cpdsolutions
transferring knowledge through excellence in training

Advanced Anaesthesia for Nurses Mini Series

Session Two: Advanced Anaesthetic Monitoring & Ventilation

Sam McMillan BSc(Hons) VTS(Anesthesia) DipAVN(Med) RVN

Sam McMillan VTS (Anesthesia), DipAVN(Med), RVN
Sammcmillan44@gmail.com

THE RVN ROLE

- Patient safety
- Recognise and minimise the risks to the patient
- Vigilance: closely observing and examining during the peri-operative period
- Reacting appropriately when a problem is identified

WHEN TO MONITOR

- Pre-med until fully recovered
- Continuously
- Record every 5 min
- Trends
- Legal document

CARDIAC OUTPUT

- Cardiac output (CO) = Heart rate x Stroke volume
- But heart rate does not correlate directly with CO and changes outside of the normal range will result in reduced CO
- Bradycardia - CO is reduced and hypotension may result
- Tachycardia does not give the heart time to adequately fill and cardiac output is reduced

ARTERIAL BLOOD PRESSURE

Mean arterial pressure = Cardiac output x systemic vascular resistance (+central venous pressure)

Normal values will vary depending on the patient's pathology but a rough guide in the anaesthetised patient is:

Systolic – 90-120mmHg

Mean – 60–90mmHg

Diastolic – 55-75mmHg

A mean of <60mmHg is the point at which perfusion to the major organs (most specifically the kidney) begins to become compromised.

Systolic pressure is the pressure generated when the left ventricle of the heart is fully contracted.

Diastolic pressure is the pressure within the left ventricle of the fully relaxed heart.

Mean arterial blood pressure (MAP) is the mean of pressures generated throughout the cardiac cycle and can be estimated as:

MAP = diastolic pressure + 1/3 (systolic pressure – diastolic pressure)

MAP gives an idea of the overall driving (perfusion) pressure to the tissues.

The body will generally try to maintain blood pressure as much as possible and can do this by:

- Increasing CO for example by increasing heart rate
- By causing vasoconstriction of the peripheral circulation
- By trying to retain fluid at the level of the kidneys

For these reasons the blood pressure may not drop initially on the monitors when haemorrhage occurs.

DIRECT BLOOD PRESSURE MONITORING

- More technically challenging
- Most accurate way to monitor blood pressure
- Gives systolic, mean and diastolic readings
- Requires arterial catheter, manometer & monitor
- Reserved for critical cases

INDIRECT BLOOD PRESSURE

Done using either the Doppler or oscillometric techniques. Both methods rely on a pressurised cuff occluding blood flow and then depressurising in a steady and measurable fashion so blood flow then returns beneath the cuff. For either method the correct cuff size should be chosen. The width of the cuff should be approximately 40% of the circumference of the site where the cuff will be placed. The cuff should be long enough so that the bladder of the cuff fully encircles the site where the cuff is to be placed. A cuff that is too wide or too tight will give measurements that are falsely low whereas a cuff that is too narrow or too loose will give falsely high readings. *If the Velcro of a cuff meets perfectly it is likely that the cuff chosen is of the appropriate size.*

CENTRAL VENOUS PRESSURE (CVP)

- Individual CVP measurements do not give an accurate assessment
- Trends should be monitored and may be more useful
- CVP is the pressure measured in the patient's cranial vena cava directly in front of the right atria and closely corresponds with the pressure in the right atrium
- CVP affects the end diastolic volume of the right ventricle and therefore influences stroke volume and cardiac output
- A central venous catheter must be placed aseptically in the right jugular vein
- Generally reserved for critical cases
- CVP is often utilised to guide fluid therapy - evidence for this technique is poor.
- Normal CVP is 0-5cmH_2O (1mm Hg =1.4cmH_2O)
- Low CVP can be indicative of decreased venous return whereas a high CVP may indicate volume overload, right sided heart failure or increased intra-thoracic pressure.

