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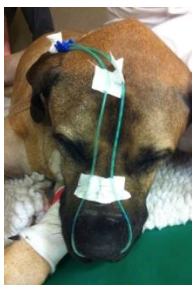
Practical Techniques for Emergency Patients Mini Series

Session One: Techniques for initial assessment of the emergency patient

Lindsay Kellett-Gregory BSc(Hons) BVetMed(Hons) DACVECC DECVECC MRCVS
RCVS Recognised Specialist in Emergency & Critical Care



Practical Techniques in the Emergency Room: Initial Assessment of the Emergency Patient

Lindsay Kellett-Gregory BSc(Hons), BVetMed(Hons), DACVECC, DECVECC, MRCVS
RCVS Recognised Specialist in Emergency & Critical Care
Lecturer in Emergency & Critical Care Medicine, Royal Veterinary College

Emergency medicine is one of the most challenging and rewarding specialities in veterinary medicine. Rapid patient assessment and stabilisation is essential. This webinar series will guide you through easily accessible practical techniques that you can apply directly to your patients to aid patient management.

Initial approach to the emergency patient

Any patient presenting to the clinic on an emergency basis should be triaged, namely assessed to determine the urgency of any problem and whether it is life threatening and hence requires immediate attention. This is performed by:

1. ABCs – does the patient have a patent airway (A), is there evidence of effective breathing (B), and is there a heart beat with pulses (C)? Problems in any of these areas require immediate attention.
2. Capsule history – a brief focused history should be obtained from the owners to determine the nature of the presenting problem. Important information to obtain includes the nature and duration of the problem. It should be made clear that a full history will follow later but this information is enough to guide initial stabilising therapies.
3. Major body systems assessment – of all of the body's systems, dysfunction of the cardiovascular, respiratory and neurologic systems may kill the patient rapidly. The initial patient assessment is therefore limited to these 3 to detect any life threatening abnormalities so they can be rapidly addressed. They can also be used to determine the degree of patient compromise to decide who requires most urgent attention when presented with more than one emergency patient. Typical stabilisation priorities and assessment parameters include:
    a. Cardiovascular: restore effective circulating volume. Assess heart rate and rhythm, along with femoral and peripheral pulse quality, mucous membrane colour and capillary refill time, mentation and temperature of extremities, and the presence of any heart murmur or gallop rhythm.
    b. Respiratory: oxygen therapy and minimise stress. Assess respiratory rate and effort, pattern of breathing, patient posture, mucous membrane colour, thoracic auscultation, and the presence of any stertor or stridor.
    c. Neurologic: ensure good perfusion, address elevations in intracranial pressure (ICP), and achieve rapid seizure control. Assess mentation (dull, stuporous, comatose), gait and ability to ambulate, presence of deep pain perception if non-ambulatory, and cranial nerve assessment to include pupil size and reactivity.

It is important that this initial patient assessment is performed rapidly and the findings acted on in a timely fashion. Prevention is always preferable, and generally less costly, than cure. Rapid treatment prevents further deterioration in the patient's organ function and reduces the incidence of further complications during hospitalisation.

Although not a major body system, assessment of the patient's abdomen and body temperature should also be performed as part of the initial assessment, to detect life threatening abnormalities and guide early correction. Specifically, the abdomen should be assessed for the presence of focal or generalised pain, any overall distension or specific organ distension (bladder, intestinal loops) or other significant abnormality. The presence of a fluid thrill should also be noted although this is an insensitive technique for assessing for the presence of peritoneal effusion requiring > 30ml/kg of fluid to be present. FAST scans are becoming more commonplace as an alternative means of assessment for free fluid where patient side ultrasound facilities are available. A rectal temperature of > 40^{o}C should also warrant concern and may be life threatening by itself, regardless of the inciting cause, if > 42^{o}C. Common causes of such severe derangements in body temperature include upper airway obstruction, prolonged seizure activity, and heat stroke from exertion or environmental temperatures. Total body cooling techniques including the use of a fan and wetting the fur with cool water, along with intravenous fluid therapy if indicated, should be used and are more effective than wetting the paw pads which have a small total surface area for rapid heat loss. Patient cooling should stop before a normal body temperature, usually around 39^{o}C, is reached to prevent inevitable iatrogenic hypothermia and the need for subsequent patient warming! The presence of hypothermia as a problem may be indicative of poor perfusion of vital organs and the cardiovascular assessment repeated. Care should be taken not to aggressively warm hypoperfused patients to prevent a worsening in their cardiovascular status due to peripheral vasodilation and distribution of circulating volume away from the major organs.
A thorough physical examination, or secondary survey, should always be performed after the initial assessment has been completed and any life threatening problems addressed.

Recognition of shock

Shock is a common presenting complaint in emergency medicine and is best defined as a decrease in oxygen delivery to, or utilisation by, the tissues, resulting in decreased cellular energy production. It is a true emergency as the longer it continues, the more tissue damage occurs, leading to potentially permanent organ dysfunction and possible death. The longer the patient remains in a state of shock the less reversible the changes are with treatment. In order to get the best possible patient outcome and minimise morbidity and mortality, it is therefore important it is rapidly recognised and treatment started to restore normal perfusion as soon as possible.

