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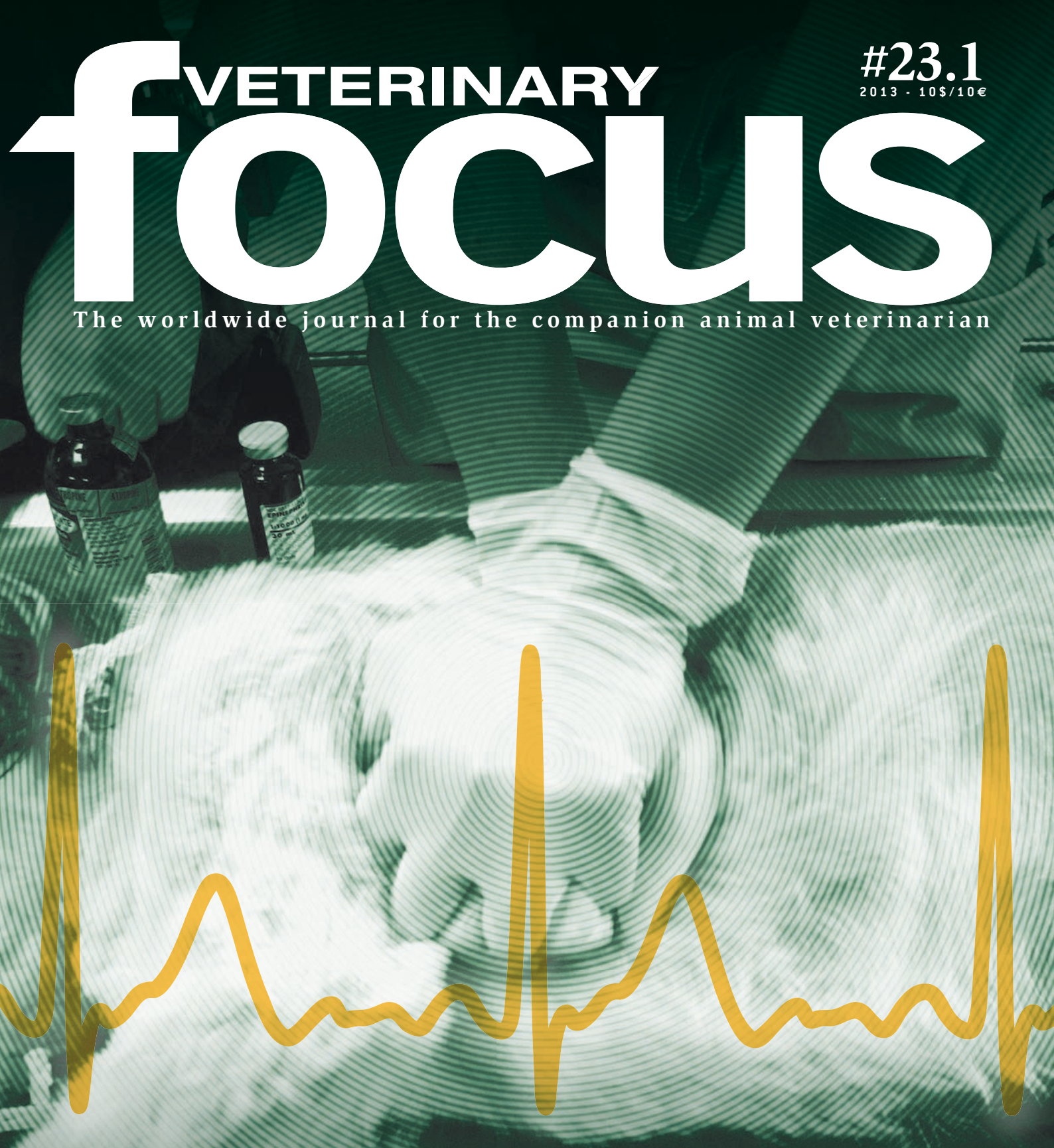

Critical care response

Pulse oximetry and capnography in emergency and intensive care • Making a difference - nutritional support in critically ill patients • Review of emergency consultations • How I approach... Anuria and acute kidney injury • Small animal blood transfusions - a practical guide • Initial assessment of spinal trauma cases • The ABC's of cardiopulmonary resuscitation • Understanding acid base imbalances in dogs and cats

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EDITORIAL

Judging by television schedules, dramas set in hospital emergency departments and critical care units make for compulsive viewing these days. The life-or-death situations, whereby one misguided move can spell disaster, or an inspirational intervention can pull a patient back from what seemed to be certain death, appeals to our sense of theater and base emotions, perhaps because we are all aware that life itself can sometimes be a frail and unpredictable entity. Yet there is no doubt that the areas of emergency and critical care have developed and diversified hugely in recent years, to the benefit of both human and veterinary patients; increased knowledge has gone hand-in-hand with technological advances, so that severely-ill patients now have a far better chance of survival than even a decade ago.

Most veterinarians – not just those employed in specialist care units – will find themselves dealing with emergencies as part of their workload on a regular basis, and many of us find this aspect of our work to be truly fulfilling. As well as being - by definition - totally unrehearsed, real-life emergencies can be less well scripted than the television but are often more dramatic; the artificial narrative and contrived plot of the screen is left far behind by the nitty-gritty of actuality where panic and uncertainty can battle with the clinician's ability to make clear-cut crucial decisions. Whether we enjoy it or not, we all recognize that in a crisis it is our actions which can be the pivotal factor that determines whether a patient lives or dies, and perhaps in this discipline more than any other the veterinarian must be able to act immediately and decisively to be effectual. In this issue of Veterinary Focus we attempt to collate many of the different aspects of critical care, with the intention that clinicians faced with an emergency situation can act sensibly, making rational assessments, intelligent interventions and worthwhile applications, without turning the whole thing into a drama.

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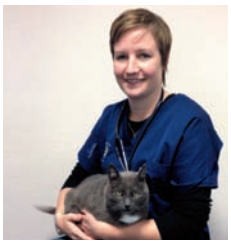
Pulse oximetry and capnography in emergency and intensive care



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Introduction

Technological advances over the last 20 years now enable the rapid and continuous monitoring of an animal's physiological parameters. These developments include pulse oximetry and capnography, and both can now play an important role to monitor companion animals in intensive care situations. Their use enables the clinician to assess and adjust the oxygen supply to the tissues and to maintain the blood pH within values compatible with good tissue function. This article outlines the advantages and limitations of pulse oximetry and capnography in emergency and intensive care to help the practitioner fully utilize these technologies in everyday practice.

KEY POINTS

- **Pulse oximetry provides continuous and non-invasive monitoring of the percentage oxygen saturation of hemoglobin; this allows estimation of arterial oxygen saturation and the arterial oxygen partial pressure.**
- **It is essential to understand the various factors involved in pulse oximetry; this will enable correct interpretation of the readouts.**
- **Capnography provides real time information about the respiratory and cardiovascular systems and enables non-invasive monitoring of the partial pressure of CO_2.**
- **Capnography allows early detection of anomalies linked to the patient or the equipment.**

Pulse oximetry

Principle

Pulse oximetry is a non-invasive method that enables the continuous monitoring of variations in hemoglobin oxygenation ***(Figure 1)***. Pulse oximeters were first developed in 1935 but did not become commercially available until the 1970's (1); the technique essentially involves an optical device that measures the difference in absorption of a wave of light between oxygenated hemoglobin (HbO_2) and non-oxygenated hemoglobin (Hb); HbO_2 absorbs more light in the infrared range (850-1000 nm) than Hb, which absorbs more light in the red wavelength (600-750 nm) (1). The pulse oximeter emits both red and infrared lightwaves across the measurement site (earlobe, interdigital spaces,

tongue, etc.) to a photodetector, which then transmits the signal to the monitor, which in turn uses an algorithm to deliver a numerical value (2).

Pulse oximetry measures the oxygenation percentage of the hemoglobin (SpO_2), which is a reliable approximation of arterial oxygen saturation (SaO_2) (3). This value is then extrapolated to produce a value for the arterial oxygen partial pressure (PaO_2) from the dissociation curve of hemoglobin ***(Figure 2)***. However, it is important to remember that the PaO_2 values corresponding to the read SaO_2 are influenced by the concentration of 2.3 diphosphoglycerate within the erythrocytes, the blood pH, and the body temperature. Normal SpO_2 values (and thus SaO_2) are 96-98%, which under normal physiological conditions corresponds to a PaO_2 of 80-100 mmHg (4).

The signal obtained depends not only on the oxygen saturation of the hemoglobin but also the amplitude of the pulse, which reflects peripheral perfusion. Thus the signal may be influenced by cardiovascular and/or respiratory dysfunction, and it is sometimes difficult to distinguish between them everyday practice (4). Many pulse oximeters now use the plethysmography technique and thus display the pulse amplitude ***(Figure 3)***, which helps the practitioner interpret the numerical values.

Practical application

Pulse oximetry was first used in healthy anesthetized animals and has now become part of the standard minimal anesthetic monitoring protocol (4). However its use is now more widespread, being employed to monitor animals under mechanical ventilation, for the evaluation of oxygenation when admitting animals in emergencies, or for the early detection of hypoxemia in animals hospitalized in intensive care.

There are different types of sensors (clip, cylinder, flat probes, etc.), but clips are the most commonly used and most practical for veterinary medicine (4) ***(Figure 4)***. The sensor should be placed on a smooth, light-colored zone of the patient, and the preferred areas are therefore the tongue, interdigital area, earlobe, axillary or inguinal folds, prepuce, or vulva (4).

To obtain the most accurate signal, it is important to follow a few guidelines (5):

- Choose a site with minimal pigmentation which is warm, thin-skinned, and without any fur. Mucosa is

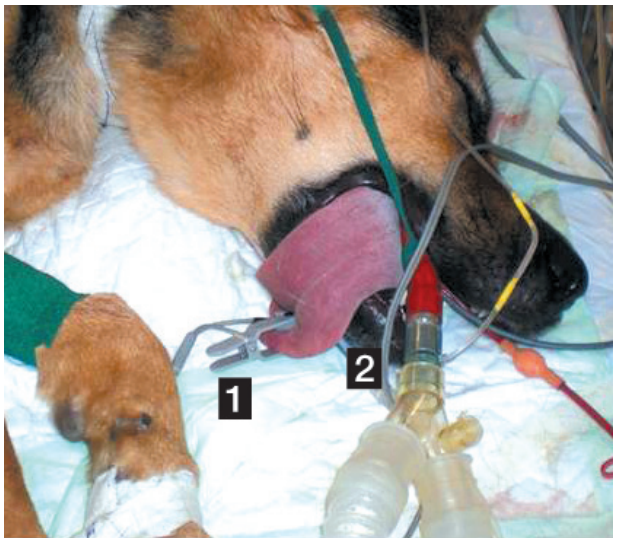

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Figure 1. Oximetry probe (1) on the tongue of an anesthetized and ventilated dog with side-stream capnography sensor (2).

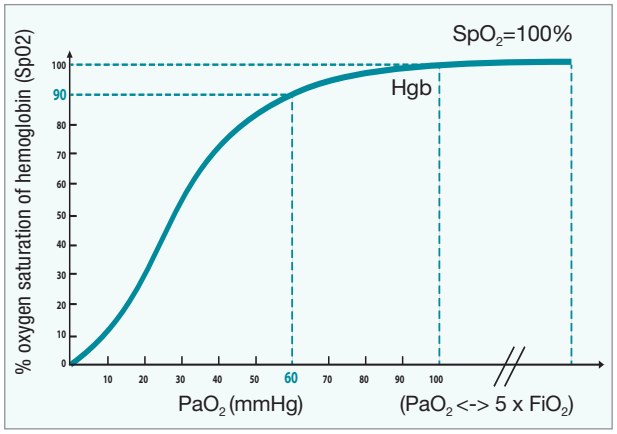

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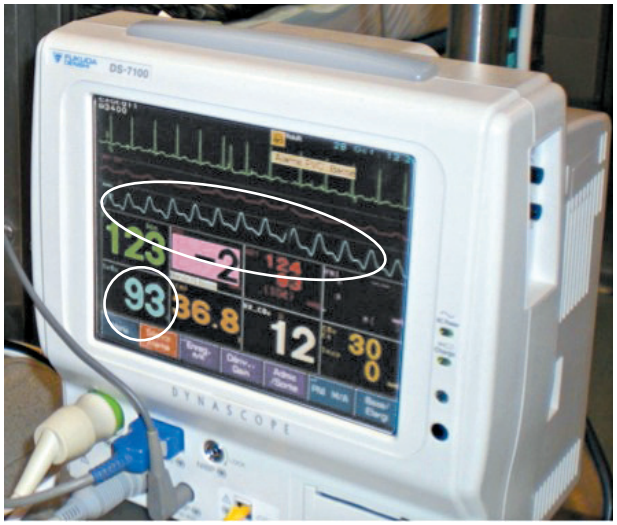
Figure 2. Dissociation curve of hemoglobin.

Figure 3. A pulse oximeter showing the plethysmographic trace (oval ring) as well as numerical values (circled).



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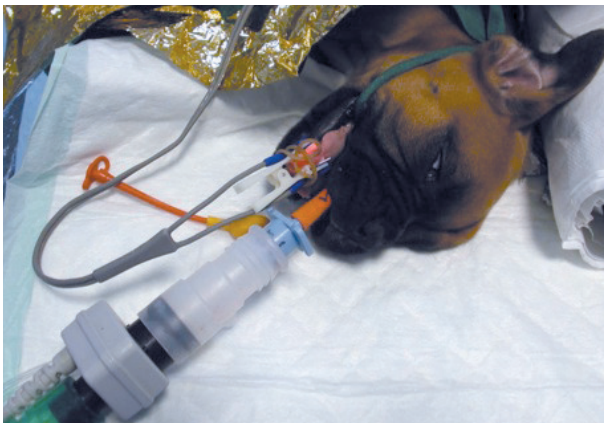

Figure 4. Clips are the most commonly used and most practical sensor for pulse oximetry in veterinary medicine.

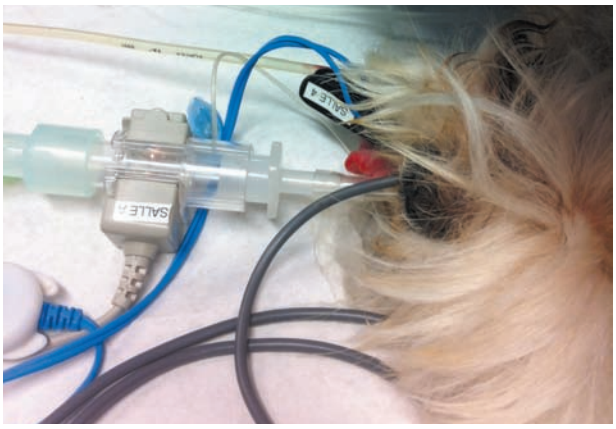

Figure 5. A mainstream capnography sensor.

ideal; if skin is used, the area can be shaved and cleaned with alcohol if necessary.

- Protect the probe from ambient light.
- Keep the animal in a quiet environment.
- Always ignore the first value displayed in pulse oximetry; perform continuous monitoring or make several measurements.
- Ensure that the heart rate given by the pulse oximeter corresponds to the actual heart beat of the patient.
- If the displayed values do not correlate with the clinical examination, confirm the values with an arterial blood gas analysis and repeat the measurements.

The most reliable values are obtained from well-perfused mucosa such as the tongue and the preputial or vaginal mucosa. These sites are very easy to access in an anesthetized animal, but such measurements become more difficult in conscious animals, especially if aggressive or painful. In such cases, it is advisable to use the axillary or inguinal skin folds and remove the probe between each measurement to avoid damage.

If the values obtained are highly variable, if the displayed heart rate is different to the animal's actual heart rate, or if the plethysmography trace has reduced amplitude, it is important to resite the probe.

Advantages and indications

Arterial blood gas measurements provide a more precise measure of blood oxygenation, but pulse oximetry offers the advantage of continuous monitoring (2). Its ease of use and good tolerance, the complete absence of risk for the animal, the low cost, the possibility of bedside monitoring, and the instantaneous display of results all make it very useful for emergency medicine and intensive care (6).

Limitations

To use pulse oximetry correctly, it is important to understand its limitations.

a. Device limitations

The size and shape of the probe can sometimes cause problems, especially in small animals such as cats (4). If the probe is left in place for several days (for example with an animal on ventilation), the resulting heat and pressure can cause tissue necrosis. The probe site, and movements of the animal, can both have a major influence on the values obtained, and as noted above its use can be complicated in conscious animals (6).

b. Technological limitations

The absorption of the light beam is altered by ambient light and by the color of the mucosa. The values obtained are not therefore very reliable in animals with black mucosa.

The technique is not very sensitive for evaluating PaO_2 in patients on oxygen therapy because of the relationship between PaO_2 and the inspired oxygen fraction (FiO_2): in animals with no alteration in gas exchange, the PaO_2 should be five times greater than the FiO_2 ***(Figure 2)*** so that an animal, intubated and ventilated with 100% oxygen, has a PaO_2 of 500 mmHg. Now, according to the dissociation curve of hemoglobin, as long as the PaO_2 is more than 100 mmHg, the SaO_2 is 100%. Thus, the SpO_2 will not detect alterations in gas

exchange if the PaO_2 value varies between 100 and 500 mmHg. The SaO_2 and SpO_2 are only affected if the PaO_2 falls below 100 mmHg (6). It is therefore important to complete the monitoring of blood oxygenation with blood gas measurements for animals on oxygen therapy. As explained earlier, the oximetry signal is highly dependent on tissue perfusion. The signal is therefore often of poor quality and potentially uninterpretable in hypovolemic and/or hypothermic animals with significant peripheral vasoconstriction. The supply of oxygen to the tissues is calculated as being the product of the arterial oxygen content (CaO_2) and cardiac output. CaO_2 depends on the Hb concentration, the SaO_2, and the PaO_2 and is calculated as follows:

$$\mathbf{CaO_2 = ([Hb] \times SaO_2 \times 1.34) + (0.003 \times PaO_2)}$$

It is clear that the hemoglobin concentration plays an essential role in the arterial O_2 concentration. Thus, in anemic animals without pulmonary disease, the SpO_2 is normal and falsely reassuring, despite a low arterial oxygen concentration (linked to the low Hb levels), thus compromising the oxygen supply to tissues.

Finally, pulse oximetry gives erroneous results in the event of qualitative hemoglobin anomalies. The commonly used probes only emit two wavelengths, making it impossible to differentiate between non-functional hemoglobins (carboxyhemoglobin, methemoglobin, sulfhemoglobin, and carboxysulfhemoglobin) and normal hemoglobin (6).

Capnography

Principles

Capnography is the measurement and graphic representation of instantaneous carbon dioxide concentrations during a respiratory cycle (6). Showing the results as a graph provides more information than capnometry alone and it is therefore preferable to choose a monitor that displays the CO_2 concentrations graphically. Capnometry is the measurement of the partial pressure of carbon dioxide (CO_2) present in the inspired and expired gases (7), with the most commonly used value being the CO_2 concentration at the end of expiration, also known as End-Tidal CO_2 ($ETCO_2$).

There are currently several methods for measuring the partial pressure of CO_2 which can be used in emergency and intensive care situations: mass spectrometry, infrared spectrophotometry, Raman spectrometry, and photoacoustic spectrometry. The most widely used is infrared spectrophotometry; this technique relies on the physical principle that gases consisting of molecules of more than two individual atoms have their own specific absorption spectrum in infrared light, which therefore represents their "identity card" (7).

Technically, capnography devices have a measurement cell either in the machine itself or somewhere along the circuit. In the first instance, the machine is known as a "sidestream" capnometer, where a sample of gas is aspirated by a small tube placed as close as possible to the patient's airways ***(Figure 1)***. With the second option, known as a "mainstream" capnometer, the reading cell is integrated into the patient's respiratory circuit, usually between the endotracheal tube and the anesthetic circuit or ventilator (8) ***(Figure 5)***.

Reading a normal capnogram

To interpret anomalies on the capnograph trace, it is important to know what a normal trace looks like. A normal capnogram can be separated into 4 phases ***(Figure 6)*** as follows:

One inspiratory phase

- **Phase 0** corresponds to inspiration. There is a sudden drop in the curve when gases without CO_2 start to enter the upper airways; the baseline then reads zero throughout inspiration.

Three expiratory phases

- **Phase I** corresponds to the start of expiration and thus to the anatomical dead space. No CO_2 should be measured during this short phase.
- **Phase II** corresponds to a mix of gas from the dead spaces and alveoli, provoking a rapid increase in the amount of expired CO_2.
- **Phase III,** or alveolar plateau, corresponds to the emptying of the gas from the alveoli. The maximal concentration achieved at the end of this plateau, shown as a red dot on ***Figure 6***, is the maximal concentration of CO_2 at the end of expiration, or $ETCO_2$, and reflects the alveolar CO_2 concentration.