VENTILATION DEFINITIONS AND BASIC CALCULATIONS

Tidal volume = the volume of gas in one breath

Patients normal tidal volume = 10 to 15 ml/kg

Minute volume = the amount of gas breathed in and out in a minute

Minute volume = respiratory rate x tidal volume (but is approximately 200ml/kg)

Respiratory cycle time = inspiratory time + expiratory time

Respiratory rate = 60 / respiratory cycle time

Ventilator inspiratory tidal volume = inspiratory time x inspiratory flow rate

Ventilation

The act of taking gas in and out of the lungs (most importantly the alveoli):

Minute ventilation (volume) (mL/min) = Tidal volume (mL) x respiratory frequency (r/min)

Where tidal volume is the volume of gas in one breath.

NEUROLOGICAL CONTROL OF BREATHING

The rhythmic pattern of breathing is controlled by nuclei in the pons and medulla (the respiratory centres) but the output can be overridden to a certain extent by the cortex, e.g. during periods of stress. These nuclei receive input from central and peripheral chemoreceptors.

Central chemoreceptors are situated near the ventral surface of the medulla and are sensitive to the partial pressure of CO_2 (PCO_2) of blood. They respond to the changes in the pH of ECF/CSF that are caused by changes in PCO_2 as CO_2 diffuses from cerebral capillaries. Peripheral chemoreceptors are situated in the carotid and aortic body. They respond to decreased PO_2 and increased H^+ (decreased pH) and PCO_2.

PCO_2 of blood is the primary factor controlling ventilation under normal circumstances. The peripheral chemoreceptor response is more rapid but less marked than the central chemoreceptor response. The hypoxic response (response to low PO_2) is small and is not really apparent until the patient is severely hypoxaemic.

Normal $PaCO_2$ in the conscious healthy patient is between 35-45mmHg- this can be considered as normocapnia. Normocapnia is maintained by minute ventilation (minute volume) and thus by the respiratory frequency and tidal volume of the patient. A number of patient or disease factors can lead to hypoventilation (elevated PCO_2 or hypercapnia) in the conscious patient:

- neurological disorders (such as brain, brainstem, cervical spinal and neuromuscular disease)
- obstructive upper airway disease (BOAS, laryngeal paralysis etc)
- pleural space disease (pneumothorax, effusions, diaphragmatic herniation)
- severe pulmonary disease

These factors will also cause hypoventilation in an anaesthetised patient but even a healthy patient will hypoventilate to a certain degree under anaesthesia because almost all anaesthetic agents have respiratory depressant effects. This is likely due to depression of the respiratory centres via the $GABA_A$ receptor effects of most anaesthetic agents ($GABA_A$ being the main inhibitory neurotransmitter in the CNS). These effects are generally dose dependant and can compromise the animal's ability to adequately ventilate their lungs leading to an increase in PCO_2. Basically they increase the level of PCO_2 that needs to be reached before a breath is taken and reduce the muscular effort of breathing. This decreases respiratory frequency and tidal volume and thus minute volume. Pain, stress, hyperthermia, light plane of anaesthesia can cause hyperventilation. NB remember that having a high respiratory frequency does not necessarily mean the patient is hyperventilating as the tidal volume may be very small.

NORMAL VENTILATION

During normal ventilation the chest wall expands and the diaphragm contracts and draws back increasing the volume of the thoracic cavity. This produces negative pressure within the chest, most importantly the airways and alveoli. This produces a pressure gradient down which gas travels from the atmosphere into the airways and down to the alveoli, which are able to expand and fill with gas.

During expiration the chest wall and diaphragm relaxes, the chest wall returns to its neutral position through elastic recoil of the thoracic wall and diaphragm and due to the action of surfactant within the alveoli. The increase in intra-thoracic pressure that occurs expels gas from the lungs.