Broadly speaking, shock may occur due to inadequate tissue perfusion, a decreased arterial oxygen content (due to severe anaemia or hypoxaemia) or metabolic derangements at a cellular level (examples include sepsis and cyanide toxicity), resulting in a failure of the tissues to use oxygen correctly). Perfusion abnormalities are the most common form of shock encountered and may be further divided into:

1. Hypovolaemic shock – decreased circulating blood volume due to loss of fluid from the intravascular space
2. Distributive (vasodilatory) shock – inappropriate systemic vasodilation secondary to systemic inflammation (SIRS, sepsis) or anaphylaxis leading to a decreased effective circulating volume
3. Cardiogenic shock – decreased cardiac output due to cardiac pump failure as seen with advanced myocardial disease, or cardiac arrhythmias
4. Obstructive shock – obstruction to blood flow which may be due to pericardial effusion, thromboembolic disease or gastric distention from gastric dilatation (volvulus)

Each of these perfusion disturbances requires different forms of therapy – for example, hypovolaemia requires aggressive fluid therapy, which could prove fatal in instances of cardiogenic shock, and thus it is important that the presence of shock and its likely cause is recognised rapidly.

Hypovolaemic shock

Hypovolaemia is associated with a loss of fluid from the intravascular space. This may occur due to haemorrhage or loss of other body fluids. Common locations for bleeding in emergency patients include body cavities (peritoneal, retroperitoneal, pericardial and pleural spaces), fracture sites, and the GI tract where bleeding may not be outwardly apparent initially. The presence of peri-umbilical haemorrhage (the Cullen sign) is suggestive of intra-abdominal bleeding. In dogs, low total solids with a normal PCV is suggestive of acute haemorrhage. Non-sanguinous fluid may be lost from the body in the form of fluid loss from the GI tract, kidneys in a polyuric patient, or third spacing of fluid (eg. peritoneal effusion) and cause hypovolaemia. Hypovolaemia is only seen in association with very severe cases of dehydration.

The normal homeostatic response to hypovolaemia involves vasoconstriction to reduce the vascular space and improve venous return to the heart, tachycardia and increased cardiac contractility. Additionally, the renin-angiotensin-aldosterone system and release of ADH stimulate renal retention of sodium and water, decreasing urine output and preserving vascular volume. The pulse quality, as determined by assessment of the pulse amplitude and pulse duration, is also affected. As hypovolaemic shock becomes more severe both mentation and the temperature of body extremities may be affected, and blood lactate levels become progressively elevated. A combination of these clinical findings can be used to assess a patient and quickly determine both the presence and severity of any hypovolaemic shock present and are shown in figure 1.

Figure 1: Clinical signs in uncomplicated canine hypovolaemic shock in dogs

Clinical sign	Mild (compensatory)	Moderate (early decompensatory)	Severe (late decompensatory)
Heart rate	130-150	150-170	170-220
Mucous membrane colour	Normal to pinker than normal	Pale pink	White, grey or muddy
Capillary refill	Rapid (< 1 sec)	Approximately normal (1-2 sec)	Slow (> 2 sec) to absent
Pulse amplitude	Increased	Mild to moderately decreased	Severely decreased
Pulse duration	Mildly reduced	Moderate reduced	Severely reduced
Metatarsal pulse	Easily palpable	Just palpable	Absent
Mentation	Usually normal	Depressed	Severely depressed
Extremities	Usually normal	Normal or cool	Cold
Plasma lactate concentration (mmol/L)	3-5	5-8	>8

When the body first detects mild hypovolaemic shock, a state of 'compensated shock' occurs while these compensatory mechanisms prove adequate to maintain perfusion. Patients with compensated hypovolaemic shock are mildly tachycardic but have adequate blood pressure and reasonably strong pulses and other perfusion parameters are normal. If hypovolaemia worsens then the body's ability to compensate becomes inadequate and decompensated shock ensues. This is can be recognised by a decrease in pulse strength, the development of hypotension, an exacerbation of the signs seen during compensated shock, and clinical evidence of vasoconstriction including changes to mucous membranes and extremities. With prolonged or severe decompensated shock, damage to susceptible organs (e.g. kidneys, brain, heart) occurs leading to organ dysfunction and eventually death if left untreated.

Hypovolaemic shock should be treated with aggressive fluid therapy delivered in the form of fluid boluses. The aim is to restore circulating volume rapidly. In cases with limited finances or those proving challenging to manage, with evidence of ongoing abdominal bleeding, placement of an abdominal wrap may be considered in an attempt to minimise bleeding prior to or instead of surgical exploration. With its application, even a small reduction in the radius of a blood vessel is translated into a reduction in flow to the power of 4, which may represent a significant reduction in bleeding. It may also produce a tamponade effect on bleeding organs, reducing the size of the peritoneal space and reducing haemorrhage volume.

A modification of this is to include both pelvic limbs into the wrap to avoid compartmentalisation of blood in the pelvic limb vessels and to avoid occluding the caudal abdominal vena cava. In dogs, wraps can be applied rapidly by circumferentially wrapping the abdomen, with or without the hindlimbs, with towels and tape, or bandaging materials. Heavy sedation and analgesia may be required for patient comfort if the hindlimbs and pelvis are included. Urinary catheter placement with a closed collection system may be required to facilitate management. Abdominal wraps should be removed gradually once the patient is stable to avoid sudden release of any tamponade on bleeding vessels. The wrap should be cut from the dorsal aspect from caudal to cranial at around 1 inch hourly and the patient's perfusion parameters assessed to detect any evidence of bleeding.