Since CO_2 is a highly diffusible gas, this value is a reliable and non-invasive approximation of the arterial partial pressure of CO_2 ($PaCO_2$) in healthy animals. Changes in the shape of this trace provide a wealth of information and enable the clinician to make an early diagnosis of cardiovascular and respiratory disorders, even before the oxygen and CO_2 start to fall in the bloodstream.

Interpreting abnormal capnograms

Analysis of the shape of the capnogram curve and the $ETCO_2$ values provides essential information regarding

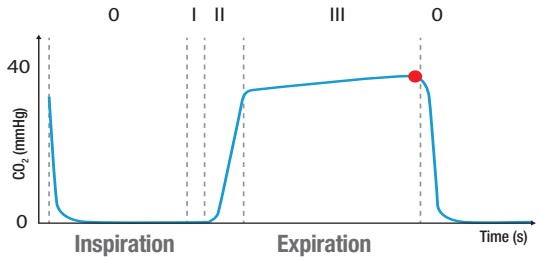

Figure 6. Example of a normal capnogram showing the four phases. The red dot denotes $ETCO_2$, the maximal concentration of CO_2 at the end of expiration.

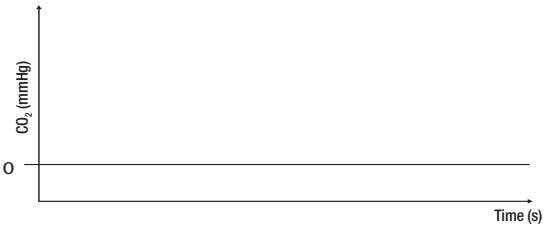

Figure 7. A flat capnograph, which could be caused by esophageal intubation, apnea, cardiorespiratory arrest, or equipment failure (poorly positioned probe, malfunctioning machine).

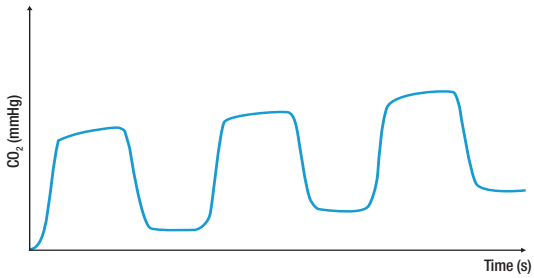

Figure 8. Capnograph demonstrating re-breathing of CO_2: note the baseline does not return to zero. In such cases, the anesthetic circuit or ventilator should be checked, as this is a technical problem.

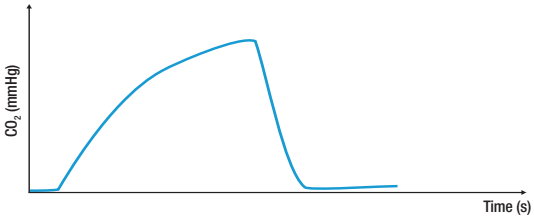

Figure 9. Prolonged duration of phase II associated with an alteration in the slope of phase III compatible with bronchospasm (*e.g.* feline asthma).

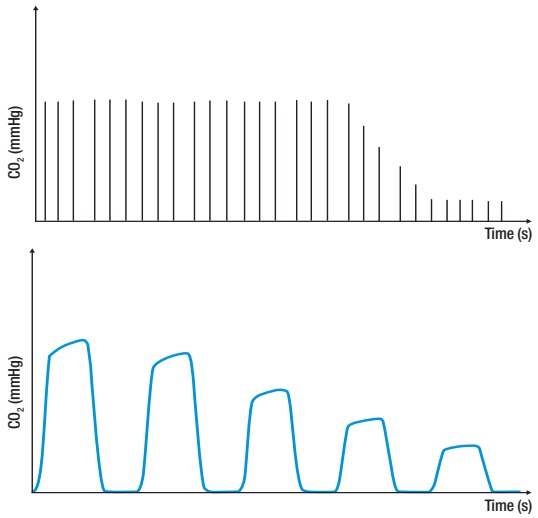

Figure 10. Exponential drop in the $ETCO_2$, *e.g* due to massive pulmonary embolism, significant pulmonary hypoperfusion, or cardiorespiratory arrest may produce capnograph readings as shown above. The general trend (top) and individual curves (bottom) are demonstrated.

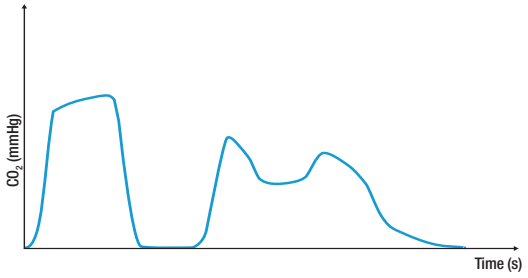

Figure 11. A sudden drop in $ETCO_2$ and irregular shape of the curve which could be caused by the patient becoming disconnected from the circuit, accidental extubation, partial obstruction of the airways, or malfunction of the ventilator.

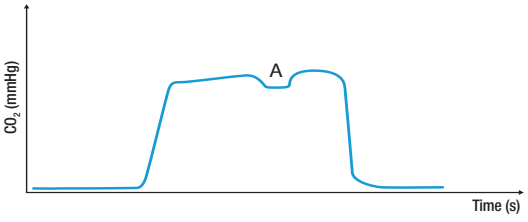

Figure 12. The "curare cleft" seen when a neuromuscular blocking agent wears off; the dip indicates the return of spontaneous inspiration (A) on the alveolar plateau.

the patient's cardiorespiratory function. Modifications can occur in individual phases or in the overall trend of the graph (8). In the majority of cases, the changes affect the alveolar plateau (phase III), the $ETCO_2$ value, or the inspiratory phase (Phase 0).

Diagrammatic representations of the most common modifications and their interpretations are given in ***Figures 7-12,*** and other examples are available at www.capnography.com.

Advantages

Capnography is a simple and non-invasive method for estimating $PaCO_2$, thus avoiding the need for repeated sampling for arterial blood gas analysis. It has thus become an important part of anesthetic monitoring or for animals under mechanical ventilation in intensive care. The $PaCO_2$ estimation provides information about CO_2 production, pulmonary perfusion, alveolar ventilation, respiratory movements, and the elimination of CO_2 by the ventilator (9). At-risk situations for the animal are also detected rapidly, such as obstruction or displacement of the endotracheal tube, respiratory or cardiac arrest, or re-breathing of CO_2 in the circuit. For animals under mechanical ventilation, $ETCO_2$ monitoring enables the detection of alterations in ventilatory parameters (particularly the respiratory rate).

The gradient (a-ET)CO_2 between the $PaCO_2$ (measured on blood gases) and the $ETCO_2$ (measured by capnography) is a good estimation of the alveolar dead space (corresponding to the alveoli that are ventilated but not perfused) (8,9). Under physiological conditions, the $ETCO_2$ is 2-5 mmHg lower than the $PaCO_2$. This normal gradient is due to the disparity in the ventilation/perfusion (V/Q) ratio in the healthy lung. An increase in the (a-ET)CO_2 gradient is indicative of an increase in alveolar dead space secondary to an overly long anesthetic circuit, hypoventilation, obstructive pulmonary disease, reduced cardiac output, pulmonary thromboembolism, or major pulmonary atelectasis (8).

Capnography is also extremely helpful during cardiopulmonary resuscitation. The Reassessment Campaign on Veterinary Resuscitation (RECOVER) emphasizes the importance of capnography in the early detection of cardiovascular deficiency, especially in anesthetized and ventilated animals ***(Figure 10)*** (10).

The $ETCO_2$ is a useful index of pulmonary perfusion and cardiac output in intubated and ventilated animals receiving constant ventilation. Combined with clinical findings, capnography can help with the early detection of cardiorespiratory arrest in these patients ***(Figure 10)*** and also enables the detection of accidental esophageal intubation ***(Figure 7)***. Capnography is also a reliable and effective indicator for cardiopulmonary resuscitation and has prognostic value; resuscitation is more likely to be successful in patients with a higher $ETCO_2$ value (10) and the routine use of capnography is advisable.

Conclusion

There is no doubt that pulse oximetry and capnography have major roles for effective monitoring of companion animals nowadays. A good understanding of the indications and limitations of these two techniques will enable practitioners to reliably monitor their patients, and thus reduce the risks of morbidity and mortality in animals admitted to intensive care.

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Making a difference - nutritional support in critically ill patients

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Introduction

For many years, the major controversy surrounding critical care nutrition was in fact "do critically ill patients actually need nutrition?" In years past (and perhaps even today) provision of nutrition to such patients typically received a very low priority. This problem was later described as "in-hospital starvation" and is most common in the care of elderly human patients (1). When the effects of malnutrition on patient morbidity and mortality were realized, a reactive movement ensued in critical care in the 1970's which coincided with the development and adoption of parenteral nutrition; many patients were fed quite aggressively, leading to the term "hyperalimentation". It was later determined that such an approach, whereby patients received calories well in excess of their needs, led to its own set of complications (2,3). Much like other aspects of critical care, our paradigm of critical care nutrition constantly changes; previously held assumptions become less relevant and new research uncovers novel strategies.

Nutritional support is now considered essential for the recovery of post-operative, critically ill, and injured human patients. Whilst there is convincing evidence of the deleterious effects of malnutrition in people (4,5) the optimal nutritional strategies for critically ill and post-operative animals remain controversial and are largely unknown ***(Figure 1)***. Because malnutrition imparts similar metabolic effects in animals, it is assumed that nutritional support is equally essential for the recovery of critically ill dogs and cats. Although definitive answers regarding the impact of nutritional support on outcome in critically ill animals are lacking, some encouraging results suggest that outcome in hospitalized animals can be enhanced with nutritional support (6-8). From these emerging advancements in veterinary nutrition, and our current understanding of metabolic response to injury, we are beginning to formulate recommendations for the nutritional management of critically ill animals. In fact, with proper patient selection, sound nutritional planning, and careful monitoring, nutritional support can be an integral part in the successful recovery of many critically ill animals.

KEY POINTS

- **Nutritional support can be an integral part in the successful recovery of many critically ill animals.**
- **The optimal nutritional strategies for critically ill and post-operative animals remain controversial and are largely unknown.**
- **Nutrition via a functional digestive system is always the preferred route of feeding.**
- **First steps in instituting nutritional support include restoring proper hydration status, correcting electrolyte or acid-base disturbances, and achieving hemodynamic stability.**

Pathophysiology of malnutrition

One of the major metabolic alterations associated with critical illness involves body protein catabolism, in which protein turnover rates may become markedly elevated (9,10). Whereas healthy animals primarily lose fat when deprived of sufficient calories (simple starvation), sick or traumatized patients catabolize lean body mass when they are not provided with sufficient calories (stressed starvation). During the initial stages of fasting in the healthy state, glycogen stores are used as the primary source of energy. Within days, a metabolic shift occurs towards the preferential use of stored fat, sparing catabolic effects on lean muscle tissue. In diseased states, the inflammatory response triggers alterations in cytokines and hormone concentrations and rapidly shifts metabolism towards a catabolic state. Glycogen stores are quickly depleted, especially in strict carnivores such as the cat, and this leads to an early mobilization of amino acids from muscle stores. As cats undergo continuous gluconeogenesis, this amino acid mobilization is more pronounced than that observed in other species. With continued lack of food intake, the predominant energy source is derived from accelerated proteolysis (muscle breakdown), which in itself is an energy-consuming process. Muscle catabolism that occurs during stress provides the liver with gluconeogenic precursors and other amino acids for glucose and acute-phase protein production. The resultant negative nitrogen balance or net protein loss has been documented in critically ill dogs and cats (11); one study estimated that 73% of hospitalized dogs (including post-operative patients) evaluated in four different veterinary referral centers were in a negative energy balance (12).

The consequences of continued lean body mass losses include negative effects on wound healing, immune function, strength (both skeletal and respiratory muscle strength), and ultimately on overall prognosis. In the context of post-operative patients, this could lead to greater risk of surgical wound dehiscence and post-operative infections (10). Due to the metabolic alterations associated with critical illness, and in part due to an inability or reluctance of many critically ill and post-operative animals to ingest sufficient calories, this patient population is at increased risk for rapid development of malnutrition. Given the serious sequelae of malnutrition, preservation or reversal of deteriorating nutritional status via nutritional support is paramount to minimize the impact of malnutrition and enhance the recovery rate.

Anorexia for as little as 3 days can produce metabolic changes in dogs consistent with those associated with starvation in people (13). However, these dogs would not necessarily exhibit any easily detectable abnormalities on clinical assessment suggestive of being malnourished. Dogs with overt signs suggestive of malnutrition usually have a more protracted period (usually weeks to months) of disease progression. Healthy cats subjected to acute starvation have detectable immune impairment by day 4 and so recommendations to institute some nutritional support in any ill cat that has had inadequate food intake for more than 3 days have been made (14). In both dogs and cats, there is some consensus that there is an urgent need to implement nutritional intervention (*e.g.* placement of a feeding tube) when an animal has not eaten for more than 5 days. The optimal timing of implementing parenteral nutrition in malnourished human patients is currently controversial (15); in animals, recommendations center on the inability to feed enterally, and in most veterinary studies parenteral nutrition has been initiated within the first 4 days of hospitalization.

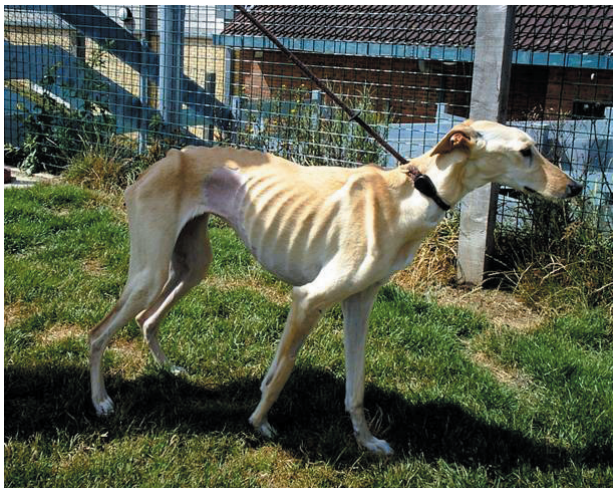

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Figure 1. Critical illness can cause significant lean muscle loss leading to poor body condition and debilitation.

Nutritional assessment and planning

An important factor related to successful management of the critically ill patient involves both selection of animals most likely to benefit from nutritional support and selection of the most appropriate route for providing nutrition. Some animals, *e.g.* obese cats (at risk for hepatic lipidosis) or growing animals may benefit from early intervention. Nutrition via a functional digestive system is the preferred route of administration, and so particular care should be taken to evaluate if the patient

can tolerate enteral feedings. Even if a patient can tolerate only small amounts of enteral nutrition, this route should be pursued. Supplementation with parenteral nutrition should occur only when enteral feeding cannot meet at least 50% the patient's nutritional needs. On the basis of the nutritional assessment, the anticipated duration of nutritional support, and the appropriate route of delivery (*i.e.* enteral or parenteral), a plan is formulated to meet the patient's nutritional needs.

First steps in instituting nutritional support include restoring proper hydration status, correcting electrolyte or acid-base disturbances, and achieving hemodynamic stability. Commencing nutritional support before these abnormalities are addressed can increase the risk of complications and, in some cases, can further compromise the patient (16). It should be emphasized that this is not counter to the concept of "early nutritional support", which has been documented to result in positive effects in several animal and human studies. Early nutritional support advocates feeding as soon as possible after hemodynamic stability is achieved, rather than delaying nutritional intervention by several days (17).

Calculating nutritional requirements

The patient's resting energy requirement (RER) is the number of calories required for maintaining homeostasis while the animal rests quietly. The RER is calculated using the following formula:

$$\mathbf{RER = 70 \times (body\ weight\ in\ kg)^{0.75}}$$

For animals weighing between 2 and 30 kg, the following linear formula gives a good approximation of energy needs:

$$\mathbf{RER = (30 \times body\ weight\ in\ kg) + 70}$$

Traditionally, the RER was then multiplied by a subjective "illness factor" between 1.0-1.5 to account for increases in metabolism associated with different conditions and injuries. Recently, there has been less emphasis on this factor, and current recommendations are to use more conservative energy estimates to avoid overfeeding. Overfeeding can result in metabolic and gastrointestinal complications, hepatic dysfunction, increased CO_2 production, and weakened respiratory muscles. Of the metabolic complications, the development of hyperglycemia is most common, and possibly the most detrimental.

Currently, the RER is used as an initial estimate of a critically ill patient's energy requirements. It should be emphasized that these general guidelines should be used as starting points, and animals receiving nutritional support should be closely monitored for tolerance of nutritional interventions. Continual decline in bodyweight or body condition should prompt the clinician to reassess and perhaps modify the plan (*e.g.* increasing the number of calories provided by increments of 25%).

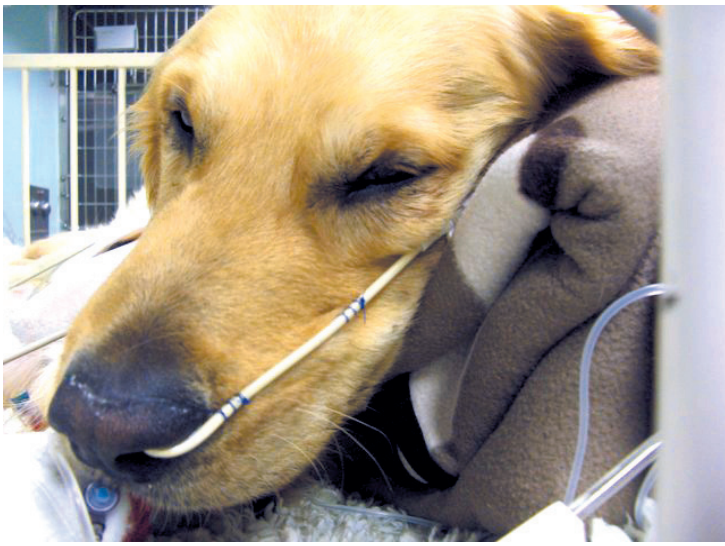

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Figure 2. Feeding tubes have become an important part of managing critically ill animals with poor food intake. This dog is being fed via a nasoesophageal feeding tube.

Implementing the nutritional plan

To implement enteral nutritional support, a feeding tube is typically required ***(Figure 2)***. Placement of a tube is recommended whenever voluntary eating by the patient is lacking in sufficient amounts to meet at least 75% of RER. Considerations for selecting one tube over another should take into account the degree of nutritional support required, the expected duration of support, and the segment of the gastrointestinal tract that must be bypassed as well as other factors such as cost and whether or not sedation/anesthesia is required. Once a feeding tube is in place, a diet preparation that is suitable to meet the nutritional needs of the patient and appropriate for the tube is chosen ***(Table 1)***. Small-bore tubes such as those typically used for nasoesophageal placement, or jejunostomy tubes, require complete liquid diets. Gruel-type diets require larger-bore esophagostomy or gastrostomy tubes and the preparation of these diets may require the use of a kitchen blender. Other considerations for choosing a diet include fat content, protein content, and caloric density of the food (taking into account the effect of dilution if water is added to the preparation). The next consideration involves the manner in which food is delivered; animals with nasoesophageal, esophagostomy, and gastro-

stomy tubes tolerate bolus feedings in which the prescribed amount of food is administered over 15 minutes up to every 4 hours. Animals with jejunostomy tubes are usually fed via constant rate infusions.

Meeting nutritional needs

There is much that remains unclear regarding the nutritional requirements of critically ill animals in general. In certain circumstances assumptions are made that nutritional requirements in animals are comparable to people afflicted with similar diseases. However, it is important to recognize that there may be significant species and disease differences that make direct comparisons or extrapolations less applicable. Experimental data suggests dramatic changes in energy requirements in animals with thermal burns, but there are virtually no clinical data to support this notion. Studies in dogs with thermal burns showed increased energy requirements, accelerated gluconeogenesis, glucose oxidation, lipolysis and increased amino acid oxidation (18). In the absence of definitive data to suggest otherwise, current recommendations are to start nutritional support as soon as it is deemed safe and initially target the RER, but to reassess the patient continually as energy requirements may be more than twice the calculated figure. The goal of nutritional support is to optimize protein synthesis and preserve lean body mass; feeding at least 6-7 g protein per 100 kcal (25-35% of total energy) may be necessary for both dogs and cats. During hospitalization, regaining normal body weight is not top priority, as this should occur once the animal is discharged to complete recovery at home.

Patients with protein intolerance (*e.g.* hepatic encephalopathy, severe azotemia) should receive reduced amounts of protein. Similarly, patients with hyperglycemia or hyperlipidemia may also require decreased amounts of simple carbohydrates and fat, respectively. Other nutritional requirements will depend upon the patient's underlying disease, clinical signs, and laboratory parameters.

When should feeding be initiated?

