A crucial goal in the critical patient is the maintenance and optimization of cellular (and organ) health. Cellular health relies on aerobic metabolism to release energy. Hypoxia occurs when tissue oxygen delivery (DO$_2$) is low enough to cause abnormal function. DO$_2$ depends on the arterial oxygen content of blood (CaO$_2$) and the delivery of the blood to the tissues (perfusion). These are vital components to cellular health, and monitoring of these components provides the clinician with a means to assess their status, and thus guide appropriate management (“what one can measure, one can manage”). Unfortunately, clinically available tools enable assessment of only aspects of this dynamic relationship. It is important, therefore, that such tools are used carefully, with their limitations in mind, and in the light of other findings.

**OXYGENATION AND VENTILATION**

The oxygen content of arterial blood is comprised primarily of O$_2$ bound to hemoglobin (Hb), with a smaller proportion dissolved in plasma, as defined by the following equation:

$$\text{CaO}_2 = (1.34 \times \text{Hb} \times \text{SaO}_2) + (0.003 \times \text{PaO}_2)$$

Hb-bound O$_2$ depends on the concentration of Hb as well as the degree of saturation of that Hb with O$_2$ (HbO$_2$ = 1.34 x Hb x SaO$_2$). The dissolved O$_2$ depends on the solubility of oxygen in plasma and the partial pressure of O$_2$ in the blood (dissolved O$_2$ = 0.003 x PaO$_2$). PaO$_2$, in turn, depends on the amount of oxygen in the inspired air (FiO$_2$), the ability of the animal to move air in and out of the pulmonary tree (ventilation), and the absorption of O$_2$ from the pulmonary tree into the blood. Since CO$_2$ is readily diffusible between the blood and alveoli, removal of CO$_2$, and thus PaCO$_2$, depends largely on ventilation.

**Arterial Blood Gas Analysis**

Partial pressure of dissolved O$_2$ (PaO$_2$) is the most common blood gas parameter measured to assess respiratory function. Normal PaO$_2$ in an animal breathing room air (21% O$_2$) is approximately 100 mmHg. A PaO$_2$ < 80mmHg is considered hypoxemic. It is possible, however, for healthy animals living at high altitudes to have significantly lowerPaO$_2$.

The efficiency of gas exchange can be calculated by the alveolar-arterial (A-a) O$_2$ gradient, PAO$_2$ - PaO$_2$. If the patient is breathing room air, at sea level, PAO$_2$ = 150 - PaCO$_2$/0.8. A normal A-a gradient is <10-15 mmHg. Gas exchange can also be assessed via the ratio of PaO$_2$ to the %O$_2$ in the inspired air. A ratio of 5 is normal. (e.g., at room air, PaO$_2$ should be approx. 100; if the patient is breathing 100% O$_2$, PaO$_2$ should be 500.)

Oxygen saturation (SaO$_2$) is a measure of the percentage of Hb in an arterial blood sample that is occupied by O$_2$ molecules. The relationship between PaO$_2$ and SaO$_2$ is sigmoidal, and represented by the oxyhemoglobin dissociation curve (Fig 1). Most blood gas analyzers do not measure SaO$_2$ directly, but rather calculate it using a nomogram. This is appropriate and accurate under most circumstances, unless there is dysfunctional Hb in the sample (e.g., carboxyHb, methHb), in which case SaO$_2$ should be measured via co-oximetry.

Ventilation is assessed by measurement of PaCO$_2$. Minute ventilation is the total amount of gas moved in and out of the lung per minute, and is a measure of the tidal volume, and the respiratory rate. Hypoventilation leads to increased PaCO$_2$; values above 50 mmHg are significant and require treatment; values above 70 mmHg are life-threatening. Hypoventilation is accompanied by decreased PaO2. As a general rule, if hypoxemia is caused solely by hypoventilation, then the decrease in PaO$_2$ is roughly equal to the increase in PaCO$_2$. 
Pulse Oximetry
Pulse oximetry is a patient-side measure of O\textsubscript{2} saturation. The pulse oximeter is a dual wavelength spectrophotometer that measures Hb saturation by transmitting light through a pulsating arterial vascular bed. By using the appropriate transmitted light wavelengths for oxyHb and deoxyHb and a microprocessor that filters nonpulsatile readings, O\textsubscript{2} saturation (SpO\textsubscript{2}) can be continually calculated. Normal SpO\textsubscript{2} is over 93%; values of 90% correlate to a PaO\textsubscript{2} of 60-70%.

