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When Things Go Wrong - Prevention and Management of Complications and Emergencies in Anaesthesia Mini Series

Session 2: Anaesthetic Complications - Things That Go Wrong and How We Deal With Them

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Equipment failure & physiological complications

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Equipment failure and error

True equipment failure is relatively rare – usually human error that causes the equipment failure.

Inability to deliver an adequate oxygen supply:

- Lack of oxygen in the cylinders
- Disconnection of piped oxygen supply
- Stuck or missing one way valve
- Leaks in the anaesthetic machine/breathing system
- Ventilator failure.

Check anaesthetic machine & breathing system thoroughly prior to use – use a checklist such as:

- Connect any electrical equipment to the mains
- Check oxygen supplies, make sure adequate back up supplies are readily available
- Check oxygen alarm
- Check flow meters
- Check vaporiser is mounted properly on back bar
- Check level of anaesthetic agent in vaporiser
- Attach breathing system to common gas outlet and to scavenging system
- Check breathing system, anaesthetic machine and vaporiser for leaks
- Check APL valve (ensure that the valve is left open)
- Check scavenging system (weigh Cardiff aldosorbers, check airbrake working etc.)
- Check monitoring equipment

Exhaustion of carbon dioxide absorber

Exhausted CO_2 absorbent will change colour but if the absorbent is not changed quickly and left within the canister, it will gradually return to its original color. This gives the appearance that it is still fully active however when exposed to CO_2 again the soda lime will rapidly change back to its used colour. This can result in the patient becoming hypercarbic due to CO_2 not being removed from the breathing system if the rapid colour change is not noted. This is easily picked up on a capnograph but if one is not available the patient may show signs of tachycardia and ventricular arrhythmias.

Endotracheal (ET) tube complications

ET tubes may become blocked prior to or during the anaesthesia. All ET tubes should be checked thoroughly (tube patency and cuff inflation) prior to use and then monitored carefully when in situ. Special care should be taken during procedures around the head and neck area to ensure that the ET tube does not become kinked or twisted. Blood, mucus, saliva, etc., may block the tube whilst in situ.

The end of an ET tube can also be blocked by it coming into contact with the tracheal wall, this is especially important to realise if the tube has no Murphy's eye. Regardless of the cause the blockage it must be quickly rectified. A complete blockage of the patient's ET tube will cause them to become dyspneic, hypoxic, hypercarbic and may progress to respiratory arrest if not recognized and dealt with rapidly. Re-intubation may be required or the solution may be as simple as repositioning the patient's head or neck to rectify a kink in the tube or suctioning the lumen to remove debris. Bronchial intubation is another possible complication (using a tube that is to long) and can cause severe respiratory compromise but can easily be avoided by ensuring that the tube used is of an appropriate length! Care should also be taken when inflating the ET tube cuff as damage to the trachea is possible especially in cats.

Adjustable pressure limiting (APL or 'pop off') valves

These can easily be accidentally left in the closed position either at the start of anesthesia where the breathing system has not been adequately checked or where the valve is left in the closed position after pressure checking or during intermittent positive pressure ventilation (IPPV). The closure of these valves causes the pressure in the breathing system to quickly build up. In some breathing systems (i.e. intersurgical disposable Bain and T-piece systems) there is a patient safety valve which allows the pressure to be released once a set limit has been reached i.e. 30-60cm H_2O (depending on the valve and flow rate). Some veterinary breathing systems do not incorporate this safety feature i.e. the mini parallel lack. This pressure causes over inflation of the reservoir bag and consequently the patient's lungs. This can cause barotrauma, prevent expiration and lead to pneumothorax, bradycardia and reduction in venous return in turn impacting on cardiac output and blood pressure. This complication can lead to respiratory arrest and then to fatality in a relatively short period of time. Treatment if this is noticed quickly is generally as simple as opening the valve. Atropine may be required to treat any bradycardia if it does not correct once intrathoracic pressure has dropped.

