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Practical Emergency Techniques for Advanced Practitioners Mini Series

Session Two: Respiratory management

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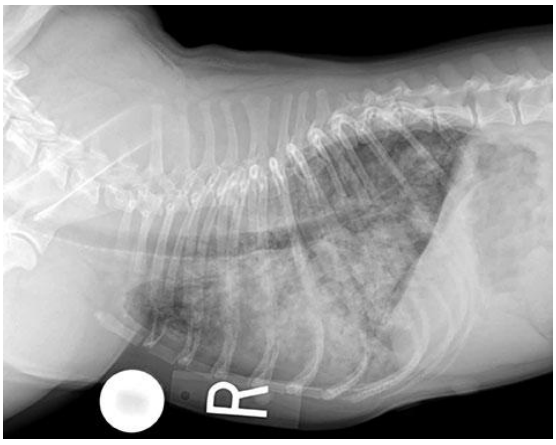

Assessment of the respiratory system

Respiratory distress is a common presenting complaint in emergency medicine and is often life-threatening. These patients can be very unstable at the time of presentation to the clinic and require immediate assessment and stabilisation. Provision of oxygen therapy, as detailed below, in addition to efforts to decrease any patient stress can help improve the balance between oxygen demand and oxygen consumption. Initial patient assessment should consist of observation of the patient's breathing to make note of the respiratory rate and pattern of breathing, such as increases in inspiratory or expiratory effort, or a rapid shallow pattern. Thoracic auscultation, assessing the patient for adventitious lung sounds such as wheezes, crackles or harsh sounds, or dull lung sounds in the case of pleural space disease, can also provide useful information. In addition, any evidence of orthopnoea (postural changes as a response to dyspnoea) should be noted. These may include extension of the neck, flaring of the nostrils, abduction of the elbows, increased abdominal movement, paradoxical abdominal movement, and an anxious facial expression and inability of the patient to settle. Open mouth breathing, positioning in lateral recumbency or a constantly changing body position in cats are seen in cats with the most severe respiratory distress and are often a precursor to respiratory arrest. Taking note of these clinical signs in combination with the patient history and signalment can help to narrow down the origin of the problem to a certain location of the respiratory system in the majority of patients (upper airway, lower airway, pleural space, pulmonary parenchyma, chest wall/diaphragm, abdominal distension or look-a-likes), permitting early appropriate stabilisation attempts.

Feline patients in particular, can be very challenging to manage when they present to the clinic with respiratory distress. A recent study looking at physical examination findings in dyspnoeic cats in a first opinion setting, showed that the presence of a gallop rhythm, rectal temperature < 37.5^{o}C, heart rate above 200 bpm and respiratory rate above 80 bpm were all useful to predict cardiac associated dyspnoea as the most likely cause. The combination of TPR data and the presence of a gallop rhythm was used in this study to produce a triage algorithm which further improved the diagnostic accuracy of these parameters when evaluating patients (JSAP 2018, Dickson et al).

<u>Patient side ultrasound techniques</u>

Any further diagnostic evaluation of unstable respiratory patients should only be performed following consideration of the risk to benefit ratio as they may result in significant increases in patient oxygen consumption and rapid patient deterioration. This challenge in patient management has, in recent times, lead to the development of patient side ultrasound techniques that can be tolerated by this patient population, provide valuable information and be performed easily.

TFAST, or thoracic focused assessment with sonography for trauma, triage (any emergency presentation) and tracking (patient monitoring), is the term used for patient side thoracic ultrasound in emergency medicine. As the term suggests, its use is not confined to any particular clinical presentation, but can be used in the assessment of any emergency patient and subsequent monitoring of patient progress whilst in the hospital. The primary objective of the TFAST examination is to rule in or out the presence of air or fluid in the pleural space, and to rule in or out the presence of fluid in the pericardial space. It may also be used to obtain information on cardiac contractility and patient volume status as detailed in the previous webinar.

An advantage of the technique is that the patient may be positioned in sternal recumbency or in a standing position if preferred by the patient, as an alternative to requiring restraint in lateral recumbency as with thoracic radiography. This means it can be well tolerated by an orthopnoeic patient in which other diagnostics are not tolerated. There is also no requirement to clip the fur. Instead it is recommended that the fur be parted over the area to be examined and either ultrasound gel or alcohol used. Alcohol should not be used if there is any possibility electrical defibrillation will be required later.

The TFAST examination involves 5 views of the thorax. These include bilateral placement of the ultrasound transducer at the chest tube site (CTS) in the longitudinal plane perpendicular to the ribs at the 7-9th intercostal spaces. The purpose of scanning at this site is to rule out pneumothorax, which is done if the real-time dynamic glide sign and/or B lines are present (see below). The transducer must

be held still against the thorax in order to maximise the chance of detecting the glide sign. The probe should be held with the marker directed towards the patient's head.

Bilateral placement of the transducer at the pericardial site (PCS) in both longitudinal and transverse planes with movement and fanning of the transducer is used to maximise the changes of detecting pleural and pericardial effusions. The fifth and final view is the subxiphoid view of the AFAST examination (also known as the diaphragmatico-hepatic view or DH) with the depth set to allow the pleural and pericardial spaces to be evaluated via the acoustic window through the liver, gall bladder and diaphragm.

Whilst pleural effusion may be seen at both the PCS and DH views, checking multiple views is advised to verify the findings. Pleural fluid is not contained, in comparison to pericardial effusion or blood within cardiac chambers. Triangulating fluid in either of these views is consistent with pleural effusion.

TFAST terminology:

