There are many conditions where oxygen may need to be supplemented for a patient for example dyspnoea, shock, sepsis, SIRs and head trauma.

Hypoxaemia can be defined as low oxygen content in arterial blood. This can be caused by reductions in arterial oxygen tension (PaO$_2$), decreases in oxygen haemoglobin saturation (SaO$_2$) and decreases in haemoglobin concentration.

Decreases in PaO$_2$ are caused by:
- Low inspired oxygen levels (FiO$_2$)
- Hypoventilation
- Diffusion barrier (i.e. pulmonary oedema/pus in alveoli)
- Ventilation: perfusion inequality
- Intrapulmonary shunt (causing blood to pass through the lungs without undergoing gaseous exchange)

Decreases in SaO$_2$ are caused by:
- Decreases in PaO$_2$
- The formation of methemoglobinemia or carboxyhaemoglobinemia

Decreases in haemoglobin concentration are caused by anaemia/ blood loss.

Hypoxia, defined as impaired oxygen delivery to tissues, can be caused by hypoxaemia but can also be caused by decreased cardiac output, decreased perfusion and increased oxygen extraction by the tissues. Systemic illnesses such as sepsis, systemic inflammatory response syndrome (SIRs) and acid-base abnormalities may also contribute to inadequate oxygen delivery.

Treatment can include oxygen supplementation, IPPV and where possible treatment of underlying disease i.e. treatment for hypoventilation, administration of packed red blood cells for anaemia et cetera.
Methods of oxygen delivery

We can compare the many techniques available for oxygen supplementation by comparing the fractional inspired oxygen concentration (FiO₂) that they provide to the patient. The FiO₂ of air is 0.21.

The method chosen will also depend greatly on the needs of the patient, patient compliance, patient size, degree of hypoxaemia, anticipated duration of delivery, skill and equipment available.

Non-invasive methods of oxygen delivery

Mask
A well fitting mask is placed over the patient's face and oxygen is delivered. Flow rates of 5-6L/min can achieve FiO₂ of up to 0.7. Masks will be poorly tolerated by some patients and another method of oxygen delivery should be employed if the patient starts to struggle or become stressed as this will increase metabolic oxygen demand. Cats especially do not tolerate masked oxygen well. Tight fitting face masks may cause rebreathing of carbon dioxide and subsequent hypercarbia. The mask should be vented periodically or a looser mask chosen. Minimal equipment is required for this technique and it allows oxygen to be administered during assessment of the patient.

Flow by oxygen
This is the simplest technique employed in an emergency setting and is achieved by simply holding the oxygen supply close to the patient's nares or mouth. An oxygen flow rate of 2-3L/min provides FiO₂ of 0.25-0.4. This is often better tolerated by the patient than a mask and is a useful technique when patients are being examined or stabilised. This technique wastes a large amount of oxygen and would not be practical or economical for long term use.

Elizabethan collar with cling film/ Oxygen hoods
The front of the Elizabethan collar is covered with cling film and taped in place. A small vent hole, 2-5cm, is made at the top to allow carbon dioxide and water to be eliminated. The collar is placed around the patient's neck insuring a snug fit. Oxygen is delivered through oxygen tubing secured inside the collar.
Some patients will not tolerate these collars and temperature should be monitored q2h to detect hyperthermia as the humidity and temperature inside the collar can increase rapidly.
A flow rate of 1-8L/min, depending on the size of the patient, will typically provide FiO₂ of 0.3-0.4.

**Incubator**
Paediatric incubators into which oxygen can be piped can be very useful for cats, small dogs and neonates. They allow the patient to be observed through the glass whilst receiving oxygen in a stress free environment. The temperature can also often be controlled on these incubators. Although expensive new these can be purchased for a more reasonable price second hand following use in human hospitals. FiO₂ of up to 0.8 can be achieved.

**Oxygen Cage**
Commercial cages are available that allow control of FiO₂, temperature and humidity. These specialised cages are vented to prevent carbon dioxide build up. Other cages are available that do not allow the control of temperature and humidity and care should be taken to ensure that the patient does not become hyperthermic. The addition of ice packs to the cage can sometimes help overcome this problem. FiO₂ can be achieved up to 1.0 depending on the cages ability to deliver pure oxygen. Large dogs may not fit in commercial oxygen chambers! The glass allows continuous observation of the patient and specialised ports allow connection of ECG, blood pressure and pulse oximetry monitoring to the patient. The major disadvantage is that auscultation and assessment of pulses is not possible without opening the door and sounds of stertor or stridor may not be heard through the glass. It should be noted that opening the cage door drops the FiO₂ to that of room air rapidly. Some commercial cages have specialised ports to put your arm through to minimise the loss of oxygen from the cage.

**Invasive Methods of Oxygen Delivery**

**Nasal Prongs**
These are manufactured for human use and penetrate approximately 1cm into the nares. Nasal prongs are minimally invasive and attach behind the ears. This technique is often useful in large breed dogs that are relatively immobile. A 6-8L/min flow rate is generally well tolerated although the FiO₂ achieved will be less than with a nasal cannula due to the superficial placement.
Nasal Cannulae
Nasal catheters can be used in dogs and cats of most sizes. 5-10 French red rubber or polypropylene catheters/ feeding tubes are utilised for this purpose depending on patient size.