As alluded to earlier, for many years conventional therapy actually ignored nutritional needs of critically ill human patients. As more and more evidence illustrated the consequences of malnutrition there was a gradual change to ensure that all patients received adequate nutrition. Typical delays in starting nutrition decreased from weeks to 10 days, and now the debate centers on how many hours nutrition should be delayed. As more and more research uncovered the benefits of enteral nutrition and the complications arising from gut atrophy, critical care specialists began feeding patients earlier and earlier in the course of hospitalization with good results (17).

In veterinary medicine a similar transition has occurred in the last 15 years, from the ineffective strategies (such as force- or syringe-feeding, warming foods, adding flavor enhancers) to more recent recommendations for early tube feeding in most, if not all, critically ill patients (19,20). While most can agree that nutritional support is important, and that early intervention is better than delayed, the question remains, how early to intervene? Is it days or hours? The most aggressive approach is to place a feeding tube as soon as possible and start feedings within hours. Is this necessary? Studies in canine patients with parvoviral enteritis, hemorrhagic gastroenteritis and acute pancreatitis support the premise that early nutritional intervention is at least well tolerated and produces few complications (19-21). The lack of any serious consequences to initiating feeding early in these patient populations dispel the myth that

Table 1. Practical considerations for tube feeding in dogs and cats.

1. Select the diet appropriate for the patient's condition. Any canned diet can be used; dry diets may also be used but require more water.
2. Put a given amount of diet in the blender, and calculate the energy content (in Kcal) it represents using manufacturer's information.
3. If necessary add sufficient water to achieve a blend consistency that can be easily pushed through the patient's feeding tube. The volume of water added must be taken into account in any fluid plan.
4. Measure the final volume (mL) of the blend and calculate the energy density of the final blend (Kcal/mL).
5. From RER calculation and feeding plan, calculate the volume (mL) the patient should receive per day, and at each meal.
6. After each meal the tube should be flushed with sufficient water to avoid clogging.
7. The blended food should be stored in a refrigerator; for each new meal the correct volume should be stirred and warmed to body temperature before administering.

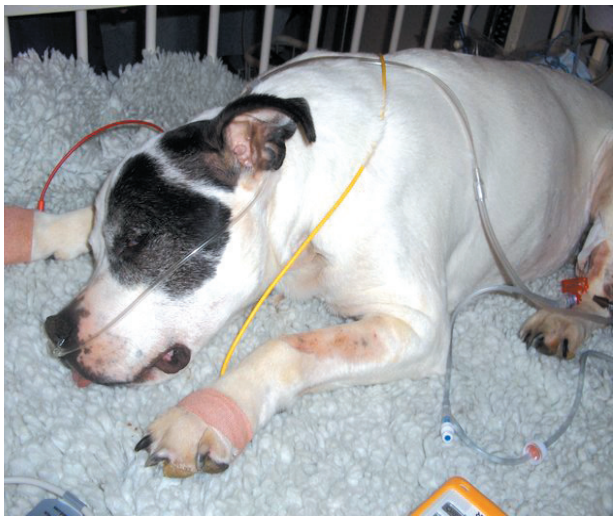

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Figure 4. Critically ill animals are at increased risk of developing malnutrition, and nutritional support can play an important role in their recovery.

feeding early is hazardous. Overall effect on survival is unfortunately beyond these small trials; however, an important point about how early to intervene is that any patient receiving nutritional support should be cardiovascularly stable and hydration, electrolyte and acid-base disturbances should be addressed before nutritional intervention. Also, feeding after tube placement should really be withheld until at least the animal has recovered from anesthesia; feeding a recumbent animal risks aspiration. Patients with compromised gastrointestinal motility (*e.g.* anesthetized patients, patients on opioids analgesics, patients with ileus) are also at risk for complications and should be monitored closely.

Monitoring and reassessment

Bodyweight should be monitored daily in all patients receiving nutritional support. However, the clinician should take into account fluid shifts when evaluating changes in body weight, and so body condition score assessment is also important. The use of the RER as the patient's caloric requirement is merely a starting point; the number of calories provided may need to be increased to keep up with the patient's changing needs, typically by 25% if well tolerated. In patients unable to tolerate the prescribed amounts, the clinician should consider reducing amounts of enteral feedings and supplementing the nutritional plan with either central or peripheral parenteral nutrition.

Possible difficulties with enteral nutrition include mechanical complications such as clogging of the tube (which should be flushed with water after each feed) or accidental removal. Metabolic problems include electrolyte disturbances, hyperglycemia, volume overload, and gastrointestinal signs (*e.g.* vomiting, diarrhea, cramping, bloating). In critically ill patients receiving enteral nutritional support, the clinician must also be vigilant for the development of aspiration pneumonia. Monitoring parameters for patients receiving enteral nutrition include bodyweight, serum electrolytes, tube patency, assessment of tube-exit site, gastrointestinal signs, and signs of volume overload or pulmonary aspiration.

Possible complications with parenteral nutrition include sepsis, mechanical problems with the catheter and lines, thrombophlebitis, and metabolic disturbances such as hyperglycemia, electrolyte shifts, hyperammonemia, and hypertriglyceridemia. Avoiding serious consequences from these complications requires early identification of problems and prompt action. Frequent monitoring of vital signs, catheter-exit sites, and routine biochemistry panels may alert the clinician to developing problems. Should persistent hyperglycemia during nutritional support become apparent, adjustments may be required (*e.g.* decreasing dextrose content in parenteral nutrition) or administration of regular insulin. This obviously necessitates more vigilant monitoring.

With continual reassessment, the clinician can determine when to transition a patient from assisted feeding to voluntary consumption of food. The discontinuation of nutritional support should only begin when the patient can consume approximately 75% of its RER without much coaxing.

Impacting outcomes

Provision of nutrition is usually only considered a "supportive measure", with other interventions (*e.g.* antimicrobials, corrective surgery, glucocorticoid therapy, fluid resuscitation) being more commonly associated in driving patient recovery. One assumption is that energy and substrates only serve to allow the body to repair itself. However, a growing number of veterinary studies have documented significant, clinically-relevant improvements in outcome measures (20,21). A pilot study to assess early enteral feeding in dogs with acute pancreatitis suggested a more rapid resolution of clinical signs compared to those fed parenterally, with both groups being fed an equivalent RER (20). In the recent study evaluating early enteral nutrition in dogs with septic peritonitis, investigators documented a shortened length of hospitalization (6). In terms of food

intake and impact on outcome, one study (7) noted that a voluntary food intake >66% (higher than RER) of maintenance energy needs was associated with a 93% hospital discharge rate whereas animals with a food intake <33% of needs had a 63% discharge rate. Although much work still needs to be done to evaluate the impact of nutritional support in critically ill patients, these studies strongly suggest a positive impact.

Conclusion

While critically ill patients are often not regarded as having urgent need of nutritional support given their more pressing problems, the severity of their injuries, altered metabolic condition, and necessity of frequent fasting place these patients at high risk of becoming malnourished during hospitalization. Proper identification of these animals with careful planning and execution of a nutrition plan can be key factors for a successful recovery ***(Figure 3)***. As our understanding of various disease processes and their interactions with metabolic pathways improve, along with the refinement of nutritional support techniques, there is indeed great optimism that nutrition can have a significant positive impact on the recovery of critically ill patients.

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Review of emergency consultations

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Dr. Lefebvre joined Banfield in 2011 as Associate Medical Advisor - Research for the Banfield Applied Research and Knowledge (BARK) team. A 2003 graduate of Ontario Veterinary College, she obtained her PhD in epidemiology through research and development of guidelines for pet visitation in human hospitals. Her most recent professional role was as scientific editor for *Journal of American Veterinary Medical Association* and *American Journal of Veterinary Research*.

Introduction

The incidence and nature of "emergency" visits in first-opinion veterinary practice is largely unknown. There are 805 Banfield Pet Hospitals which provide general services seven days a week during normal working hours with a focus on preventive care, although patients are also seen on an emergency basis; approximately 0.2% of all visits to the clinics are classified as "emergency" or "urgent". The purpose of the present study was to characterize these emergency visits and determine the annual incidence of specific ailments requiring immediate care.

Method of analysis

Records for all cats and dogs seen at Banfield hospitals in 2011 were used to identify appointments designated as "emergency/urgent" and patients with a diagnosis suggesting an ailment requiring urgent care. Data were summarized by species. When sufficient sample sizes existed (n ≥10,000), associations between various emergency-related diagnoses and species, breed or sex were evaluated with the Fisher exact test (1). Confidence intervals (CIs) were calculated for all estimates, and odds ratios (ORs) were calculated for comparisons.

Results

In 2011 429,682 cats and 2,021,849 dogs were treated at Banfield hospitals; of these 13,658 dogs and 2,150 cats (0.7% and 0.5% respectively of all patients) were admitted on an emergency basis. Dogs were 40% more likely to be brought in for an emergency visit than cats (OR, 1.4; 95% CI, 1.3 to 1.5). The most common presenting complaint for dogs was unspecified poisoning, whereas that for cats was a bite or fight wound ***(Table 1)***.

Several canine breeds were identified as being significantly (P <0.05) more likely to have a diagnosis of animal bite than others, namely the rat terrier (OR, 2.6; 95% CI, 1.0 to 5.4), fox terrier (OR, 2.3; 95% CI, 1.4 to 3.5), Boston terrier (OR, 2.9; 95% CI, 1.0 to 3.4), and Jack Russell terrier (OR, 1.9; 95% CI, 1.0 to 3.3). Sexually intact dogs (OR, 2.6; 95% CI, 2.2 to 3.2) as well as fox terriers (OR, 2.7; 95% CI, 1.7 to 4.0) and pit bulls (OR, 1.5; 95% CI, 1.1 to 2.3) were more likely to been seen after being hit by a vehicle. Of the sexually intact dogs, males were 40% more likely to present than females for automobile injury (OR, 1.4; 95% CI, 1.0 to 1.8).

Breeds most likely to have a vaccine reaction diagnosis were dachshund (OR, 4.2; 95% CI, 2.5 to 6.6), Chihuahua (OR, 2.5; 95% CI, 1.6 to 3.7), and pug (OR, 2.3; 95% CI, 1.0 to 4.7). Only 15 cats seen at an emergency appointment were diagnosed with a vaccine reaction, and there was no significant difference between the proportions of the neutered and sexually intact cats (P=0.20) for this diagnosis.

Various types of toxicoses or poisonings were diagnosed during the emergency appointments ***(Table 2)***. In dogs, chocolate toxicosis was most common, while in cats, it was pyrethroid/pyrethrin toxicosis.

Table 1. Top 5 presenting complaints for canine and feline emergency appointments at Banfield hospitals in 2011.

Complaint by species	No. with complaint	% of all emergency patients	95% CI
Dogs			
Poisoning	1,595	11.7	11.2-12.2
Vomiting or diarrhea	1,166	8.5	8.0-9.0
Bite or fight wound	899	6.6	6.2-7.0
Seizures	857	6.3	5.9-6.7
Hit by car	701	5.1	4.7-5.5
Cats			
Bite or fight wound	122	5.7	4.7-6.7
Vomiting or diarrhea	114	5.3	4.4-6.3
Lameness	105	4.9	4.0-5.8
Poisoning	97	4.5	3.6-5.4
Hit by car	81	3.8	3.0-4.6

Table 2. Top 5 toxicoses diagnosed in all canine and feline patients at Banfield hospitals in 2011.

Toxicosis by species	No. with toxicosis	Cases per 10,000 patients seen	95% CI
Dogs			
Chocolate	1,922	9.5	9.1-9.9
Rodenticide	1,316	6.5	6.2-6.8
Pyrethroid/ pyrethrin	220	1.1	1.1-1.2
Acetaminophen (paracetamol)	115	0.8	0.7-0.9
Plant	94	0.5	0.4-0.6
Cats			
Pyrethroid/ pyrethrin	185	4.3	3.7-4.9
Plant poisoning	33	0.8	0.5-1.1
Rodenticide	29	0.7	0.5-1.0
Ethylene glycol	19	0.4	0.2-0.6
Organophosphate	17	0.4	0.2-0.6

The number of dogs seen as emergencies with foreign bodies was surprisingly low; per 10,000 dogs, there were 39 oral, 6 ocular, and 2 respiratory cases. In cats, the incidence of oral foreign bodies was considerably higher (240 cases/10,000 cats seen), but the number of ocular (five) and respiratory (one) cases were similar to dogs.

Conclusion

The findings reported here are useful in understanding the most common types of emergency visits in general veterinary practice and to help practices prepare for the unexpected. Readers should keep in mind that Banfield hospitals are not open 24 hours a day, so the data reported here cannot be generalized to 24-hour practices or emergency clinics.

Although small numbers of affected patients precluded controlling for potential confounders through multivariate analysis, the univariate comparisons revealed some information that may be useful for client counseling or future hypothesis exploration. The reason certain small breeds were more at risk of vaccine reactions than other breeds is unclear, but these observations are supported by findings in a recent study (2) of vaccine reactions in dogs involving Banfield data. Unlike the results in that study, no significant difference was found between the proportion of sexually intact versus neutered dogs with a diagnosed vaccine reaction in the present study. However, it should be noted that the earlier study included other diagnostic codes such as urticaria and allergic reaction in its outcome definition, and the records used were not restricted to those of emergency appointments.

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HOW I APPROACH...

Anuria and acute kidney injury

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Introduction

Reduced or no urine production (oliguria and anuria respectively) is always an acute, life-threatening situation and can be indicative of, or occur during, other disorders. Oliguria is defined as urine production of <0.5 mL/kg/h or <2 mL/kg/h on infusion therapy; anuria is defined as urine production <0.2 mL/kg/h or unmeasurable.

Emergency diagnosis and therapy

Reduced urine production can be due to pre-renal (shock), renal (acute kidney injury) or post-renal (obstruction) conditions. It is essential that the emergency clinician identifies the source of the oliguria in order to deliver targeted treatment. Hypovolemia, vasodilation or cardiogenic shock are the most likely pre-renal causes. Pre-renal and renal anuria can be differentiated by determining the urine specific gravity (USG); for solely pre-renal cases, canine USG is >1.030 and feline USG >1.035. With renal causes the USG is typically in the isosthenuric range (1.008-1.012). Differentiation is difficult when both pre-renal and renal processes are present, or if therapy has been initiated. Post-renal causes must be identified by subsequent examination.

Clinically, pain is frequently noted on kidney palpation with acute renal processes. In post-renal cases the kidneys or bladder are frequently painful and/or feel distended. When a uroabdomen is present, an ascitic thrill can be detected if there is >40 mL/kg free fluid (1).

Metabolic acidosis, hyperkalemia and anemia are particularly life-threatening for anuric patients. Blood gas analysis with electrolytes and hematocrit (Hct) measurement should be performed.

Hypovolemia

Since pre-renal causes accelerate direct hypoxic kidney damage and acute kidney injury, emergency therapy for these cases consists of treatment of shock and, where applicable, dehydration. In addition to oxygen, patients in shock should receive a bolus of crystalloid solutions (~10-20 mL/kg for 10 minutes), and repeated until the perfusion parameters have been restored. Colloidal infusion solutions are effective in increasing circulating blood volume in the medium term, and medium molecular weight solutions such as hydroxyethyl starch (HAES) are increasingly popular. The

KEY POINTS

- **Pre- and post-renal causes of oliguria/anuria should be identified by means of clinical examination and urine specific gravity.**
- **Depending on the condition, emergency therapy includes treatment for shock, acid-base neutralization and balancing of electrolyte.**
- **After rehydration, diuresis may be stimulated by forced infusion or osmotic diuresis; furosemide for hyperkalemia and overhydration is also recommended.**
- **Vital parameters, including blood pressure, urine production, electrolyte and acid-base status must be closely monitored.**
- **Peritoneal or hemodialysis should always be considered if 12-24 hours of therapy is unsuccessful.**

Table 1. Therapy for hyperkalemia.

Plasma potassium level	Therapy	Mechanism and notes
5.5-6.0 mmol/L	Potassium-free infusion	Dilution + possibly diuresis
6.0-6.5 mmol/L	Additionally • Furosemide 2.0-4.0 mg/kg IV • Hydrochlorothiazide 2.0-4.0 mg/kg IV	Potassium diuresis in the loop of Henle and/or distal tubules
6.5-7.0 mmol/L	Additionally • Insulin 0.1-0.25 IU/kg IM + glucose 1-2 g IV per unit of insulin • Sodium bicarbonate 8.4% 1.0-2.0 mmol/kg IV for 10-15 min	Transports potassium into the cell; regular blood glucose monitoring is required Transports potassium into the cell by increasing pH; regular blood pH monitoring is required
In addition, if there are ECG changes, 10% calcium gluconate can be given at 0.5-1.0 mL/kg IV for 10 minutes, with concomitant ECG monitoring.		

recommended dose is matched to the severity of the shock and should be administered in boluses of 5-10 mL/kg for 10 minutes. Note high molecular weight HAES solutions may cause acute kidney failure; this is less likely with medium molecular weight solutions.

Where higher fluid volumes (~90 mL/kg) are employed but the vital parameters do not significantly improve, additional problems (*e.g.* sepsis) may be present; where applicable, positive inotropes or vasopressors should be given. If dehydration is present (usually from vomiting or anorexia), it should be assessed by mucosal moisture and skin elasticity, and depending on the speed of onset, balanced out over 4-24 hours using crystalloid infusion solutions.

Hyperkalemia

Hyperkalemia can often accompany acute kidney damage. Serum potassium levels >7-8 mmol/L may produce cardiac arrhythmias, bradycardia and even cardiac arrest. Hyperkalemia treatment depends on the severity ***(Table 1)***; initially, an attempt should be made to lower the potassium level using potassium-free infusion solutions. If potassium concentration is 6-7 mmol/L, diuretics that promote potassium excretion (*e.g.* furosemide or hydrochlorothiazide) should also be administered (2). To transport potassium into the cell, short-acting insulin can be given, along with IV glucose to prevent hypoglycemia. Bicarbonate will increase the blood pH and transport potassium into the cells, but if end-tidal carbon dioxide remains high, or alkalosis is already present, bicarbonate is contraindicated. If an ECG indicates bradycardia, an absence of P-waves, widened QRS complex or tall, tented T-waves due to hyperkalemia, calcium gluconate should be given whilst monitoring the ECG. Particularly severe hyperkalemia can be reduced with the aid of hemodialysis or peritoneal dialysis.

Acidosis

Metabolic acidosis is frequently found in acutely uremic patients, and hypovolemia can contribute to this. If bicarbonate levels are <12 mmol/L and blood pH is <7.2 after reversing the hypovolemia, bicarbonate therapy can be given. Initially, a third of the bicarbonate deficit is infused for 20 minutes; if severe acidosis is still present after a subsequent acid-base check, the second, and where necessary, the remaining third of the bicarbonate deficit can be infused ***(Table 2)***.

Anemia

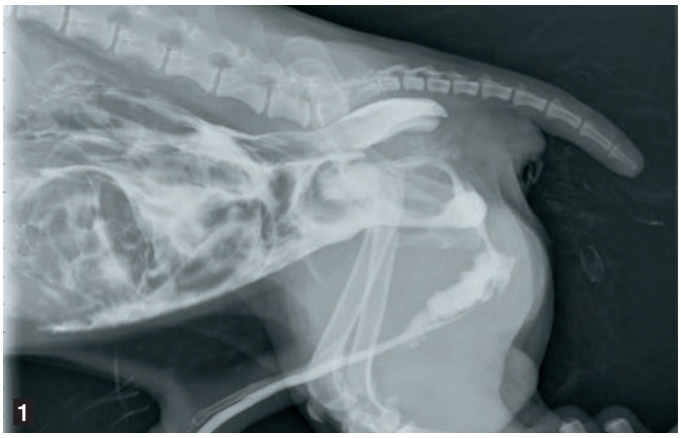
Severe anemia should be treated by transfusing whole blood or erythrocyte concentrate. The transfusion trigger is controversial, but a Hct <20%, tachycardia, polypnea or hypothermia after reversing hypovolemia are all suggested triggers. An average of 2 mL whole blood per kg is required to increase the recipient's Hct by 1%.

Further diagnosis

Initial emergency therapy is followed by additional diagnostic investigations; an in-depth history should be taken, checking for potential access to toxins or drugs and the possibility of leptospirosis or babesiosis, as well as a thorough clinical examination, laboratory diagnosis and imaging investigations, with the aim of identifying the specific cause of the anuria.

Post-renal causes of anuria

Imaging is generally necessary to diagnose post-renal



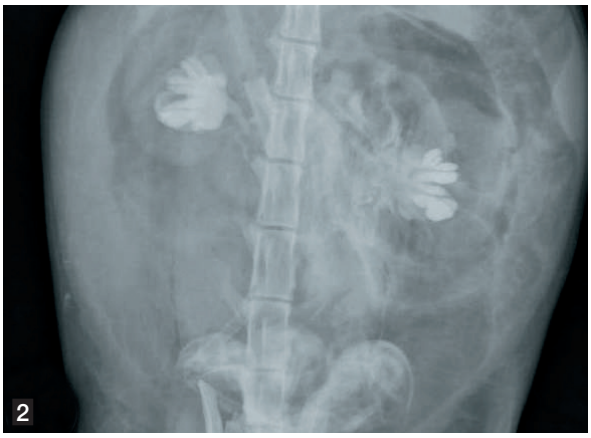


Figure 1. Retrograde urethrography in a dog with a ruptured urethra.
Figure 2. Contrast radiography of a cat with renal damage; note the leakage of contrast agent into the abdomen.