Distributive (vasodilatory) shock

The most common cause of inappropriate systemic vasodilation is the systemic inflammatory response syndrome (SIRS) whereby widespread inflammation is triggered by disease elsewhere in the body. Examples of underlying conditions include sepsis, pancreatitis and neoplasia. The vasodilation results in peripheral pooling of blood and a decrease in effective circulating volume to the core organs and signs of shock (this may be considered as a relative hypovolaemia). The clinical signs of vasodilatory shock are different to that of hypovolaemic shock in that although patients will be tachycardic, the mucous membranes appear congested with a rapid (< 1 second) capillary refill time. Femoral pulses are also often hyperdynamic (taller and narrower compared to normal).

Although this type of shock is treated in the first instance with fluid therapy to supplement circulating volume and restore perfusion to vital organs, it is important it is distinguished from hypovolemic shock since the underlying causes are different and it is important sepsis is addressed as a matter of urgency. Recent studies have shown an improved outcome in sepsis with early antibiotic therapy with mortality increasing significantly for every hour delay in the administration of appropriate antibiotic therapy. If fluid therapy alone fails to correct cardiovascular instability, then early vasopressor therapy could be considered to specifically target the vasodilation.

Cardiogenic shock

Cardiogenic shock can result from failure of the myocardial pump mechanism and a reduced cardiac output secondary to intrinsic cardiac disease, or a severe arrhythmia where the ventricular rate is either too fast to permit sufficient cardiac chamber filling, or too slow to achieve adequate cardiac output over time. Examples of specific conditions include dilated cardiomyopathy, hypertrophic obstructive cardiomyopathy, chordae tendinae rupture in advanced mitral valve disease, ventricular tachycardia, sick sinus syndrome or 3rd degree atrioventricular block. Cardiogenic shock is treated with myocardial support and/or heart rate or rhythm control rather than fluids which would likely be detrimental to the patient. Indicators on physical examination that cardiogenic shock is likely to be present include extremes of heart rate (a heart rate > 200 bpm is more likely to be cardiac in origin rather than compensatory in the case of hypovolaemic shock in dogs), an irregular pulse pattern with pulse deficits, and the presence of a heart murmur or gallop rhythm in cats. History and signalment may also provide invaluable clues that cardiogenic shock should be strongly considered.

If the origin of the shock is still unclear after initial assessment, patient side thoracic ultrasonography may be used to briefly assess cardiac function and chamber size as an initial screen. If ultrasound is not available and/or the results ambiguous, a small isotonic crystalloid fluid bolus should be given and respiratory rate and effort as well as thoracic auscultation closely monitored for any deterioration in respiratory status as well as observing cardiovascular perfusion parameters for evidence of improvement.

Obstructive shock

Obstruction to forward blood flow most typically occurs secondary to gastric dilatation (and volvulus) or pericardial effusion. In both cases the clinical presentation is typically suggestive of the underlying condition making this form of shock a consideration. After initial stabilisation as appropriate for the condition, gastric decompression or pericardiocentesis should be performed to relieve the obstruction and allow normal blood flow to resume.

What about cats with shock?

There are several differences in the clinical presentation and progression of shock in dogs and cats. Most notably, cats with shock tend to show bradycardia (HR < 120 bpm) more frequently than tachycardia and a normal or low heart rate in a systemically unwell cat should raise concern for perfusion abnormalities. Cats also do not tend to show the hyperdynamic phase of compensated shock as seen in dogs, typically progressing rapidly to decompensated shock and ongoing hypoperfusion. Hypothermia, poor pulse quality and a depressed mentation are common findings in a cat with shock. The mucous membranes tend to be pale and may be slightly icteric in some cases with vasodilatory shock.

<u>Vascular access</u>

Vascular access is essential in the management of cases with hypovolaemic shock but may be challenging in patients with collapsed blood vessels with severe hypoperfusion. If possible, short, large bore catheters are preferable to allow a rapid rate of fluid administration. If intravenous access is difficult to delayed due to the patient's condition, a cut-down approach to the blood vessel or intra-osseous access may provide rapid patient access for the administration of fluids. Setting a short defined time limit (< 5 minutes) on any attempt at percutaneous catheterisation, such an alternative approach should be considered.

Vascular cut down

In dogs the lateral saphenous vein is preferred owing to its size and location under relatively thin skin. The hair should be clipped from the site and the skin prepared as much as time allow. A scalpel blade is used to incise the skin immediately adjacent to the blood vessel, running in a line immediately adjacent to the vessel. Once the incision is made, the skin should be retracted to expose the vein. Haemostats are then used to bluntly dissect around the vessel, applying pressure to the tips directly onto the vein to strip away perivascular fascia.

The vein should be freed from its fascia in all directions so the haemostats can be passed underneath the vein and the instrument positioned such that the vein is stretched out over the two handle shafts. The vein is now exteriorised, free from fascia, and occluded at the top and bottom by the pressure of the handles. It is also immobilised to facilitate catheter placement using a smaller than normal gauge over-the-needle catheter. The stylet is removed, a connector attached and the haemostats removed. The wound margins should be apposed over the catheter, the skin wound temporarily closed, and the catheter secured. In cats, the medial saphenous vein is often used in preference, using the same technique as described above.

Catheters inserted using this technique should always be considered to be contaminated and should be removed as soon as the patient has been stabilised and more appropriate longer-term vascular access secured. Until this time, the would should be cleaned as for any laceration and any remaining hair removed around the site. The wound should be flushed liberally while the catheter remains in place. Next, the site should be compressed using sterile swabs, the catheter removed and direct pressure applied to provide haemostasis. Once any bleeding has stopped, the wound may be dressed and left to heal by secondary intention. If the size of the incision is considered too large, skin sutures may be placed but it is essential that the distal third of the wound be left open to drain.