- Glide sign: at the chest tube site, the pulmonary-pleural line is visible as a white line running between 2 ribs, usually just distal to the ribs. This combination of findings is also referred to as the 'gator-sign'. To-and-fro motion of the pleural line as the parietal and visceral pleura slide over one another during inspiration and expiration is referred to as the glide sign. This is a key finding that indicates that the parietal and pleural linings are in contact with normal aerated lung. The glide sign is a dynamic finding and seen as horizontal movement or shimmering over the pulmonary-pleural line. Its presence rules out the presence of a pneumothorax at that location. The glide sign may be difficult to see if the ultrasound echoes strike the air interface too directly (for example at 90 degrees). Pivoting the probe dorsally or ventrally so the echoes are interpreted more obliquely by the ultrasound machine can help produce a better image for interpretation. It is also advised that a patient should be observed for the presence of a pneumothorax following a change in position
- A-lines: are horizontal lines of decreasing echogenicity, visible in the far field of the image, similar to and equidistant from the pleural line in the chest tube site view. They should not be confused with the pulmonary-pleural line. They are present as a result of reverberation artefact and may be seen in patients both with and without pneumothorax.
- Lung point: The severity of any pneumothorax present may also be ascertained using the TFAST examination. The ultrasound probe is moved from a dorsal to ventral position whilst the patient is in sternal recumbency, noting the point at which there is no longer air between the chest wall and the lung (return of the glide sign). The point at which the glide sign returns is known as the 'lung point'. In cases of massive pneumothorax, there will be no lung point and no return of the glide sign will be seen as the transducer is moved. The distance of any lung point from the chest tube site can be used to track the worsening or resolution of the pneumothorax during hospital treatment.
- B-lines: are also seen in the chest tube site view. They are hyperechoic vertical lines extending from the pleural line to the edge of the far field image, passing through any A lines without fading. They are reverberation artefacts from the visceral pleura. They move in a to-and-fro fashion with inspiration and expiration synchronously with the glide sign. They are created when small amounts of fluid are next to air in the peripheral lung tissue (usually in the outer 1-3 mm). Whilst occasional B-lines are considered to be normal, excessive B-lines or those occurring closely together are indicative of an interstitial or alveolar lung abnormality. When B-lines are close together they are termed a B-pattern or ultrasound lung rockets. They have previously been referred to as comet tails. When they are seen in a trauma patient they are considered diagnostic for pulmonary contusions. In non-traumatic cases, other causes of pulmonary parenchymal disease should be considered (pneumonia, cardiogenic oedema, non-cardiogenic oedema etc). B-lines are not seen in the presence of a pneumothorax, hence the presence of B-lines can also be used to rule out the presence of a pneumothorax at that site. The term 'wet lung' is also used to describe the lung of a patient with an increased number of B-lines.

Vet BLUE

In the past, there has been a reluctance to use ultrasound for the assessment of the lung owing to the belief that the presence of air provides a barrier to imaging. Recent evidence now suggests that lung ultrasound has a higher sensitivity than lung auscultation and supine thoracic radiography for many

acute and potentially life-threatening conditions in people. Ongoing research into this in veterinary patients would, at the present time, suggest this may also the case in veterinary patients.
Lung ultrasound is based on the observation of ultrasonographic artefacts looking for evidence of 'dry lung' (A-lines with a glide sign), versus 'wet lung' (ultrasound lung rockets or B-lines). Ultrasound does not transmit through aerated lung and the presence of ultrasound lung rockets represents forms of interstitial oedema. Whilst the standard TFAST protocol looks for these artefacts, it is only performed at the 'chest tube site' on each side the thorax.

The Veterinary Bedside Lung Ultrasound Examination (VetBLUE) has been developed to provide a more comprehensive lung ultrasound survey. The VetBLUE examination consists of four bilaterally applied lung views (eight total acoustic windows), referred to as the caudodorsal lung lobe region (Cd, CTS view, upper third, 8-9th intercostal space), the perihilar lung lobe region (Ph, 6-7th intercostal space, middle third), the middle lung lobe region (Md, 4-5th intercostal space, lower third), and the cranial lung lobe region (Cr, 2-3rd intercostal space, lower third). The ultrasound probe is placed horizontally at each site, with the starting point of the caudodorsal lung lobe region, the equivalent of the chest tube site of the TFAST assessment.

Each of these sites is observed for lung ultrasound artefacts using the same wet versus dry lung principal as described above. Each site is given a score based on the number of B-lines seen. The maximum number of ultrasound lung rockets over the respective single intercostal space at each view is recorded. The counting system is as follows: 1, 2, 3, >3 (when the ultrasound lung rockets are still recognised as individuals) and infinity ∞ (when the ultrasound lung rockets blend into one another becoming confluent).

It is important to note that VetBLUE detects peripheral lung changes only, and is therefore not a replacement for other diagnostic testing in all cases. The VetBLUE sites also are regions and do not correlate to a specific lung lobe.

A recent study showed that lung ultrasound using the VetBLUE protocol had good diagnostic accuracy to identify cardiogenic pulmonary oedema in dogs and that it may be helpful in staging dogs with chronic valvular heart disease. Further research into the application of VetBLUE in veterinary patients is needed, but it shows initial promise as a readily available, non-invasive, patient-side diagnostic tool for patients with respiratory dysfunction.

Additional lung ultrasound terminology

- The step sign is caused by a disruption in the normal linear continuity of the pulmonary-pleural lines and is identified by a deviation in the glide sign from its expected course. Its presence indicates there is an abnormality present along the thoracic wall or pleural space. Such conditions include pleural effusion, intercostal tear(s) and rib fracture(s), diaphragmatic hernia, severe left atrial enlargement, anterior mediastinal mass and others.
- The shred sign is seen in patients with lung consolidation with aeration. It is identified by a deviation from the expected linear continuity of the lung line (pulmonary-pleural line) by hypoechoic, echo-poor tissue. Within the 'shred' sign, hyperechoic (white) foci (partially aerated lung) and B-lines are typically present. The shred sign is comparable to an air bronchogram on thoracic radiography.
- The wedge sign is a subset of the shred sign, which represents an infarct of the lung and pulmonary thromboembolism at the lung surface.
- The tissue sign is more severe consolidation or infiltration of the lung in which no air movement is present and the lung is likened to liver. This is often referred to as 'hepatisation'. Distinguishing the shred sign from the tissue sign may be difficult to determine and clinically irrelevant in many cases. As with the other lung ultrasound findings, these changes are only apparent if the lung consolidation occurs at the periphery of the lung. Centrally located (deeper) lung consolidation will be missed using lung ultrasound.
- The nodule sign is characterised by an anechoic circle (nodule) often with acoustic enhancement through the far field (typically as a lung rocket) and represents lung nodules in the periphery. Nodules may range from a few millimetres to several centrimetres and may occur singly or in large numbers. If the nodules are associated with nearby inflammation (such as oedema or haemorrhage) there will likely be B-lines in the surrounding lung tissue.

Patient-side thoracic ultrasound may also be of use to determine a patient's left atrial to aortic ratio (LA:Ao) as an indicator of the likelihood of the presence of congestive heart failure. Although currently the subject of discussion amongst cardiologists, the consensus appears to be that it is best measured in the right parasternal short axis echo view, and a value of < 1.5 is considered to be normal. If the ratio obtained is above this value, then the patient can be considered to have left atrial enlargement and further cardiac evaluation is advised, or the patient should at least receive continued appropriate treatment for probably congestive heart failure. A left atrial diameter that is twice the aortic diameter is very suggestive of left-sided heart failure in an animal with consistent clinical signs. This test may be performed briefly at the patient-side, thereby minimising stress yet providing valuable clinical information.