Vaporiser complications

- Filling the vaporiser with the wrong anesthetic agent – not so common with newer key fill vaporisers such as the TEC 4
- Sticking of the dial - previously associated with the thymol preservative in halothane
- Under filling, overfilling and tilting of the vaporizer (more of a problem with older techs) - latter two will result in the patient receiving an overdose of inhalant agent regardless of the percentage on the dial whilst under filling will cause the patient to not receive any anesthetic agent and potentially wake up.
- Incorrectly seated and locked onto the back bar of the anesthetic machine - may not be immediately obvious but will cause the patient to become lighter and lighter despite turning the percentage up on the vaporiser dial. The patient will wake up if this error is not recognized and rectified quickly.

Patient Related Anaesthetic Complications

Assessment Method	What can we assess?	Parameters recorded
Observation/ see	Reservoir bag, chest wall movement, respiratory rate and effort, mucous membrane colour, eye position, patient movement.	Respiratory rate, effort and pattern MM colour Depth of anaesthesia
Palpation/ touch	Pulse rate and quality, capillary refill time, palpebral & pedal reflexes, jaw tone, muscle relaxation.	Pulse rate and quality Capillary refill time Depth of Anaesthesia
Auscultation/ hear	Heart rate and rhythm, abnormal heart sounds, lung sounds, leaks in the breathing system, incomplete ET tube seal, leaks in the anaesthetic machine.	Heart rate and rhythm Abnormal heart and lung sounds Equipment failure

Respiratory System

Regurgitation & Aspiration

Aspiration can occur following vomiting or regurgitation of stomach or oesophageal contents or due to aspiration of saliva, blood or mucus caused as a consequence of either the anaesthetic, surgical procedure or disease process.

Particularly hazardous during induction and recovery as the patient's airway is not protected. Aspiration, especially following regurgitation, may be a silent process and probably occurs more often than is realized.

Initial problem caused by aspiration is airway obstruction - dyspnoea, cyanosis and bronchospasm. Aspiration pneumonia may develop, signs include dyspnea, tachypnea, cough, pyrexia, and increased lung sounds and will require treatment but this is a more gradual process as it develops usually 1-2 days post anaesthesia.

Treatment in the anaesthetized patient - a cuffed endotracheal tube ensures that the risk is relatively small, the patient should be placed with the head lower than the body and the oral cavity may need to be lavaged and suctioned.

In the unconscious patient without a protected airway, intubation should be attempted. If this is not possible the patient should be positioned as before to allowing drainage of fluid from the oral cavity and suction should be utilized.

The conscious patient that has the ability to swallow should have the ability to protect its own airway and the anaesthetist should just ensure that the head and neck are extended with the head lowered.

Apnoea/ Respiratory Arrest

Commonly seen at induction when drugs such as propofol are given too rapidly - may also be caused by an overdose of anaesthetic drugs. Following induction breath holding may also be observed due to an inadequate depth of anaesthesia.

Other causes of respiratory arrest:

- Brainstem injury in head trauma patients
- Hypoxia
- Severe pulmonary disease
- Cardiac arrest
- Iatrogenic hyperventilation of the patient
- Administration of NMBAs
- Neuromuscular diseases
- Equipment failure (i.e. APL valve left closed).

Where a drug has been administered too quickly the apnea is often transient and spontaneous respiration will return as the initial effect of the drug wears off. SpO_2 and cardiovascular monitoring of the patient through periods of apnea is required to ensure that haemoglobin desaturation and cardiovascular complications do not occur. If the apnea is prolonged and SpO_2 begins to fall then IPPV can be initiated. IPPV must be initiated when apnea is secondary to administration of NMBAs. Treatment of the underlying cause must be established. Anaesthetic depth should be evaluated and adjusted accordingly. In the case of respiratory arrest then resuscitation should be commenced.

Airway Obstruction

Most likely to occur in the pre-anaesthetic period or during recovery when the patient does not have a protected airway.

Signs of airway obstruction include: inspiratory stridor, dyspnea, paradoxical thoracic wall movement (full airway obstruction), increased respiratory effort with prolonged inspiratory time, little or no movement of the reservoir bag (if anaesthetised) and cyanotic mucous membranes.

Causes of upper airway obstruction may include:

- Soft tissue entrapment – common in brachycephalic patients
- Laryngeal spasm
- Laryngeal paralysis
- Laryngeal edema (often caused by trauma due to intubation)
- Laryngeal or tracheal collapse
- Foreign material i.e. blood, mucus, debris, gastric and oesophageal contents
- Mechanical obstruction i.e. kinked or blocked ET tube, endobronchial intubation or a head or neck bandage that is too tight.