Intra-osseous catheterisation

Intra-osseous catheterisation involves placing a needle into a bone. The technique is simple to perform, does not require any specialised equipment, and can be very useful. Intra-osseous catheters may be placed in the trochanteric fossa of the femur, the wing of the ileum, the proximal humerus, and the tibial tuberosity. Of these sites, the trochanteric fossa of the femur is the preferred site in dogs and cats. In small or neonatal animals, a standard 22-gauge hypodermic needle is used. Bone marrow needles can be used in larger patients whose bones have already ossified. Intraosseous drills can also be used to facilitate insertion of an intraossous catheter in an ossified bone (see below). In smaller patients whose bones have not yet ossified, the shaft of the hypodermic needle may become clogged with cortical bone debris during placement. This may be avoided by using a spinal needle containing an inner stylet than can be removed following correct placement, or using a piece of surgical wire in the shaft of the needle to prevent clogging. In the event the needle does become clogged, it may be removed and replaced with an identical one through the hole that was created.

The site of insertion should be clipped and cleaned. Local anaesthetic may be injected down to the level of the periosteum to decrease any discomfort associated with catheter placement. The stifle should be adducted so it is pushed toward the central midline and the trochanteric fossa is rotated laterally. This positioning helps to decrease the risk of trauma to the sciatic nerve. The needle tip is pushed through the skin, and using a simultaneous pushing and twisting motion, it is pushed into the groove in the intertrochanteric fossa, through the periosteum, and into the shaft of the femur. There will be a loss of resistance felt as the needle enters the shaft of the femur. Once the needle has been placed correctly, you should be able to push the hub of the needle back and forth and move the leg.

Aspiration of bone marrow also confirms correct placement. Ideally radiographs should be taken to confirm correct placement. A T-connector should be attached to the hub of the needle and the catheter flushed with saline. Non-heparinised saline should be used on very small patients to prevent the development of a coagulopathy. There should be very little resistance felt when flushing the catheter. If the fluid does not flow freely, the needle should be rotated 90 to 180 degrees to ensure the bevel has not become lodged against the wall of the bony cortex, causing an occlusion. The catheter should be secured using tape or suture. The catheter may then be used to deliver fluid therapy (crystalloid, colloids and blood products), drugs and parenteral nutrition. Also very fast rates may be reached, rapid infusion rates can cause discomfort in some patients.

Relative contraindications include fracture of the proposed catheter site, bacterial infection or sepsis, or the presence of a skin wound or infection over the proposed site of catheterisation.

EZ-IO catheter placement

The EZ-IO system (www.vidacare.com) provides a new technique for performing intraosseous catheterisation in patient with ossified bones. A hand-held, battery-powered drill is used to rapidly place (< 10 seconds) a purpose-made intraosseous catheter. Any of the anatomic sites listed above may be used for intraosseous catheterisation using the technique, although the greater tubercle of the humerus, or tibial tuberosity are often preferred.

The patient should be placed in lateral recumbency for catheter placement. If the greater tubercle of the humerus is being used for placement, the landmarks include the scapular spine and the acromium. The site should be clipped and skin prepared prior to infiltration with local anaesthesia as described above. A stab incision may be made to facilitate placement of the catheter. A catheter is selected based on the size of the patient and is loaded onto the drill. The tip of the needle is pushed through the skin and placed directly into the periosteum of the greater tubercle. Forward pressure is then applied, taking care that the needle does not slip off the cortical bone. The power button is depressed on the drill and the catheter placed directly into the bone. The stylet is removed and connector attached. Correct placement of the catheter should be confirmed, as described above. If there is any pain associated with infusion of high fluid rates, an infusion of lidocaine may be used (1-2mg/kg, 2% solution) to improve patient comfort. Drug doses for intraosseous administration are the same as for intravenous doses. The catheter may be used until the patient is stable and alternate vascular access has been secured.

Approach to the patient with respiratory distress

Respiratory distress is a common presenting complaint in dogs and cats and may be life-threatening. These patients require immediate assessment to ascertain the most likely cause of their respiratory compromise in order to provide appropriate stabilising and definitive management strategies.

However, this may be easier said than done as the degree of respiratory dysfunction may limit further diagnostic evaluation, making it impossible to attain a specific diagnosis, and further diagnostics may worsen the patient's condition. These notes describe a logical approach to the initial management and assessment of these patients, in order to immediately narrow down the most likely possible causes and subsequent therapeutic interventions. This can be done from critical evaluation of readily available and free of charge clinical data and a targeted efficient approach can also prove money saving.

Initial assessment and stabilisation

Although patients with severe respiratory distress may be daunting to manage, there are a finite number of possible underlying causes than can arise from even smaller number of anatomic locations of the respiratory tract.

1. Upper airways
2. Lower airways
3. Pulmonary parenchyma
4. Pleural space
5. Chest wall and diaphragm
6. Abdominal distension
7. Look-a-likes

The goal of the initial patient assessment is to identify any respiratory distress, determine the most likely location of the problem and subsequently stabilise the patient's condition. Careful assessment of the respiratory rate, effort, pattern of breathing and respiratory sounds can help locate the affected portion of the respiratory tract and narrow down the list of possible causes, avoiding unnecessary diagnostics and treatment.

Patient assessment should begin with observation of the patient from a distance. Common signs of severe respiratory distress include anxiety and restlessness, abducted elbows, extended head and neck, and open mouth breathing, in addition to tachypnoea. Affected dogs tend to prefer to stand meaning that lateral recumbency can be a late and concerning sign. In contrast, severely affected cats typically prefer to sit in sternal recumbency and the change to lateral recumbency can be a sign of impending respiratory arrest.

