Practical Emergency Techniques for Advanced Practitioners Mini Series

Session Three: Metabolic challenges

Lindsay Kellett-Gregory BSc(Hons), BVetMed(Hons), DACVECC, MRCVS
Lecturer in Emergency & Critical Care Medicine

Systematic approach to venous blood gas analysis

Alterations in acid base status are common in emergency medicine and in hospitalised critically ill patients. They can be easily assessed via a venous blood gas analysis, and can provide vital information on patient stability, the underlying disease process and subsequent treatment options. With the more widespread use of portable patient side blood gas analysers, the application of venous blood gas analysis in veterinary medicine is becoming more common.

Significant alterations in acid base status can be life threatening. Blood pH is closely regulated to remain within a narrow range (7.35-7.45) as any derangement can cause damage to proteins and alter normal cellular function. This can manifest in marked patient deterioration, and additional morbidity and mortality. Patients with acid base alterations frequently have altered cardiovascular function, changes in the neurologic status, changes in respiratory function, and even an altered response to various drug therapies. The signs of acid/base disturbances are usually vague and unable to be detected clinically, making blood gas analysis essential.

An additional benefit of running a venous blood gas analysis in an emergency patient is that the results can directly inform the clinician of the most likely differential diagnoses. The results are also typically obtained very rapidly to allow case progression quickly.

Basic Definitions

An acid is a molecule that donates a hydrogen ion, while a base accepts one. A buffer is a weak acid or base, and helps to protect against large changes in pH. The primary extracellular buffer is bicarbonate. Intracellular buffers are phosphate, proteins and hemoglobin, and bone also acts as a buffer. pH is the measure of acidity/alkalinity, and is equal to negative the log of the H^+ concentration. Acidaemia is a blood pH less than 7.35, while alkalaemia is a blood pH of greater than 7.45. Acidosis and alkalosis refer to the process that is causing the pH disturbance. There are 4 basic types of disturbances:

1. Metabolic acidosis – a primary gain in acid or loss of base
2. Metabolic alkalosis – primary gain in base or loss of acid
3. Respiratory acidosis – retention of CO_2 due to alveolar ventilation not keeping up with CO_2 production
4. Respiratory alkalosis – removal of more CO_2 by ventilation than is being produced

There are a few other key terms to remember when interpreting blood gases. PaO_2 is the partial pressure of oxygen dissolved in the arterial blood. It is a measure of oxygenation, not ventilation. Normal PaO_2 is 90-100 mm Hg. A PaO_2 of less than 80 mm Hg is considered to represent hypoxemia, and less than 60 mm Hg represents a severe compromise to tissue oxygenation.

$PaCO_2$ is the partial pressure of carbon dioxide dissolved in the arterial blood. It gives the best measure of the patient's ability to ventilate. It is important to remember that CO_2 is approximately 20 times more diffusible than O_2, making it much easier for a patient to maintain normal CO_2 concentrations. The $PaCO_2$ is evaluated to determine if there is a respiratory acidosis or alkalosis. A $PaCO_2$ of greater than 45 mm Hg is a respiratory acidosis and a $PaCO_2$ of less than 35 mm Hg is a respiratory alkalosis. The base excess/deficit (BE) is a calculated value that reflects the metabolic portion of the acid/base balance. It takes into account all of the body's buffer systems and is an estimate of how much base needs to be added or taken away from the system to achieve a normal pH at a normal temperature. It is used to evaluate for a metabolic acidosis or alkalosis. For dogs, if the BE is greater than 4 mmol/L, a metabolic alkalosis is present, if the BE is less than –4 mmol/L, a

metabolic acidosis is present. Cats have been reported to have slightly lower values for their PCO_2 and a wider range for BE than dogs.