Assessment of patient oxygenation

Respiratory function can be defined as the patient's ability to oxygenate and ventilate. Although pulse oximetry can be used to detect hypoxaemia, arterial blood gas analysis remains the gold standard. Physical examination findings in patients with hypoxaemia can include cyanosis, but this is not present until hypoxia is severe and may only become evident just prior to death, making it an unreliable indicator of adequate oxygenation. Respiratory rate and effort can be difficult to interpret as they are affected by many other factors such as pain, excitement, fear, and metabolic derangements, and can be masked by sedation or anaesthesia. Arterial blood gas analysis allows us to accurately assess respiratory function and diagnose hypoxia and respiratory failure.

An arterial blood sample can be easily performed with practice. Although several sites may be considered for sample collection in dogs and cats, the dorsal pedal artery (or dorsal metatarsal artery) is commonly used as a superficial artery of a good size. The fur over the intended site of sampling should be clipped of fur and the skin lightly prepared to avoid spasm of the underlying artery which impairs attempts to collect a blood sample from the site. The pulse should be palpated using 1 or 2 fingers of the non-dominant hand as it will not be possible to visualise the artery. A 25- or 22-Ga needle is usually used, along with a vented arterial blood gas syringe. The plunger should be pulled back to the volume of blood to be collected prior to sampling. All equipment necessary to run the sample should be prepared in advance as the sample will need to be analysed immediately once collected to prevent inaccurate and misleading results due to artefact.

The needle should then be inserted, initially only through the skin, allowing the patient time to react to any discomfort without damage to the artery. Using the palpating finger tips as a guide, the needle should then be inserted parallel to where you feel the arterial pulse with the needle angled at around 45 degrees. Successful arterial puncture should result in a flash of blood in the hub of the needle and subsequent filling of the syringe until it reaches the volume preset by the plunger. The needle should then be withdrawn, and pressure applied to the site for 5 minutes. The site should be monitored for bleeding. Arterial sampling is contraindicated in patients with known coagulopathy due to the increased risk of severe bleeding as a complication of this procedure. Further details on interpretation of the results obtained will be presented in the notes accompanying webinar 3.

Airway sampling techniques

Collection of a sample from the airway and lung may be considered in patients with disease localising to the pulmonary parenchyma requiring further investigation. In cases where a community acquired pneumonia is suspected, empiric broad-spectrum antibiotic therapy is warranted initially and typically results in full clinical resolution in the majority of cases. Collection of an airway sample may be considered in cases with a poor response to therapy, a history of prior antimicrobial therapy, concern for a potentially resistant hospital-acquired infection, or an open diagnosis based on available information. Samples may be collected by an endotracheal wash (sometimes called a transoral wash), transtracheal wash, or a bronchoalveolar lavage (BAL). An endoscopic guided BAL may be preferable to obtain a sample directly from the affected lung tissue, thereby increasing diagnostic yield, but is often unavailable on an emergency basis and requires additional equipment and expertise that may further limit its availability. The endotracheal and trans-tracheal washes are both performed blindly but require no special equipment or expertise, and can be performed readily in the emergency room. Both procedures involve the instillation of sterile saline into the airway to help retrieve airway secretions. It is important to note that either may therefore result in hypoxia and possible

bronchospasm that may worsen patient respiratory function for several hours following the procedure. They should only therefore be performed in patients in which a thorough risk to benefit ratio assessment has been performed. In patients with pre-existing respiratory distress and hypoxaemia, a wash should only be performed if the facilities are present for additional support afterwards should the patient's condition further decline.

Endotracheal wash (ETW)
The endotracheal wash is preferred over the trans-tracheal wash in all cats, dogs of < 10 kg, brachycephalic dogs (owing to the smaller tracheal diameter and thicker necks which can make tracheal palpation more difficult), and larger breed dogs in which the length of available through-the-needle catheter may limit sample collection with a trans-tracheal wash. Brief general anaesthesia is required in order for the patient to permit brief intubation whilst the wash is performed, as well as to limit coughing which can adversely affect the quality of the sample retrieved. Propofol is commonly used to facilitate intubation, has the potential benefits of a rapid recovery and is a good choice if concurrent evaluation of laryngeal function is planned. Patients should be pre-oxygenated prior to induction. A sterile ET tube should then be placed cleanly. Consideration should be given to using slightly smaller sized tube than would normally be used for a given patient so as to avoid the need for application of lubricant to the tube which may affect interpretation of the fluid cytology. If lubrication is required, a small amount only should be applied to the outside of the tube.

A 5- to 8-French red rubber catheter is passed down the ET tube to approximately the level of the carina (at the heart base). Sterile isotonic saline is then infused down the tube; volumes of 3 ml (small dogs and cats), 5 ml (medium sized dogs, 10-20 kg) and 10-15 ml (large dogs, > 20 kg) and followed with air to ensure all saline is instilled within the tube. The fluid is then manually aspirated and collected into a sterile container. Concurrent coupage focused over the area of lung affected may help increase fluid yield, if not contraindicated. Drainage of fluid may also be facilitated by lifting the patient's hindquarters. Alternatively, a suction catheter or closed suction trap device may be used for sample collection. Suction devices may permit better recovery of airway cells but are less widely available than red rubber catheters.

The patient should be extubated once the procedure has been completed and is sufficiently awake to be able to protect their airway and hence minimise the risk of aspiration pneumonia. They should be closely monitored afterwards to ensure a good recovery. The clinician should be prepared to provide more aggressive respiratory support than was required prior to the procedure owing to the hypoxia and possible bronchospasm that may occur.

Trans-tracheal wash (TTW)
A trans-tracheal wash may be performed in medium to large breed dogs and has the potential advantage over the ETW as it can be performed in the awake or occasionally slightly sedated patient. The procedure involves the placement of a catheter directly between the tracheal rings to access the tracheal lumen and airway. Saline is infused as for the ETW to help retrieve airway secretions. Potential risks of the procedure include hypoxia as previously discussed, as well as subcutaneous emphysema, tracheal laceration and haemorrhage, although these are considered uncommon with good technique.