MONITORING VENTILATION

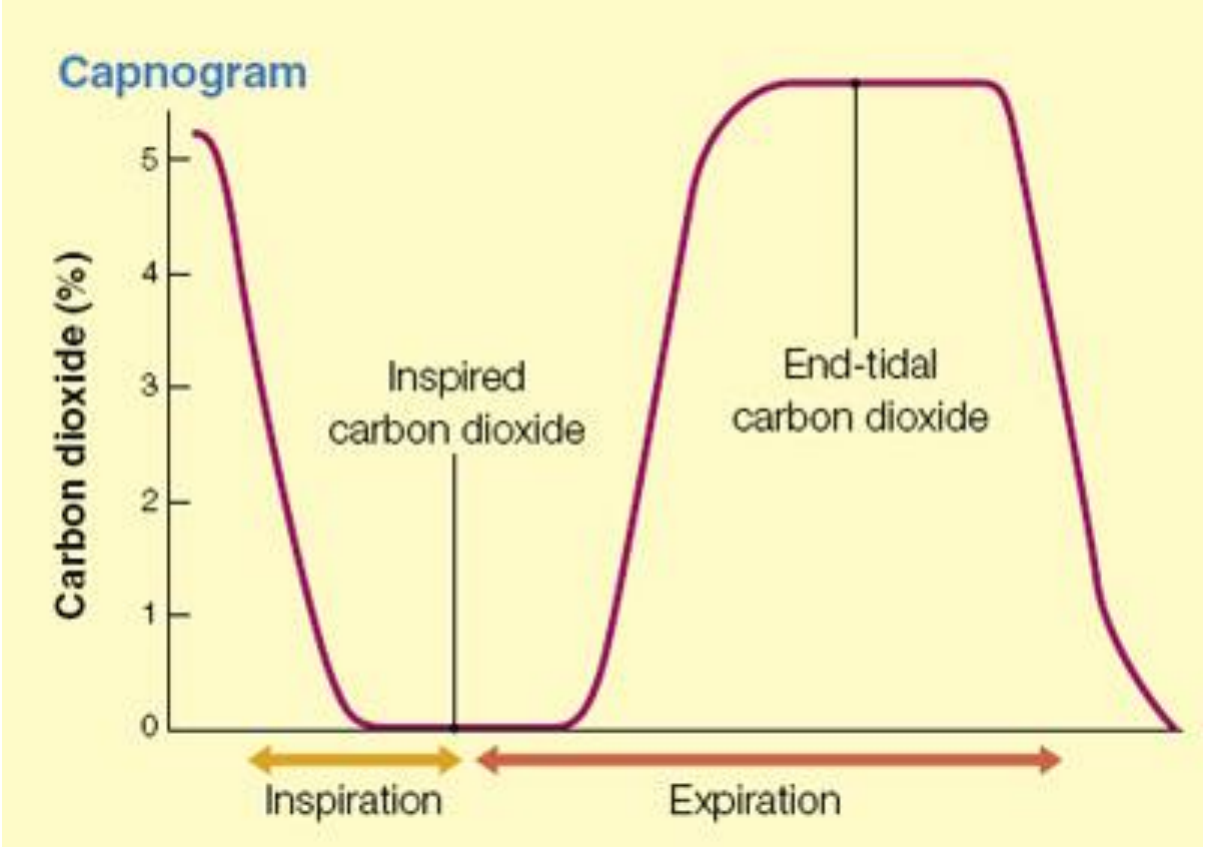
As CO_2 diffuses so rapidly from the blood into the alveoli it can be used as a measure of the adequacy of alveolar ventilation. To get a true indication of how well an animal is ventilating an arterial blood gas is required for the measurement of the partial pressure of arterial CO_2 ($PaCO_2$). However arterial CO_2 levels are approximately equal to alveolar CO_2 levels which can be more easily measured. Alveolar gases are the last gases to be expelled during expiration and therefore end tidal CO_2 ($ETCO_2$) monitoring via capnography will give a breath to breath approximation of $PaCO_2$ and therefore the adequacy of ventilation. Hypoventilation can be classified as $PaCO_2$ (or $ETCO_2$) over 45mmHg and some anaesthetists would instigate intermittent positive pressure ventilation (IPPV) for any patient whose $PaCO_2$ rose above this level. Other anaesthetists would argue that the adverse effects of IPPV are worse than the adverse effects of mild hypoventilation, and the subsequent hypercapnia, so would instigate IPPV at a $PaCO_2$ (or $ETCO_2$) of 50-55mmHg. This is termed permissive hypercapnia.

CAPNOGRAPHY

The capnograph measures end tidal carbon dioxide concentration throughout the respiratory cycle. This can give us an indirect estimation of ventilation status. $ETCO_2$ can give us an assessment of $PaCO_2$ which is usually ~ 5mmHg higher than $ETCO_2$ 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 $PaCO_2$ with the $ETCO_2$ to see how well they correlate. The $ETCO_2$ can then be monitored for trends and an estimated $PaCO_2$ calculated.