Normal Values for Blood Gases

Canine:

	Arterial	Venous
pH	7.35-7.45	7.35-7.45
PO_2 (mm Hg)	90-100	30-42
PCO_2 (mm Hg)	35-45	40-50
HCO_3^- (mmol/L)	20-24	20-24
BE (mmol/L)	-4-+4	-4-+4

Feline:

	Arterial	Venous
pH	7.34 ± 0.1	7.30 ± 0.08
PO_2 (mm Hg)	102.9 ± 15	38.6 ± 11
PCO_2 (mm Hg)	33.6 ± 7	41.8 ± 9
HCO_3^- (mmol/L)	17.5 ± 3	19.4 ± 4
BE (mmol/L)	-6.4 ± 5	-5.7 ± 5

Interpreting Blood Gases

Step by step process for interpreting blood gas samples:

1. Determine if it is a venous or arterial sample. Ideally, a venous sample should be taken from a central catheter or direct jugular venipuncture, to get the best representation of the global acid-base and respiratory status. Either can be used to evaluate the overall acid-base status with the exception of severe shock and post-arrest situations, in which cases large discrepancies may exist between arterial and venous samples. Poor tissue perfusion can result in huge increases in CO_2 and secondary decreases in pH on the venous side despite low to normal CO_2 on the arterial side. Although some general information can be gained about respiratory function from a venous sample, only an arterial sample can be used to truly assess respiratory function. If unable to obtain an arterial sample, use pulse oximetry to measure oxygen saturation and a venous sample to evaluate acid-base status and estimate ventilation.
2. By evaluating the pH, determine if acidaemia or alkalaemia is present.
3. If acidaemia is present, determine if it is respiratory or metabolic in origin. If the $PaCO_2 > 45$ mm Hg, it is respiratory. If the BE < -4 mmol/L (or $HCO_3^- < 21$ mmol/L), it is metabolic.
4. If alkalaemia is present, determine if it is respiratory or metabolic in origin. If the $PaCO_2 < 35$ mm Hg, it is respiratory. If the BE > 4 mmol/L (or $HCO_3^- > 27$ mmol/L), it is metabolic.
5. Evaluate for any compensatory changes that may have occurred. For example, if a primary metabolic acidosis is present, a compensatory respiratory alkalosis may be present. Remember the rules of compensation:
    a. A change in the respiratory or metabolic component of the acid-base status will normally induce an opposite compensatory response in the other to try to bring the pH back to normal.
    b. The lungs can compensate very quickly by changing minute ventilation in a matter of minutes.
    c. The kidneys compensate much slower, starting after a few hours with maximum compensation requiring 4-5 days.
    d. The absence or presence and degree of compensation gives some idea of the chronicity of the disturbance. See Table 1.
    e. Overcompensation does not occur!

6. Assess the oxygenation – the PaO_2 should equal approximately 5 times the inspired oxygen concentration (FiO_2). The FiO_2 of room air is 21%.

These rules apply to the normal values listed above for dogs. For cats, the reported normal values for PCO_2 and BE should be substituted into steps 3 and 4.

Table 1: Expected compensatory changes:

Disorder	Primary Change	Compensatory Response
Metabolic acidosis	↓ HCO_3^-	0.7mm Hg decrease in PCO_2 for each 1mEq/L decrease in HCO_3^-
Metabolic alkalosis	↑ HCO_3^-	0.7mm Hg increase in PCO_2 for each 1mEq/L increase in HCO_3^-
Acute resp acidosis	↑ PCO_2	1.5mEq/L increase in HCO_3^- for each 10mm Hg increase in PCO2
Chronic resp acidosis	↑ PCO_2	3.5mEq/L increase in HCO_3^- for each 10mm Hg increase in PCO2
Acute resp alkalosis	↓ PCO_2	2.5mEq/L decrease in HCO_3^- for each 10mm Hg decrease in PCO2
Chronic resp alkalosis	↓ PCO_2	5.5mEq/L decrease in HCO_3^- for each 10mm Hg decrease in PCO2

Other Useful Equations for Interpreting Blood Gases

Other important equations for evaluating blood gases include a derivative of the Henderson-Hasselbalch equation, the equation for calculating the alveolar (A) to arterial (a) oxygen difference, or A-a gradient, and the anion gap (AG) equation. The derivative of the Henderson-Hasselbalch equation is the following:

$$HCO_3^- + H+ \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$$

This equation is essential to remember for two reasons. The first is that the concentration of HCO_3^- will be affected by CO_2, which is why evaluating the BE is the preferred method for determining the metabolic portion of an acid/base disturbance. For example, a patient with a respiratory acidosis (increased CO_2) will have an increase in HCO_3^- because of this equation. This could be misinterpreted as metabolic compensation. However the BE, because it is independent of CO_2, is a better way to evaluate the acid/base status, and may show no compensation or even a concurrent metabolic acidosis that would have been missed by evaluating the HCO_3^- alone. The second is that administration of exogenous bicarbonate, as in sodium bicarbonate, will cause an increase in CO_2, which can lead to a respiratory acidosis if the patient is unable to increase minute ventilation.