To perform a TTW, the patient is adequately restrained on the floor or a treatment table. It can be helpful to position the patient in a corner on the floor to prevent backwards movement. An area of the ventral neck, centred over the cricothyroid ligament or first couple of tracheal rings, is clipped and prepared. After carefully palpating the intended catheter placement site, a local anaesthetic block (lidocaine) is infused and the area re-prepared. It is often easier to isolate the cricothyroid ligament in dogs with thicker necks (for example Labrador Retrievers). With the needle bevel of the catheter directed downwards, the catheter is inserted through the skin and into the tracheal lumen. The catheter will typically be directed perpendicular to the trachea for the insertion. The stylet is removed once the catheter is in the desired location, taking care not to kink the catheter at the point of entry into the skin. A sample catheter of an appropriate size is fed through this catheter lumen and sterile isotonic saline aliquots of 5 to 10 mls are instilled into the airway and followed with air before the sample is collected. The instillation of saline and its aspiration may be repeated up to 3 times. Gentle coupage may help to improve the volume of sample obtained as described above.

Airway sample analysis
Regardless of the technique used for airway fluid collection, the recovered fluid should appear cloudy with occasional flecks of mucus. Cytologic examination, looking for evidence of suppurative inflammation and intracellular bacteria, should be performed in-house immediately after sample collection. Ideally, the sample should also be submitted to a veterinary clinical pathologist for interpretation, as well as aerobic culture and sensitivity testing. Anaerobic culture should also be requested if a pulmonary abscess is suspected, and mycoplasma testing also requested separately if clinically suspected. Consideration of atypical causes of respiratory distress, such as Toxoplasmosis and Pneumocystis, will also guide the submission request to the laboratory.

Samples should be evaluated cytologically for the presence of oropharyngeal contamination, which is a relatively common occurrence during airway sampling. It can be recognised in cytologic preparations by the presence of squamous cells, a mixed bacterial population that is predominantly extracellular in location, as well as unusually large bacteria (Simonsiella sp.) that normally inhabit the oral cavity and pharynx. The subsequent results obtained from contaminated samples should be interpreted with caution.

Deep oral swabs
Deep oral swabs (DOS) have been considered as an alternative means of airway sampling in patients with suspected pneumonia. They have the potential benefit of being performed in the conscious or lightly sedated patient, without the risk of worsening hypoxaemia. A recent study compared the results obtained from DOS with ETW in both puppies and adult dogs with community or hospital acquired pneumonia. Positive cultures were obtained from all samples taken, but there was no agreement between the sampling techniques in puppies. Whilst there was complete agreement in 2 adult dogs with hospital acquired pneumonia, the results obtained from cases of community acquired infections were completely different. At this time, the DOS cannot be used as an alternative to either the ETW or TTW based on the results of this study.

Advanced oxygen supplementation techniques

There are several methods of oxygen supplementation that are used in veterinary patients, including flow-by, mask, oxygen cages or tents, and nasal prongs, to name a few. Nasal cannulae are a useful alternative means of providing a high level of inspired oxygen in a practice setting (up to 60-70% with bilateral cannulae), that can also be used for longer term oxygen supplementation in hospitalised in-patients. In a minority of patients, a light sedation is required for stress-free placement although topical anaesthesia of the nose with proxymetacaine in advance of the cannula placement is sufficient for most. An 8-French (or 5-French for smaller dogs) red rubber catheter or similar feeding tube is used and is pre-measured from the nose to the medial canthus of the eye to determine the desired length of tube to be inserted. The tube is placed into the ventral nasal meatus to the desired point; this is facilitated by aiming the tube ventro-medially. The tube should pass with minimal resistance when it is in the correct location. A 20-guage needle is then placed in the skin immediately lateral to the nare being used to place the tube. Non-absorbable suture in then threated through the needle, placing the suture through the sharp end of the needle first. The needle can then be removed leaving the suture in situ, available to secure the tube in place at the point it exits the nare. A finger-trap suture pattern should be used to secure the tube. A second site of attachment is also recommended around the base of the ear, using a single knot or finger-trap pattern. Alternatively, the tube can be sutured to the top of the head although this may cause the tube to fall in front of the eyes, distressing the patient and increasing the chance of early removal by the patient. One or 2 catheters may be placed depending on the desired level of oxygen supplementation. Oxygen flow rates should not exceed 2L/min for a single cannula or 4L/min for bilateral cannulae. Consideration should be given to the placement of an Elizabethan collar to the patient to prevent early removal by pawing at the tubes. The tube(s) should be attached to a source of humidified oxygen for ongoing oxygen therapy.

If necessary, the ultimate method of oxygen supplementation is intubation and ventilation using an anaesthetic circuit or anaesthesia ventilator in practice. This does require general anaesthesia (rapid induction) and is very labour intensive. Such short-term ventilation may be considered in some patients with readily treatable disease as a life-saving measure. In patients with imminent respiratory fatigue and arrest it is preferable to take control of the patient's breathing in this manner rather than deal with respiratory arrest once it has happened. It may also mean the patient can undergo a more

thorough diagnostic evaluation than would be tolerated conscious in cases where the initial response to therapy has been poor and more information is needed for ongoing treatment. Further details on mechanical ventilation are provided in the notes below.

In some rare cases, having made the decision to intubate the patient, it may prove difficult to place an endotracheal tube. Possible causes include: laryngeal masses or inflammation, complete upper airway obstruction (foreign body, neoplasia), oral and pharyngeal masses, an inability to open the jaw (tetanus or masticatory myositis), or a lack of normal anatomy (following extensive trauma). It is important to stay calm in this situation and work through a number of possible solutions to gain airway access as described below. Ensuring optimal patient positioning, with the head held up and the neck in a straight line, is essential and can make a significant difference. Using a laryngoscope for direct visualisation of the larynx is recommended, alongside suction in case of secretions blocking the entrance to the airway. Changing to a much smaller endotracheal tube can also be helpful, and/or using a urinary catheter or similar narrow bore rigid tube as a guide, over which to feed the ET tube to access the trachea. Retrograde intubation is a technique described in people whereby a wire is placed between tracheal rings and directed cranially. It is then used as a guide to enter an endotracheal tube through the larynx and into the airway. A time limit should be set as to how long attempts to gain an airway in this way is made before considering an alternative strategy.

Where none of these techniques are possible, a needle cricothyroidotomy can be considered as a life-saving short-term alternative. This is a temporary airway that may be used to provide oxygenation until a better option becomes possible. It involves the placement of an over-the-needle catheter through the cricothyroid membrane which is then connected to a high pressure oxygen delivery system. Owing to the small diameter of the catheter, it is not possible to use a standard ventilation circuit. Instead, the catheter should be connected to a 3-way tap and a high pressure delivery system such as the oxygen flush on an anaesthetic machine. An oxygen flow rate of 12-15 litres/min is used for this purpose in people. The open limit of the tap is occluded to provide ventilation. A ratio of 1:4 for inspiratory time: expiratory time should be used to prevent air trapping. This is especially important in cases with complete upper airway obstruction where the chest excursion needs to be monitored closely and breaths given less frequently if necessary.