Placement of a Nasal Cannula:
- Desensitise the nares using a few drops of Proxymetacaine or 2% lidocaine placed into the ventral meatus. Wait 5 mins.
- Pre-measure the tube holding the tip at the medial canthus of the eye and marking the tube at the level of the nostril.
- Direct the tube ventro-medially beyond the medial canthus to ensure correct placement and then retract to the medial canthus so the mark on the tube is at the entrance to the nostril. The tube should now be in the ventral meatus.
- Suture or glue the catheter carefully in place adjacent to the nostril and again n the side of the face or top of the head

Two nasal cannulae may be placed in severely compromised patients with an oxygen source to supply each. This will achieve a higher FiO₂.
An Elizabethan collar can be utilised to prevent patient interference.
Oxygen delivered through a nasal cannula should always be humidified. High flow rates may be irritant and cause sneezing. Sneezing and patient intolerance may be alleviated by reapplying local anaesthetic to the nares or advancing the cannula into the nasopharyngeal region.
Table to Show Suggested Flow Rates for Supplemental Nasal O2 (adapted from Oxygen Supplementation, Matthews K, Veterinary Emergency and Critical Care, 2nd edition, 2006, Lifelearn, Canada.)

<table>
<thead>
<tr>
<th>Weight (Kg)</th>
<th>Flow rate (L/min) to achieve FiO₂ 0.5</th>
<th>Flow rate (L/min) to achieve FiO₂ 0.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5</td>
<td>0.3</td>
<td>0.5</td>
</tr>
<tr>
<td>5</td>
<td>0.6</td>
<td>1.25</td>
</tr>
<tr>
<td>10</td>
<td>1.0</td>
<td>2.0</td>
</tr>
<tr>
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</tr>
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<td>2.8</td>
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<td>10.0</td>
</tr>
<tr>
<td>60</td>
<td>6.5</td>
<td>N/A</td>
</tr>
</tbody>
</table>

Humidification of Oxygen

Any method of oxygen supplementation that is going to be used for more than a few hours should be humidified i.e. saturated with water vapour. This prevents drying and dehydration of the nasal mucosa which can lead to desiccation of the respiratory epithelium, impaired ciliary clearance and poses an increased risk of infection.

Specialised humidifiers can be purchased that both heat and humidify the oxygen however in practice where one may not be available humidification may be achieved by bubbling oxygen through a tube submerged in sterile saline. Humidified oxygen then collects above the surface of the saline. This can then be delivered to the patient via a second length of tubing. A container should be used that allows an air tight seal to be achieved around the entrance site of each tube.

Oxygen Toxicity

High concentrations of oxygen FiO₂ >0.6 administered for more than 12 hours can be associated with oxygen toxicity and resulting damage to the pulmonary epithelium. The timing and severity of the damage is dependent on the duration of exposure and the FiO₂.
Inflammation injury is caused by toxic metabolites of oxygen including free radicals, superoxide and peroxide molecules. A massive release of inflammatory mediators causes increased tissue permeability and therefore pulmonary oedema develops.

It is often difficult to maintain FiO$_2$ over 0.6 with many methods of oxygen delivery with the exception of oxygen cages and mechanical ventilation so the risk of oxygen toxicity is lowered.

In cases which are receiving FiO$_2$ > 0.6 for severe dyspnoea it may not be possible to decrease the FiO$_2$ without causing severe respiratory distress.

Other Complications of Oxygen Therapy

Hypercapnia is the overriding stimulus for respiration in healthy animals. In patients with chronic respiratory disease and hypercapnia the hypercapnic respiratory drive is greatly reduced or lost. This means that hypoxaemia becomes the main respiratory stimulant in these patients. By administering oxygen to a chronically hypercapnic patient we are removing the hypoxaemic respiratory drive. In some cases this can result in severe hypoventilation and respiratory failure. However this is not commonly seen in veterinary patients and shouldn’t be seen as a reason not to supplement with oxygen.

Assessment of Oxygenation and Ventilation

Arterial Blood Gases

Measurement of partial pressure of oxygen (PaO$_2$) and carbon dioxide (PaCO$_2$) in arterial blood is the gold standard for determining lung function as it gives us information about oxygenation as well as ventilation.

Animals with normal lung function should have a PaO$_2$ > 85mmHg when breathing 21% oxygen (room air). A PaO$_2$ <80mmHg can be considered hypoxaemia and are usually treated by oxygen supplementation and addressing the underlying cause. Values less than 55mmHg are imminently life threatening and require immediate action. Increases in FiO$_2$ lead to increases in PaO$_2$ with a general rule of thumb being that PaO$_2$ should equal roughly five times FiO$_2$ e.g. a patient receiving 100% oxygen via an ET tube should have a PaO$_2$ of approximately 500mmHg. The PaO$_2$ to FiO$_2$ ratio (where FiO$_2$ is expressed as a decimal) should be over 500. If this ratio is under 400 this is indicative of moderate pulmonary dysfunction. When the ratio falls below 200 severe pulmonary dysfunction is indicated.