The A-a gradient equation (A-a gradient = $P_AO_2 – P_aO_2$) is useful in determining lung function, or the ability to oxygenate normally, while removing the effects of changes in minute ventilation. This gradient is measured by calculating the ideal alveolar PO_2 (P_AO_2), then subtracting the measured arterial PO_2 (P_aO_2) taken from our arterial blood gas. The value for P_AO_2 is calculated using the ideal alveolar gas equation:

$$P_AO_2 = [PB – PH_2O]\ FiO_2 \ – \ (P_aCO_2/ R)$$

PB = atmospheric pressure
PH_2O = the partial pressure of water
FiO_2 = the concentration of oxygen in inspired air
R = the respiratory quotient (approximately = 0.8)

A simplified equation can be used for patients breathing room air at sea level:

$$P_AO_2 = 150 – 1.1[P_aCO_2]$$

It is very important to remember the limitations of this equation – if the patient is at an elevation that causes significant reduction in atmospheric pressure, or is on supplemental O_2, this short-cut cannot be used. Normal values for the A-a gradient are 5-15 mm Hg. Values above 20-25 mm Hg are

definitely abnormal and signify ventilation/perfusion mismatch in the lungs. Hypoventilation will not cause an abnormal A-a gradient.

Another equation of value for interpreting lung function is the P_aO_2/FiO_2 ratio. This allows arterial blood gases on different concentration of inspired oxygen to be evaluated and compared. A normal P_aO_2/FiO_2 ratio is approximately 500 (P_aO_2=100 mm Hg, FiO_2=0.21). If a patient is on 100% O_2, their expected P_aO_2 would be 500 mm Hg.

Calculation of the anion gap (AG) is helpful in determining the presence of a mixed disturbance, and is helpful in differentiating the causes of metabolic acidosis. The anion gap is the difference in the sum of the commonly measured cations and the commonly measured anions.

$$AG = (Na^+ + K^+) – (Cl^- + HCO_3^-)$$

In the patient, there is no anion gap because electroneutrality is always maintained. This is due to the presence of unmeasured anions (UA) and cations (UC) that maintain the balance. Therefore:

$$AG = UA – UC = (Na^+ + K^+) – (Cl^- + HCO_3^-)$$

The majority of the unmeasured anions are the plasma proteins, with albumin contributing a larger portion than the globulins. The unmeasured cations are calcium and magnesium, which normally do not vary enough to cause appreciable changes in the AG. Decreases in AG are most commonly associated with hypoalbuminemia. Increases in AG are seen with many causes of metabolic acidosis, such as ethylene glycol or salicylate intoxication, ketoacidosis, uremia, and lactatic acidosis. Normal anion gap acidosis is often referred to as hyperchloremic acidosis as the AG is maintained by increases in the Cl^- when HCO_3^- is decreased. This is seen with diarrhoea, renal tubular acidosis, administration of carbonic anhydrase inhibitors and acidifying agents such as NH_4Cl and oral $CaCl_2$.

Treatment of acid base disorders

Treatment of a metabolic acidosis should always be aimed at correcting the underlying problem. In patients with an increased anion gap acidosis, unmeasured organic anions such as ketones or lactate can be metabolised to HCO_3^- during recovery. If the metabolic acidosis is due to lactate, appropriate volume resuscitation should be sufficient to correct the pH. Bicarbonate administration should be considered when there is a severe persistent metabolic acidosis (pH < 7.1) due to a cause other than lactate. Severe acidosis can lead to vasodilation and hypotension, arrhythmias, decreased cardiac contractility, increased respiratory effort, mental dullness, and insulin resistance. Sodium bicarbonate administration should only be considered in cases of refractory acidosis or in cases with evidence of cardiovascular compromise secondary to the metabolic acidosis. Caution should be taken when administering bicarbonate as it can cause a secondary respiratory acidosis if the patient is unable to blow off excess CO_2 that will form. Sodium bicarbonate administration is also associated with hypernatremia, paradoxical CNS acidosis caused by diffusion of CO_2 into the CSF, development of alkalosis as organic anions are metabolised, hypokalemia as K^+ ions shift back into the intracellular spaces as H^+ ions move out, shifting of the oxyhaemoglobin curve to the left (making it more difficult for haemoglobin to unload oxygen to the tissues), hyperosmolality, and decreased serum ionised calcium concentration caused by increased binding of Ca^{2+} to albumin.