As an alternative, the Manujet III may be used to provide tracheal jet ventilation. The jet ventilator machine is hand-held and connected to either an oxygen cylinder or wall oxygen supply. It has an adjustable dial to limit the maximum jet pressure delivered to a preset desired level. There is also a separate manually controlled trigger to control the respiratory rate. The jet ventilator kit also comes with tracheal catheters that have lateral holes to improve airflow in the trachea, a flange which helps secure the catheter to the patient more easily, as well as a luer lock connector to prevent accidental disconnection. Although this represents a financial investment for a (hopefully) infrequent indication, the system is used on an elective basis on people during rigid bronchoscopy or in patients that are predicted to have a difficult extubation.

The final option for patients in which endotracheal tube placement is not possible, is the placement of a temporary tracheostomy tube. This requires rapid induction of general anaesthesia, most commonly achieved with either propofol or alfaxalone as an induction agent. The patient is positioned in dorsal recumbency with the neck stretched gently over a sandbag to place it in full extension. The patient should be positioned as straight as possible to facilitate finding of the correct anatomic landmarks and subsequent placement of the tracheostomy tube. The animal is rapidly clipped and the skin prepped. A skin incision is made in the midline of the neck, long enough to allow exposure of the trachea during the later stages of surgery. The tracheostomy tube will usually be inserted 2-3 tracheal rings below the cricothyroid membrane. Following skin incision, the paired strap muscles of the neck are identified on the midline and separated by a combination of blunt and sharp dissection as necessary. This exposes the trachea. Placement of paired Gelpi retractors can aid visualisation of the surgical field prior to tube placement. The standard tracheostomy incision is an incision between tracheal cartilage rings, extending no more than 50% across the diameter of the trachea, to prevent damage to adjacent structures and destabilisation of the tracheal integrity. This is large enough to allow the tracheostomy tube of an appropriate size for the patient to be easily placed. Once an airway is secured, stay sutures should be placed on either side of the incision, and clearly labelled ‘cranial’ and ‘caudal’ to allow easy replacement of the tube in an emergency. A monofilament suture should be used for this purpose and incorporate 1-2 tracheal rings on either side. An anaesthetic breathing circuit or Ambu-bag can be

attached to the tracheostomy tube as soon as it is in situ, taking care to prevent accidental dislodgement before it is secured in place.

The tracheostomy tube is suctioned shortly after placement to remove any blood in the airway associated with the procedure. It is then suctioned as needed based on the volume and nature of any airway secretions. Ideally the patient should be pre-oxygenated prior to each suction attempt to minimise any hypoxaemia associated with the procedure. If the tracheostomy tube has an inner cannula, this should be removed and cleaned on a regular basis to prevent accumulation of respiratory secretions that may otherwise block the tube. If a specific tracheostomy tube is unavailable, an endotracheal tube of an appropriate size for the patient can be cut down and used.

Oxygen provided by any of these routes after initial assessment should be routinely humidified (saturated with water vapour) to prevent desiccation of the airway mucosa and impairment of normal airway defences. This is especially important if the nasal turbinates are bypassed with nasal or tracheal oxygen catheters. Inspired oxygen is humidified by bubbling through a chamber of sterile distilled water prior to delivery to the patient.

Oxygen toxicity

Although maintenance of oxygen delivery to the tissues is of paramount importance in the emergency patient and oxygen therapy is indicated in the management of respiratory distress, oxygen is a highly reactive and potentially damaging substance that has the potential to cause significant injury to our patients. This is due to the formation of reaction oxygen species (ROS) which are very damaging to tissues and can cause cellular death and organ dysfunction.

Oxygen toxicity is thought to occur when animals are exposed to excessive oxygen which overwhelms the body's endogenous antioxidant systems. The clinical manifestations of oxygen toxicity are primarily pulmonary damage, but also include retinal dysplasia in young animals. Within the lung, the damage caused by the production of ROS is severe, diffuse and similar to that seen in acute respiratory distress syndrome (ARDS). High levels of inspired oxygen can also rapidly cause collapse of low ventilation/perfusion regions of the lung. The resultant atelectasis can be a significant cause of worsened hypoxia.

In dogs and cats, there is limited evidence as to both the level of oxygen and duration of exposure required for oxygen toxicity to occur. There is marked variation between studies but also variation in susceptibility between individual animals. Cats are perhaps more susceptible than dogs as their anti-oxidant systems are likely more easily overwhelmed. As oxygen toxicity is not just a product of the level of oxygen administered but also the duration of exposure, the general recommendation for both dogs and cats is to avoid the administration of 100% oxygen for greater than 12-24 hours. In situations of long-term oxygen therapy, the fraction of inspired oxygen should be maintained at less than 60% where possible. This is a general guideline as it is known that critical illness can lead to depletion of endogenous antioxidant levels, and hence it is possible that inspired oxygen levels of less than 60% could result in toxicity in some patients. This, combined with the fact that there is no way to predict an individual animal's susceptibility to toxicity, leads to the recommendation that the inspired oxygen level should always be titrated to the lowest level a patient can tolerate. In some cases, however, it may not be possible to reduce the inspired oxygen concentration due the presence of severe respiratory distress and inability to decrease inspired oxygen levels without worsening patient stability. There is no evidence to suggest that antioxidant therapy is useful in the prevention of oxygen toxicity in people or animals.

At the present time, there is no way to positively diagnose oxygen toxicity in our patients. It should be suspected in any patient that develops significant pulmonary change whilst receiving what can be considered to be a toxic exposure to oxygen. A deterioration in pulmonary function may be difficult to ascribe to oxygen toxicity rather than a progression in the primary disease process that lead to the requirement for supplemental oxygen in the first instance.

Mechanical ventilation

Mechanical ventilation using specialist ventilator machine, is currently not commonly used in veterinary medicine, with its use largely restricted to academic hospitals and large specialty practices. With the development of critical care medicine in the future, this may well change and its use may become more commonplace. Anaesthetic ventilators may be used successfully as an alternative for shorter time ventilation and where referral is not possible. Successful delivery of positive pressure ventilation (PPV), the most common form of mechanical ventilation, requires appropriate patient selection, necessary equipment, an understanding of the ventilator, and most importantly intensive supportive care.