Normal Values are 35-45mmHg. $PaCO_2$>60mmHg is suggestive of excessive respiratory acidosis and these patients should almost certainly be ventilated.

Capnography has typically only been utilised in intubated patients or in patients with tracheostomy tubes. But recently nasal $ETCO_2$ taken from a sidestream capnograph has been shown to be a reasonable estimator of $PaCO_2$. Nasal $ETCO_2$ is normally lower than $PaCO_2$ due to dilution by atmospheric gases. Nasal $ETCO_2$ can be taken via nasal prongs or from a nasal catheter.



The normal capnograph trace – Anaesthesia UK©

INTERMITTENT POSITIVE PRESSURE VENTILATION

Intermittent positive pressure ventilation (mechanical ventilation or controlled ventilation) is the act of applying a positive pressure into a breathing system in order to produce a pressure gradient into the chest. As the pressure in the breathing system is greater than that in the chest gas flows into the chest and the lungs expand. When this pressure is ceased expiration occurs in the normal manner. Pressure in a breathing system can be elevated in a number of ways:

- Closing the expiratory APL valve and squeezing the rebreathing bag (manual ventilation)
- Intermittently occluding the exhaust from circuit allowing the pressure of fresh gas flow to inflate the lungs. Known as artificial or mechanical thumbs (Vetronic services ventilator, Penlon with paediatric or Newton valve)
- Increasing the pressure within an air-tight canister that contains a gas filled bellows. The bellows is attached to the breathing system and when the pressure in the canister is increased the bellows empty- in effect this is squeezing the bag hence the name “bag squeezers” (Hallowell)
- Providing a flow of gas into the expiratory limb of the circuit (Penlon, Pneupac Ventipac with patient valve)
- Taking a volume of gas from the anaesthetic machine and driving or squeezing it into the patient (minute volume dividers such as Manley ventilators or electronic ventilators such as the MERLIN)

IPPV is not a benign act. Increasing intra-thoracic pressure during inspiration can decrease venous return to the heart and therefore have detrimental effects on cardiac output. It is not unusual to detect a transient decrease in pulse profile amplitude immediately after inspiration. This can be often be detected audibly on a Doppler flow detector or visually on a pulse oximeter’s plethysmograph (pulse trace) or on a direct arterial blood pressure trace. Changes are often small within the range of pressures and inspiratory times generally used for IPPV but the effects may become clinically significant the higher the pressure and the longer the inspiratory time. The cardiovascular effects of IPPV can be exaggerated by hypovolaemia or in animals with cardiac disease. Consequently the pressure which IPPV exerts should be carefully controlled.

In addition to this over inflation of the lungs during IPPV can:

- Cause bradycardia secondary to a reflex mediated by pulmonary stretch receptors and the vagus nerve.
- Lead to barotraumas. The lung injury caused can lead to pulmonary oedema and other adverse pulmonary events (probably pressures above 30cmH_2O)
- Cause uneven ventilation in the lungs which can affect pulmonary function especially in animals with pre-existing pulmonary disease
- hyperventilate patients if not monitoring $ETCO_2$

IPPV is indicated when the patient's spontaneous ventilation is inadequate to maintain normocapnia or if there is ineffective gas exchange in the lungs. There are a number of patient and anaesthetic factors for which IPPV is always required and some where IPPV is recommended or advisable.

Absolute indications for IPPV:

- Diaphragmatic rupture
- Open chest
- Neuromuscular blockade
- Raised intracranial pressure
- Respiratory arrest

Relative indications for IPPV:

- Increased pressure on diaphragm secondary to abdominal enlargement (obesity, GDV, insufflation of abdomen during laparoscopy, ascites and abdominal effusions, horses and other large animals in dorsal recumbency)
- Debilitated animals (muscle weakness)
- Long duration anaesthesia
- When using potent respiratory depressants e.g. fentanyl
- Animals breathing erratically
- Hypoventilation

Other advantages of IPPV

- More accurate control of respiratory variables
- Constant arterial gas tensions create stable plasma pH and potassium concentrations
- Regular rhythm depresses ventilation, augments narcosis and improves operating conditions
- Mechanical ventilator frees anaesthetist (or nurse) for other duties