Table 2. Drugs frequently used in uremic/anuric animals.

Indication	Drug	Dose	Notes
Diuresis	Mannitol	0.5-1.0 g/kg IV	For 20 min; do not repeat if anuria persists
Diuresis, hyperkalemia	Furosemide	2.0-4.0 mg/kg IV every 6-12 h	
Hyperkalemia	Insulin	0.1-0.25 IU/kg IM + glucose 1.0-2.0 g IV per unit of insulin	Glucose and potassium monitoring required
	Sodium bicarbonate 8.4%	Dose in mls = base excess (BE) x 0.3 x kg BW	For 15-20 min
	Calcium gluconate 10%	0.5-1.0 mL/kg IV	For 10 min under ECG supervision
Non-regenerative anemia	Darbepoetin alpha	0.45-1.0 µg/kg every 7 days SC	
Vomiting	Maropitant	1 mg/kg SC every 24 h	
	Ondansetron	0.1-0.2 mg/kg every 8-12 h IV	
Gastrointestinal ulcers	Famotidine	0.5-1.0 mg/kg every 12-24 h IV/PO	
	Omeprazole	0.7 mg/kg every 24 h IV/PO	
	Misoprostol	1-5 µg/kg every 6-12 h PO	
	Ranitidine	0.5-1 mg/kg every 8-12 h IV/PO	Effectiveness in dogs questionable
	Sucralfate	Dog: 0.5-1.0 g every 6-10 h Cat: 0.25-0.5 g every 6-12 h	
Prokinetic/anti-emetic	Metoclopramide	0.1-0.4 mg/kg SC every 8 h	Cumulative effect possible
Analgesia	Buprenorphine	0.01-0.02 mg/kg IV every 6-8 h	
	Methadone	0.1-0.3 mg/kg IV/IM/SC every 4 h	
	Fentanyl	0.002-0.005 mg/kg/h IV	
Ethylene glycol intoxication	4-methylpyrazole (Fomepizole)	Dog: 20 mg/kg IV then 15 mg/kg after 12 and 24 h and 5 mg/kg after 36 h IV Cat: 125 mg/kg followed by 31.25 mg/kg every 12 h 3 times IV	Alcohol dehydrogenase inhibitor
	Ethanol (30%)	1.3 mL/kg followed by 0.42 mL/kg/h	Maintain blood alcohol level at one part per thousand
Hypertension	Amlodipine	0.125-0.25 mg/kg every 24 h PO	Ensure blood pressure monitoring

causes. Radiographs should allow measurement of the kidneys and bladder, and any calcium-rich calculi (*e.g.* oxalate) in ureters, bladder or urethra will be visible. Ultrasonography will identify congestion of the renal pelvis or ureters if there is an obstruction, and will image the bladder and urethra, using retrograde contrast as necessary. Contrast radiography studies should accurately identify any ureteral obstructions (3) ***(Figures 1 & 2)***. In the event of a suspected uroabdomen, free fluid should be noted on ultrasound. Where no ultrasound is available, abdominocentesis is performed via a paraumbilical puncture; fluid is usually obtained if the ascitic fluid is more than 10 mL/kg in ~ 80% of patients (4) but a four-quadrant technique, (with punctures left and right, anterior and posterior to the umbilicus) is more sensitive (5). Any fluid should be analyzed; creatinine in ascitic fluid that is twice the blood level is 100% specific and 86% sensitive for uroabdomen (6). The treatment of post-renal anuria depends on the cause and may include catheter management, retrograde urohydropropulsion, surgery, or fluoroscopy with stenting of the urethra or ureter.

Diagnosing renal causes

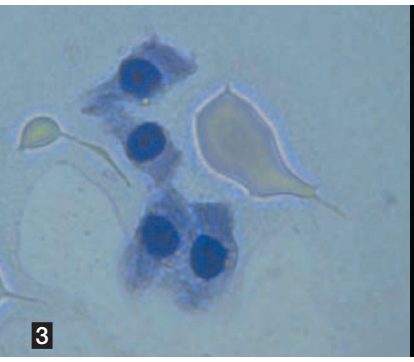
Diagnosing the cause of acute renal anuria or oliguria is more resource-intensive and includes differentiating potentially reversible acute kidney injury (AKI) from irreversible chronic kidney disease (CKD), as well as identifying the cause of the kidney damage ***(Table 3)***. History-taking should identify the duration of symptoms, any weight loss, previous laboratory investigations, polyuria polydipsia (PU-PD), vaccination status and any foreign travel. During the clinical examination particular note should be made of any ulcers, melena, arrhythmia or increased respiratory sounds, as well as body condition score. The core body temperature in severely uremic animals is usually low; if a uremic animal is normothermic, infectious or neoplastic causes should be considered.

Urinalysis

The urine examination is the principal component of diagnosis. Glucosuria when blood glucose is <10 mmol/L (180 mg/dL) (dog) or <15 mmol/L (270 mg/dL) (cat) indicates a disturbance in the proximal tubules.

Table 3. Differentiation between acute kidney injury (AKI) and chronic kidney disease (CKD).

Parameter	AKI	CKD
Duration clinical signs	<2 weeks	Usually >2 weeks
Polyuria/polydipsia	Occasional, often anuria/oliguria	Frequent, occasionally oliguric in final stage
Nutritional state	Usually unchanged	Often reduced
General state of health	Usually clearly reduced	Often not reduced until kidney values have risen sharply
Coat quality	Usually unchanged	Usually dull and unkempt
Abdominal palpation	Frequently painful around kidneys	Occasional cranial abdominal discomfort if vomiting or secondary disease, otherwise unremarkable
Body temperature	Sometimes (particularly with infections or neoplasia) within reference range or above	Usually hypothermia
USG	1.008-1.015, occasionally above	Typically 1.008-1.015
Glucosuria	Common	Rare
Proteinuria	Frequent; UPC frequently <3	UPC frequently >3 *e.g.* with glomerulonephritis
Urinary sediment	Frequent (blood leukocytes, casts, epithelial cells, bacteria)	Frequently absent
Ultrasound	Often unremarkable; may be capsular edema and moderately hyperechogenic renal cortices	Small kidneys with irregular surface, increased cortical echogenicity, indistinct or obscured corticomedullary junction
Hematocrit	Often normal, occasionally low	Often reduced, occasionally normal
Blood leukocytes	Frequently increased	Typically within reference range



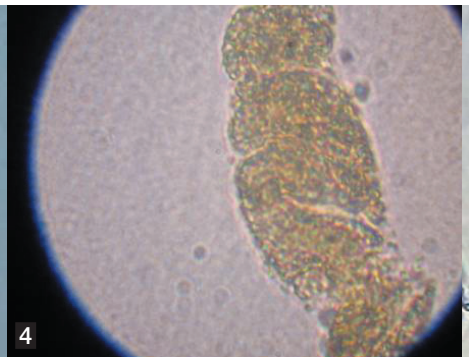
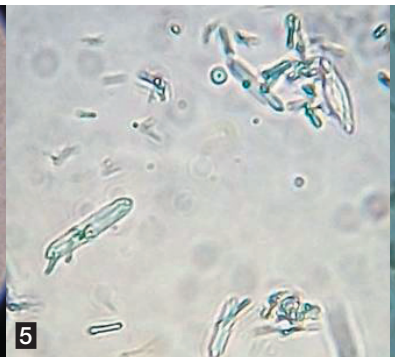


Figure 3. Tubular epithelium in stained urinary sediment (x1000).
Figure 4. Granular cast in the urine (x1000).



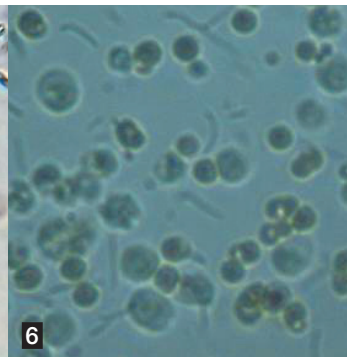


Figure 5. Oxalate crystals in the urine following ethylene glycol intoxication (x400).
Figure 6. Urinary sediment with leukocytes and *Leptospira* bacteria (x400).

Glucosuria is reported in 23-92% of animals with acute kidney injury (7-11), and has a 90% specificity in differentiating AKI from CKD (10).

Proteinuria is found in 66-95% of all uremic dogs (7-9, 11). Protein levels may be influenced by urinary infection or bleeding, and proteinuria is only significant when the sediment is unremarkable. Protein content should always be interpreted along with the USG or the urine protein-creatinine (UPC) ratio (12).

Microscopic examination of a stained sediment smear assists rapid and easy identification of bacteria and other cells in the urine (13) ***(Figure 3)***. More than 3 leukocytes per high power field in the sediment (when obtained by cystocentesis) suggests an inflammatory urinary tract process. Granular casts in the urinary sediment ***(Figure 4)*** have been reported in 11-33% of dogs with AKI (7-10), but also in 8.1% of dogs with CKD (10). Calcium oxalate monophosphate crystals are observed with problems such as ethylene glycol or lily poisoning (13) ***(Figure 5)***. Intracellular bacteria in urine (recovered via a sterile method) concur with infection ***(Figures 6 & 7)***.

Hematology

Initially non-regenerative anemia is found in ~30% of uremic dogs with AKI (6). This is due to reduced erythropoietin formation, reduced erythrocyte longevity in uremic plasma, iron deficiency from malnutrition, erythropoiesis-inhibiting substances in the uremic plasma, blood loss from gastrointestinal ulcers, and myelofibrosis (14). An increase in leukocyte count, mostly with left shift, is observed in AKI due to infection. In severe cases of uremia, a thrombocytopathy is frequently present (14). Thrombocytopenia is often observed with leptospirosis; one study noted thrombocytopenia in 55% of infected dogs (8). Many neoplasias (*e.g.* perianal tumors, malignant lymphoma) and bone marrow diseases (*e.g*. acute lymphoblastic or lymphocytic leukemia) can lead to AKI from hypercalcemia and some of these disorders may also cause thrombocytopenia.

Clinical chemistry

Severe uremia with anuria is characterized by an increase in plasma concentrations of urea, creatinine and phosphate. High urea values along with mild to moderately increased creatinine indicate pre-renal processes like dehydration, cardiac disease, Addison's disease or gastrointestinal bleeding. Hypoproteinemia and hypoalbuminemia are often present in patients with AKI due to infectious or neoplastic causes. The low levels are due to increased renal excretion, uremic gastroenteritis, reduced absorption and albumin formation (15). Renal protein losses also occur with some forms of CKD (14).

In uremic dogs, ionized calcium is often low (11,15). If hypercalcemia is detected, hyperparathyroidism or paraneoplastic syndrome (*e.g.* malignant lymphoma, tumors of the perianal region, lymphoma, adenocarcinomas, multiple myeloma) may be present. Further tests, including measurement of parathormone and parathormone-related peptide, as well as bone marrow cytology, are appropriate.

Leptospirosis diagnosis

Every dog with suspected AKI should be regarded as potentially infected with leptospirosis until proven otherwise. The diagnosis can be made using an antibody titer test or a urinary PCR test; note antibodies only form after 1-2 weeks, and if the initial test is negative, a second titer should be taken 3 weeks later. Identifying the pathogen via PCR testing can be more reliable,

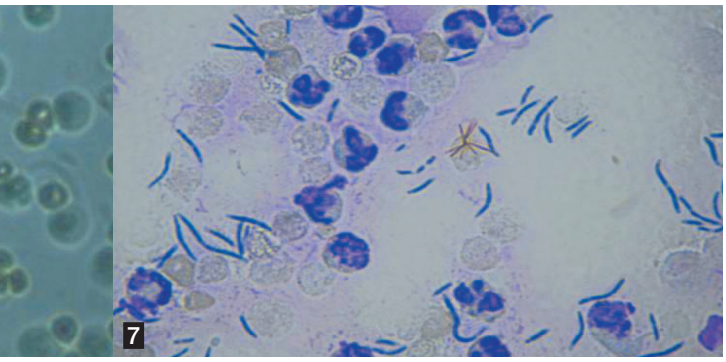


Figure 7. Stained urinary sediment with leukocytes, rod bacteria and bilirubin crystals (x1000).

but since bacteria are not always excreted, this test is only significant if positive.

Diagnostic imaging

Ultrasound examination of a patient with AKI may demonstrate normal or enlarged kidneys, hyperechogenic renal cortices, or subcapsular edema ***(Figure 8)***. With ethylene glycol poisoning, a clear increase in cortical echogenicity and a subsequently distinct corticomedullary interface is often noted (16) ***(Figure 9)***. With pyelonephritis the renal pelvis is frequently dilated. Doppler technology allows intrarenal blood flow to be assessed (the Pourcelot Resistive Index) which may also differentiate between AKI and CKD (17).

Problem-oriented therapy

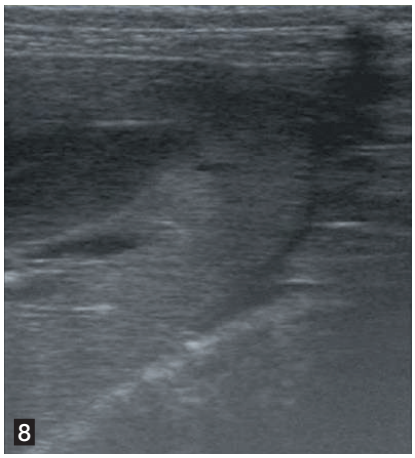
Fluid status

Most animals with AKI cannot adequately excrete excess fluid. As a result, overhydration can occur following rehydration. This is difficult to verify clinically, but a distended subcutis, edema of the jaw or lower limbs, nasal discharge, and respiratory signs (from pulmonary edema) may be noted; if present, diuretics or ultrafiltration during dialysis are indicated. When a patient is rehydrated, maintenance infusion should be matched to urine production. Given that the maintenance requirement is approximately 2 mL/kg/h for most of our patients, and around 1/3 of this volume is lost via metabolism, respiration and the gastrointestinal tract, approximately 0.7 mL/kg/h should be infused. The volume of urine produced should be added to the previously calculated infusion volume; note this does not include additional fluid loss such as that from vomiting or diarrhea.

Anuria

Urine production should be monitored closely to check for response to therapy. For this, urine should be collected, using an atraumatic catheter if possible. If no catheter is used, urine volume must be estimated, which may involve weighing bedding, etc.

After rehydration, attempts can be made to increase urine production by forced infusion therapy at about 6-8 mL/kg over a 4-hour period. If this is not successful, this therapy should be stopped to avoid overhydration. Osmotic diuresis, using substances such as mannitol, may also increase urine production. Mannitol is filtered by the glomeruli and its osmotic effect means that fluid is retained in the tubules; it reduces cellular and interstitial edema in the kidney and flushes detritus out of the tubules, and may also have an antioxidative effect. Mannitol should be infused at 0.5-1 g/kg IV for about 20 minutes. If there is good urine production after one bolus, this can be repeated up to 4 times daily, but if anuria persists, hypervolemia and osmotic kidney damage may develop and so additional doses should not be given.



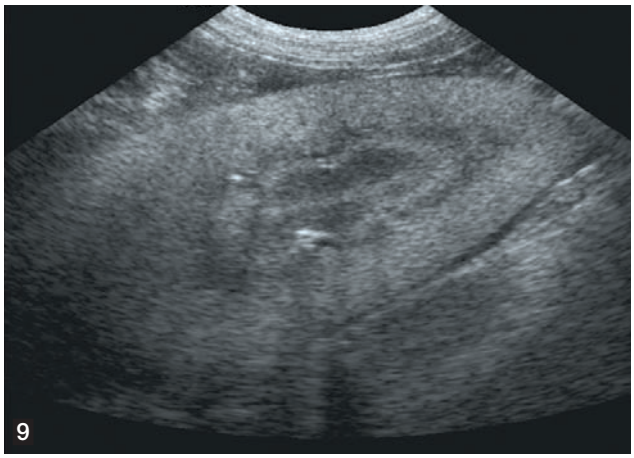


Figure 8. Ultrasound image of a dog with acute kidney damage and capsular edema.

Figure 9. Ultrasound image of a dog's kidney with ethylene glycol intoxication.

The loop diuretic furosemide increases the final urine volume and thus facilitates management of overhydrated patients. In addition, potassium and calcium excretion are promoted. Note furosemide must undergo glomerular filtration to work, which restricts its impact on anuria, and I find that in most cases the glomerular filtration rate (GFR) shows no increase (and may even decrease) following furosemide. However its use appears justified, especially for overhydrated, hyperkalemic or hypercalcemic patients.

In small doses (1-2 µg/kg/min) dopamine may improve kidney perfusion; however, this effect is distinct, individual and species-specific, and it is questionable whether this mechanism works in animals with acute kidney injury. Higher doses lead to vasoconstriction and a reduction in kidney perfusion, and so dopamine to increase urine production at normal blood pressure does not appear to be beneficial.

If an animal fails to display increased urine production or lowered renal plasma values within 12-24 hours, despite infusion therapy and diuresis, dialysis should be considered ***(Figure 10)***.

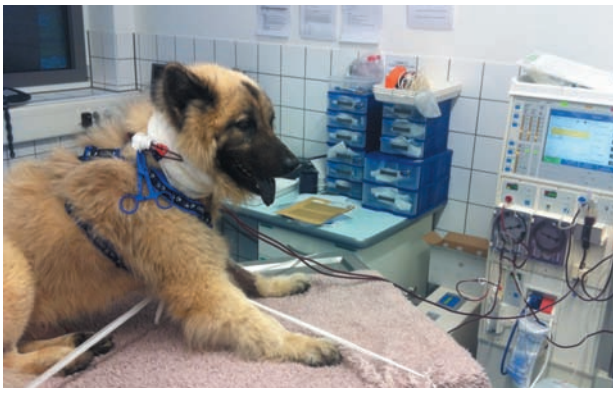


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Figure 10. Dog on hemodialysis.

Cardiovascular changes

Up to 81% of dogs with AKI develop systemic hypertension (18) which can further damage the kidneys as well as other organs. With systolic blood pressure >160 mmHg, therapy (*e.g.* amlodipine 0.25-0.5 mg/kg) is advisable, subject to strict monitoring of blood pressure and renal parameters.

Uremia toxins produced in AKI (particularly with infectious causes) can reduce cardiac output and myocardial contractility. This may result in hypotension, and dysrhythmias in the form of ventricular extrasystoles. In addition, an increase in vagal tone (and hence bradycardia) frequently develops. If there is marked ventricular extrasystole, lidocaine therapy is appropriate. In conjunction with vomiting, increased vagal tone can lead to the "vomit and die syndrome"; glycopyrrolate can be administered preventively if bradycardia is noted.

Gastrointestinal complications

Animals with AKI are often anorexic due to both uremia toxins and oral and gastrointestinal ulcers. Reduced gastric emptying further promotes nausea and anorexia. This impacts negatively on metabolism, the immune system and wound healing. Antiemetics, prokinetics and gastric protectants are all worthwhile ***(Table 2)***. Early enteral nutrition is essential and helps ease the catabolic situation, and can be provided by means of palatable food (ideally with a commercial renal diet), appetite stimulants and tube feeding. Where vomiting persists, central parenteral feeding should also be provided. In animals with oral ulcers, the mouth can be cleaned several times a day with chlorhexidine and local anesthetic applied to the ulcers. For systemic analgesia, an opioid (as a bolus or constant rate infusion) is useful.

Anemia

Any anemia should be treated with blood transfusions until regeneration has successfully been achieved. Erythropoietin can also be considered for non-regenerative anemias, dosing at 100 IU/kg every 2-3 days. Antibody formation can result, particularly with recombinant human erythropoietin, but with the longer-acting darbepoetin alpha the dosage interval can be extended and antibody production is therefore reduced (19).

Central nervous system

High levels of uremic toxins can lead to uremic encephalopathy. Convulsions can be treated with midazolam (0.2-1 mg/kg as a bolus, or 0.05-0.2 mg/kg/h as a constant rate infusion). If there are further seizures propofol or alfaxalone may also be used. In addition, attempts should be made to lower the toxin load with hemo- or peritoneal dialysis.

Lung

Lung damage can occur as a result of the actual uremia, overhydration, aspiration or because of the underlying disorder. Uremic toxins lead to chemical irritation of the lung and development of uremic pneumonitis; the latter can lead to acute respiratory distress syndrome.

Acute pulmonary hemorrhage (which is increasingly being described with leptospirosis, although the cause has not yet been fully clarified) can lead to respiratory distress and death. Oxygen therapy is generally the only available option; if this is insufficient, artificial ventilation may be required.