Indications for mechanical ventilation
Mechanical ventilation may be considered in veterinary patients with severe hypoxemia or severe hypoventilation despite therapy, and in patients with excessive respiratory effort or work of breathing. Another possible indication is 'thinking about mechanical ventilation' for a given patient.

Hypoxemia is defined as an arterial partial pressure of oxygen (PaO_2) < 80 mmHg, corresponding to a haemoglobin saturation (SpO_2) < 95%. Severe hypoxemia is defined as a PaO_2 < 60 mmHg, corresponding to a SpO_2 < 90%. Patients with severe hypoxaemia, in which standard therapy has already failed to provide adequate oxygenation, may be considered for mechanical ventilation. Most patients with respiratory failure requiring ventilation have primary lung diseases which may include infectious or aspiration pneumonia, pulmonary contusions, cardiogenic and non-cardiogenic pulmonary oedema or acute respiratory distress syndrome. As inspired oxygen concentrations of greater than 60% over a prolonged period of time may lead to oxygen toxicity and further pulmonary damage, ventilation may also be considered in any patient requiring a high concentration of inspired oxygen for longer than 24 hours to achieve adequate oxygenation to avoid this possible complication.

Hypoventilation is defined as an elevation in the arterial partial pressure of carbon dioxide ($PaCO_2$) and is due to a decrease in alveolar ventilation. Severe hypoventilation is defined as a $PaCO_2$ > 60 mmHg and can be an indication for mechanical ventilation should the patient fail to improve with standard therapy for the underlying disease. Possible causes of hypoventilation (or ventilatory failure) requiring mechanical ventilation include brain disease, high cervical spinal cord disease, peripheral neuropathies, neuromuscular junction diseases, or primary myopathies. Using a cut-off of a $PaCO_2$ of 60 mmHg to guide decision making as to the need for ventilation may work in the majority of patients although the degree of patient distress should be taken into consideration at any elevation of carbon dioxide. Similarly, patients with brain disease may not tolerate even small elevations in $PaCO_2$ and mechanical ventilation should be considered in this patient group if the $PaCO_2$ > 45 mmHg. Other causes of hypoventilation include upper airway obstructive disorders, increased breathing apparatus dead space and sedative overdose, although these conditions are more likely to respond to specific medical management alone. It is important to note that patients with hypoventilation can become significantly hypoxemic as the elevated carbon dioxide levels in the alveoli dilute out the alveolar oxygen present. Oxygen supplementation should therefore be provided to all patients with hypoventilation while the underlying cause is addressed.

Positive pressure ventilation is also indicated in patients that are clinically believed to be in danger of impending respiratory arrest due to respiratory fatigue, regardless of the results of the arterial blood gas analysis. In these animals, extreme muscle effort associated with the work of breathing augments energy expenditure which increases tissue oxygen consumption and may negate the effects of any supplemental oxygen provided. As there are no objective measures of respiratory fatigue, assessment is based on clinical judgement alone.

Further indications for ventilation include management of fractious animals with respiratory distress that cannot otherwise be safely restrained for diagnostic tests or therapy, and provision of adequate analgesia in patients having undergone major trauma or surgery. Ventilation may also be considered in patients with multiple organ failure or those which are unstable following resuscitation from cardiopulmonary arrest for the provision of control over the airway and respiratory function. Overall, the decision to use mechanical ventilation is based on the clinical condition of the patient, arterial blood gas results if available, knowledge of the underlying disease process and the owner's wishes. It is important to note that the decision to ventilate should not be taken lightly as it is not necessarily a benign process and is highly labour intensive requiring specialised machinery and intensive nursing

care. It is recommended however, that mechanical ventilation be initiated early when indicated to minimise patient distress and any further worsening in their clinical condition and prognosis.

Initiation of ventilation

The ultimate goal of mechanical ventilation is to maintain adequate arterial blood gas values (PaO_2 80-100 mmHg and $PaCO_2$ 35-45 mmHg) using the least aggressive ventilator settings possible so as to limit further injury to the lungs. Most ventilators can generate a breath either by providing a pre-set airway pressure for a given inspiratory time (pressure controlled ventilation) or by delivering a pre-set tidal volume over a given inspiratory time (volume control ventilation). When using each of these breath types, it is essential to monitor the patient's tidal volume and airway pressures respectively, as well as the patient's clinical parameters to ensure an adequate ventilation has been delivered to the patient. Ventilator breaths may be spontaneous, assisted or controlled. Spontaneous breaths occur when the patient determines the respiratory rate and tidal volume, assisted breaths when the patient determines the respiratory rate but the tidal volume is generated by the machine, and controlled breaths when the machine determines both the respiratory rate and tidal volume. There are also a number of settings that can be modified for the individual patient. These include the respiratory rate, tidal volume, peak inspiratory airway pressure, inspiratory time, inspiratory to expiratory ratio, trigger sensitivity and positive-end-expiratory pressure (PEEP). By modifying these settings, effective, safe and comfortable ventilation for the patient can be delivered. Some ventilators also provide pressure support, which is the augmentation of spontaneous breaths with increased airway pressures during inspiration. This may be useful in weaning patients off the ventilator as the work of breathing can be gradually transitioned back to the patient away from the ventilator.

Prior to initiating ventilation, the ventilator should be set-up with appropriate starting settings for the patient and all the necessary anaesthesia and monitoring equipment prepared. It should be anticipated that patients with disease lungs and respiratory failure will require more aggressive ventilator settings and higher inspired oxygen concentrations than those with normal lungs and ventilatory failure. For any given patient, the settings will need to be adjusted to suit them at any point in time; finding the ideal ventilator settings may be described as a process of trial and error. Ventilation should be started using an inspired oxygen concentration of 100% to reverse any hypoxemia and help stabilise the patient. Once adequate oxygenation has been established, the oxygen concentration should be gradually decreased to the lowest possible to achieve the goals of ventilation to minimise the risk of oxygen toxicity and further lung injury.

Mechanical ventilation can recruit collapsed alveoli and improve gas exchange so patients may achieve acceptable blood gas values on lower levels of inspired oxygen with PPV compared with spontaneous breathing, thereby reducing the risk of oxygen toxicity. This is achieved by the use of PEEP (positive end-expiratory pressure) in which small amounts of positive pressure are applied to the airways to prevent complete expiration. As the lung is held at a higher volume and pressure during expiration, previously collapsed alveoli may be recruited and others prevented from collapsing, serving to increase the functional lung tissue present. Appropriate use of PEEP may also decrease ventilator associated lung injury by preventing the shear injury caused by cyclical reopening and collapse of the alveoli with each breath. PEEP also has a role in lung-protective strategies when used in conjunction with lower tidal volumes, and in some instances permissive hypercapnia, to minimise overdistension of alveoli causing further inflammatory injury to the lung.

