Example: Pyloric obstruction with vomiting of stomach acid

Primary Disturbance: ↓pH, ↑ CO₂
Compensation: ↑HCO₃
Example: drugs/sedatives, severe lungs disease, Coonhound paralysis, neuro disease (cervical IVDD)

Primary Disturbance: ↑pH, ↓CO₂
Compensation: ↓HCO₃
Example: pain, nervousness, seizures, hyperthermia, any other causes of panting

And there are 3 main steps in evaluation:

1. **Determine the primary disorder.** Evaluation of the pH will reveal if the patient is acidotic or alkalotic according to the above normals. For an acidosis, determine if it is metabolic or respiratory in origin. If the PaCO₂ is elevated, the acidosis is respiratory and you have just diagnosed hypoventilation. If the PaCO₂ is normal or decreased, but the HCO₃⁻ is decreased, the acidosis is metabolic.

2. **Evaluate the expected compensation.** This is where things get a mite ugly. Let’s start with the compensation – this is how the body attempts re-establish homeostasis, and the compensation always occurs in the body system not responsible for the primary disturbance: the compensation for a metabolic disorder is respiratory and vice-versa. If the pH is normal, or near normal, then compensation is said to have occurred. Over-compensation does not occur – unlike in those people you see driving monster jacked-up trucks. They must be over-compensating for something. Here are the expected compensations:

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Primary Change</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>↓HCO₃</td>
<td>0.7mm Hg decrease in PCO₂ for each 1mEq/L decrease in HCO₃⁻</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑HCO₃</td>
<td>0.7mm Hg increase in PCO₂ for each 1mEq/L increase in HCO₃⁻</td>
</tr>
<tr>
<td>Acute resp acidosis</td>
<td>↑PCO₂</td>
<td>1.5mEq/L increase in HCO₃⁻ for each 10mm Hg increase in PCO₂</td>
</tr>
</tbody>
</table>
3. Evaluate for mixed disorders: If the expected compensation has not occurred, then there is a mixed disorder. Mixed disorders can both be in the same direction (e.g. a metabolic and respiratory acidosis) or in opposite directions (e.g. metabolic alkalosis and respiratory acidosis). Overcompensation does not occur.

**Example:**

Cat, DKA and pancreatitis. On a fentanyl CRI for pain. Venous sample.

\[
\begin{align*}
\text{pH} & = 7.05 \\
\text{PCO}_2 & = 40 \\
\text{HCO}_3^- & = 14
\end{align*}
\]

Evaluation of the pH reveals an acidosis. The \( \text{PCO}_2 \) is normal and the \( \text{HCO}_3^- \) is low, so the primary disorder is a metabolic acidosis, which is consistent with his primary disease. Expected compensation for the metabolic acidosis is through the respiratory system (by blowing off \( \text{CO}_2 \)), so you would expect a drop in \( \text{PCO}_2 \) of 0.7 mm Hg for each 1 mEq/L decrease in \( \text{HCO}_3^- \). That means the expected drop in \( \text{PCO}_2 \) should be:

\[
0.7 \times (\text{Normal HCO}_3^- - \text{patient HCO}_3^-) \quad \text{-OR-}
\]

\[
0.7 \times (24 - 14) = 7 \text{ mmHg}
\]

Leading to an expected \( \text{PCO}_2 \) of 40 minus 7, or 33. Since the actual \( \text{PCO}_2 \) is higher than the expected level, compensation has not occurred and we should look for a secondary disorder. Since we also know that elevated \( \text{PCO}_2 \) causes respiratory acidosis, the patient also has a concomitant respiratory acidosis.
**SIMPLE(R) BLOOD GAS ANALYSIS:**

1) Evaluate the PAO2 to determine if the patient needs supplemental oxygen

2) Evaluate the pH and determine if there is an alkalosis or acidosis present

3) Evaluate the PaCO2 to determine if it explains the pH findings – if YES, then there is a respiratory condition, if NO then the condition is not primarily respiratory

4) Evaluate the HCO3 to determine if it explains the pH findings – if YES then there is a metabolic condition, if NO then the condition is not primarily metabolic.

5) Evaluate the condition NOT responsible for the abnormal condition (example – look at the CO2 for a metabolic condition) to assess for compensation.