Causes of lower airway obstruction may include:

- Anaphylactic reaction
- Bronchospasm
- Bronchitis
- Asthma
- Chronic obstructive pulmonary disease
- Tumour
- Foreign body

Treatment:

- Depends on the underlying cause
- Upper airway obstruction - extend the head and neck and pull the tongue forward
- Supplemental O_2
- Rapid intubation or even emergency tracheostomy may be necessary
- Further treatment, especially for lower airway obstruction, may be required - IPPV and/or drug administration e.g. steroids, antihistamines or bronchodilators.

Planning for the recovery of patients with obstructive airway disease must be thorough and include having essential equipment close to hand e.g. laryngoscope, ET tubes, induction drugs, tracheostomy tubes. Brachycephalic and other at risk patients should be recovered in sternal recumbency with the head and neck extended and tongue pulled forward.

Laryngospasm

Occurs in cats when the laryngeal tissues are irritated during intubation - results in airway obstruction due to reflex closure of the laryngeal cartilages.

Prevention:

- Spray the larynx with local anaesthetic prior to intubation
- Ensuring adequate anaesthetic depth before attempting intubation
- Use a gentle intubation technique.

Treatment:

- Delay further attempts at intubation
- Provide supplemental O_2
- Spray local anaesthetic onto the larynx
- Deepen plane of anaesthesia

This may be enough to encourage the laryngeal cartilages to relax and allow intubation. If this does not work a NMBA could be considered to paralyze the larynx however the patient will need rapid intubation and ventilation. In severe cases a needle can be placed into the trachea, percutaneously, to provide oxygen or an emergency tracheostomy may need to be performed. Intubation should never be forced as damage to the larynx can result in oedema and may lead to upper airway obstruction when the patient is extubated.

Hypoventilation

Hypoventilation - reduced minute volume due to a reduction in tidal volume/respiratory rate. Leads to hypercarbia - an increased level of CO_2 in blood and can simultaneously cause hypoxaemia. Hypercarbia will cause depression of the central nervous system (CNS) if left untreated. Retention of CO_2 causes respiratory acidosis and acidaemia.

Causes of hypoventilation may include:

- Overdose of anaesthetic agents i.e. 'too deep'. Anaesthetic agents depress the respiratory centers in the brain and therefore the normal ventilatory response to hypercarbia, hypoxia and acidosis.
- Pleural space disease e.g. pleural effusion.
- Pulmonary disease e.g. pulmonary edema, pneumonia
- Accidental endobronchial intubation
- Abdominal distension increasing pressure on the diaphragm
- Diaphragmatic hernia/ rupture
- Severe hypotension leading to reduced cerebral perfusion
- Hypothermia depressing the respiratory centers
- Decrease in functional residual capacity during anaesthesia can lower alveolar ventilation: perfusion ratios and result in ventilation: perfusion mismatch, it can also expand atelectatic areas which can increase intrapulmonary shunting
- Cervical disease where compression of the spinal cord at the level of the phrenic nerve will compromise respiration
- Paralysis of the muscles of respiration by NMBAs or neuromuscular disease
- Restrictive chest bandages or pain following thoracotomy

Treatment:

- Reduction in anaesthetic depth
- Reversal of drugs contributing to hypoventilation (e.g. NMBA)
- IPPV
- Thoracocentesis
- Treatment of underlying causes (i.e. abdominal distension, hypotension, hypothermia)
- Analgesia
- Check equipment – exhausted soda lime, one way valves
- Supplemental oxygen

- Sternal recumbency - minimises effects of atelectasis, ventilation perfusion inequality and intrapulmonary shunting.
- Check thoracic bandages

Hyperventilation

Hyperventilation - an increase in minute volume due to an increase in tidal volume/respiratory rate.

Common causes:

- Pain
- Inadequate anaesthetic depth (i.e. 'too light')
- Surgical stimulation
- Overzealous ventilation
- Hypoxia
- Hypotension
- Pyrexia
- Hyperthermia.

Hyperventilation causes a decrease in $PaCO_2$ and results in a drop in hydrogen ions; this causes respiratory alkalosis and the patient to become alkalemic.