Total intravenous anaesthesia is required rather than inhalant agents for anaesthesia of the ventilator patient. There is not a standard protocol that can be used, rather the anaesthetic plan is tailored for each individual depending on the anticipated duration of ventilation and concurrent disease. A multi-modal approach to anaesthesia is beneficial to decrease the dosage and hence minimise the adverse effects of any single anaesthetic agent. Pre-oxygenation prior to a rapid induction is recommended. Potential induction agents include a combination of drugs including opioids and benzodiazepines, propofol or alfaxalone. Mask inductions should be avoided. To maintain an adequate plane of anaesthesia, constant rate infusions of combinations of opioids, benzodiazepines and propofol or alfaxalone can be used with good effect. The level of sedation should be frequently assessed and adjusted based on the patient's cardiovascular stability and plane of anaesthesia achieved. The use of neuromuscular blockade may be considered in some patients but is not a substitute for adequate anaesthesia and introduces concerns for incomplete recovery and prolonged effects during the weaning process. A controlled decrease in the anaesthetic drugs used is needed for weaning from ventilation to restore the patient's own respiratory drive and function.

Patient-ventilator dysynchrony is also referred to as 'bucking the ventilator' and occurs when there is any mismatch of the patient's own breathing pattern and the machine settings resulting in the patient breathing against the ventilator. Such dysynchrony can severely impair ventilation and oxygenation and is a sign of patient distress that must be addressed immediately. Causes include an insufficient depth of anaesthesia or analgesia, hypercarbia, hypoxemia (e.g. secondary to airway obstruction, pneumothorax or inadequate ventilator settings) and hyperthermia amongst others.

Complications associated with ventilation
Mechanical ventilation is not a benign process and there are a number of complications which may arise. Cardiovascular compromise secondary to an increase in intra-thoracic pressure may occur. This may impair venous return and hence decrease cardiac output. These effects may be particularly severe in patients with intravascular volume depletion and pre-existing cardiovascular instability, or when particularly aggressive ventilator settings are required to maintain adequate oxygenation and ventilation. Close cardiovascular monitoring is essential for all ventilated patients, and especially those at particular risk of adverse cardiovascular effects such as those requiring high PEEP or aggressive ventilator settings.

Upper airway damage may occur secondary to the placement of the endotracheal tube and the damage caused by instillation of oxygen under pressure, resulting in excessive drying of the mucous membranes. A large volume of tenacious mucus is frequently produced which can result in clogging and occlusion of the endotracheal tube. Use of a mucolytic agent, such as N-acetylcystine given at a dose of 70 mg/kg IV q6, may be considered if this is a recurrent problem for a particular patient.

Ventilator-induced lung injury is a significant concern and is associated with volutrauma, or overdistension of the alveoli. This can be a particular problem as the lung disease initially present is usually not evenly distributed throughout the lung tissue. This means relatively normal areas may receive a disproportionate amount of the tidal volume resulting in overexpansion in comparison to the less compliant diseased areas. Repetitive expansion and collapse of alveoli leads to shearing injury and the release of inflammatory mediators and secondary lung injury. Pneumothorax is another complication, which may be seen in any ventilated patient although perhaps more frequently in those requiring aggressive settings such as high airway pressures (> 30 cmH_2O) or large tidal volumes. Efforts should be made to minimise the settings wherever possible to decrease the risk although the use of higher pressures may be unavoidable in the abnormal lung. The presence of a pneumothorax should be considered in any patient with a sudden decline in oxygen saturation and/or tidal volume, elevation in end-tidal carbon dioxide, sudden cardiovascular instability, patient-ventilator dysynchrony or a decrease in chest wall movement, lung sounds on thoracic auscultation or pulmonary compliance. Management of a pneumothorax may require the placement of unilateral or bilateral thoracic drains and continuous pleural drainage given the requirement for ongoing PPV. Without timely treatment, the development of a tension pneumothorax would likely be rapidly fatal.

Ventilator patients are also at significant risk of infection, with ventilator-associated pneumonia (VAP) being extremely common in both people and veterinary patients. There are numerous risk factors for the development of VAP including the compromise of the normal airway protective mechanisms due to endotracheal intubation, and potential for introduction of bacteria into the airways through intubation and suctioning, patient immunosuppression, pulmonary atelectasis secondary to recumbency, and gastric regurgitation and prior histamine 2-antagonist or proton pump inhibitor therapy decreasing gastric acid production. Ventilated patients should be closely monitored for any worsening in pulmonary function, development of fever, new alveolar infiltrates on thoracic radiographs, inflammatory changes on complete blood count monitoring, or any other evidence of sepsis. Thoracic imaging along with and culture and sensitivity testing of airway samples obtained from an endotracheal wash or bronchoalveolar lavage should be performed in cases where an infection is suspected and early intravenous antibiotic therapy administered. Consideration should be given to the suspected pathogens when choosing an antibiotic given local resistance patterns may vary significantly between hospitals. Despite the high risk of a patient developing VAP, antibiotic therapy should not be routinely started unless an underlying infectious aetiology has been previously identified or there is suspicion for the development of VAP so as to limit the development of bacterial resistance.

There are several other possible complications of mechanical ventilation. PPV and the use of narcotic sedatives also increase the secretion of anti-diuretic hormone (ADH), thereby decreasing urine

output, resulting in patient fluid retention and subsequent oedema formation. This may further worsen pulmonary function and compromise other organ function. Close attention to patient fluid balance and intermittent diuretic therapy may be required during the period of ventilation. The development of limb compartment syndrome has been reported in human medicine in association with mechanical ventilation and has also seen at the author's hospital in two dogs, both of which required emergency fasciotomy to preserve limb function. Other possible complications include corneal ulceration, oral ulceration, muscle atrophy, the development of pressure sores and accidental disconnection from the ventilator or machine failure.