Dialysis

For symptomatic therapy of uremia with concomitant reduction of uremic toxins, correction of the acid-base and electrolyte balance, as well as the fluid status, hemo- and peritoneal dialysis are both suitable. Once an abdominal catheter is in position, peritoneal dialysis is simple to implement, although it is more labor-intensive and only moderately effective; hemodialysis is superior, but there are few specialist animal dialysis centers. Indications for dialysis include AKI that is non-responsive to conventional therapy within 12-24 hours, patients with serum creatinine concentration >500 µmol/L (5.7 mg/dL), and intoxication cases and refractory hypercalemia.

Causal therapy

Where the cause of the kidney failure is known, specific treatment should be instigated. Leptospirosis is treated with amoxicillin, ampicillin or other penicillin derivatives. For urinary tract infections an antibiotic should be chosen on the basis of urine cytology whilst awaiting an antibiogram. Where cocci are present, I use amoxicillin/ clavulanic acid (20 mg/kg every 8 h IV), and with rods I prefer marbofloxacin (4 mg/kg every 24 h).

For ethylene glycol intoxication, effective therapy within 8 hours of ingestion can prevent kidney failure (20). The conversion of ethylene glycol to oxalic acid by alcohol dehydrogenase must be reduced and ethylene glycol elimination increased. Enzyme inhibition can be achieved with 4-methylpyrazole (Fomepizole), or where this is unavailable, ethanol may be given as a constant rate infusion (21). To promote ethanol excretion, diuresis can be enforced using a balanced electrolyte solution (at least 6 mL/kg IV) and furosemide (as a bolus or constant rate infusion).

Conclusion

Animals with acute kidney damage should always be regarded as intensive-care patients. Adequate, round-the-clock monitoring should be available; routine monitoring of our patients consists of clinical examination and assessment of vital parameters and urine production every 4 hours, with blood pressure taken to check for hypo- or hypertension every 4-12 h. We closely monitor electrolytes, glucose, renal parameters, Hct and albumin, and perform daily weight checks and central venous pressure measurement. This is all essential to allow optimum treatment and maximize the chances of recovery.

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Small animal blood transfusions - a practical guide

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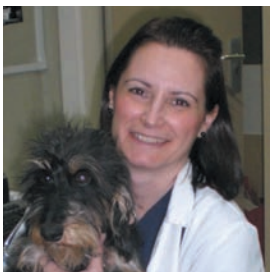

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KEY POINTS

- **Blood and blood component transfusions can provide red blood cells, platelets, clotting factors and plasma proteins, and may be critical for the survival of many critically ill patients.**
- **There are several different blood components available for dogs and cats. The veterinarian should know how to select the product for each patient that will afford the greatest benefits and lowest risks.**
- **Blood typing and/or cross-matching should be performed from the very first transfusion in cats, and from at least the second transfusion in dogs.**
- **Adverse reactions can be prevented by selecting the right donor, correctly managing blood products during collection, storage and administration, and monitoring the recipient during and after a transfusion.**

Introduction

Blood and blood component transfusions play an important role in small animal medicine, especially in emergency clinical care. This article offers a practical review of the most relevant aspects to enable effective transfusions whilst minimizing risks. In many countries commercial blood banks now offer whole blood and blood components for transfusions in both dogs and cats; it is always important to select the product that will provide maximum benefits for the patient. The different blood products, their characteristics and indications are described in ***Tables 1 and 2*** (1,2).

Transfusions in anemia

Hemoglobin is the main component involved with oxygen transport to tissues. It is hard to categorically define the hemoglobin (Hb) or hematocrit (Hct) value below which a patient needs a transfusion (the transfusion trigger), because a patient's capacity to compensate for the anemia can vary (3). Chronic anemia is better compensated for than acute anemia, because the intra-erythrocyte levels of 2,3-diphosphoglycerate (2,3-DPG) increase, facilitating oxygen transfer from the hemo-

Table 1. Blood components used in transfusion.

Product	Collection	Storage/shelf life	Active components
Fresh Whole Blood (FWB)	Blood collected straight from the donor, administered within 8 hours of collection	Room temperature, 8 hours	Red blood cells, platelets White blood cells (very low viability) Clotting factors (all) Albumin, other plasma proteins
Stored Whole Blood (SWB)	Whole blood more than 8 hours after collection	2-6 °C, 28-35 days	Red blood cells Albumin
Packed Red Blood Cells (pRBC)	Sediment of WB centrifugation, 5,000 G for 5 min or 2,000 G for 10 min (4 °C)	2-6 °C, 28-35 days (up to 42 days if nutrient solution is added)	Red blood cells
Plasma	Supernatant of WB centrifugation, 5,000 G for 5 min or 2,000 G for 10 min (4 °C)	• Fresh Plasma: administered within 6 hours of blood collection. Store at room temperature. • Fresh Frozen Plasma (FFP): frozen (and stored) at -30 °C within 6 hours of blood collection. • Frozen Plasma (FP): frozen after 6 h of blood collection; FFP thawed and refrozen; FFP frozen for more than 1 year; supernatant plasma from cryoprecip itate. Store at -30 °C for up to 5 years after blood collection.	• Fresh and fresh frozen plasma: all clotting coagulation factors, albumin and other plasma proteins. • Frozen plasma: albumin (2 years). Clotting factors: less activity in FV, FVIII and v.Willebrand factor. Other plasma proteins (2 years).
Platelet-Rich Plasma (PRP)	Supernatant from slow centrifugation of FWB, 1,000 G for 4-6 min (22 °C)	Room temperature, under constant agitation: 24 hours	Platelets Clotting factors and other plasma proteins
Platelet Concentrate (PC)	Sediment from PRP centrifugation at 2,000 G for 10 min (22 °C)	Room temperature, under constant agitation: 3-5 days	Platelets
Cryoprecipitate	Insoluble precipitate after thawing FFP at 4-6 °C.	-30 °C, 1 year	vWF, fibrinogen, fibronectin FXIII and FVIII

globin to the tissues. Normovolemic anemia is better tolerated than hypovolemic anemia because cardiac output (CO) can be increased efficiently. The underlying cause of the anemia and the possible presence of other disease also influence a patient's capacity to compensate. Consequently the decision on whether to administer a transfusion ***(Figure 1)*** should not be based solely on the Hct value. The degree of tissue hypoxia caused by the anemia which can be determined by the presence of tachycardia/tachypnea, reduced level of consciousness/stupor, syncope, and increased blood levels of lactate (lactic acidosis is an indicator of tissue hypoxia; normal value is <2.5 mmol/L) - is a major factor. In general, in acute hypovolemic anemia (such as hemorrhage) the Hct should not be allowed

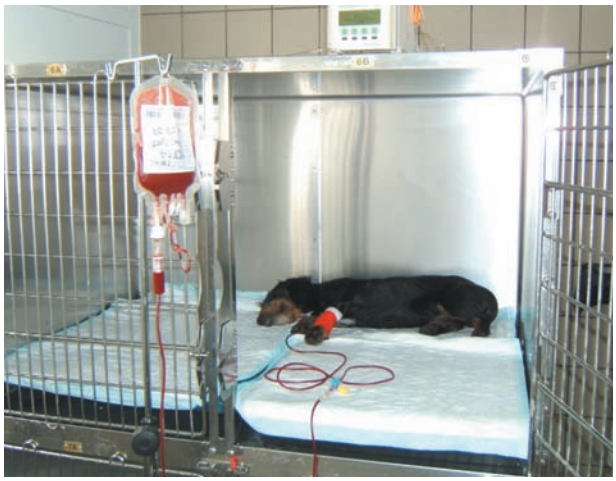

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Figure 1. pRBC transfusion in an anemic dog.

Table 2. Indications for blood products and dosages.

Condition	Indicated product	Dosage
Hypovolemic anemia (hemorrhagic)	FWB (or pRBC + FFP)	• Acute hemorrhage: 10-20 mL/kg • Formula 1*: 2.2 mL/kg, increases recipient Hct by 1% • Formula 2*: - Dogs: (Target Hct - current recip Hct/donor Hct) x weight (kg) x 90 = mL WB to be transfused - Cats: (Target Hct - current recip Hct/donor Hct) x weight (kg) x 60 = mL WB to be transfused • pRBC 10 mL/kg + FFP 10 mL/kg
Normovolemic anemia (hemolytic, hypoproliferative)	pRBC	• Acute hemorrhage: 6-10 mL/kg • Formula 1*: 1.1 mL/kg, increases recipient Hct by 1% • Formula 2*: 50% of the volume calculated for WB
Thrombocytopenia Thrombocytopathies	Platelet Concentrate Platelet-rich plasma If not available, FWB	• 1 unit/10 kg, every 8-12 h • FWB: 12-20 mL/kg every 24 h (10 mL/kg increases platelets by approx. 10,000/µL)
Coagulation disorders	All disorders; fresh plasma or FFP For disorders due to Vit K antagonists or liver failure, FP (or if not available, FWB) is also suitable	• 8-12 mL/kg/6-8 h (until bleeding/clotting times are controlled) • FWB: 12-20 mL/kg every 24 h
Hypoalbuminemia	FP, FFP, fresh plasma	• 8-12 mL/kg/6-8h
Hemophilia A Von Willebrand disease Hypofibrinogenemia	Cryoprecipitate If not available, FFP (fresh plasma)	• 1 unit per 10 kg (until clotting times return to normal) • 8-12 mL/kg every 8-12 h (until clotting times return to normal)

* Target hematocrit (Hct) in recipient: Dogs: 25-30%; Cats: 20-25%.

to fall below 25-30% in dogs or 20-25% in cats. However, in chronic normovolemic anemia, patients can compensate and will not usually require a transfusion until a lower Hct value (12-15% in dogs or 10-12% in cats) (2). Suitable products that provide Hb/RBC are whole blood and packed red blood cells, as follows:

1. Whole Blood (WB): this is blood that has not been separated into its various components. In dogs, one unit (1U) of WB is the volume obtained when a commercial human blood bag is filled (about 450 mL of blood and 63 mL of anticoagulant). In cats, 1U of WB is about 60 mL. It is called Fresh Whole Blood (FWB) for up to 8 hours after collection, when all the blood components remain viable; Stored Whole Blood (SWB) is WB that has been stored in a refrigerator for >8 hours after collection. After this time, the platelets are no longer viable and the most labile clotting factors (FVIII, von Willebrand Factor) gradually lose their activity.

2. Packed red blood cells (pRBC) in dogs: centrifugation of 1U of canine WB will give 200-250 mL of pRBC (=1U of pRBC) and 200-250 mL of supernatant (=1U of plasma) separated in a satellite bag without anti-coagulant ***(Figure 2)***. A pRBC transfusion achieves the same increase in Hb and RBC as a WB transfusion but using a much smaller volume, and so is mainly indicated for normovolemic anemia. Since pRBCs have a very high hematocrit (60-80%), 0.9% NaCl (70-100 mL) should be added to the bag before transfusion to reduce its viscosity and facilitate administration. In hemorrhagic anemia, if FWB is not available, pRBC (10 mL/kg) can be transfused together with fresh frozen plasma (FFP) (10 mL/kg).

3. Hemoglobin-based oxygen carriers (HBOCs): These solutions are based on polymerized/recombinant stroma-free human or bovine hemoglobin, and are capable of capturing and carrying oxygen. The advantages of HBOCs are that they do not require special

storage conditions and that the risk of adverse reactions due to incompatible blood group is avoided. They are indicated in anemia and certain states of shock (2,4).

Transfusions in clotting factor deficiencies

The following coagulation disorders require transfusion most frequently in small animals: disseminated intravascular coagulation (DIC, deficiency of all clotting factors), rodenticide intoxication (deficiency/inactivity of vitamin K-dependent factors II, VII, IX, X), liver failure (deficiency of all factors except FVIII), von Willebrand disease (vWF deficiency), hemophilia A (FVIII deficiency) and hemophilia B (FIX deficiency). A transfusion is indicated when the clotting factor deficiency causes significant hemorrhage or if the patient is to undergo a surgical procedure that entails a bleeding risk. Plasma is the most indicated blood product for coagulation disorders (5,6). If plasma is not frozen it gradually loses the clotting factor functions after 8-12 hours. If it is frozen at -30 °C within 6 hours of blood collection, it is known as Fresh Frozen Plasma (FFP) and will retain viable clotting factors and other plasma proteins for at least one year. If frozen after 6 hours, it is called Frozen Plasma (FP); this name is also given to plasma that has been thawed and refrozen, to FFP that was collected/frozen more than a year previously, and to plasma collected from the supernatant of a cryoprecipitate. FP retains the viability of albumin, globulins and all but the most labile clotting factors. Blood typing is not strictly necessary for plasma transfusions in dogs, but is required in cats.

The initial dose for plasma is 8-12 mL/kg/6-12 h until bleeding is controlled or clotting times return to normal values. It is always preferable to transfuse fresh plasma or FFP, because both products provide active clotting factors. FP is also acceptable for coagulation disorders induced by Vitamin K antagonists (*e.g.* rodenticides) and in coagulation disorders associated with liver failure. In the case of DIC, which also involves thrombocytopenia, FWB is also indicated (because it supplies clotting factors and platelets). In the case of hemophilia A or von Willebrand disease, the most indicated product is cryoprecipitate (CRYO) - where available - at a dose of 1U/10 kg (7). If plasma is not available, FWB can be transfused (10-20 mL/kg/24h).

Transfusions in other plasma protein deficiencies

Plasma protein deficiencies can result from conditions

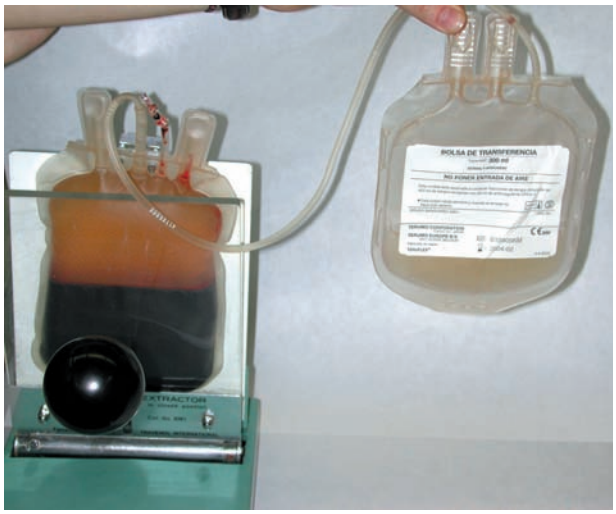

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Figure 2. Separation of plasma and packed red blood cells after whole blood centrifugation.

such as hypoalbuminemia, pancreatitis and parvovirus. Hypoalbuminemia causes a reduction in plasma oncotic pressure, leading to edema. It can also cause hypercoagulability, delay wound healing, and alter the transport or action of certain drugs, and is associated with a significant increase in morbidity/mortality in severely ill/critical patients (8). It is well recognized that transfusions of albumin solutions alone do not fully guarantee a good clinical outcome for hypoalbuminemia; the correct approach is to treat the primary cause (and in particular to resolve any inflammatory processes), and it is also extremely important to ensure that enteral nutrition provides adequate protein intake. A blood transfusion should be considered if the hypoalbuminemia causes edema or if there is a high risk of edema developing (plasma albumin <1.5-2 g/dL). Plasma is indicated in this situation, and it is calculated that 45 mL/ kg is needed to increase plasma albumin by 1 g/dL. Due to its high cost, many authors recommend administering plasma until albumin values of ≥1.5 g/dL are attained, and then combine it or replace it with synthetic colloids (20 mL/kg/day) to maintain oncotic pressure (9).

Another option is to administer a highly concentrated (20-25%) human albumin solution. Its oncotic pressure is >100 mmHg, and therefore a small amount increases the oncotic pressure and circulating volume very efficiently. However in dogs its use causes antibody production that can trigger severe immediate or delayed anaphylactic reactions, especially with a second infusion (10,11). Canine albumin solutions are now commercially available (7), but due to their marked oncotic effect they should not be used in patients with heart failure, renal failure or chronic normovolemic hypoalbuminemia.

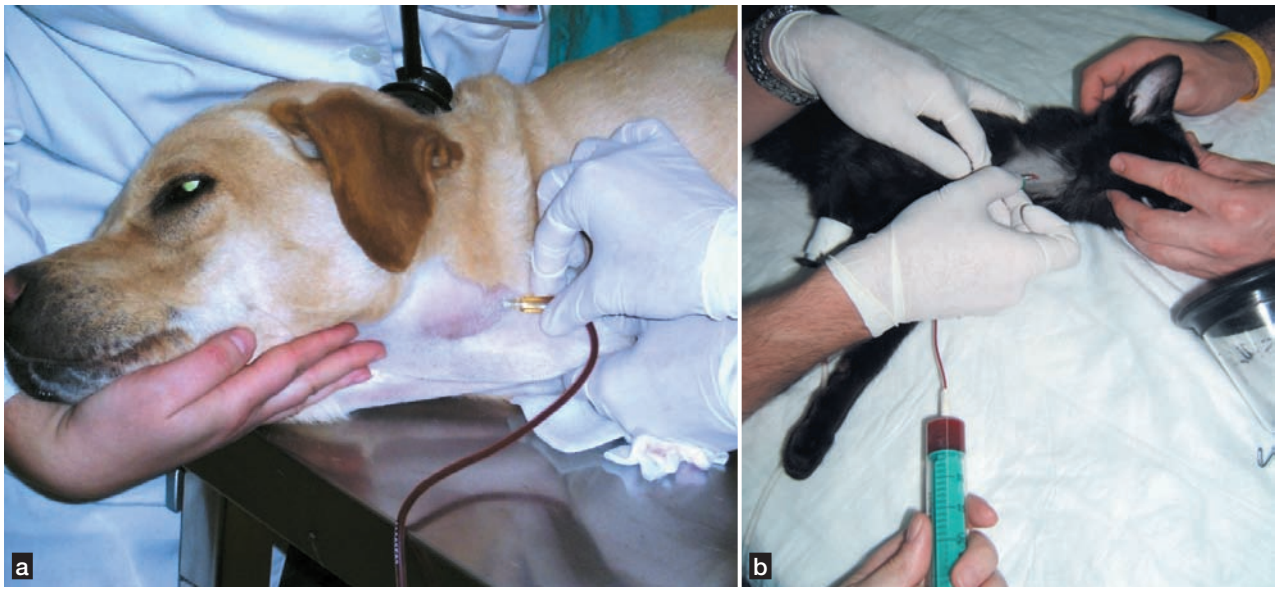

Figure 3. Blood collection from the jugular vein in a canine donor (a) and a feline donor (b).

The usual transfusion regimen for human albumin is 0.5 g/kg over 2-4 hours, followed by an infusion rate of 0.05-0.1 g/kg/h (maximum 2 g/kg/day) until serum albumin levels of ≥1.5 g/dL are achieved (9,12). It is recommended that a small test dose of 0.25 mL/kg/h should be given for 15 minutes, discontinuing the transfusion if any sign of anaphylaxis is detected. The transfusion should not take longer than 72 hours and (in order to reduce the risk of reaction from antibody formation) should not be repeated. Very little data are available regarding albumin transfusions in cats.

The administration of FP or FFP in patients with acute pancreatitis has been proposed as a source of albumin, antithrombin, alpha-2-macroglobulin and alpha-1-antitrypsin, and clotting factors. There are no conclusive results in human or veterinary medicine on the potential benefits; one retrospective study in dogs concluded that there were no significant differences in mortality/outcome of patients who received plasma compared with those who did not (13). It has also been suggested that FFP/FP transfusions in dogs with parvovirus infection may be beneficial (due to a possible passive transfer of antibodies and albumin), but there have been no controlled studies to support this assertion.

Transfusion in thrombocytopenia and thrombocytopathies

Platelet transfusion may be necessary to stop/prevent hemorrhage (14,15); this is known as therapeutic transfusion if there is active bleeding due to thrombocytopenia or thrombocytopathy (generally, there is no serious risk of bleeding until the platelet count is <10,000-20,000/µL), and prophylactic transfusion (recommended when the platelet count is <10,000/µL in the absence of factors that increase platelet requirements, such as surgery, or <20,000/µL in the presence of such factors). Indicated products are platelet concentrates (PC) and platelet-rich plasma (PRP). These products can be obtained by apheresis or by slow centrifugation of FWB, so that the platelets remain in the supernatant (PRP). To obtain the PC, the PRP is centrifuged again, and the platelets (sediment, PC) is separated from the plasma (supernatant, FFP) and transferred to another satellite bag. One unit of PC contains approximately $60x10^9$ platelets in 40-60 mL of plasma. PC has very limited use in veterinary medicine because it is hard to obtain a sufficient volume to be therapeutic, and because of storage challenges. An alternative is the PRP resulting from slow centrifugation of the FWB, which should be transfused within 24 hours of collection. 1U of PC or PRP increases the platelet count by 10,000/µL in a 30 kg dog. Lyophilized or cryopreserved canine platelets are now available in some markets, and they have the advantage of being readily available and easy to store, although more research is needed on their effectiveness (15). 10 mL/kg of FWB can increase the platelet count by about 10 x 10^3/µL.