An increase in respiratory effort may also be observed, secondary to recruitment of additional muscles of respiration (intercostal and abdominal wall muscles) in addition to the standard diaphragmatic activity. It is important to evaluate the phase of respiration in which any additional effort occurs as this can provide vital clues as to the most likely underlying causes. In severe cases, paradoxical respiration may be seen, characterised by a loss of the normal synchronous movement of the chest and abdominal walls as the diaphragm, caudal intercostal and abdominal muscles collapse inwards and forwards during inspiration.

The presence of paradoxical respiration is a clear indicator of severe respiratory distress or dyspnoea, representing an increase in the work of breathing and fatigue of the respiratory muscles.

Cyanosis is another clinical sign that may be observed in patients with severe respiratory compromise. Cyanosis indicates the presence of desaturated haemoglobin. Due to the nature of the oxygen-haemoglobin dissociation curve, this desaturation will only occur in the most severe cases of respiratory compromise where life-threatening hypoxaemia exists (less than 80% saturation of arterial blood). It is also important to note that cyanosis cannot be observed in markedly anaemic animals as at least 5g/dL of desaturated haemoglobin (corresponding to a PCV < 15%) must be present for cyanosis to be visible.

Patients with respiratory distress are often most 'fragile' shortly after presentation to the hospital, owing to the stress of transportation and handling. Additional stress due to handling and diagnostic testing should therefore be kept to a minimum to prevent a potentially fatal decompensation in the patient's condition. In many cases, initial stabilisation should consist simply of minimal handling and supplemental oxygen therapy. Sedation may be considered at this stage in patients with severe distress or anxiety.

While the temptation exists to obtain a diagnosis immediately in the dyspnoeic patient, aggressive pursuit of diagnostic tests can be fatal. Simply positioning an animal for radiographs may cause sufficient stress and loss of postural adaptive responses sufficient to cause respiratory arrest. In such cases, careful observation and auscultation of the patient may usually be performed with minimal stress. This can be performed with concurrent oxygen administration and will allow the clinician to shorten his/her differential diagnosis list. The benefits of employing empirical treatments, such as bronchodilators and diuretics almost always outweigh the risks in such situations. If pleural space disease is suspected, then thoracocentesis (with or without ultrasound guidance) may also be life saving and has a low complication rate if performed appropriately.

In patients with marked respiratory distress, intubation to provide 100% inspired oxygen, ventilatory support and to bypass any upper airway obstruction, can be life saving. While this is a big step, to is immensely preferable compared to intubating a patient after respiratory arrest has occurred and it can usually be achieved with minimal doses of anaesthetic/sedative drugs, buying the clinician more time for diagnostics and treatment.

Supplemental oxygen therapy

Oxygen therapy should be provided to all patients with respiratory distress until it is no longer required. The simplest method is to use a face mask however as the majority of animals do not like having their face covered this may prove too stressful for most. Any stress will negate any potential benefit of oxygen therapy and may worsen the patient's condition further. In such cases it is preferable to hold the circuit or tube without the face mask close to the mouth or nose of the patient.

Holding the tube at right angles to the patient is usually better tolerated than having it angled directly at the animal's face. This method is easy, cheap and effective and may allow initial assessment to be carried out.

Nasal catheter placement or nasal prongs provide another simple method although the former can take longer to institute and isn't suitable for oxygen provision during initial patient assessment. For nasal catheter placement, the nasal mucosa should be anaesthetised using topical proxymetacaine 5-10 minutes prior to placement if possible. The tube (red rubber catheter or feeding tube) should be pre-measured to the medial canthus of the eye before insertion into the ventral nasal meatus, as if placing a naso-gastric feeding tube. Once successfully placed, it should be sutured as close to the nose as possible to prevent displacement. The amount of oxygen delivered via this method will depend on the oxygen flow rate and whether the animal is open-mouth breathing but may be up to 60%. Unilateral catheter placement may be sufficient, or bilateral catheters can be considered in severely affected cases.

Personal oxygen tents can be constructed using buster collars and cling-film. It is important that space is left to prevent carbon dioxide build up or overheating, although the space around the collar is often sufficient. Although purpose built oxygen cages may provide even high levels on inspired oxygen, their use is often limited by expense and the physical space they take up. Polyethylene cage door fronts may be used to make a lower cost yet practical alternative. A cage also has the possible benefit of providing a quiet space for the patient in which they are not being handled which may limit their stress and subsequent personal oxygen consumption. The limiting factor however with most oxygen cages or tents is that they can take time to fill with oxygen and levels subsequently rapidly decrease when they are opened for patient handling or treatments. Considering nasal methods of oxygen delivery, which can be provided continuously, may be more cost efficient and practical in most settings once patients are more stable if continued oxygen therapy is required.

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, and 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 more thorough diagnostic evaluation than would be tolerated conscious in cases where initial response to therapy has been poor and more information is needed for ongoing treatment.

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 such as those listed below.

Where none of these techniques are possible, a needle cricothyroidotomy can be considered as a life-saving measure. 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 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. 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 as 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. The inspired concentration of oxygen should be kept as low as is necessary to maintain patient comfort and oxygenation. Long-term therapy with high concentrations of oxygen ($FiO_2 > 0.6$ for more than 12 hours) may cause lung injury due to free radical damage, worsening respiratory function further. In some cases, 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.

Anatomic origin of respiratory distress

If the initial history does not provide specific information as to the cause of the respiratory distress (e.g. trauma, history of previous medical conditions such as cardiac or respiratory disease, etc), the approach should be to treat the patient according to the likely site of the respiratory pathology.