Management of the veterinary ventilator patient

Intensive nursing care is essential to minimise the occurrence of the possible complications described above. Diligent airway management is an essential part of ventilator patient care. The endotracheal tube used should be sterile and ideally have a high-volume low-pressure cuff, appropriately inflated to prevent tracheal necrosis. The cuff should be temporarily deflated and the tube repositioned frequently to decrease the risk of pressure necrosis to the trachea. A non-porous material, such as intravenous tubing, should be used to secure the tube in place and the tie also frequently repositioned to minimise pressure injuries to the oral tissues. The tube should also be routinely changed every 24-48 hours depending on the volume and nature of any airway secretions. The tubing of the ventilator circuit should also be changed daily if possible to limit nosocomial infections in the patient. Prior to any such airway management, a period of pre-oxygenation with 100% oxygen should be performed to prevent patient hypoxia and clinical deterioration. As anesthetised patients are unable to cough to clear respiratory secretions, occlusion of the endotracheal tube lumen may occur resulting in a potentially life-threatening situation. Humidification of the inspired oxygen is helpful to prevent loss of heat and moisture, and make the secretions less viscous and more easily removed by suctioning the airway.

Patients undergoing ventilation for longer periods, those with neurological dysfunction and brachycephalic dogs may benefit from a temporary tracheostomy tube for airway management. This helps to decrease anaesthetic drug requirements and may facilitate successful weaning from the ventilator. The tracheostomy tube should ideally have an inner cannula which should be cleaned every 4 hours and the entire tracheostomy tube replaced every 24-48 hours. If no inner cannula is present, the tube should be suctioned as required and the tube changed every 24 hours or sooner if needed. Any tracheostomy tube used also requires a cuff to be inflated to protect the airway and allow PEEP and PPV to be delivered.

Regular eye care is necessary to prevent corneal drying and secondary ulceration. An artificial tear product should be applied to both eyes every 2 hours or more frequently if required. Fluoroscein staining should be performed regularly to check for the presence of ulceration and treatment instituted if necessary. A temporary tarsorrhaphy may be considered to prevent corneal injury if ulceration develops despite preventative treatment and in dogs considered at risk due to their ocular conformation or previous history of ulceration. Ensuring patients are not positioned directly under air-vents or fan devices is another useful means or decreasing the incidence of ocular complications. Regular oral care is also necessary to prevent oral ulceration and lingual swelling which could subsequently lead to bacterial colonisation of the oropharynx and an increased risk of pneumonia, in addition to difficulty weaning from the ventilator. Oral care consists of preventing mechanical injury by frequent repositioning of monitoring equipment, protecting the tongue from compressive injury from the teeth by use of a mouth gag and from drying by the application of a glycerin socked gauze. Regular oral rinses using a chlorhexidine solution should also be performed to decrease bacterial colonisation and the subsequent risk of infection.

Placement of a urinary catheter and measurement of urine output can be a useful patient monitoring aid and to minimise soiling and urine scald. The colon should also be palpated on a daily basis and warm water enemas administered as necessary to prevent build-up of faeces. Careful attention to fluid balance is also important taking into consideration the patient's underlying disease process, degree of cardiovascular compromise caused by the ventilator settings used, and the tendency for peripheral oedema formation. Particular attention should be paid to management of any intravenous catheters and other indwelling devices, and body temperature control. The patient should be repositioned frequently to avoid pressure necrosis of the extremities and regional oedema secondary to poor lymphatic drainage. The body and all limbs should be well padded and passive range of motion exercises performed frequently.

In patients undergoing prolonged ventilation and/or those with a prior history of inadequate nutritional intake, provision of nutritional support is an important part of their management. Enteral nutrition may be challenging due to the frequent presence of ileus and the significant risk of regurgitation and subsequent aspiration. Post pyloric feeding may be associated with a decreased risk of aspiration events. The use of promotility drugs such as metoclopramide, ranitidine or erythromycin, may be considered in patients receiving enteral nutrition. Alternatively, the use of parenteral nutrition may be considered.

Ventilated patients require close cardiovascular monitoring in a similar fashion to any other anaesthetised patient. Required monitoring equipment includes that needed for arterial blood pressure measurement, continuous electrocardiography, continuous temperature measurement, pulse oximetry and capnography. An arterial catheter, usually placed in the dorsal pedal artery, can be useful in dogs for direct arterial blood pressure monitoring and sampling for arterial blood gas analysis which should be performed regularly. If obtaining arterial blood gas samples is not possible, a combination of venous blood gas analysis and pulse oximetry readings can be used to assess the effectiveness of ventilation. Ventilator settings including inhaled oxygen concentration, pressure and volume settings, mode of ventilation and respiratory rate should be recorded regularly to monitor trends and assess patient progression. Ventilator waveform analysis may also be performed to provide information on pulmonary compliance and aid in trouble-shooting any problems. Routine monitoring of the underlying disease process is also necessary and further investigations into any patient deterioration performed as indicated.

Weaning from mechanical ventilation
In order for weaning to be successful, the patient must have a sufficient respiratory drive, adequate neuromuscular function to achieve a sufficient tidal volume, and should no longer be dependent on significant ventilator support to maintain adequate gas exchange. The patient should also be haemodynamically stable with an improvement in the primary disease process, and the absence of major organ failure. The weaning process involves a gradual reduction in the work of breathing being performed by the ventilator with a proportional increase in the work performed by the patient. This may be performed using a variety of ventilator modes. The final stage of weaning is the disconnection of the patient from the ventilator and extubation. The anaesthetic plan should be modified as necessary depending on the drugs used to allow for weaning. The patient should be closely monitored at every stage of the process for any sign of weaning failure including tachycardia, respiratory distress, hypoxemia, hypoventilation, hypertension or patient distress. Given that mechanical ventilation is not a benign process, it can be said that the weaning process begins at the time the patient is put on the ventilator as settings are continually changed to provide the minimum possible level of support to maintain adequate arterial blood gas results.

Prognosis following mechanical ventilation
The prognosis for successful weaning of the patient from the ventilator is largely dependent on the primary disease process leading to the initiation of ventilation. The survival rates reported in veterinary medicine are currently much lower than those in human critical care medicine, although this may be in part explained by financial limitations, the effect of euthanasia and lack of availability and experience with the therapy in veterinary patients. Both human and veterinary clinical studies consistently show a lower survival to discharge rate for patients ventilated for pulmonary parenchymal disease (respiratory failure) as compared to those ventilated for neuromuscular disease processes (ventilatory failure). In animals ventilated because of respiratory failure survival rates are reported to vary from 14-30%. In animals with ventilatory failure reported survival rates vary from 71% (dogs) to 33% (cats). The incidence of ventilator related complications is not well characterised in veterinary patients and the effect on patient morbidity and mortality currently unknown.