Pulse oximeters have been validated to be accurate in dogs and cats. The oximeter probe can be placed on the tongue, or shaved skin over the lip, ear pinna, axilla, inguinal fold, hock, interdigital web, or prepuce. Pulse oximetry provides an inexpensive, non-invasive means for assessing oxygenation, that is easy, generally well tolerated, and reliable. It provides the ability to continually monitor trends between blood gas analyses, or in place of blood gas analysis where the latter is unwarranted, infeasible or unavailable. There are, however, drawbacks. Readings may not be possible, or accurate, in some patients as the oximeter is susceptible to artifact. Readings may be affected by dark skin, vasoconstriction, hypothermia, hypoperfusion, tachycardia, severe anemia, hyperbilirubinemia, oxyglobin, and ambient light. The pulse oximeter also cannot distinguish between normal and abnormal Hb. CarboxyHb will absorb light similar to oxyHb providing falsely high readings. MetHb, on the other hand, absorbs both light wavelengths equally well, and the pulse oximeter will default to a reading of 85%, reading high or low based on the patient’s saturation.

Even when accuracy is assured, there are limitations in the use of O\textsubscript{2} saturation to assess oxygenation. Given the nonlinear shape of the dissociation curve, oxygenation can drop precipitously with little change in SO\textsubscript{2}. Moreover, oximetry provides little information regarding gas exchange efficiency; an SpO\textsubscript{2} of 100% in a patient breathing 100% O\textsubscript{2} does not evaluate whether the PaO\textsubscript{2} is 100 mmHg or 500 mmHg.

Capnography
Capnography measures the amount of CO\textsubscript{2} in air. End-tidal capnography refers to the amount of CO\textsubscript{2} in exhaled air at the end of exhalation (ETCO\textsubscript{2}). At this point in the respiratory cycle, the air is almost entirely composed of alveolar air. And, because of the diffusability of CO\textsubscript{2}, the amount of CO\textsubscript{2} in alveolar air is almost exactly that of pulmonary capillary blood. That is, ETCO\textsubscript{2} reflects PaCO\textsubscript{2}, and provides a continual and noninvasive assessment of ventilatory status. There is normally a small gradient between ETCO\textsubscript{2} and PaCO\textsubscript{2} (usually < 5 mmHg). This gradient, however, is variable, and can change dramatically under a variety of patient- and apparatus-related factors. As such, the instrument is best used to follow trends, and interpreted together with intermittent blood gas analysis.

Capnography can be used during spontaneous ventilation, in awake or anesthetized patients, or during mechanical ventilation. Two types of capnographs are available: mainstream and sidestream. Mainstream capnography measures CO\textsubscript{2} concentration directly in the breathing circuit. Advantages include: fast response time, ease of calibration, and few disposables. Disadvantages include: the need to place the sensor at the ET-tube junction, increase in apparatus dead space, potential for leak/disconnection/obstruction, and exposure of the sensor to damage. Sidestream capnography samples gas from the breathing system, suctions it via a sampling tube to the main unit, and measures CO\textsubscript{2} therein. Advantages include: minimal dead space, lightweight patient interface, and the ability to measure multiple gases (e.g., anesthetic). Disadvantages include: delayed responsiveness to scavenge gas, need for calibration, and potential for gas mixing in the sampling tubing.