A combination of the history, signalment and physical examination is often sufficient to determine which areas of the respiratory tract are involved. The respiratory tract may be divided into several sections as outlined below.

1. Upper airways

The upper airways include the nasal passages, larynx, pharynx, and the extra-thoracic trachea. Common differential diagnoses for upper airway conditions causing respiratory distress include laryngeal paralysis or spasm, collapsing trachea, BOAS (brachycephalic obstructive airway syndrome), nasopharyngeal polyp, pharyngeal oedema or inflammation, retropharyngeal lesions (abscess, mass, haematoma), foreign body and neoplasia. Clinical signs of upper airway disease include the presence of stridor or stertor, increased respiratory effort over the inspiratory phase of breathing with normal expiration, and possibly a history of dysphonia (a change in bark). On auscultation, upper airway noise is loudest over the trachea although referred sounds may be audible throughout the thorax which may limit detection of additional respiratory noises such as crackles in the presence of concurrent aspiration pneumonia. Most dogs with dynamic upper airway obstructions (brachycephalic airway disease, laryngeal paralysis) have stridor or stertor primarily on inspiration. Animals with fixed upper airway obstructions (such as masses, foreign bodies or abscesses) tend to display signs on both inspiration and expiration.

Dyspnoea in animals with an upper airway obstruction will likely be worsened significantly by any exercise, excitement or anxiety. Any increase in inspiratory drive results in enhanced negative inspiratory pressures causing more severe narrowing of the airway. All stress should therefore be minimised and consideration given the administration of a sedative medication such as butorphanol (0.2 mg/kg IM/IV) or acepromazine (0.01 mg/kg IM/IV) to prevent further deterioration in the patient's condition.

In addition to the reduction in ventilation and hence oxygenation, upper airway obstructions may lead to additional complications. These include hyperthermia due to the loss of a major thermoregulatory mechanism, non-cardiogenic pulmonary oedema due to the extreme negative pressure that the alveoli are subjected to and patient stress, all of which may further compromise respiratory function. Patient cooling may be necessary to prevent complications of heatstroke and further worsening of respiratory function due to an additional increase in respiratory drive. This may be best achieved by wetting the patient's coat with water and the application of a fan. Very cold water and ice should not be used as this may cause peripheral vasoconstriction and limit heat loss from the body. Intubation of patients with upper respiratory dyspnoea may be particularly beneficial as a way of temporarily bypassing any obstruction and allowing return of normal ventilation. Emergency tracheostomy may be necessary if an airway cannot be secured by alternate means.

2. Lower airways

Disease of the lower airways resulting in respiratory distress typically involves the small bronchi and bronchoconstriction, and is inflammatory in origin. Clinical signs consistent with lower respiratory tract involvement include acute onset respiratory distress, often with a history of cough, intermittent respiratory signs and possibly exercise intolerance. The cough is usually harsh and non-productive. If the cough should be found to be productive (soft and moist, followed by expectoration or swallowing when material is expectorated into the pharynx), a different localisation of the problem should be considered and include possible pneumonia. On auscultation, patients with lower airway disease will frequently have wheezes and increased expiratory effort to their breathing.

The most common cause of lower airway disease is feline asthma although dogs may rarely present with chronic bronchitis, defined as a persistent cough in the absence of any other pulmonary disease. Initial therapy includes the provision of oxygen and bronchodilators (terbutaline 0.01 mg/kg IM/IV, aminophylline 5-10 mg/kg IV dogs). As most affected animals are thought to have an allergic/inflammatory component to their bronchoconstriction, a single anti-inflammatory dose of corticosteroid (dexamethasone 0.1 mg/kg IM/IV) may also be considered.

3. Pleural space

Accumulation of air, fluid or soft tissue in the pleural space may result in respiratory compromise as lung expansion becomes reduced, decreasing tidal volume and resulting in atelectasis. Patients with pleural space disease may be observed to have a short, rapid, shallow respiratory pattern as they attempt to maintain normal ventilation with a reduced lung capacity. Alternatively, a dysynchronous respiratory pattern, combined with dull lung sounds may be present.

Pleural effusions, such as pyothorax, chylothorax, congestive heart failure or neoplasia, may be suspected based on respiratory pattern and the presence of dull or absent sounds when ausculting the ventral thorax. By contrast, pneumothorax may be suspected if lung sounds are dull dorsally. In some cases of diaphragmatic herniation gut sounds may be ausculted within the thorax. Depending on the underlying cause, additional physical examination findings such as pyrexia may be present.

In any patient with suspected pleural space disease, thoracocentesis may be both diagnostic and life saving. There is a low complication risk, although cardiac/vena cava puncture may occur or a pneumothorax may develop (usually subclinical). Thoracocentesis may usually be performed successfully with minimal or no sedation in animals with enough pleural space disease to cause overt respiratory distress. To perform a thoracocentesis:

- Oxygen should be provided during the procedure
- The thorax should be clipped ventrally for fluid and dorsally for air
- The area should be cleaned with surgical scrub and spirit although surgical asepsis is not necessary
- The choice of needle is dictated by patient size and space occupier; in cats a butterfly needle is usually sufficient whereas in larger dogs it may be easier, and quicker, to use a longer larger gauge catheter or needle

- The needle or catheter should be inserted at rib space 7-8, 1/3 way from the bottom for fluid and 1/3 way down from the top for air (or at the point of dullness or as guided by ultrasound)
- The needle should be placed cranial to the rib, to avoid blood vessels and nerves running along the caudal aspect of each rib
- A 3-way tap should be used to allow repeated drainage into a syringe while not allowing air to leak into the chest

Fluid samples should be stored in a sterile container for culture (aerobic and anaerobic) and in EDTA for cytology. If air or fluid is rapidly re-accumulating in the chest after thoracocentesis has been performed, then chest drains may be placed to allow for repeated or continuous drainage. MILA chest drains tend to work well in the majority of patients and can be placed under sedation allowing a more rapid recovery in comparison to trocar type drains that require general anaesthesia for placement. The MILA chest drains are placed using a Seldinger technique (over the wire approach), can be maintained for several days and as one size can be used in many patients limit the need to have several sizes of chest drain in stock at any time.