To calculate the amount of sodium bicarbonate to give, the following equation is used:

$$\text{Bicarbonate deficit} = \text{BE} \times \text{body weight (kg)} \times 0.3$$

Usually one quarter to one third of the dose is given, either as a slow bolus over 5-10 minutes or in IV fluids over 1-2 hours. Alternatively, 1-2 mEq/kg of sodium bicarbonate can be given over a similar time period if a severe metabolic acidosis is strongly suspected and blood gas analysis is not available. Careful monitoring of pH, HCO_3^-, BE, and CO_2 should occur when bicarbonate is administered.

Treatment of metabolic alkalosis should also be aimed at correcting the underlying cause. The most common causes of metabolic alkalosis in veterinary patients are gastrointestinal obstruction with loss of H^+, K^+, and most importantly Cl^- in the vomitus, and frusemide administration. In patients with GI losses of Cl^-, treatment should be aimed at correcting intravascular volume and Cl^- concentrations and eliminating the GI obstruction. These patients are often hypokalemic as well. The fluid therapy of choice is 0.9% NaCl because of its high Cl^- concentration with KCl supplementation once any boluses, if indicated, have been completed. If the alkalosis is due to diuretic administration, it will usually correct itself once the diuretic therapy has been discontinued or reduced to lower levels and the patient is eating again.

Respiratory acidosis can rapidly become a life-threatening disorder and needs to be recognised and treated immediately. Causes can range from an airway obstruction to any impairment of the normal respiratory pathway from the respiratory center in the brain stem, the cervical spinal cord, neuromuscular disease, restrictive disorders preventing lung expansion such as pleural space disease, to severe primary pulmonary disease such as pneumonia. Treatment is aimed at correcting the underlying problem, particularly airway obstruction or restrictive disease, but in the meantime, intubation and support with positive pressure ventilation is often indicated. Respiratory alkalosis can be caused be a variety of disorders including hypoxaemia, pulmonary disease, central nervous system disease, as well as exercise, stress, and pain. Treatment consists of correcting the underlying cause.

Nutritional support of the critically ill patient

Identification of patients in need of nutritional support
Hospitalised veterinary patients are often malnourished or found to be receiving inadequate diets. One study of veterinary patients showed that provision of adequate calories was only achieved 27% of the time in 276 hospitalised dogs over a total of 821 days. Malnutrition in people is associated with increased complication rates, an increased length of hospitalisation and an increased mortality. Similarly, poor outcomes in veterinary patients receiving inadequate nutritional support can be expected. Compared with that in human medicine, there is a dearth of data describing the impact of nutritional support on hospitalised veterinary patients. However, one study showed that hospitalised dogs and cats that received less than one third of their target energy requirements were more likely to have a poor outcome.

There are some guidelines in the literature as to when we should start nutritional support in our patients. Nutritional support should be considered for any animal that has been anorexic or has had inadequate voluntary caloric intake for ≥ 3 days, has lost ≥ 10% of their body weight or has other signs of malnutrition (e.g. poor hair coat, muscle wasting, poor wound healing, hypoalbuminemia, or lymphopenia). Nutritional support also should be considered in patient with predisposing conditions such as vomiting, diarrhoea or liver disease, prior to the development of overt malnutrition. Pre-emptive feeding tube placement is also recommended in patients where complete or partial anorexia can be expected (e.g. facial or jaw surgery, invasive surgery etc.) and can often be done at the time of general anaesthesia for initial therapeutic or diagnostic procedures. It is strongly recommended that no patient go without food for a period of time greater than 5 days. It is also notable that it is not necessary for a patient to have a full clinical diagnosis prior to the provision of nutritional support which can be considered a supportive therapy along the same lines as fluid therapy for example.

The provision of adequate nutritional support of even more important in critical illness where patients are usually in a hypermetabolic state. This is characterised by increases in circulating cytokines, catecholamines, and other stress mediators. These ultimately result in an inflammatory

response with undesirable effects including increased protein catabolism and impaired healing ability. There is also a decrease in immune function and impaired gastrointestinal tract integrity which may increase the likelihood of bacterial translocation and sepsis. There may also be serious adverse effects of intermediary drug metabolism and ultimately there may be impaired overall recovery from the illness.