Blood collection

Blood donors should be young adults weighing at least 25 kg (dogs) or 4 kg (cats). They must be healthy,

up to date with vaccinations/deworming, not have received transfusions, and free of blood-borne disease (which varies by geographical area) (16). Sedation is not usually necessary in dogs although it is generally required in cats (ketamine 5-10 mg/kg and diazepam 0.5 mg/kg IV is recommended); drugs that cause hypotension/bradycardia should be avoided. The blood can be collected in single commercial bags (whole blood), in double bags (main bag with anticoagulant and another satellite bag without anticoagulant for separation of plasma or platelet-rich plasma) or triple bags (main bag with two satellite bags for separation of cryoprecipitate and/or platelet concentrate) (2,6).

In dogs: the jugular vein is the best site for collecting blood from a donor. Place the animal in lateral recumbency, shave the neck, clean the area aseptically and perform venipuncture with a needle attached to the bag collection system ***(Figure 3a)***. Place the bag below the patient so that blood flow is aided by gravity, and continuously move the bag manually or mechanically. Check the bag weight regularly until the desired volume (approximately 450 mL) has been collected.

In cats: specific feline collection bags can be used, or a butterfly needle attached to a 20 mL syringe previously filled with CPDA1, sodium citrate 3.8% (1 mL per 9 mL of blood) or sodium heparin (5-10 IU/mL of blood) can be used ***(Figure 3b)***. Blood collected by syringe can be directly administered by attaching a neonatal transfusion filter ***(Figure 4)***, or transferred to a human collection bag that has had the anticoagulant removed. Blood collected using this open collection method should not be stored for more than 24 hours due to risk of bacterial growth; it should not be stored for more than 12 hours if sodium citrate or heparin is used.

Dogs can donate up to 20 mL/kg every 4 weeks; fluid replacement is not required to replace the blood taken. In cats, 10 mL/kg can be collected every 4 weeks, or up to 60 mL/cat if donation is not a regular occurrence, but it is advisable to replace the withdrawn volume with an isotonic crystalloid. When blood collection has been completed, the bag must be hermetically sealed (by heat or tight knots) and then centrifuged if blood components are to be separated.

Blood groups and compatibility testing

Canine blood groups are classified using the DEA (Dog Erythrocyte Antigen) system: DEA-1.1, DEA-1.2, DEA-3 to DEA-8. The ideal donor is DEA-1.1 negative,

Table 3. General recommendations for transfusion administration.

Preparation of the blood product
• Visual inspection of the product: clots? Hemolysis? Abnormal color?
• SWB and pRBC: reheat in a bath or in warm water to 25-35 °C (never exceed 37 °C; never use a microwave). Keep the bag connection ports covered to prevent water contamination.
• pRBC: add 70-100 mL of normal saline to the bag and mix (to reduce hyperviscosity).
• FFP: thaw in a water bath at 37 °C (in an emergency, thaw in a microwave at low power (700 W) at 10-second intervals).
• Cryoprecipitate: thaw in a water bath at 37 °C.
Administration
• Administer through a peripheral or central vein, or intraosseous route.
• Always use infusion sets with filters (even for plasma). A syringe and neonatal transfusion filter (40 µm) may also be used.
• Do not mix with any fluids or drugs, only normal saline.
• Maintain a constant temperature throughout the transfusion (never >37 °C).
• Complete the transfusion in ≤4 hours (to avoid contamination risk).
• Administer slowly for first 30 minutes (0.3-3 mL/kg/h); if there is no adverse reaction the remainder can be given at 10 mL/kg/h (dogs) and 5 mL/kg/h (cats).
• For hemorrhagic shock give up to 20 mL/kg/h (or faster if necessary).
• In animals with heart disease: do not exceed 3 mL/kg/h.
Monitoring during administration and for 1-2 hours post-transfusion
• Pulse, heart and respiratory rate and auscultation, temperature, color of mucosae, capillary refill time.
Hct determination (if transfusion is due to anemia)
• Pre-transfusion (baseline Hct).
• 1-2 h after end of the transfusion (check if target Hct has been reached: usually 25-30% in dogs, 20-25% in cats).
• 24-48 h post-transfusion (when the infused blood has been completely distributed).
• In the absence of complications, 70% of transfused RBC are viable after 24 hours, with half-life of ~ 21-50 days.

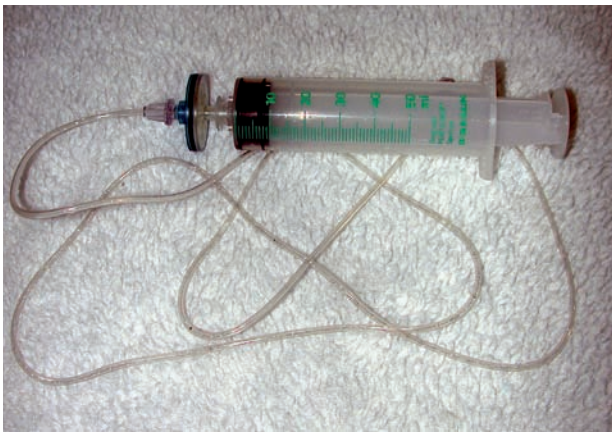

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Figure 4. Syringe coupled to neonatal transfusion filter.

because the DEA 1.1 group has the highest antigenic potential. A new dog erythrocyte antigen, the Dal antigen, has been described, but its clinical significance is unknown. Dogs do not have significant levels of alloantibodies against other blood groups unless the patient has received a transfusion and has developed antibodies against the donor's blood group. Severe adverse reactions in the first transfusion are therefore highly unlikely, but the recipient will produce significant numbers of antibodies against other blood groups 3-4 days after receiving the transfusion, which means that compatibility testing should always be carried out after that period (2,17,18).

Cats have three blood groups: A, B and AB. A is dominant over B. Prevalence of these groups varies greatly by breed and geographic region, but A is the most common and AB is the least common. The newly-discovered *Mik* blood group can also cause incompatibility reactions. Cats have naturally-occurring alloantibodies against other blood groups; tests must therefore be performed even for the first transfusion to check for compatibility. The most severe reaction (which is often fatal) occurs when group A blood is transfused to a group B recipient. Cats with type AB blood can receive group A blood. The alloantibodies present in cats can cause neonatal isoerythrolysis if a group B female is mated with a group A (dominant) or AB male; group A (or AB) kittens ingest maternal anti-A antibodies in the colostrum, which can cause severe hemolysis, weakness, tail necrosis, hemoglobinuria, jaundice, severe anemia and sudden death. If an affected kitten requires a transfusion, this can be given using maternal washed red blood cells (or another B-group cat) at a dose of 5-10 mL/kitten administered over several hours.

There are several commercial tests available to determine if a dog is DEA-1.1 antigen positive or negative, and if a cat has group A or B blood ***(Figure 5)***.

Compatibility testing

Blood typing detects the presence of antigens of a certain blood group in the red blood cell membrane, while cross-matching determines the presence of antibodies in the plasma of the donor and recipient which may cause incompatibility reactions (17,18). Cross-matching must always be performed if the blood type cannot be determined, or in any dog or cat that has already received a transfusion. *Major* cross-matching verifies if the recipient's plasma has antibodies to the donor's red blood cell antigens, while *minor* cross-matching checks if the donor's plasma contains antibodies to the recipient's red blood cell antigens. A *control* should also be performed (using the recipient's red blood cells and plasma). If hemolysis and/or agglutination occurs with major cross-matching, the transfusion cannot be performed (because the recipient has antibodies against the donor's red blood cells). If hemolysis and/or agglutination occurs with minor cross-matching, the transfusion may be performed whilst closely monitoring the patient (because the donor has antibodies to the recipient's antigens, but the quantity present in the transfused blood does not confer serious risk). If there is underlying auto-

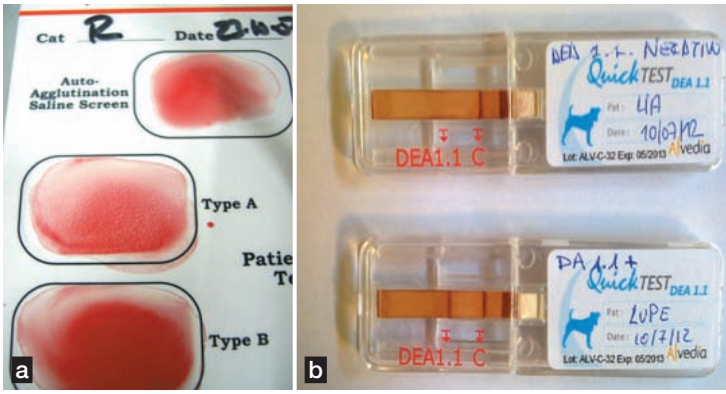

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Figure 5. Examples of feline and canine commercial blood typing kits.

a: Feline blood group test kit ; agglutination can be seen in the well marked "Type A".

b: DEA-1.1 negative dog (top) and DEA-1.1 positive dog (bottom): the red line on the lower slide denotes the positive result, whilst the control line on each slide indicates that the tests were performed correctly.

agglutination and/or hemoglobinemia in the recipient, such tests will be inconclusive. The correct procedure for cross-matching involves washing the donor and recipient red blood cells several times (by centrifugation with 0.9% NaCl).

In emergencies, simplified (but less reliable) compatibility test can be performed: centrifuge the donor and recipient blood, dilute the RBC to 5% (1 drop RBC + 20 drops normal saline) and perform the three tests (major, minor and control) on three slides, mixing each one with one drop of plasma and one drop of RBC. Incubate for 2-5 minutes, and check for agglutination under the microscope. There are also commercial kits for performing cross-matching quickly and reliably.

Conclusion

The actual administration of blood and blood products is summarized in ***Table 3***. Adverse reactions to transfusion can occur and may be immunological or non-immunological in origin, and either acute (occurring during the transfusion or within 24 hours) or delayed (occurring >24 hours after the start of the transfusion) (3,19). However risks may be minimized by carefully selecting the donor and blood product, and applying the most appropriate techniques in terms of collection, storage, handling and administration. Performed properly, blood transfusions can be a fundamental factor in treating various critical care cases, and a good knowledge of the different options is a key requisite for the emergency veterinarian.

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Initial assessment of spinal trauma cases

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KEY POINTS

- **Spinal injury can result directly from primary damage (fractures, disc herniations, bleeding or edema) or may be a secondary consequence from biochemical and metabolic alterations caused by primary trauma.**
- **It is essential to determine if instability is present with any spinal trauma patient.**
- **Diagnosis requires both a neurological-orthopedic evaluation and appropriate diagnostic imaging.**
- **Immediate management of the spinal patient requires stabilization of vital functions, immobilization of the patient, good pain control and therapy with methylprednisolone (if appropriate).**

Introduction

The most common causes of spinal trauma (ST) are road traffic accidents, falls, gunshot wounds, bite wounds and injuries from large falling objects, and the trauma may involve various anatomical structures: vertebrae, intervertebral discs, meninges, spinal cord, or any combination of these. A preliminary approach should assess spinal injury lesions as follows:

- Primary damage: vertebral dislocations and fractures, traumatic herniation of the intervertebral discs, bleeding (intramedullary, epidural and subdural), bone marrow edema.
- Secondary damage: biochemical and metabolic alterations produced by the primary damage that ensue some hours or days following trauma.

Primary damage can be caused by compression, flexion, rotation and/or extension forces acting on the vertebral column: these forces can interact causing a variety of injuries, and it is essential to determine if trauma has

caused instability (1). There are two methods for determining the presence of instability. The first is to subdivide the vertebrae into three sectors ***(Figure 1)***:

- The dorsal area, which includes the articular processes, lamina, pedicles and spinous processes;
- The intermediate area, which includes the dorsal longitudinal ligament, the dorsal portion of the vertebral body, and the dorsal portion of the annulus fibrosis;
- The ventral area, which includes the ventral longitudinal ligament, the lateral and ventral portions of the annulus fibrosis, the nucleus pulposus, and the remaining portion of the vertebral body (2).

If at least two sectors are involved there will be instability of the spine.

The second method entails evaluating the number and location of lesions to the vertebral body, facet joints and intervertebral discs (2,3) as follows ***(Figure 2)***:

A. Lesions of the intervertebral disc: instability may be present due to the loss of ability to control rotation, flexion and extension of the column.
B. Lesions of only one joint facet: slight instability during rotation.
C. Lesions of the vertebral body only: fractures are often unstable, particularly when bending, and the vertebral body may collapse if the spinal column is compressed axially.
D. Lesions involving two or three anatomical structures simultaneously: severe instability with any and all spinal movement.

When evaluating the spinal cord to determine lesion severity, three parameters should be considered (4);

- The duration of bone compression;
- The extent to which the bone is affected;
- The force exerted by the compression.

Secondary damage is caused by a series of biochemical and metabolic abnormalities, and from perfusion deficit. The reduced blood perfusion of the spinal cord reduces the energy available, the neuronal membranes lose their polarization releasing excitatory neurotransmitters (*e.g.* glutamate) which, by binding to specific neuronal receptors, activate voltage-gated calcium channels. Calcium and sodium ions enter the neurons and glia, increasing the osmotic gradient which causes edema and cell death. The neuronal membrane injury also triggers the inflammatory cascade, the release of

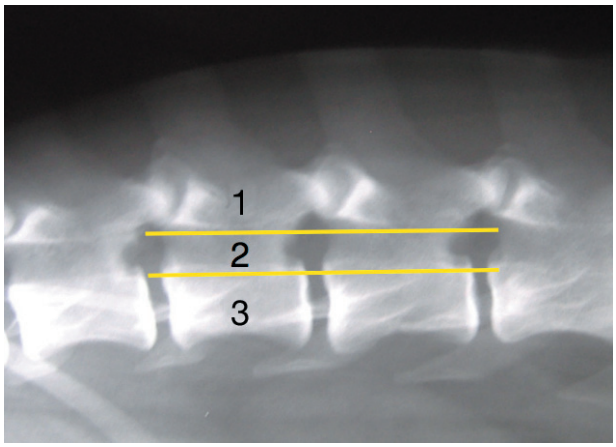

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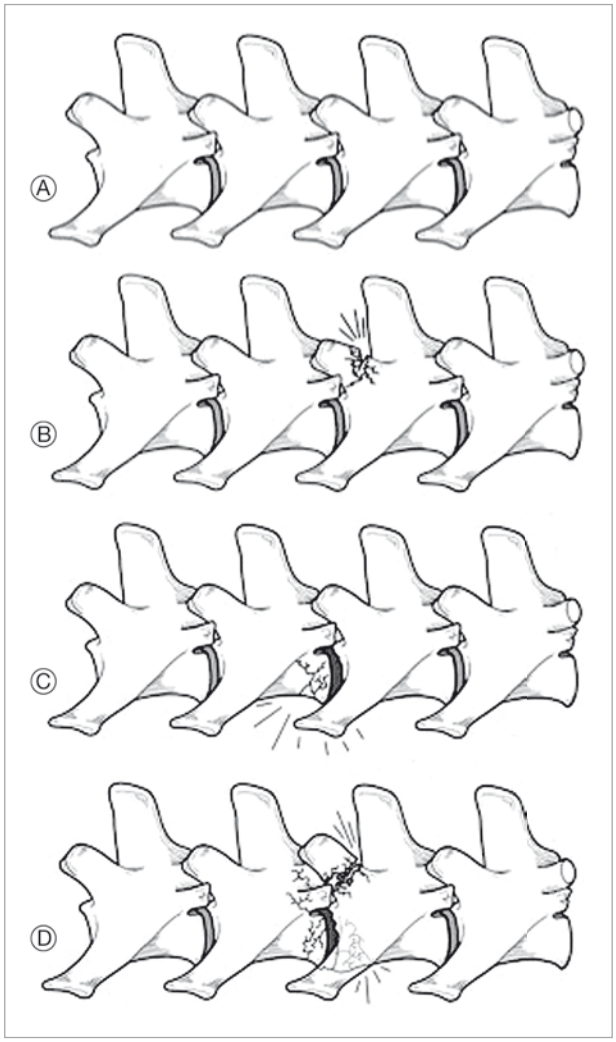
Figure 1. When assessing for spinal damage, the vertebrae can be subdivided into dorsal (1), intermediate (2) and ventral (3) sectors as shown.

Figure 2. Vertebral damage resulting from spinal trauma:
A) lesions of the intervertebral disc without fracture (no bony damage);
B) fracture of the articular facets;
C) fracture of the vertebral body;
D) multiple fractures.



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free radicals and reactive oxygen species (ROS) that help perpetuate neuronal damage. A secondary lesion may resolve within a few hours or can permanently affect the neurological tissue. Damage to the gray matter is always more serious than white matter damage, as axonal damage is more easily repairable than that in the perikaryon; the degree of perikaryon involvement affects the severity of the clinical symptoms (5).

Initial management

The management of a patient with spinal trauma can start by offering telephone advice to the owner or rescuer before the animal reaches the veterinary facility. If spinal instability is suspected, the patient must be transported secured to a rigid board that is at least as long as the spine and supports the limbs, with restraint applied to the shoulders and pelvis (6). ST patients are usually polytraumatic and require a rapid assessment of vital functions in order to perform life-saving procedures and reduce the consequences of secondary injury. Bladder integrity should always be verified by abdominal palpation, and contrast radiography or ultrasound performed in doubtful cases. Repeated hematocrit tests or an abdominal ultrasound may suggest traumatic organ rupture (*e.g.* liver, kidney and spleen). Only after the assessment of vital signs (perfusion, blood pressure and oxygen parameters at minimum) is it possible to investigate spinal cord lesions. As the emergency ST patient almost always arrives in a state of hypovolemic shock it is difficult, if not impossible, to make any immediate assessment regarding mobility and the presence or absence of deep and superficial pain sensation (7).

Diagnosis

Diagnosis requires both a neurological-orthopedic evaluation and diagnostic imaging; these procedures must be performed only once the patient is stabilized.

Orthopedic and neurological evaluation

The patient, even if able to walk, may have suffered severe neurological lesions and have an unstable column; careful handling is imperative to avoid any additional trauma. The assessment starts with gentle palpation of the column ***(Figure 3)***, from the base of the skull to the base of the tail, in order to detect vertebral instability, skeletal dislocations, crepitus or pain, although the absence of such findings do not exclude the presence of instability (7). The evaluation of a functional spinal cord is performed by assessing:

- Posture
- Level of consciousness and cranial reflexes
- Spinal reflexes of the four limbs

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Figure 3. The spinal trauma patient should be examined gently, palpating the column from the base of the skull to the base of the tail.

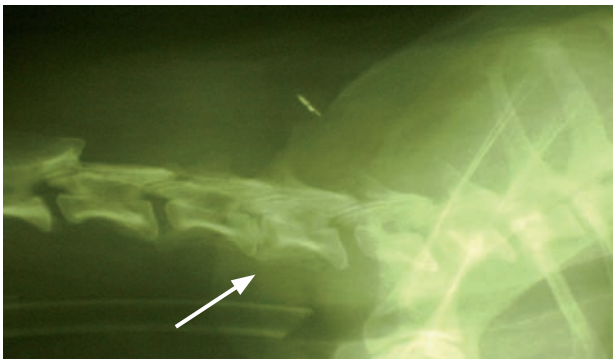

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Figure 4. Cervical disc protrusion may be apparent on radiography.

- Deep and superficial pain sensation
- Panniculus reflexes

Functional evaluation of the spinal cord permits the neuroanatomical site of the lesion to be ascertained and to identify early prognostic signs. As well as applying standard neurological tests to localize a spinal lesion, the presence of certain characteristics should be noted: *e.g.* the Schiff-Sherrington posture, with characteristic forelimb rigidity and paraplegia, symptomatic of trauma at the level of T2-L4 (5), where injury causes increased activity of the forelimb extensor muscles. This posture should not be confused with the rigidity of all four limbs seen with decerebration, or the decerebellar posture characterized by rigid extension of the forelegs associated with flexion of the hind limbs, both produced by head trauma and accompanied by altered cranial reflexes (8). Hemiplegia with asymmetrical neurological deficits, poor nociception on the affected side and loss of sympathetic function are signs that may typically indicate traumatic disc herniation with a dorso-lateral protrusion of the nucleus pulposus (9).

The panniculus reflex is lost approximately one or two segments caudal to the spinal lesion, and whilst it may be used to precisely locate lesions between T1 and L3, it is not reliable for more caudal lesions. Note that a double spinal cord injury can be difficult to recognize: *e.g.* a lesion at the level of the caudal lumbar vertebrae may mask a problem around T3-L3. In terms of prognosis, absence of hindlimb deep pain sensation is the only definitive sign that indicates a functional recovery is unlikely.