4. Pulmonary parenchyma

Pulmonary parenchymal disease is a common cause of respiratory distress and includes cardiogenic and non-cardiogenic pulmonary oedema, pulmonary contusions, bacterial bronchopneumonia (aspiration or infectious), neoplasia, pulmonary thromboembolism, inflammatory lung disease and acute respiratory distress syndrome (ARDS).

Clinical signs may include increased respiratory rate and effort, nasal discharge, cough (which may be productive) and signs of hypoxaemia (pale or cyanotic mucous membranes, tachycardia, weakness and lethargy). Patients with pulmonary parenchymal disease will often show a restrictive pattern to their breathing with short shallow breaths or a specific pattern may not be evident. Depending on the severity of hypoxaemia, there may be paradoxical respiration present. Careful systematic auscultation may reveal harsh lung sounds and/or pulmonary crackles. The location of any abnormal lung sounds can be informative in narrowing down the most likely underlying cause. Examples of this include:

- Non-cardiogenic oedema (caused by electrocution, prolonged seizure activity, choking) is usually most evident in the caudodorsal lung fields
- Aspiration or other infectious pneumonia tends to affect the cranioventral lung fields and right middle lung lobe most commonly
- Cardiogenic pulmonary oedema in dogs is typically most evident in the peri-hilar region initially, before progressing to affect all lung fields. In contrast, cardiogenic pulmonary oedema in cats tends to be more diffuse.
- Pulmonary haemorrhage, contusions and lungworm all tend to vary in their location.

Careful evaluation of the heart for the presence of a murmur and pulse palpation to detect arrhythmias are useful to suggest congestive heart failure as a cause in dogs. A heart rate over 200 beats per minute would also be consistent with the presence of a cardiac arrhythmia, warranting cardiac evaluation. Cats may present with significant cardiac dysfunction and with no evidence of a cardiac murmur or gallop rhythm. Cardiac disease in cats can therefore not be excluded as a cause of respiratory distress by the absence of these clinical findings. Nasal discharge may be present in patients with pneumonia or in cases of severe pulmonary oedema. A thorough history can also be very helpful in determining the origin (e.g. history of trauma, electrocution, history of vomiting etc). Should cardiogenic oedema be suspected, frusemide should be administered at a dose of 1-2 mg/kg IM/IV. A clinical effect should be seen within 20-30 minutes and the dosing repeated up to a maximum of 10 mg/kg in 24hrs and renal function assessed when possible. A continuous rate infusion of frusemide may be considered in refractory cases or those receiving long term diuretic therapy (0.1-1 mg/kg/hr). Attention should also be paid to body positioning in patient with pulmonary disease. Simply changing the position of the patient from lateral into sternal recumbency will significantly improve oxygenation and may make the difference between a case that can be managed in practice vs one where referral for more aggressive care is required. Antimicrobial therapy should be started early if there is strong clinical indication of an underlying infective aetiology although there will be a lag between treatment and clinical improvement. There are no proven treatments for pulmonary contusions or non-cardiogenic pulmonary oedema beyond supportive care. Cases suspected to have lungworm should be treated with a licenced product and consideration given to the administration of a single anti-inflammatory dose of dexamethasone to limit worsening in respiratory status associated with inflammation from larval die-off.

Thoracic radiographs will frequently reveal alveolar disease, the distribution of which can be very informative as to the origin of the problem as described above. In some cases, airway sampling via tracheal wash or bronchoalveolar lavage (BAL), or further imaging, may be necessary to confirm the diagnosis. Whilst airway sampling may provide valuable information as to the presence of an infectious underlying cause and associated antimicrobial sensitivity results, the procedure may temporarily worsen patient oxygenation. This may increase morbidity significantly and prolonged or more intensive oxygen therapy be required at additional cost. It may be preferred to reserve airway sampling for cases that fail to respond to typical medical management following discussion with the owner.

5. Chest wall disease

Chest wall disease resulting in respiratory distress is most frequently due to trauma and is evident on initial physical examination. Solitary or multiple rib fractures may be present. When there are multiple rib fractures, a (pseudo)flail chest may result with a freely mobile area of the chest wall which moves paradoxically to the rest of the chest. Whilst this has the potential to impair a patient's ventilatory function and oxygenation, in most cases concurrent pulmonary contusions or pneumothorax may be the greater cause of respiratory distress.

Treatment consists of oxygen therapy and adequate analgesia to ensure normal chest excursions, and thoracocentesis as necessary. Rarely does the flail segment require specific intervention although surgical stabilisation may be required in severe cases.

Any disease otherwise affecting the integrity of the chest wall or neuromuscular function may also result in respiratory distress. Penetrating the thoracic wounds should be managed with initial oxygen therapy and coverage of the defect with a sterile dressing that still allows air to escape on inspiration. Following initial stabilisation, surgical exploration of penetrating wounds is necessary to repair the defect and treat concurrent thoracic trauma present.