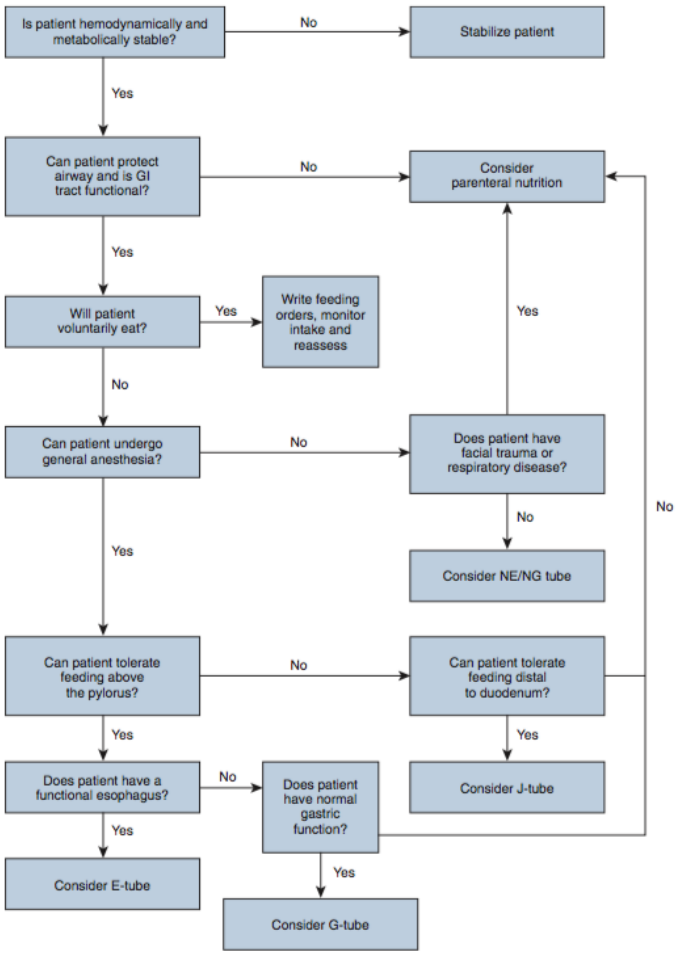
Whilst there are few veterinary studies assessing the effect of nutritional support on outcome following critical illness, those published provide good support for its provision. A recent randomised controlled clinical trial investigated the effect of early enteral nutrition on intestinal permeability, intestinal protein loss, and outcome in canine parvoviral enteritis. Dogs were randomised into 2 groups: 15 dogs received no food until vomiting had ceased for 12 hours (mean 50 hours after admission; NPO group), and 15 dogs received early EN by naso-oesophageal tube from 12 hours after admission (EEN group). All other treatments were identical. The results of the study showed that body weight increased insignificantly from admission in the NPO group whereas the EEN group exhibited significant weight gain and there was earlier clinical improvement in the EEN group. Although there was no significant difference in survival in groups, the study was likely inadequately powered to be able to show any difference present between groups given the overall high survival rate. There was also some evidence that patients in the EEN group had improved gut barrier function which could limit bacterial or endotoxin translocation.

Two additional retrospective studies also provide evidence for nutritional support in veterinary medicine. One study demonstrated a positive association between energy intake and hospital discharge. Animals with a lower body condition score had a greater mortality in this study. The second study made a positive association between dogs with septic peritonitis receiving early (defined as within 24 hours post-operatively) enteral or parenteral support and a shorter length of hospitalisation.

Enteral feeding

Overall, when the gastrointestinal tract is functional, enteral nutrition is usually preferable to parenteral nutrition, as it is a simpler, more economical, and has fewer complications. There are also the advantages that it directly supports enterocytes and preserves the mucosal barrier thereby 'preventing' bacterial translocation, preserves normal gastrointestinal immunologic function, and is more physiologically sound by stimulating motility, secretions and neuroendocrine pathways. There are several routes by which enteral feeding may be provided in the hospitalised patient in which voluntary intake is inadequate. Force or syringe feeding is not recommended due to concerns for causing food aversion, increasing the risk of aspiration pneumonia, and ultimately failing to provide adequate daily calories requirements due to a lack of patient tolerance. The enteral feeding route in general should not be used whilst a patient is haemodynamically or metabolically unstable. Efforts should instead be directed towards patient stabilisation to improve patient outcome and increase the likelihood of tolerance of enteral feeding when it is introduced.