Diagnostic imaging

After performing a full neurological evaluation, radiographs can be taken: lateral views are generally obtained first, then (with the patient still in lateral recumbency) an oblique beam may be used to identify any fracture of the facet joints. For ventro-dorsal views a horizontal beam may be considered to reduce the risks involved when positioning the animal in dorsal recumbency. Deep sedation or general anesthesia is not recommended because this can remove spasm of the para-vertebral muscles and cause instability (1). Radiography has a sensitivity of 72% for vertebral fractures and 77.5% for subluxations, but has a negative predictive value of 51% in identifying fracture fragments within the vertebral canal. Diagnosis of herniated intervertebral discs ***(Figure 4)*** by radiography has a 64-69% sensitivity while the positive predictive value is 63-71% (7). In many cases it is therefore necessary to employ other diagnostic methods such as myelography, computerized axial tomography (CT) and nuclear magnetic resonance imaging (NMR).

These imaging techniques are indicated if the radiographic lesions do not correlate with the neurological assessment and can also be useful to guide decisions such as whether or not to operate on a traumatized patient (*e.g.* bone damage may be irreversible, making surgery pointless); when to intervene and which surgical approach is best; the type of operation required (decompression, fixation, or both); and whether or not bone fragments or disc material in the vertebral canal need to be removed.

Myelography

Myelography requires general anesthesia, which, as noted, may lead to column instability. Injecting contrast medium can also put the patient at risk; this must be balanced against the potential benefits (1), namely that compressive pathologies may be identified by deviation, attenuation or obstruction of the contrast medium ***(Figure 5)*** and more expensive imaging may not be required. Myelography is especially useful in cases where there has been a temporary dislocation of the vertebra but no bony displacement is evident on radiography.

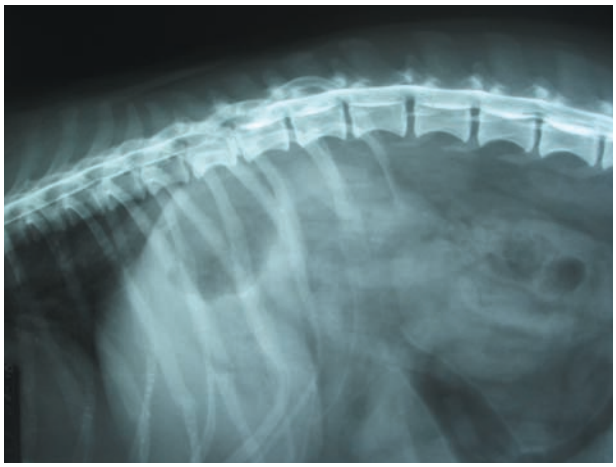

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Figure 5. Myelography can demonstrate spinal trauma spinal trauma.

Computerized axial tomography

CT scanning requires general anesthesia with the above-mentioned risks, but permits 2- or 3-D visualization of the lesion. Because of this the degree of instability of a vertebral fracture can be evaluated, and the scan may provide useful information when considering surgical options (*e.g.* the preferred approach to a fracture site). CT scans are more accurate than radiology in detecting fractures of the articular facets and can even identify small bone fragments in the spinal canal. CT is also becoming increasingly popular because it can distinguish between spinal cord edema (which is potentially reversible) and intramedullary hemorrhage (which has a poor prognosis) (10). The disadvantages of the technique include the necessary handling of the patient and the risk that the degree of cord compression may be underestimated, so ideally CT should be supplemented by NMR (10).

Nuclear magnetic resonance

Nuclear magnetic resonance is an expensive technique and requires a longer procedure time. However it provides better soft tissue imaging and is the gold standard for evaluating lesions of the spinal cord, which may be underestimated by CT scans.

Management

In the first 24-48 hours after the patient arrives at the veterinary institution it is necessary to:

- Stabilize vital functions.

- Immobilize with external hard supports (*e.g.* splint, stretcher).
- Manage pain control for at least 96 hours after the injury.
- Institute therapy with methylprednisolone sodium succinate (if the clinician thinks it is appropriate).

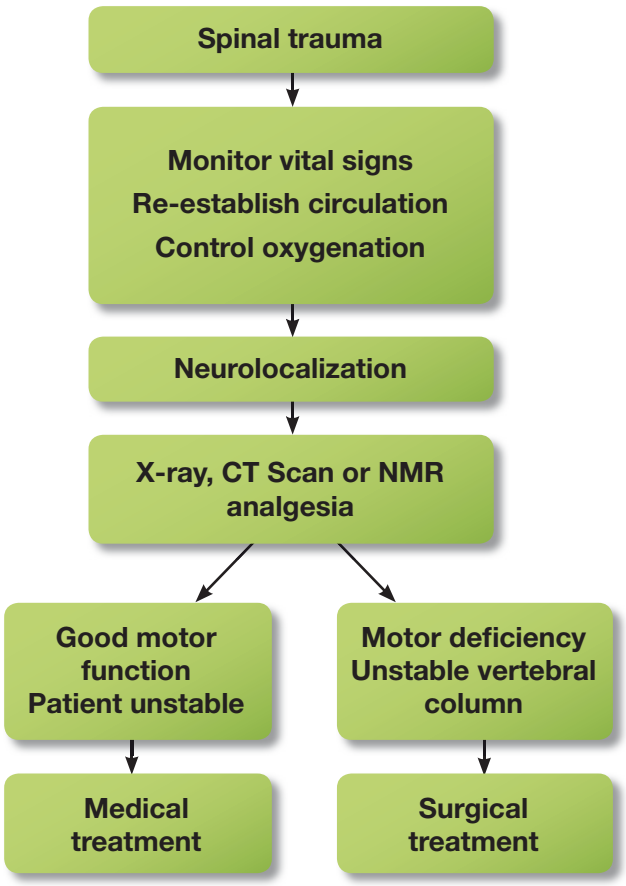
Consequently it is possible to perform the necessary diagnostic tests and decide on a definitive treatment regime ***(Figure 6)***.

Primary damage

Vertebral fractures and spinal dislocations

These lesions can be treated surgically with decompression and internal or external fixation, or non-surgically, with external rigid supports (*e.g.* splint). There is much debate in the literature as to the best approach for these cases, and it is ultimately the subjective opinion of the surgeon and the choice of the owner who together decide on the preferred treatment. There are, however, specific indications that suggest the surgical option (11,12);

Figure 6. The approach to the patient with spinal trauma.



- Presence of minimal motor voluntary function or complete paralysis.
- Clinical or radiological evidence of very unstable fractures.
- Progressive worsening of neurological signs despite medical treatment.
- Absence of deep pain sensation (with an intact spinal cord).
- Severe compression of the spinal cord.

The essential factor for surgery is that there should be no obvious severe, irreversible spinal cord lacerations. Non-surgical management of vertebral fractures or dislocations involves rigid supports made of fiberglass, thermoplastic materials, plaster or metal rods ***(Figure 7)*** and enforced cage rest for at least 6-8 weeks. Splints must conform to the animal's body to ensure complete immobilization of the entire column and be secured to the patient with elasticated bandage to maintain column stability without compromising normal breathing. Dressings should be checked daily for soiling, and any abrasions (*e.g.* caused by pressure from bandages) treated as early as possible. Non-surgical management is generally cheaper and enables owners to treat the animal at home without a team of specialized professionals, but care can be time-consuming, with long recovery periods and a high probability that neurological deficits may persist.

The discharge instructions given to the owner are very important, especially for larger dogs, because more people are needed to provide nursing (13). Where good homecare may be difficult, hospitalizing the patient at a specialist rehabilitation center may be recommended.

Analgesia, as well as the welfare aspect, is important as it reduces cardio-respiratory changes (tachycardia, tachypnea, vasospasm) and immunosuppression (which can lead to increased risk of infection), and may obviate the delayed tissue repair, increased catabolic phase and reduced appetite that can all result from severe pain. Suitable options include:

- Opiates such as fentanyl (2 µg/kg bolus, then continuous rate infusion (CRI) at 2-4 µg/kg/h), morphine (0.05 mg/kg bolus then CRI 0.1 mg/kg/h or as a bolus 0.2 mg IV/IM every 4-6 hours), or buprenorphine (10-20 µg/kg IV/IM tid). In cases of very severe pain, a combination of opiates plus ketamine may be given (0.2-0.3 mg/kg/IV as a loading dose, followed by 5-10 mcg/kg/min as a CRI) for 24 hours before re-evaluating the patient to decide if this regime needs to be continued.

- Anti-inflammatory drugs such as ketoprofen (2 mg/kg IV/SC/IM as a loading dose then 1 mg/kg PO sid), or carprofen (4 mg/kg IV as a loading dose then 2.2 mg/kg SC/IM/PO bid). These may be combined with opiates but only if corticosteroids are not administered.

When anti-inflammatory drugs or corticosteroids are employed, drugs such as ranitidine (2 mg/kg/IV or PO bid/tid), or famotidine (0.5 mg/kg PO sid or bid), or diosmectite (1.5 g per patient PO tid for small dogs and cats, 3 g tid for medium and large-sized patients) should be administered.

Traumatic disc herniation

The indications for surgical treatment of traumatic disc herniations are the same as described above; as an alternative to surgery it is possible to opt for enforced cage rest (6-8 weeks) in order to reduce the inflammation.

Spinal cord contusion

Bruising of the spinal cord rarely occurs alone and is usually the result of a compressive lesion or spinal instability. Surgery or medical treatment may be necessary to limit secondary damage.

Extra axial hematoma

Extra axial hematoma (bleeding outside the spinal cord) as a result of trauma is rarely diagnosed in veterinary medicine, but surgical decompression is recommended if present.

Secondary damage

There are many different opinions as to the best therapeutic approach, and the clinician must assess the patient's needs and the latest scientific knowledge and choose the most suitable protocol. ST patients, as already noted, need good perfusion, normal blood pressure and optimal oxygenation in order to reduce secondary damage.The ST is frequently associated with respiratory and cardiovascular problems, bleeding and head trauma. The high risk of ischemia and frequent marrow hypoperfusion often require aggressive fluid therapy with crystalloid isotonic, hypertonic or synthetic colloids. In particular, hypertonic saline solution increases cardiac contractility, reduces endothelial cell inflammation and protects the blood-brain-spinal cord barrier (14). Severe blood loss (>15%) may require blood transfusion (10-20 mL/kg) - or alternatively polymerized hemoglobin (HBOC 10-15 mL/kg/h) - whilst trying to maintain a hematocrit value of ≥20-25% to ensure good tissue oxygenation. In patients who do not respond to fluid therapy, vasoactive amines by CRI (*e.g.* dobutamine (5-15 µg/kg) or dopamine (3-10 µg/kg)) may be used; if there is excessive vasodilation, first-line drugs are dopamine or norepinephrine (1-10 µg/kg) whilst dobutamine is the preferred treatment for shock with reduced cardiac output. Oxygen therapy is indicated in all trauma patients until hypoxia can definitely be ruled out.

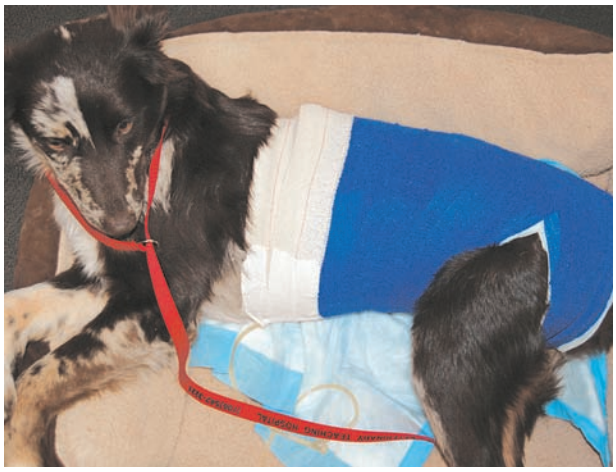


Figure 7. Example of a spinal splint in a dog.

The use of corticosteroids to reduce secondary damage in ST is debatable in both human and veterinary medicine. In particular methylprednisolone sodium succinate (MPSS) is used to reduce secondary damage produced by free radicals and to increase local blood flow. Trials suggest that the most important protective effect is that afforded against free radicals, an effect not achieved by other corticosteroids (7). One human study suggested that a bolus of MPSS followed by an continuous infusion for 48 hours resulted in the treated group showing a moderate neurological improvement six weeks after treatment compared to a placebo group (14) but the improvement was not sustained in the majority of patients; 6-12 months later some patients developed severe pneumonia or had an increased risk of sepsis. The most common side effects of corticosteroids are gastritis, ulcers and hyperglycemia. Many veterinary professionals regard the MPSS protocol as standard for neuroprotection (even though there is no scientific evidence for it), using a 30 mg/kg bolus followed by CRI at 5.4 mg/kg/hr (or a 15 mg/kg bolus repeated every 6 hours) for 24-48 hours (11). Other corticosteroids (prednisone at 1-2 mg/kg or dexamethasone at 0.15-0.3 mg/kg daily for 1-2 weeks) may be

given for anti-inflammatory purposes only. The use of dexamethasone has been reported in dachshunds that underwent surgery for herniated discs (15); this study also noted an increased incidence of GI side effects with the use of MPSS.

A brief note on polyethylene glycol (PEG) is appropriate; a hydrophilic polymer surfactant, it can seal damaged nerve fibers, aiding restoration of nerve conduction as well as repair of damaged cells and preventing the release of excitatory neurotransmitters and other cytotoxic substances that cause secondary damage. One study, involving dogs suffering from disc herniation, employed two doses of PEG given at 2 mL/kg/IV over 15 minutes, 4 hours apart (16). The treated dogs showed improvements 72 hours after the onset of clinical symptoms compared to the control group, but to date no studies have been undertaken using PEG for dogs with ST.

Prognosis and conclusion

This paper covers the immediate assessment and management of spinal trauma, but it is appropriate to comment on the prognosis for these cases. The loss of pain sensation at examination carries a poor prognosis for the recovery of normal neurological function, although this possibility cannot be completely ruled out. Patients with vertebral fractures or dislocations have a worse prognosis than patients with disc herniation. Dogs with fractures of the cervical vertebrae, inability to walk or where surgery is delayed for more than 5 days have a poor prognosis. Surgical stabilization of the vertebrae has a peri-operative mortality rate of 36%; dogs with a favorable surgical outcome have a good prognosis for neurological recovery (11). Hypoventilation is one of the principle complications at surgery, but positive pressure ventilation and aggressive supportive therapy can improve surgical outcome (17).

The prognosis in cats is similar, although one study (18) reported a high incidence of myelomalacia observed during surgery and at necropsy in cats that had lost deep pain perception after ST; it may be that cats are predisposed to irreversible neurological lesions with ST, especially where trauma is so severe that pain perception is lost. The clinical signs include progressive lower motor neuron signs, leading to death from respiratory failure after 2-4 days.

Ultimately in all cases the veterinarian must clearly explain the advantages and disadvantages of the various treatment options with the owners and agree a treatment plan once the patient is stabilized.

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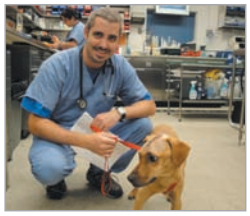
The ABC's of cardiopulmonary resuscitation



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Introduction

Cardiopulmonary resuscitation (CPR) is a series of emergency procedures aimed at restoring and optimizing perfusion to the brain and heart during a period of cardiopulmonary arrest (CPA) in an effort to not only achieve return of spontaneous circulation (ROSC) but, more importantly, to promote a good neurologic outcome for the patient. Although the incidence of CPA in small animal patients is unknown, survival to discharge is poor with the best outcomes occurring in patients that arrest while under anesthesia (1). In contrast to humans, CPA in animals frequently involves hypoxia and poor tissue oxygenation which may complicate resuscitative measures and may explain the low incidence of survival to hospital discharge.

KEY POINTS

- **Rapid recognition of cardiopulmonary arrest is essential and immediate basic life support, including securing a patent airway and provision of ventilation and chest compressions, is the foundation for successful cardiopulmonary resuscitation.**
- **Once basic life support measures have been instituted, advanced life support may be considered.**
- **Monitoring end-tidal carbon dioxide during CPR may be useful in evaluating efficacy of resuscitative measures.**
- **Re-arrest is common during the post-resuscitative period and survival to discharge following cardiopulmonary arrest is poor; therapy aimed at optimizing perfusion, oxygenation and ventilation will help to improve patient outcome.**

Until recently, veterinary CPR guidelines were mostly adopted from the human literature despite some notable differences in the physiology and pathophysiology of arrest. The first comprehensive, evidence-based consensus guidelines for CPR in small animals were recently published (2). This article reviews the central tenets of CPR, including basic and advanced life support, as well as post-resuscitative care, which may be utilized to help prevent unexpected death in small animal patients.

Recognition of CPA

Rapid recognition of CPA is an essential step in the initiation of CPR. Common signs of impending arrest include cessation of spontaneous ventilation, the presence of agonal breaths, acutely deteriorating mentation, fixed and dilated pupils, or sudden change in heart rate or rhythm (3). Arrest should be suspected in any patient that is unresponsive, apneic, or when

an agonal breathing pattern is observed. Palpating for peripheral pulses or attempting to confirm CPA via absence of Doppler pulse signal is not recommended as this inevitably delays CPR initiation. In patients that are already intubated, a sudden decrease in end-tidal carbon dioxide ($ETCO_2$) may suggest an acute decrease in pulmonary perfusion such as with CPA. Electrocardiography (ECG) should not be used as a sole method of diagnosing CPA as some arrest rhythms, especially pulseless electrical activity (PEA), may be mistaken for a perfusing rhythm (4). When in doubt, CPR should be initiated as soon as possible; there is little evidence to suggest that basic life support measures will harm patients that are not experiencing CPA.

In small animal patients, the most common arrest rhythms on ECG include asystole or PEA (3), although the sudden development of a bradyarrhythmia may precede cardiac arrest ***(Figure 1a)***. Monitoring a continuous ECG in "at risk" patients is useful and may allow life-saving interventions before CPR is required. Continuous ECG monitoring may also help to identify cardiac arrhythmias that are best treated with electrical defibrillation, including pulseless ventricular tachycardia (VT) and ventricular fibrillation (VFib) ***(Figure1b)*** (4).

Obtaining client consent for CPR at the time of admission or prior to anesthetic procedures is recommended; it may be beneficial for clinicians to discuss with clients the potential need for CPR (as well as outcomes to be expected) whenever a patient is admitted. If the patient is then coded accordingly (*e.g.* via a color system) this ensures that all team members know what action should be taken should CPA occur (*e.g.* red (do not resuscitate), yellow (basic life support only, +/- administration of resuscitative drugs), or green (advanced life support, including open-chest CPR)).

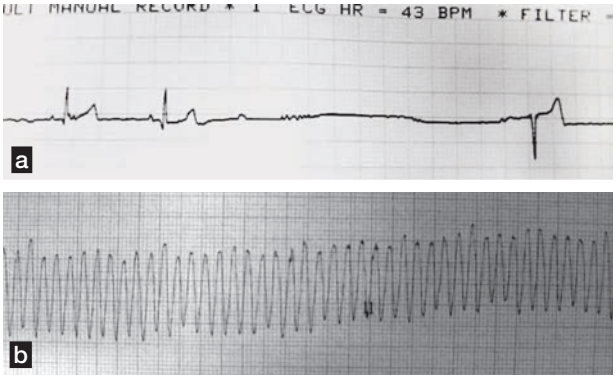

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Figure 1. An ECG can be of value in detecting CPA.
a. The sudden development of a bradyarrhythmia, such as sinus arrest with a ventricular escape beat as seen on the ECG, may precede cardiac arrest.
b. ECG monitoring may help identify cardiac arrhythmias such as ventricular fibrillation that are best treated with defibrillation.

Basic life support

Basic life support is the foundation for successful CPR and in the event of CPA clinicians are advised to follow the "ABC" mnemonic (airway, breathing, compressions) (3). A patent airway should be secured via endotracheal intubation as soon as possible. In some circumstances, intubation may be complicated by a presence obstructing the upper airway. Ready access to endotracheal tubes of various sizes, laryngoscopes, stylets and instruments for suction is recommended ***(Table 1)***. A laryngoscope can improve larynx visualization and its use may be facilitated by an assistant holding the mouth open and extending the tongue. When intubation is not possible due to complete upper airway obstruction, placement of a percutaneous tracheal catheter or urgent tracheostomy to bypass the obstruction may be required to allow for ventilation and oxygenation. The procedure for performing a surgical

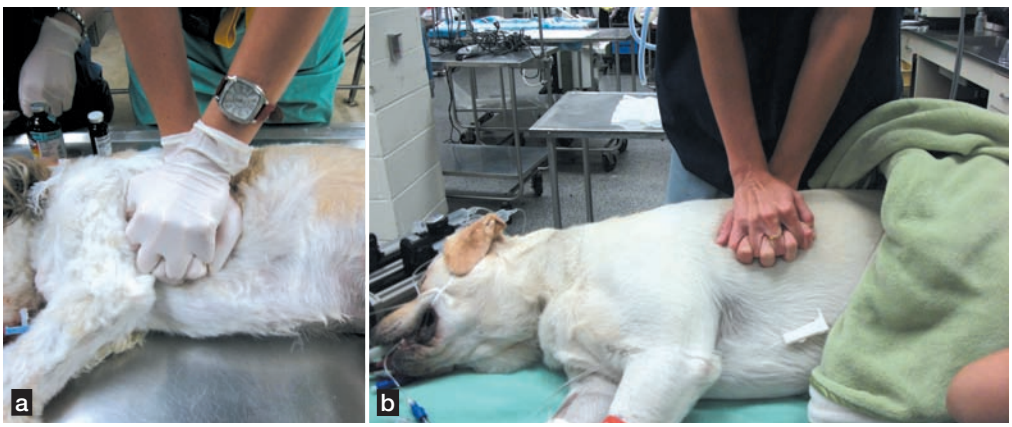

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Figure 2. Closed chest compressions with the patient in lateral recumbency. For small patients the hands are placed directly over the heart at the 5th intercostal space (a).