Generally IPPV will be performed at a tidal volume of between 10-15ml/kg, a pressure of 10-20cmH_2O and a respiratory rate that maintains $PaCO_2$ (or $ETCO_2$) at a set level. This level is normally within the normal range or within the range of permissive hypercapnia that the anaesthetist has set. In patients with brain disease, especially if raised intracranial pressure is suspected, patient's can benefit from being ventilated more than this. Increasing $PaCO_2$ levels increase cerebral blood flow and therefore can further increase intracranial pressure. In these circumstances the current recommendations are to ventilate the patient to a $PaCO_2$ (or $ETCO_2$) level of 35mmHg. Short term hyperventilation can be used to further decrease intracranial pressure in the emergency situation but long term hyperventilation is not advisable as it reduces cerebral perfusion to a level that can lead to cerebral hypoxia.

HAND VENTILATION

The simplest way of achieving positive pressure ventilation is by intermittent, controlled squeezing of the reservoir bag against a closed or semi-closed APL valve. This is labour intensive and it is difficult to keep consistent inspiratory pressures and volumes for long periods. Only certain breathing systems are suitable for long term IPPV; the T-piece, the Bain, and the circle (those with the bag on the expiratory limb). Other breathing systems such as the Magill and the Lack can be used in the short term with increased fresh gas flow but should be changed for a more suitable circuit for longer term IPPV. The reason for this is that these breathing systems (those with the reservoir bag on the inspiratory limb) tend to cause rebreathing of expired air.

MECHANICAL VENTILATION

As already discussed, in order for a ventilator to cause inspiration and then allow expiration it must intermittently force air into the lungs down a positive pressure gradient. Different types of ventilator perform this task in different ways; this helps classify the different types of ventilator.

- The first way of classification is by describing how the flow of gas is delivered. Most ventilators are volume controlled or pressure controlled. Volume controlled ventilators provide a constant flow of gas during inspiration. Pressure controlled ventilators provide a constant pressure of gas during inspiration.
- The second way of classifying ventilators is by how the ventilator switches from providing gas in inspiration to not providing gas in expiration. Ventilators can be time cycled, pressure cycled, or volume cycled. Time cycled ventilators switch from inspiration to expiration after a set time. Pressure cycled ventilators switch from inspiration to expiration once a set pressure is met. Volume cycled ventilators change from inspiration to expiration once a set volume is met.