A decision-making tree for selecting the most appropriate enteral feeding route is provided below.



Naso-oesophageal (NO) and nasogastric(NG) feeding tubes

NO feeding tubes have historically been recommended over NG tubes because the risk of gastric reflux has been thought to increase if the tube passes through the lower oesophageal sphincter, compromising sphincter competence. However, one small retrospective study failed to demonstrate a difference in recorded complication rates between a group of dogs fed via NO versus NG tubes. NG tubes have the additional advantage that they may allow for gastric decompression and measurement of gastric residual volumes and are being use more commonly at the present time. A silicone or polyurethane tube of an appropriate size (3.5-5 Fr in cats, 5-8 Fr in dogs) is used. Nasal feeding tube placement is a good option for patients that are poor candidates for general anaesthesia as they are usually placed conscious or rarely require a light sedation. Facial trauma or coagulopathy may preclude placement of this type of tube, and it should not be used in patients with respiratory disease because it may exacerbate respiratory compromise by occluding a nares. Any patient that receives enteral nutrition must have a functional GI tract and the ability to guard the airway if vomiting or regurgitation occurs.

A nasal feeding tube is easy to place. The tube is premeasured from the nose to the last rib (NG) or xiphisternum (NO) before it is placed into the ventral nasal meatus as previously described when placing nasal oxygen cannulae. If difficulty is encountered when the tube has been advanced to the level of the pharynx, the patient may be encouraged to swallow by repositioning the neck ventrally and stroking the area. The tube should be secured in place as previously described.

Radiography is considered to be the gold standard and radiographic confirmation of correct placement for all tube types and is highly recommended to prevent inadvertent delivery of nutrition into the respiratory tract. It is important to note that even properly placed nasal feeding tubes can appear to be in the airways on a lateral radiograph because the oesophagus overlies the trachea ad bronchi on caudal cervical and thoracic radiographs. Evaluating other areas can help in the event of any difficulty interpreting the images. Additional measures that may be considered should radiography be unavailable include measurement of $ETCO_2$ from the tube (should be zero from the GI tract), suction to check for negative pressure or retrieval of gastric contents if the tube is correctly placed in the GU tract, and/or visualisation of the course of the tube using a laryngoscope (although this step likely requires anaesthesia). The presence of a cough following tube placement and even after instillation of a small volume of sterile saline is not a reliable marker to determine whether the tube is placed correctly. The tube should not be used for the provision of nutritional support until the correct placement has been verified.

Because only small-bore tubes are used, diet selection is limited to liquids. NO and NG tubes are best for short-term (7 to 14 days) feeding because they can be irritating to some patients and they are not as versatile as other feeding tube types where prolonged nutritional support is required. An Elizabethan collar and close patient monitoring are both required to reduce of the risk of premature tube removal by the patient. In addition, sneezing or vomiting may dislodge the tube, requiring reassessment for correct placement. Complications associated with NO or NG tubes include epistaxis, rhinitis, sinusitis, dacryocystitis, inadvertent placement or dislodgement of the tube into the airway, oesophageal irritation, reflux, or clogging of the tube, and small intestinal diarrhoea secondary to feeding of a liquid diet. In general complications are not common.

Gastric decompression

Gastric decompression may be performed following placement of a NG tube. A NG tube may be placed primarily for this purpose in patients with severe ileus and a fluid filled distended stomach seen on abdominal ultrasound in order to improve patient comfort and decrease the occurrence of vomiting and regurgitation which may lead to aspiration pneumonia. Decompression may be the only effective intervention in patients with ileus whose clinical signs have failed to improve with standard medical management including pro-kinetic therapy. NG tube placement also permits the measurement of gastric residual volumes (GRV), defined as the amount of contents aspirated from the stomach via the tube prior to feeding. The practice of monitoring GRV and using it as a measure of gastric emptying and tolerance of enteral feedings has become commonplace in many human and veterinary intensive care units. Drainage should be performed with the primary goal of improving patient comfort and is likely unnecessary unless the GRV exceeds 10 ml/kg. If the patient GRV is large, it is recommended that half the volume be given back each time to avoid severe electrolyte and acid base disturbances occurring. It is common practice that the GRV be checked prior to bolus feeding but should also be checked very 4 hours even in a patient receiving feeding via constant infusion. Electrolytes should be checked in these patients also.