PaCO$_2$ gives us a picture of how well the patient’s alveoli are being ventilated. Normal PaCO$_2$ is 35-45mmHg. In simple terms if the PaCO$_2$ is greater then there is either reduced ventilation of the perfused alveoli or there is an increase in CO$_2$ production. Conversely if
there is a decrease in \( \text{PaCO}_2 \) then either alveolar ventilation is increased or there is a
decrease in \( \text{CO}_2 \) production.

There are several sites for arterial sampling: dorsal pedal artery, digital artery, auricular
artery, lingual artery and femoral artery. The dorsal pedal artery is most commonly used for
arterial sampling. If serial sampling is required an arterial catheter can be placed. Pre-
heparinised syringes should be used for arterial sampling. The patient should be suitably
restrained. The area over the dorsal pedal artery is clipped and gently prepped. The artery is
calculated so the pulsations can be felt whilst guiding the needle at a 60° angle towards the
artery. When the needle penetrates the artery a flash of blood will be seen in the needle hub
and the sample can be collected. All bubbles should be removed immediately, the sample
tightly capped and run immediately if possible.

Common Errors
- If the sample is not capped or there are bubbles in the sample \( \text{PaCO}_2 \) will be decreased
  and the \( \text{PaO}_2 \) increased as the sample equilibrates with room air. Samples run within 90
  seconds are unlikely to be affected by air bubbles.
- Too much heparin compared to sample volume will decrease \( \text{PaCO}_2 \).
- Samples not run immediately or held on ice and run within 2 hours will show changes to
  \( \text{pH} \), \( \text{PaO}_2 \) and \( \text{PaCO}_2 \) due to cellular metabolism.

Venous Blood Gases
More easily obtained than arterial blood gases. Useful for the assessment of ventilation the
\( \text{PCO}_2 \) of venous blood is usually 4-6mmHg higher than that of arterial blood. Venous \( \text{PO}_2 \)
values are not representative of arterial oxygen values however a venous \( \text{PO}_2 \) of less than
30mmHg may suggest poor tissue oxygenation and should be investigated.

Pulse Oximetry
This can be used to determine the arterial haemoglobin saturation with oxygen (\( \text{SpO}_2 \)). In a
healthy patient the haemoglobin should be >95% saturated with oxygen. The relationship
between \( \text{PaO}_2 \) and \( \text{SpO}_2 \) forms a sigmoid curve meaning that below 93% \( \text{SpO}_2 \) a small
decrease in \( \text{SPO}_2 \) will result in a large decrease in \( \text{PaO}_2 \). Therefore pulse oximeter readings
of 93% or higher are acceptable in non-anaemic critically ill patients. But as 90% \( \text{SpO}_2 \)
correlates to a \( \text{PaO}_2 \) of 60-70mmHg (severe hypoxaemia) oxygen should be supplemented
where readings on the pulse oximeter are less than 93%. As a rule of thumb below 90%
\( \text{SpO}_2 \) \( \text{PaO}_2 \) is approximately \( \text{SpO}_2 \) minus 30. Always try moving the probe to another
location before assuming hypoxaemia as the place where you have located the probe may merely be poorly perfused.

This uses a simple principle that oxygenated blood is a different colour than blood that is not well oxygenated. Light is passed through a pulsating arterial vascular bed and the pulse oximeter can detect the oxygen saturation within that artery. It disregards absorption from tissues that are not pulsating i.e. venous blood, skin and muscle. Oxygenated and deoxygenated hemoglobin give different light wavelengths which allow the microprocessor to detect the saturation. Pulse oximeters cannot distinguish dysfunctional hemoglobin such as methaemoglobin or carboxyhemoglobin.

The probe can be placed on various sites including the tongue, pinna, lip, toe web and tail but is sometimes not well tolerated in conscious patients. It is however minimally invasive and where arterial blood gases are not available it can be a useful tool in monitoring trends and disease progression in hypoxaemic patients and for tailoring oxygen therapy.

The major limiting factor of pulse oximetry is tissue perfusion. Conditions such as shock and hypotension which reduce peripheral blood flow will prevent the pulse oximeter from accurately reading hemoglobin saturation.

It should be noted that fluorescent lighting, pigmentation, compressed tissue (from leaving the probe in one place for too long); cold extremities and patient movement can all interfere with pulse oximeter readings.

Pulse oximetry is not accurate below an oxygen saturation of 75%.

**Capnography**

This technique is generally only utilised in intubated patients. The capnograph measures the amount of carbon dioxide in exhaled air (ETCO₂) and can give us an indirect estimation of ventilation status. ETCO₂ can give us an assessment of PaCO₂ which is usually ~ 5mmHg higher than ETCO₂ where the patient’s lungs are normal and the patient is otherwise stable.

In the non-stable patient or where the lungs are considered not to be normal an arterial blood gas can compare the PaCO₂ with the ETCO₂ to see how well they correlate. The ETCO₂ can then be monitored for trends and an estimated PaCO₂ calculated.
References and Further Reading