Treatment:

- Increase anaesthetic depth
- Analgesia
- reduce IPPV (rate and/or tidal volume)
- Treatment for hypoxia, hypotension, pyrexia and hyperthermia if present

Monitoring Ventilation

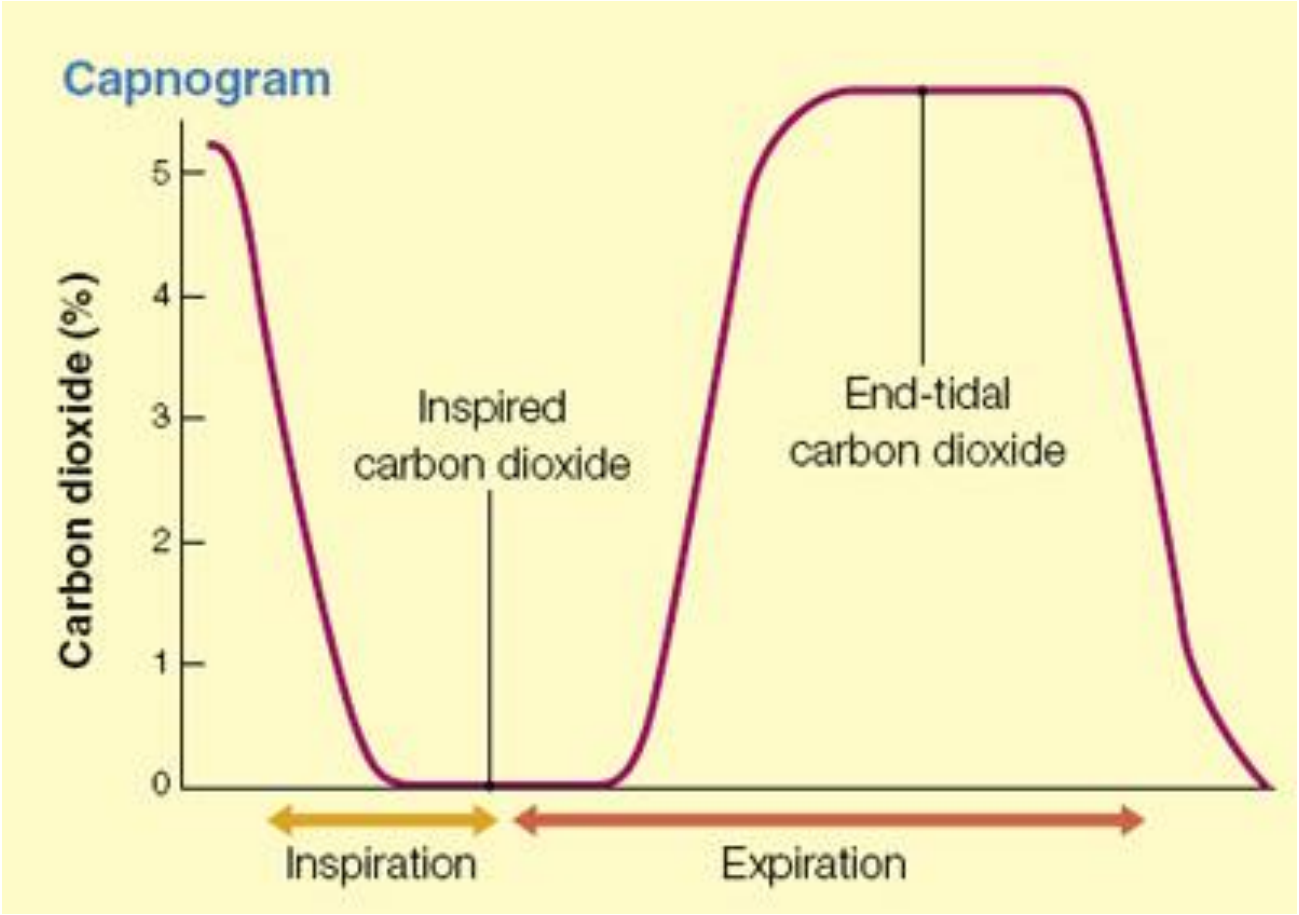
As CO_2 diffuses so rapidly from the blood into the alveoli it can be used as a measure of the adequacy of alveolar ventilation. To get a true indication of how well an animal is ventilating an arterial blood gas is required for the measurement of the partial pressure of arterial CO_2 ($PaCO_2$) (see below). 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 (IPPV)

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 intrathoracic 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

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, patients 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.