6. Abdominal distension

Any disease resulting in abdominal distension may cause anterior displacement of the diaphragm, leading to respiratory compromise and possible pulmonary atelectasis. Common causes of marked abdominal distension include gastric-dilation or volvulus, large volume ascites, intra-abdominal masses and heavy pregnancy. Where possible, abdominal decompression should be performed via treatment of the underlying condition to improve respiratory function.

7. Respiratory look-a-likes

Panting or increased respiratory rate/effort is a commonly encountered clinical sign. It is important to remember that this sign does not always equate to an abnormality in the respiratory system and may represent a central increase in respiratory drive. Potential reasons for this include anaemia, compensation for metabolic acidosis, steroid or opioid administration, fear/pain/stress/anxiety, pyrexia, cardiovascular instability or central nervous system disease. These patients will have normal oxygenation as assessed by pulse oximetry or arterial blood gas analysis, and have no abnormalities on either thoracic auscultation or radiography.

Additional diagnostics

Whilst there are many options for further diagnostics in the workup of patients with respiratory distress, a targeted approach may negate their need or focus in on that with the most relevance to a particular patient. There should always be a risk to benefit ratio assessment performed when considering performing tests in a dyspnoeic patient given the potential to worsen their condition. A diagnostic thoracocentesis should always precede and likely replace the need for radiographs in patients suspected to have pleural space disease.

The use of patient-side thoracic ultrasound (TFAST) is gaining popularity and may be a cost effective, minimally invasive, means of patient assessment. The 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 rule out the presence of fluid in the pericardial space. Despite variation in sensitivity and specificity between experienced and less experienced sonographers, the negative predictive value of the TFAST examination is high, meaning the detection of the glide sign rules out the presence of a pneumothorax at the site of the transducer.

The TFAST examination can be performed with the patient in right, left or sternal recumbency, which is of benefit in a patient with respiratory distress. There is also no requirement for the patient to be clipped prior to the assessment. The TFAST examination involves 5 views of the thorax. These include bilateral placement of the ultrasound transducer at the chest tube site 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. The transducer must be held still against the thorax in order to maximise the chance of detecting the glide sign. Bilateral placement of the transducer at the pericardial site 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 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.

TFAST terminology:

- Glide sign: at the chest tube site, the pleura line is visible as a white line running between 2 ribs, usually just distal to the ribs. 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 pleural line. Its presence rules out the presence of a pneumothorax at that location.
- 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 pleural line. They are present as a result of reverberation artefact and may be seen in patients both both and without pneumothorax.
- 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 the A lines without fading. They are reverberation artefacts from the visceral pleura. The move in a to-and-fro fashion with inspiration and expiration synchronously with the glide sign. 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 lung rockets. When these are seen in a trauma patient they are considered diagnostic for pulmonary contusions unless proven otherwise. B lines are absent in the presence of a pneumothorax.

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 when 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.

If available, patient-side 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 congestive heart failure as a cause of the respiratory distress. Although currently the subject of discussion amongst cardiologists, the consensus appears to be that it is best measured in the right parasternal short axis 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. This test may be performed briefly at the patient-side, thereby minimising stress yet providing valuable clinical information.

N-terminal pro-brain natriuretic peptide (NT-proBNP) is a cardiac biomarker that is commercially available as a patient test. Increases in NT-proBNP are seen in cases of myocardial stretch and in renal disease and blood concentrations have been shown to be useful for differentiating cardiac disease from respiratory disease in cats with respiratory distress. This assay may be useful in increasing clinical confidence in cardiac disease as the underling cause of dyspnoea but requires venepuncture in an otherwise unstable patient. It is likely a case where the risk versus benefit ratio should be carefully considered.

Additional diagnostic testing may be considered depending on the case and the most likely differential diagnoses based on clinical assessment. These may include coagulation testing, lung worm testing (snap test, faecal smear or faecal Baermann), or infectious disease testing depending on the patient's travel history.

A simple approach

Although the logical anatomic approach presented here will likely provide an economical short cut to the most likely cause of respiratory distress, it is probable that some cats will prove difficult to localise and their condition will limit further diagnostic evaluation. In these cases, it is recommended to treat them empirically for the most common and most treatable underlying causes with the aim being to stabilise their condition somewhat to allow further assessment. The conditions that are covered with this approach are congestive heart failure and feline allergic airway disease. Drug therapy that should be administered to these cases is described as 'triple combination therapy' and includes:

- Frusemide 1-2 mg/kg IM
- Dexamethasone 0.1 mg/kg IM
- Terbutaline 0.01 mg/kg IM

Cats presenting with marked respiratory distress may also be administered a low dose of sedation in order to decrease anxiety and stress, and so decrease respiratory drive and oxygen consumption. A low dose of an opioid such as 0.1 mg/mg morphine IM or 0.1 mg/kg butorphanol IM could be considered.

Should the patient's condition fail to improve in anyway following drug administration, the likelihood of these more treatable conditions as an underlying cause decreases and the situation warrants a serious discussion with the owner regarding the other possibilities and the medical and financial involvement that will be required to pursue treatment further.

Monitoring the respiratory patient

When treating a patient with respiratory distress, the most useful monitoring tools include physical examination findings including respiratory rate and effort, respiratory auscultation, patient comfort and postural reactions, and mucous membrane colour. Whilst, in addition to these findings, other monitoring techniques may be employed to determine a patient's response to treatment including pulse oximetry, end-tidal capnography, and arterial blood gas analysis results, these should be considered in relation to the overall clinical picture.