Interpretation of ETCO\textsubscript{2} should consider both patient- and equipment-related causes, and include intermittent arterial blood gas analysis. Hyperventilation results in a decreased ETCO\textsubscript{2} normal waveform, and a normal PaCO\textsubscript{2}-ETCO\textsubscript{2} gradient. Hypoventilation results in opposite effects. Decreased transport of CO\textsubscript{2} to the lungs (e.g., decreased cardiac output, pulmonary thromboembolism) results in pathologic dead space, manifested as decreased ETCO\textsubscript{2}, and an increased PaCO\textsubscript{2}-ETCO\textsubscript{2} gradient. Inadequate seal around the ET-tube can also result in low ETCO\textsubscript{2} and an increased PaCO\textsubscript{2}-ETCO\textsubscript{2} gradient.
**PERFUSION**

**Arterial Blood Pressure**

Arterial blood pressure (BP) is a product of cardiac output and systemic vascular resistance. Each left ventricular ejection creates a pressure pulse whose peak is systolic BP. Normal values for systolic BP are 110-170 mmHg in dogs, and 120-170 mmHg in cats. Blood ejected in systole is partly stored in distended arteries that rebound to create diastolic BP. This is influenced by the duration of diastole, blood volume, and arterial elasticity. Normal values for diastolic BP are 55-110 mmHg in dogs and 70-110 mmHg in cats. Mean arterial pressure (MAP) is the integrated mean of pressures throughout the cardiac cycle, and is calculated as follows: \[ \text{MAP} = \frac{1}{3} \left( \text{systolic pressure} - \text{diastolic pressure} \right) \].

BP is an important determinant of tissue perfusion, and cannot be reliably estimated from digital palpation of the pulses. Pulse quality (amplitude) reflects stroke volume and is not well correlated with BP. Pulse pressure is the difference between systolic and diastolic BP; a large difference creates a high amplitude pulse, but is weakly correlated to BP. Measurement of BP, therefore, is important. Hypotension is defined as a systolic BP < 80 mmHg, or a MAP < 60 mmHg.

BP, however, is not the sole determinant of perfusion, and should not be excessively relied upon for clinical decision making. Perfusion is the difference between arterial blood pressure (BP) and intra-organ pressure. Adequate BP, therefore, does not necessarily equate to adequate perfusion; vasoconstriction increases vascular resistance and thus BP, but results in decreased perfusion. Conversely, low BP does not always mean inadequate perfusion; if intra-organ pressure is low, adequate flow can be obtained even with subnormal BP.

**Indirect (noninvasive) BP monitoring.** Noninvasive BP monitoring is based on inflation of a cuff to occlude arterial flow, followed by measurement of the pressure at which flow returns. While these methods are technically not difficult, and relatively inexpensive, they are prone to error – both operator error and related to the methodology. Proper cuff selection and placement is essential. The cuff width should be 40% of the circumference of the site where it will be placed in dogs, and 30% of the circumference in cats. Use of a cuff that is too wide will give falsely low readings, while a too narrow cuff will give falsely high readings. The cuff should be snugly applied sufficient to allow insertion of a small finger between the cuff and the site. Too tight placement will result in erroneously low readings, while loose placement will result in erroneously high readings. To minimize inter-reading variability, it is advisable to take 5 measurements, discard the highest and lowest, and average the remaining three.

The ultrasonic Doppler method uses an inflatable cuff attached to a manometer, and a 10-MHz ultrasound probe to detect arterial blood flow distal to the cuff. Sounds become audible when the pressure in the cuff is less than the pressure in the artery. Typically, the Doppler method is considered to measure only systolic BP. A study in cats, however, suggested that Doppler readings tend to be 10-15 mmHg lower than systolic BP, and more closely resemble MAP.

The oscillometric technique uses a cuff that alternately inflates and deflates; during deflation alterations in cuff pressure are sensed by the transducer in the main unit. Systolic BP is the pressure at which oscillations are first detected, and diastolic BP when oscillations disappear. The peak amplitude equals MAP. Some units measure MAP only, and calculate systolic and diastolic pressures, making MAP most accurate. Oscillometric methodology offer the advantage of enabling continuous monitoring, but is generally less accurate in small patients and those with hypotension or dysrhythmias. Some newer models (e.g., Cardell), however, appear to be more accurate in these situations.

**Direct (invasive) BP monitoring.** Direct BP monitoring is considered the gold standard. An arterial catheter is connected to a pressure transducer and monitor, allowing for continuous monitoring of systolic, diastolic and mean pressures. The arterial catheter is connected to a bag of heparinized saline (1 unit heparin per ml 0.9% NaCl) via primed semirigid tubing. The bag is maintained at a pressure of 300 mmHg to prevent back flow of arterial blood. A 3-way stopcock connects the tubing to a pressure transducer that is connected to a cable placed at the level of the patient’s heart. The transducer converts pressure changes to an electrical signal, then displayed by waveform and numerically on the monitor.
Erroneous results can occur even with direct BP monitoring due to compliant or kinked tubing, blood clots, or air bubbles.