Hypoxaemia and hypoxia

Hypoxaemia - low oxygen content in arterial blood 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 edema/pus in alveoli)
- Ventilation: perfusion inequality

- Intrapulmonary shunt (causing blood to pass through the lungs without undergoing gaseous exchange) (or right to left cardiac shunting)

Decreases in SaO_2 are caused by:

- Decreases in PaO_2
- The formation of methaemoglobinaemia or carboxyhaemoglobinaemia

Decreases in haemoglobin concentration are caused by anaemia and haemorrhage.

Hypoxia - 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.

Treatment:

- O_2 supplementation
- IPPV
- Where possible treatment of underlying disease i.e. treatment for hypoventilation, administration of packed red blood cells for anaemia et cetera.

Monitoring Oxygenation

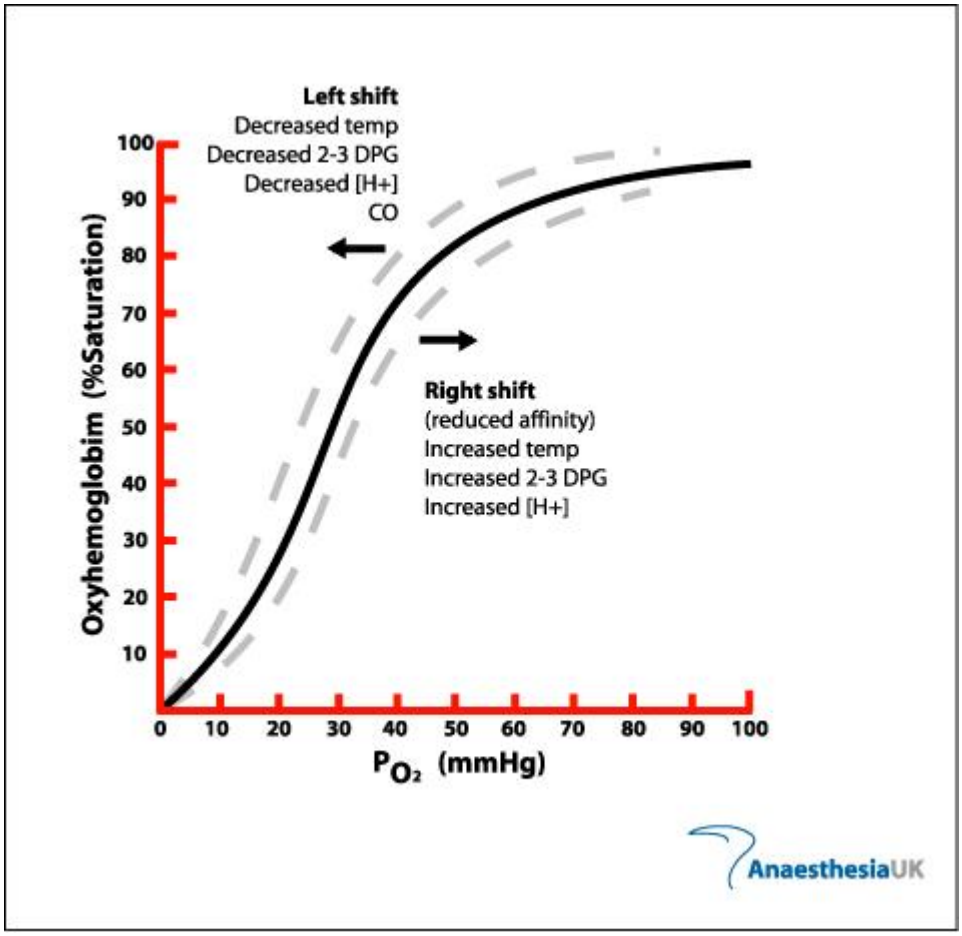
Pulse Oximetry

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.

Cardiovascular System

Hypotension

Hypotension - a mean arterial blood pressure of less than 60mmHg in small animals. A pressure of greater than this is required to ensure adequate perfusion and oxygen delivery to the brain, heart and kidneys. Hypotension can lead to signs of shock, ischemia of vital organs resulting in organ dysfunction and ultimately organ failure.