Monitoring Oxygenation

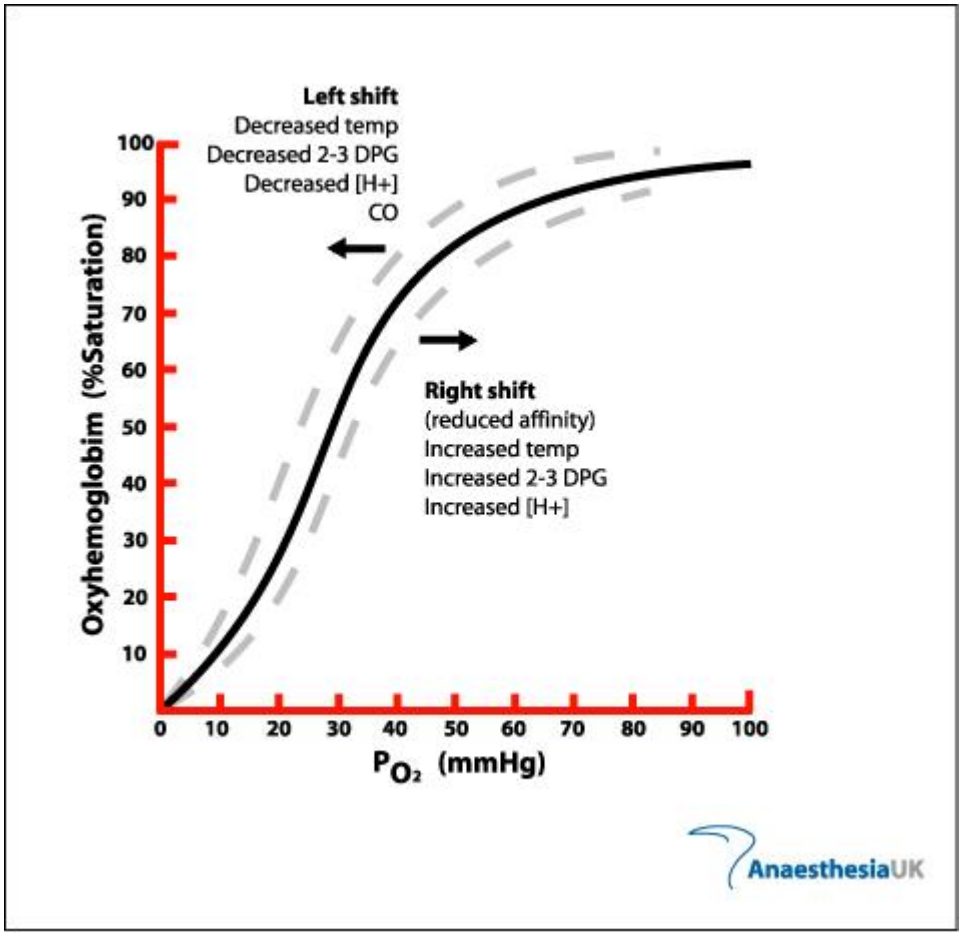
Pulse Oximetry

This can be used to determine the SpO_2. In a healthy patient breathing room air the haemoglobin should be >95% saturated with oxygen. We can expect the anaesthetised healthy patient breathing 100% oxygen to have a SpO_2 reading closer to 100%. The pulse oximeter is a monitor that mainly becomes useful in the recovery stages of anaesthesia and later in intensive care.

The relationship between PaO_2 and SpO_2 forms a sigmoid curve (see diagram) meaning that below 93% SpO_2 a small decrease in SPO_2 will result in a large decrease in PaO_2. Clinically however below a SpO2 of 90% the curve is roughly linear so:

PaO2 mmHg = SpO_2 (if <90%) - 30

Therefore pulse oximeter readings of 93% or higher are acceptable in non-anaemic critically ill patients breathing room air. But as 90% SpO_2 correlates to a PaO_2 of 60-70mmHg (severe hypoxaemia) oxygen should be supplemented where readings on the pulse oximeter are less than 93% and the cause investigated and treated. 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.



The oxyhaemoglobin dissociation curve - Anaesthesia UK©

Pulse oximetry 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. Oxyhaemoblobin and deoxyhaemoglobin give different light wavelengths which allow the microprocessor to detect the saturation. Pulse oximeters cannot distinguish dysfunctional haemoglobin such as methaemoglobin or carboxyhaemoglobin.

Pulse oximetry can work by detecting absorbency from light that travels through a tissue to a sensor (transmittance) or by measuring absorbance of light that is refracted or reflected back to a sensor (reflectance). Both transmittance and reflectance systems are now available for use in veterinary medicine. It is likely that now reflectance probes are becoming more widely available that pulse oximetry will become more and more useful in conscious patients. “Reflectance” pulse oximetry allows SpO_2 readings to be taken from places that would not be possible when using traditional finger/ear lobe probes meaning the probes are better tolerated by patients. Continuous pulse oximetry for conscious patients is likely to be possible within the near future.

Probes can be placed on various sites including the tongue, pinna, lip, toe web and tail, it is 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 post anaesthesia.

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 haemoglobin 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%.

It should also be noted that the pulse oximeter gives us no indication of the oxygen content of the blood, the amount of oxygen dissolved in the blood (PaO_2), ventilation, cardiac output or blood pressure.

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 FiO2 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 $PaCO_2$ then either alveolar ventilation is increased or there is a decrease in 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 (see vascular access). 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 palpated 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 $PaCO_2$ will be decreased and the 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 $PaCO_2$.
- Samples not run immediately or held on ice and run within 2 hours will show changes to pH, PaO2 and $PaCO_2$ due to cellular metabolism.

Venous Blood Gases

More easily obtained than arterial blood gases. Useful for the assessment of ventilation the PCO_2 of venous blood is usually 4-6mmHg higher than that of arterial blood. Venous PO_2 values are not representative of arterial oxygen values however a venous PO_2 of less than 30mmHg may suggest poor tissue oxygenation and should be investigated.