Direct BP monitoring is indicated for critically ill patients who will benefit from continuous or frequent BP monitoring over a period of time. It is very useful for guiding vasopressor or antihypertensive therapy.

**Central Venous Pressure**

Central venous pressure (CVP) is the hydrostatic pressure in the intrathoracic vena cava and, in the absence of vascular obstruction, is approximately equal to right atrial pressure. When the tricuspid valve is open, right atrial pressure equals right ventricular end-diastolic pressure. CVP is an estimate of the relationship between blood volume and blood volume capacity (vascular filling). It is also a measure of the relative ability of the heart to pump the volume of fluid being returned to it. CVP is indicated in situations in which it is difficult to define an end-point of fluid resuscitation from other parameters, or where relatively large fluid volumes are indicated in patients with some degree of cardiac insufficiency or anuria/oliguria.

CVP is measured via a central catheter placed with the tip in the cranial vena cava, close to the right atrium. (Catheters placed in the caudal vena cava via the femoral or saphenous vein may also be used in cats and puppies but tend to give less accurate and predictable results.) The CVP is measured via electronic pressure transducer or a water manometer. A pressure transducer is advantageous in that it provides continuous readings and allows assessment of a waveform. Where a transducer is not available, intermittent readings can easily be obtained with readily available and inexpensive equipment (manometer, or infusion tubing and ruler, 3-way-stopcock, 60 ml syringe). A point on the parameter that is estimated to be on a horizontal level with the tip of the catheter (manubrium for patients in lateral recumbency, or point of the shoulder for patients in sterna recumbency) establishes the “zero reference” point.

Normal CVP is 0-4 mmHg or 0-5 cmH₂O, but can vary in individual patients, making evaluation of trends more significant than absolute values. A low CVP implies inadequate vascular filling (hypovolemia or vasodilatation). A high CVP implies intravascular volume overload, increased intrathoracic pressure, or right-sided cardiac dysfunction (myocardial failure, pericardial effusion, tamponade, restrictive pericarditis). The change in CVP following fluid bolus can be an extremely useful guide to fluid resuscitation, especially if fluid overload is a concern. If CVP is low, increases following bolus, and rapidly returns to pre-bolus levels, then additional fluid therapy is indicated. If it remains high, adequate vascular volume is implied, and alternative therapy is indicated.

<table>
<thead>
<tr>
<th>Monitoring tool</th>
<th>Parameter (unit)</th>
<th>Dogs</th>
<th>Cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood gas</td>
<td>PaO₂ (mmHg)</td>
<td>102 +/- 7</td>
<td>107 +/- 12</td>
</tr>
<tr>
<td></td>
<td>SaO₂ (%)</td>
<td>95 - 100</td>
<td>95 - 100</td>
</tr>
<tr>
<td></td>
<td>PaCO₂ (mmHg)</td>
<td>37 +/- 3</td>
<td>31 +/- 6</td>
</tr>
<tr>
<td>Pulse oximetry</td>
<td>SpO₂ (%)</td>
<td>95 - 100</td>
<td>95 - 100</td>
</tr>
<tr>
<td>Capnography</td>
<td>ETCO₂ (mmHg)</td>
<td>0-5 lower than PaCO₂</td>
<td></td>
</tr>
<tr>
<td>Arterial blood pressure</td>
<td>Systolic (mmHg)</td>
<td>100 - 160</td>
<td>120 - 160</td>
</tr>
<tr>
<td></td>
<td>Diastolic (mmHg)</td>
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<td>Central venous pressure</td>
<td>CVP (mmHg)</td>
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<td>0 - 4</td>
</tr>
<tr>
<td></td>
<td>CVP (cm H₂O)</td>
<td>0 - 5</td>
<td>0 - 5</td>
</tr>
</tbody>
</table>
Figure 1.

OxyHemoglobin Dissociation Curve

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