Causes in anaesthetised patients include:

- Overdose of anaesthetic agents (i.e. ‘too deep’)
- Relative or absolute hypovolemia (hemorrhage, fluid loss, sepsis, systemic inflammatory response syndrome [SIRS])
- Drug effects.

There are three main mechanisms that cause hypotension:

- Decreased cardiac output which can be due to myocardial depression, cardiac arrhythmias or decreased venous return.
- Reduced systemic vascular resistance due to vasodilation. Vasodilation is commonly caused by many anaesthetic drugs including barbiturates, propofol and all the volatile agents. Vasodilation is also present in patients with SIRS and sepsis.
- Hypovolaemia – this can be relative due to vasodilation or can be absolute caused by factors such as hemorrhage, existing fluid deficits, third spacing of fluid.

Treatment:

- Determine the underlying cause
- Reduce the amount of injectable/inhalational anaesthetic agents administered
- Volume restoration to correct hypovolemia
- Rectify oxygenation/ventilation problems to correct hypoxia/hypercarbia,
- Correction of acid-base and electrolyte abnormalities
- Treatment of arrhythmias
- Control of haemorrhage
- Cardiac support (dobutamine, dopamine) if necessary and vasopressor (dopamine, phenylephrine) therapy.

Hypertension

Causes:

- Inadequate depth of anaesthesia
- Pain
- Hypercarbia
- Hypoxia
- Fever
- Metabolic acidosis
- Drug effects (catecholamines, ketamine)
- Systemic hypertension secondary to metabolic disease
- Secondary to raises in intracranial pressure (Cushing reflex)

Treatment:

- Identify the underlying cause and correcting this where possible i.e. administering analgesia, increasing depth of anaesthesia, IPPV treatment of intracranial pressure et cetera
- Where treatment of the underlying cause is not possible it may be necessary to administer drug therapy this may include beta blockers or calcium channel blockers.

Haemorrhage

Blood loss quickly builds up especially in smaller patients. Try to estimate blood loss where you can. Techniques include weighing and count swabs and checking the volumes in any suction bottles (remember to minus any flush used). Treatment requires replacement of the lost blood with fluid therapy. Crystalloids such as Hartmann's (lactated Ringers solution or compound sodium lactate) or 0.9% NaCl (normal saline) don't stay in the circulation for long tending to redistribute from the intravascular space into the interstitial space. Books tend to describe giving 2-3 times the volume of crystalloid as compared to blood lost whereas an equal volume of colloid is generally required. If estimation of blood loss is not possible then administration of 10ml/kg boluses of crystalloid or 2ml/kg of colloid can be administered until the patients cardiovascular parameters are returned to normal.

0-10% blood volume minimal blood loss

10-20% blood volume moderate blood loss

20-30% blood volume severe blood loss

>30% blood volume extreme blood loss

Remember blood volume is 90ml/kg in a dog and 60-70ml/kg in a cat

Colloids and blood transfusions should be considered as blood loss becomes more severe but up to 30-40% blood loss can probably be treated appropriately with isotonic crystalloids if necessary.

Bradycardia

If the heart rate becomes too slow i.e. bradycardia, cardiac output (CO) is reduced and hypotension may result. This may occur at heart rates below 40-60 beats per min (bpm) but this depends on the size of the animal. Bradycardia may be caused by:

- Excessive anesthetic depth
- Drug induced (α_2 agonists, μ opioids, anticholinesterases)
- Increased vagal tone & vagal stimulation during surgery
- Severe hypoxia
- Pre-existing heart disease
- Hyperkalemia
- Intracranial disease causing an increase in intracranial pressure
- Hypothermia
- Severe systemic hypertension.