Oesophageal feeding tubes

Oesophageal tubes are likely the most versatile and important feeding tube in small animal patients and are placed from the cervical to caudal oesophagus. Their larger bore (typically 12-20 Fr) in comparison to nasal feeding tubes permits the use of both liquid and blenderised soft foods and some medications. They do require, albeit a short, full general anaesthesia which may not be ideal or practical in some emergency patients. They are more indicated for long term (>1 week to 3-4 months) in hospital or at home nutritional support.

The technique for correct tube placement is described as follows:

- The patient must be under general anaesthesia with endotracheal intubation and in right lateral recumbency
- Clip and aseptically prep the left cervical region from the angle of the mandible to the mid-cervical region and the wing of the atlas to the trachea
- Choose a 12 (small cats), 14 (cats and small dogs), or 16 to 20-French (dogs > 15 kg) feeding tube
- Determine the length of the tube to be inserted by measuring the tube from the 7th intercostal space (tip of the tube) to the point where the tube will exit the skin (just distal to the hyoid apparatus and dorsal to the jugular groove)
- Advance curved forceps (such as curved Carmalts) through the mouth into the proximal oesophagus and direct the curved tip laterally
- Palpate the tip of the forceps externally in the mid-cervical region, over the site of intended insertion. Using a number 11 scalpel blade, make a small (5 mm) skin incision over the tip of the curved forceps.
- Push the forceps laterally to expose the oesophagus over the tips of the forceps through the skin incision and using the scalpel blade make a very small nick in the oesophagus over the tip of the forceps
- Gently push the tip of the forceps through the oesophagus and skin incision
- Grasp the distal end of the tube with the forceps and pull it into the oesophagus and out of the mouth such that the distal end of the tube extends out the oral cavity and the proximal end out the cervical incision
- Being careful not to pull the proximal end of the tube through the skin incision, redirect the distal end of the tube posteriorly (down the oesophagus) with fingers or forceps. The proximal end of the tube will rotate in a cranial direction as the distal portion of the tube moves down the oesophagus. Gentle traction on the proximal end may be needed in order for the necessary ‘flip’ of the tube to occur
- Advance the tube to the premeasured length and secure in place with a purse-string suture (around the incision and tube) and "Chinese finger trap" suture. The skin around the tube insertion site should be cleaned with dilute chlorhexidine to prior to suturing to reduce the risk of infection of the insertion site with bacteria from the lumen of the oesophagus that the tube has previously come into contact with
- Place a small amount of antibiotic ointment over the tube exit site and cover with a light dressing. The end of the tube should be capped to prevent aerophagia and the tube clearly labelled as being an oesophageal tube.

More recently, a normograde minimally invasive technique for oesophagostomy tube placement in cats has been described. In this technique, angled or curved forceps are introduced through the mouth into the proximal oesophagus under general anaesthesia. The jaws of the forceps are opened and the tips palpated under the skin in the middle cervical area dorsally to the jugular vein. A 14 G over-the-needle catheter is then inserted following a normograde direction in between the forceps tips directly into the oesophagus. The needle is then removed and a small diameter feeding tube introduced through the sheath and pushed into the distal third of the oesophagus. The feeding tube is secured in place as described above.

Once an oesophageal tube has been placed, and prior to recovery from anaesthesia, a lateral thoracic radiograph should be taken to ensure the distal end of the tube lies in the distal third of the oesophagus and does not encroach on or cross the lower oesophageal sphincter. The tube should also be checked along its length to ensure there are no kinks or twists in the tube path that may cause patient discomfort or difficulties during feeding.

The most common complication of oesophageal tube placement, is inflammation or infection of the stoma site. The insertion site should be inspected daily. Minor redness and discharge around the stoma site is normal, especially for the first 1-2 days after placement. Premature or inadvertent tube dislodgement is also common. Vomiting may displace the distal end of the tube into the oral cavity. If the patient vomits, a lateral thoracic radiograph is recommended to confirm tube placement prior to resumption of tube feeding. Animals have been known to bite off and swallow the end of a dislodged tube. Alternatively, patients may dislodge tube through biting, rubbing or scratching at stoma site or circumferential wrap. If clinical signs of oesophagitis or vomiting are observed, a lateral thoracic radiograph is indicated to evaluate for potential distal migration of the tube across the lower oesophageal sphincter, in which case repositioning of the tube may be required.