The bradyarrhythmias commonly seen under anaesthesia are:

- Sinus bradycardia - < 100bpm in cat or 60-70bpm in dog
- Atrioventricular (AV) block (1^{st}, 2^{nd} and 3^{rd} degree) – occurs when there is a disruption in conduction between the Sinoatrial (SA) node and the ventricles.
    - 1^{st} degree AV block – prolonged P-R interval – treatment often not necessary but monitor for deterioration.
    - 2^{nd} degree AV block – P waves that lack QRS complexes
    - 3^{rd} degree AV block – P-wave and QRS complex occur completely independent of each other.
- Sinus arrest
- Atrial standstill

Bradycardia may not need to be treated in all cases if other cardiovascular parameters such as blood pressure are acceptable. Many bradyarrhythmias can initially be treated by administration of an anticholinergic (atropine/glycopyrolate) although this will not work in some cases i.e. where the

bradycardia is caused by hypothermia or 3rd degree AV block– sometimes the problem can worsen before it gets better. The underlying cause should be corrected (warm the hypothermic patient, reverse opioids with naloxone etc.). Anticholinergics are contraindicated if bradycardia is caused by α_2 agonist administration, hyperkalemia or hypertension (such as with raised intracranial pressure). Where bradycardia is caused by α_2 agonist's reversal can be achieved by the administration of atipamezole if required. Hyperkalemic patients can have their myocardium stabilised with calcium gluconate and the hyperkalaemia treated with fluid therapy, dextrose and insulin administration or potentially sodium bicarbonate. Patients with increased intracranial pressure may require treatment e.g. mannitol.

Tachycardia

If the heart rate becomes too fast i.e. tachycardia (>180bpm in dogs and >240bpm in cats) the heart does not have time to adequately fill and cardiac output is also reduced. As the heart has a short diastolic filling time coronary artery perfusion is also affected and if left untreated can result in cardiac dysfunction. Tachycardia may be caused by:

- Inadequate anaesthetic depth
- Hypovolaemia
- Hypotension
- Pain
- Hypoxaemia
- Hypercapnia
- Hyperthermia
- Sepsis
- Drugs (anticholinergics, ketamine, thiobarbituates, sypathomimetics, pancuronium)
- Hyperthyroidism
- Pheochromocytoma
- Anaemia
- Shock
- Heart disease
- Hypokalaemia and other electrolyte abnormalities

Supraventricular tachyarrythmias

- Sinus tachycardia - associated with systemic abnormalities such as hypotension, hypovolemia or hypoxia and can normally be rectified by correcting the underlying problem. It is also associated with patients in chronic heart failure (CHF).
- Atrial tachycardia
- Atrial fibrillation – Fast irregular rhythm with no discernible P waves and a trembling baseline. Normal QRS.

Supraventricular complexes arise from an ectopic focus above the ventricles. The ventricles will then depolarize in the normal way resulting in a normal QRS complex. Runs of supraventricular complexes are termed supraventricular tachycardia and this can greatly reduce cardiac output and consequently tissue perfusion. Where these arrhythmias are severe and diastolic filling is compromised along with myocardial perfusion and an underlying cause is not obvious it may be necessary to treat with beta blockers (e.g. propanolol, esmolol).

Ventricular arrhythmias

- Ventricular premature complexes (VPCs) – a contraction originating in the ventricle before a contraction is expected i.e. prematurely! Wide and bizarre QRS complex.
- Ventricular tachycardia
- Ventricular fibrillation

These rhythms can be associated with primary cardiac disease or a non-cardiac cause. Common causes include:

- GDV
- Trauma
- Sepsis
- Acid-base disturbances
- Hypercarbia
- Electrolyte imbalances
- Drugs
- Hypoxia and myocardial ischemia
- Splenic, hepatic or atrial haemangiosarcoma
- Pancreatitis
- Traumatic myocarditis.

VPCs arise from an ectopic focus within the ventricles. Depolarization conducts in an abnormal direction across the myocardial cells without using the normal conduction pathways. The complex will appear abnormal, usually wide (due to the prolonged depolarization) and bizarre with the p wave often hidden by the VPC (since the VPC occurs prematurely). Infrequent VPCs will not normally compromise cardiac output and specific treatment is often unnecessary but identification of the underlying cause is appropriate to prevent deterioration. May result in pulse deficits as there is a reduction in ventricular filling.
A run of three or more VPCs is termed ventricular tachycardia and can cause a significant decrease in cardiac output. Ventricular tachycardia may deteriorate into ventricular fibrillation which is an arrest rhythm (see CPCR). Treatment for ventricular tachycardia will include treatment of the underlying cause, supplemental oxygen and may require drug treatment i.e. lidocaine.