Tube feeding can occur as a continuous infusion (generally liquid diets to hospitalised patients) or as 4-6 bolus feedings per day. Bolus feedings should be warned to body temperature by resting the filled syringe in a warm water bath. For bolus feeding through oesophagostomy (or gastrostomy tubes), the tube is aspirated with a syringe prior to instillation of food. If residual food is aspirated it is returned to the patient and the volume of the scheduled feeding decreased by an equivalent amount. If residual food volumes are persistent or prevent feeding full RER, pro-motility agents should be considered. A total volume of 5-10 mL/kg/feeding is usually well tolerated, although animals that were eating normally before tube placement (e.g. post-op facial surgery) or animals that have been chronically tube fed may tolerate slightly larger volumes. The tube should be flushed with 5-10 mL of water after each feeding to reduce the risk of clogging and blockage.

Alternative feeding tubes

Gastrostomy or jejunostomy feeding tube are alternative feeding tubes that can be used to provide enteral nutrition to small animal patients with dysfunction of the GI tract proximal to the site of placement. Neither are typically placed on an emergency basis, are more invasive to place requiring more technical skills, and can be associated with more severe complications.

Calculating energy requirements

For any kind of nutritional support provided, a patient's resting energy requirement (RER) should be calculated. This is calculated using either of the following formulae:

$$70 \text{ (body weight kg)}^{0.75}$$

$$30 \text{ (body weight kg)} + 70$$

Whilst historically illness factors (multiples of the RER typically ranging from 1.1-2.5 x RER) were applied to the calculated RER when deciding how many calories to give a patient, there is no proven benefit of doing so and this is no longer recommended. The application of illness factors in most patients is now considered to increase the risk of complications. The exception to this is the nutritional planning of patients with severe catabolic states (such as those with generalised tetanus or severe burns) where the energy requirements are often in excess of those calculated by the RER formulae.

Calculation of the RER is important both to ensure that adequate nutrition is provided, as well as to prevent complications of overfeeding. These are numerous and may include hyperglycaemia (and possible infectious complications related to this), hyperlipidaemia, increased BUN (which can worsen uraemia) or hyperammonaemia, increased carbon dioxide production and respiratory muscle weakness and possible refeeding syndrome depending on the patient's prior history and nutritional intake.

Parenteral nutrition

In patients where enteral feeding is not tolerated due to a dysfunctional gastrointestinal tract, parenteral nutrition may be considered whereby nutrition is provided intravenously, thereby avoiding the gastrointestinal tract completely. Other indications for parenteral nutrition include any other contraindication to enteral feeding such as an inability to protect the airway or cardiovascular instability. It may also be used in patients where enteral nutrition is poorly tolerated and only a low percentage of RER may be delivered in order to increase the level of nutritional support a patient receives until such as time the enteral route may be used more. Parenteral nutrition solutions typically contain amino acids, dextrose and lipid solutions. They are also usually more expensive that enteral feeding alternatives, may be more challenging to manage in practice and are thus usually reserved for cases of convincing gastrointestinal tract failure.

Parenteral nutrition may be either total or partial. Total parenteral nutrition (TPN) solutions are tailor made to an individual patient and meet all that patient's nutrient requirements. They are highly hypertonic (hyperosmolar) and need to be delivered through a central vein (usually jugular catheter) to prevent phlebitis. The production of TPN solutions typically requires access to a nutrition specialist and is therefore rarely applied in veterinary clinical patients at the present time.

Partial parenteral nutrition (PPN) solutions may only meet 40-70% of a patient's energy requirements but have the advantage that they are of a lower osmolality and can be delivered via a dedicated peripheral vein. It is therefore a much more practical option to administer to veterinary patients. The catheter used to deliver PPN must only be used for this purpose and should be placed for this purpose alone. It should also be handled aseptically at all times to reduce the risk of catheter site infections and should not be disconnected from the patient for the same reason. Ready to mix preparations of PPN (such as Kabiven) are now available that are cost effective and have a long shelf life. Although they are not tailor made to the specific requirements of the patient as TPN is, they appear to work well for the majority of patients and are particularly useful when used in conjunction with low levels of enteral nutrition.