It should be considered that many arrhythmias cannot be distinguished without an ECG but that pulse deficits are often indicative of reduced cardiac output due to a cardiac arrhythmia.

Monitoring Arrhythmias

Electrocardiography (ECG)

Electrocardiography (ECG) gives us a trace of the electrical activity of the heart. It does not indicate that there is contractility, cardiac output or perfusion. ECG is helpful to monitor arrhythmias and response to treatment and in patients where arrhythmias may be expected e.g. splenic torsion and GDV. It also gives us a heart rate value. Many of the drugs we use in anaesthesia predispose the patient to cardiac arrhythmias and stress before induction and inadequate oxygenation can often exacerbate this.

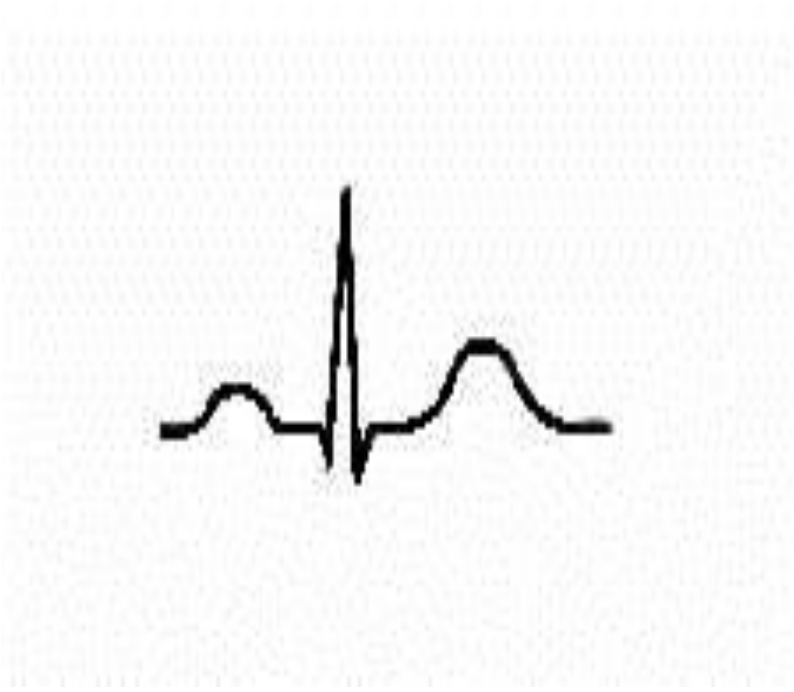
The electrical impulse is normally conducted through the heart as follows:

- The impulse starts in the sinoatrial (SA) node located in the wall of the right atrium.
- The impulse travels across the right and left atria to the atrioventricular node located in the septum between the atria and the ventricles.
- The impulse passes along purkinje fibres located in the ventricular septum and ventricular walls.
- The impulse travels across the ventricles.

An ECG trace of a normal complex should consist of the following:

- P Wave - Atrial depolarization (contraction) where blood is pumped from the atria into the ventricles. P wave should be positive (above the baseline).
- QRS complex – Ventricular depolarization where blood is ejected from the ventricles.
- T Wave – ventricular repolarization (relaxation). T wave can be positive or negative.

Familiarise yourself with what a normal ECG looks like:



You do not necessarily need to be able to name an abnormal rhythm but by learning what is normal you will recognise what is abnormal! Be aware that you may get electrical interference in some cases i.e. diathermy, EMG, clippers, the interference may look like an arrhythmia but will normally disappear when the interference is removed!

ECG lead placement RED: Right Fore, YELLOW: Left fore, GREEN: Left hind, BLACK: Right hind.

Evaluation of the ECG:

What is the rate? (bradycardia, normal, tachycardia)
Is the rhythm regular (normal sinus rhythm), regularly irregular (normal e.g. sinus arrhythmia) or irregularly irregular (abnormal)?
Is the PQRST waveform consistently present with a normal appearance?
Is there a P wave for every QRS?
Is there a QRS for every P?
Are the QRS and the P consistently and reasonably related?
Can you feel a pulse for every complex on the ECG?
Any abnormalities noted on the ECG should be recorded for review by a veterinary surgeon.

By using this simple list of questions you will be able to begin to identify arrhythmias.

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