For larger patients, the hands are placed over the widest portion of the chest (b).

tracheostomy is outside the scope of this article and can be found elsewhere (5).

Proper placement of an endotracheal tube can be confirmed by visual assessment, intraoral palpation, or palpation of the tube within the trachea; the clinician can also auscultate the thorax for respiratory sounds. The tube should be secured in place and the cuff inflated to prevent fluid or foreign material from entering the airway. $ETCO_2$ is frequently not a useful indicator of endotracheal intubation in patients that have experienced CPA as delivery of carbon dioxide to the lungs may be diminished due to poor perfusion. However, a high $ETCO_2$ reading confirms proper intubation as little CO_2 is expected in the stomach or esophagus (4). When possible, intubation in lateral recumbency is preferred as this allows for concurrent initiation of chest compressions.

Once an airway is secured, ventilation with 100% oxygen is initiated at a rate of 10-12 breaths/min (6). This can be accomplished either by the use of an ambu-bag or the oxygen reservoir on an anesthetic machine. Ambu-bags have a built-in pressure relief valve to prevent barotrauma during ventilation; when an anesthetic machine is used, end-inspiratory pressure should be kept <20 cm H_20. Higher pressures may be needed for patients that are overweight or have poor thoracic compliance due to pulmonary disease, but excessive pressure should be avoided as it may cause pulmonary parenchymal injury or pneumothorax. Arterial partial pressure of carbon dioxide is a major determinant of cerebral vascular tone and therefore cerebral blood flow (7); consequently, care should be taken to avoid both hyper- and hypo-ventilation during CPR. Hypocapnea due to hyperventilation leads to vasoconstriction which can contribute to cerebral ischemia (7). Excessive ventilation may also cause positive intrathoracic pressure which can impede venous return of blood to the heart and decrease coronary perfusion pressure (8). When multiple rescuers are available, it can be advisable to nominate one individual to deliver a breath every 6 seconds.

Chest compressions should start as soon as possible and are best performed in lateral recumbency, with the clinician above the patient using upper bodyweight and straight arms to compress the chest; fatigue occurs quickly when triceps muscles are used with cyclical flexion and extension of the elbows. For patients weighing <15 kg, hands are placed directly over the heart at the 5th intercostal space, directly compressing the cardiac ventricles to promote forward flow of blood into the great arteries ("cardiac pump") ***(Figure 2a)***.

Table 1. Emergency cart checklist.

Airway/intubation
• Endotracheal tubes (various sizes from 2-12mm ID) • Laryngoscope with functional light and blades (various sizes) • Stylets for endotracheal tubes • Gauze sponges (to use when extending tongue) • Muzzle gauze (to secure endotracheal tube) • Air syringe or cuff inflator (to inflate endotracheal tube cuff)
Arrest drugs and other medications
• Epinephrine • Atropine • Vasopressin • Lidocaine • Sodium bicarbonate • Calcium gluconate • Dextrose • Naloxone • Flumazenil • Atipamezole
Defibrillation
• Defibrillation paddles • Posterior paddle adaptor (to place under patient) • Defibrillation contact gel
Surgical equipment
• Sterile surgical blades (for venous cut-down or other procedures) • Small surgical pack (for open-chest CPR)
Miscellaneous
• IV catheters and butterfly catheters (various sizes) • Suction catheters and canister • ECG patches • Tape • Syringes of various sizes • 3-way stopcocks • Suture material

For patients >15 kg hands are placed over the widest portion of the chest; direct compression of the thorax raises intra-thoracic pressure, driving blood forward ("thoracic pump") ***(Figure 2b)***. The thorax should be compressed by approximately 1/3-1/2 of its width and 100-120 compressions/min is recommended. Allowing for full elastic recoil of the chest following compression is essential – the negative intrathoracic pressure is necessary for return of blood to the great veins and heart (6). There is limited evidence to support interspersed abdominal compressions but this may help promote bloodflow back to the heart (6).

External chest compressions may generate approximately 25% of normal cardiac output. The person performing compressions should be rotated every two minutes to avoid fatigue (6) and the brief pause in compressions at this point is an appropriate time to assess for a heart beat or evaluate the ECG; otherwise, interruption of compressions should be kept to a minimum as it may take several minutes to re-establish adequate coronary perfusion after a pause (9).

Advanced life support

Advanced life support measures are an extension of basic life support in an effort to achieve ROSC. Ideally, both basic and advanced measures occur simultaneously, but if personnel are limited, the importance of ventilation and accurate chest compressions cannot be over-emphasized and pharmacologic therapy should only be utilized once these measures are initiated.

Coronary perfusion pressure is determined by aortic diastolic pressure and pressure in the right atrium. Likewise, cerebral perfusion pressure is the difference between mean arterial pressure and intracranial pressure (10). Vasopressor medications are used in CPR to increase peripheral vascular resistance which elevates aortic pressure; when used in conjunction with properly performed chest compressions one may optimize perfusion to the heart and brain.

Epinephrine (adrenaline) is a mixed adrenergic agonist acting at both α- and β-receptors. Epinephrine stimulates myocardial β_1-receptors increasing heart rate, myocardial contractility, and myocardial oxygen demand. β_2-mediated effects include relaxation of vascular smooth muscle and bronchodilation. Epinephrine is used in CPR primarily for its effect on vascular receptors, causing peripheral vasoconstriction and improving perfusion centrally in the brain and heart (11). In the literature both low and high-dose are discussed, but there is some evidence that high-dose epinephrine may have deleterious side-effects due to its adrenergic activity including increased myocardial oxygen demand in the face of poor perfusion and propagation of cardiac arrhythmias. The current recommendation is to administer low-dose epinephrine (0.01 mg/kg IV) every 3-5 min ***(Table 2)*** (12).

Arginine vasopressin (antidiuretic hormone) is an endogenous peptide vasopressor that has been investigated as an alternative to epinephrine in CPR. Vasopressin exerts an effect on vascular smooth muscle V_{1a} receptors resulting in peripheral vasoconstriction while seeming to preserve coronary and cerebral blood flow. Adrenergic receptors may not function properly in severe acidemia, as one might expect during CPA; conversely, vasopressin does not appear to be compromised in an acidemic environment and it has a longer half-life than epinephrine (11). The evidence for vasopressin use in veterinary CPR is mixed and limited mostly to research; one randomized, prospective study (13) comparing administration of epinephrine to vasopressin in dogs during CPR reported no difference in ROSC between the two groups. However, current guidelines support the use of vasopressin (0.8 U/kg IV) repeated every three

Table 2. Emergency drug dosages and defibrillator quick reference chart.

	Weight (kg)	2.5	5	10	15	20	25	30	35	40	45	50
Drug	Dose	mL	mL	mL	mL	mL	mL	mL	mL	mL	mL	mL
Epinephrine low-dose (1 mg/mL)	0.01 mg/kg IV	0.03	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
Epinephrine high-dose (1 mg/mL) *	0.1 mg/kg IV	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Vasopressin (20 U/mL)	0.8 U/kg IV	0.1	0.2	0.4	0.6	0.8	1	1.2	1.4	1.6	1.8	2
Atropine (0.54 mg/mL)	0.04 mg/kg IV	0.2	0.5	0.8	1.1	1.5	1.9	2.2	2.6	3	3.3	3.7
Lidocaine (20 mg/mL)	2 mg/kg IV (dogs)	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Naloxone (0.4 mg/mL)	0.04 mg/kg IV	0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Flumazenil (0.1 mg/mL)	0.02 mg/kg IV	0.5	1	2	3	4	5	6	7	8	9	10
Atipamezole (5 mg/mL)	0.1 mg/kg IV	0.05	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
	Energy level	J	J	J	J	J	J	J	J	J	J	J
Defibrillator - external	4-6 J/kg**	10	20	40	60	80	100	120	140	160	180	200
Defibrillator - internal	0.2-0.4 J/kg**	1	2	4	6	8	10	12	14	16	18	20

** High-dose epinephrine should be used with great care.** Energy listed is for a monophasic defibrillator (see reference 15).*

to five minutes in addition to, or instead of, epinephrine ***(Table 2)*** (12).

Atropine sulfate is an antimuscarinic parasympatholytic agent that blocks the effects of vagal afferents at the cardiac sinoatrial and atrioventricular nodes, increasing sinus rate and conduction velocity (14). Atropine (0.04 mg/kg IV) repeated every 3-5 minutes is the resuscitative drug of choice in animals with vagally-mediated arrest ***(Table 2)*** (12). This might be suspected in critically ill animals with high resting vagal tone, especially if bradycardia follows a period of retching, vomiting, coughing or straining to urinate/defecate. Continuously monitoring the ECG for sudden bradyarrhythmias in these patients is prudent, as pre-emptive atropine administration may prevent CPA.

Other pharmacologic agents that may be useful include anesthetic reversal agents, lidocaine and sodium bicarbonate. Naloxone may be given to reverse opioids, flumazenil reverses benzodiazepines, and atipamezole is the reversal for α_2-agonists ***(Table 2)***. Pulseless VT may be best treated with early electrical defibrillation but when defibrillation is not immediately available, lidocaine (2 mg/kg IV in dogs), a class Ib antiarrhythmic that blocks fast sodium channels, may be administered. Given the increased susceptibility to toxicity, lidocaine should be used cautiously in cats (0.2 mg/kg IV). Sodium bicarbonate, a buffer, is not recommended for routine administration; however, during a prolonged arrest (>10-15 min) it may be employed (1 mEq/kg IV) to treat acidemia (12).

Central venous administration of medications is recommended when a jugular catheter is already in place to provide the highest level of drug possible to the myocardium. However, central venous access may be difficult to obtain and compressions should not be interrupted for placement of a catheter. If a peripheral vessel is used, medications should be followed by administrating at least 10-20 mL of sterile saline to promote delivery of the medication centrally. Several medications, including epinephrine, vasopressin and atropine may be administered via an endotracheal tube. The drug can be diluted with saline and administered via a long catheter fed down the tube between breaths; in this situation epinephrine should be administered at the high-dose. There is some debate about the correct dosing of atropine and vasopressin when given intratracheally, but many clinicians give double the typical IV dose with the medication diluted.

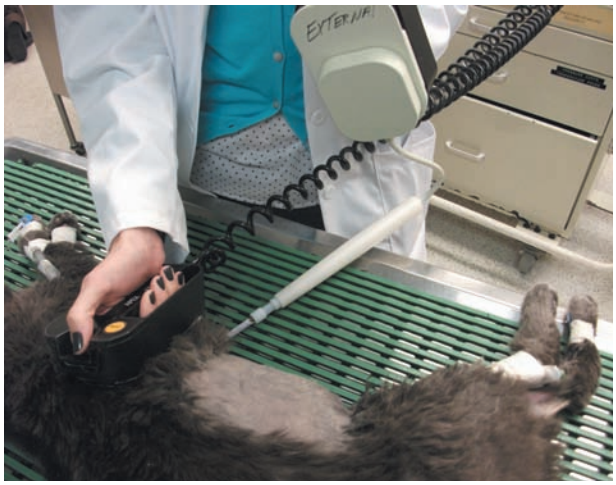

Figure 3. Defibrillation: if the patient is in lateral recumbency, a posterior paddle adapter may be placed under the patient to achieve electrode contact on the dependent side.

Electrical defibrillation is the treatment of choice for certain cardiac arrhythmias that may be noted during CPR, namely pulseless VT and VFib. Ventricular fibrillation results from random and uncoordinated electrical activity in the cardiac ventricles; electrical defibrillation attempts to globally depolarize the myocardium so that most myocardial cells enter a refractory period, which allows the sinoatrial node to resume function as the cardiac pacemaker. Monophasic defibrillators generate a unidirectional current that flows from one electrode to the other, while biphasic defibrillators develop a current that flows in both directions between the electrodes (15). The latter are preferred as these allow for lower defibrillation energy and may cause less myocardial injury. Good contact between the patient and electrodes is essential and defibrillator paste or gel should be applied prior to use; clip the fur as necessary. Electrodes should be placed with gentle pressure on either side of the thorax over the heart at the level of the costochondral junction. For patients in lateral recumbency, a posterior paddle adapter may be placed under the patient to achieve electrode contact on the dependent side ***(Figure 3)***. Otherwise, defibrillation in dorsal recumbency may be done with electrodes placed on either side of the thorax; use of a V-shaped trough may facilitate positioning. A starting energy of 4-6 J/kg for monophasic defibrillators and 2-4 J/kg for biphasic defibrillators is recommended for external defibrillation (15). Once the electrodes are charged, the operator must ensure that no one is in contact with the patient or any metal, including the exam table, as this may lead to severe personnel injury. If ROSC is not achieved after a shock, chest compressions should

be resumed for two minutes before evaluating the ECG to determine whether further defibrillation is necessary. If the first shock is unsuccessful, increasing the defibrillator energy by 50% is suggested (15). If VFib develops during CPR and electrical defibrillation is unavailable, a precordial thump may be attempted, although this is unlikely to be of much benefit.

Open-chest CPR via a lateral thoracotomy may be warranted in several circumstances. For very large patients, it is unlikely that external chest compressions will develop sufficient cardiac output to perfuse the brain and heart. External chest compressions may be ineffective when intrathoracic pressure is high due to accumulation of fluid, air or tissue within the pleural space, or when pericardial effusion is present. Thoracic wall injuries with broken ribs may preclude the use of external chest compressions as rib fragments may lacerate underlying pulmonary or cardiovascular tissue. For patients with significant intra-abdominal bleeding with subsequent CPA, open-chest CPR offers the theoretical advantage of occluding the descending aorta preventing further hemorrhage and allowing for preferential perfusion of the heart and brain. Finally, open-chest CPR is suggested for any patient where prolonged (>10 minutes) external chest compressions have failed to achieve ROSC.

When open-chest CPR is elected, a lateral thoracotomy is performed by rapidly clipping and aseptically preparing the thoracic wall at the 5th intercostal space. An incision is made from the dorsal chest wall to the costochondral junction on the cranial aspect of the rib and the underlying tissue is sharply dissected down to the level of the pleura. Blunt dissection with a finger or hemostat is used to enter the pleural space between breaths to avoid lung injury. The ribs are retracted (manually or mechanically) and the heart is compressed directly. For patients with pericardial effusion, the pericardium can be incised at the sternopericardial ligament, taking care not to damage the phrenic nerve. The descending aorta can be identified along the dorsal thoracic wall and may be occluded digitally, with sterile umbilical tape, or a Penrose drain (16). Internal defibrillation may be utilized if necessary with an initial energy of 0.2-0.4 J/kg; prior to this, sterile saline-soaked gauze should be placed between the paddles and the heart. Successful open-chest CPR must be followed by sterile flushing of the thoracic cavity and closure of the incision with placement of a thoracostomy tube. However, open-chest CPR should only be offered when 24-hour critical care is readily available for the post-operative period.

Monitoring during CPR

Of all the monitoring devices available, end-tidal capnography is probably the most useful for CPR situations. As stated previously, $ETCO_2$ is often initially very low or zero, due to pulmonary hypoperfusion and lack of CO_2 delivery to the lungs. An increase in $ETCO_2$ during CPR suggests that chest compressions are generating forward blood flow and $ETCO_2$ has been positively correlated with coronary perfusion. In a sense, end-tidal capnography allows for real-time assessment of cardiac output, and failure to increase $ETCO_2$ should prompt rescuers to re-evaluate their CPR strategy (4). In humans, $ETCO_2$ >10 mmHg within the first 20 minutes of CPR is predictive of ROSC with high sensitivity and specificity (17). When $ETCO_2$ monitoring is unavailable, palpation for peripheral pulses, auscultation for heart sounds and assessment of the ECG during pauses in CPR may help identify ROSC.

Post-resuscitative care

Many patients that achieve ROSC will experience re-arrest hours or days following their first CPA episode (1). Patients often develop a post-arrest syndrome that involves release of inflammatory cytokines, dysfunction of the microcirculation, increased vascular permeability with fluid loss into the interstitium, and myocardial stunning with subsequent decreased systolic function (18). Additionally, patients may succumb to whatever underlying pathology initially led to CPA. Successful CPR is only the first step in ensuring a good outcome for these patients, and intensive care and monitoring is generally required in the post-resuscitative period.

Hemodynamic optimization with use of intravenous fluid therapy, inotropes, and vasopressors should be instituted with goal-directed end-points including arterial normotension and resolution of lactic acidosis. Pulmonary contusions from chest compressions, pulmonary edema and pneumonia are common findings in the post-arrest period and patients may require supplemental oxygen titrated to maintain hemoglobin oxygen saturation (SpO_2) between 94-98% (15). Hyperoxia should be avoided as this may lead to generation of reactive oxygen species which can damage DNA, proteins and cellular membranes exacerbating injury (19,20). Blood glucose should be monitored and euglycemia is maintained by judicious dextrose supplementation or insulin therapy. Hypertonic saline or mannitol may be useful to treat cerebral edema secondary to prolonged ischemia. Patients may develop critical illness-related corticosteroid insufficiency (CIRCI) and will benefit from corticosteroids administered at a physiologic dose; however,

there is no evidence to support the routine use of higher doses of glucocorticoids in patients post-CPA (21).

Anesthetic arrests

Anesthetic arrests are considered separately here as these often have the best outcome, because many patients under general anesthesia already have an established airway, venous access, and cardiopulmonary monitoring equipment in place. In one study involving 204 patients with CPA, only 12 survived to discharge from the hospital; 75% of these patients were under anesthesia at the time of arrest (1). End-tidal capnography is a useful tool for monitoring for CPA in anesthetized patients; a sudden decrease in $ETCO_2$ can indicate pulmonary hypoperfusion as one would expect with CPA. If CPA is confirmed via auscultation, the endotracheal tube should be investigated for kinking or obstruction and the pop-off valve on the anesthesia machine should be inspected to ensure that it is not closed. All volatile gas anesthetics should be turned off with anesthetic reversal agents given as necessary. If the peritoneal cavity is opened for laparotomy an incision through the diaphragm allows for access to the heart for open-chest compressions. An easy-to-read emergency drug dosage chart ***(Table 2)*** placed in clear view where anesthetic procedures are performed is useful.

Conclusion

CPA is commonly encountered in small animals and should be suspected in any patient presenting as unresponsive, apneic or with an agonal breathing pattern. Basic life support measures, including establishing an airway for oxygenation and ventilation and accurately performing chest compressions, should be initiated promptly when CPA is suspected. Resuscitative drugs and defibrillation when an appropriate cardiac rhythm is present should ideally be performed early in the course of CPR but when a limited number of rescuers are available ventilation and chest compressions are a priority. After successful CPR, many patients will experience re-arrest and intensive care is required after resuscitation to optimize patient outcome.

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CUT-OUT AND KEEP...

Understanding acid-base imbalances in dogs and cats

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ACID-BASE

The body keeps pH within narrow limits, necessary for normal cellular function.

pH 7.35-7.45

Acidemia: Blood pH <7.35
Acidosis: Process causing an increase in acid or decrease in base.

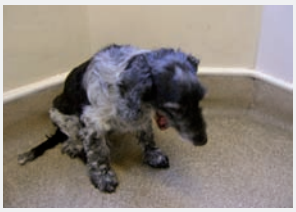
Alkalemia: Blood pH >7.45
Alkalosis: Process causing a decrease in acid or increase in base.

Proteins denatured – enzymes lose the ability to function.

Acid-base balance is composed of a respiratory and a metabolic component.

RESPIRATORY

- The respiratory component of an acid-base disorder depends on PCO_2, the amount - or partial pressure (P) - of CO_2 dissolved in the blood.
- CO_2 is produced as a by-product of cellular carbohydrate and lipid metabolism.
- CO_2 dissolves in blood, combining with water to form carbonic acid which lowers blood pH.
- The amount of CO_2 dissolved in the blood is controlled by alveolar ventilation in the lungs.



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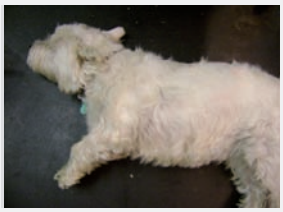
Increased ventilation (Hyperventilation)
→↓PCO_2 →↑pH
= **Respiratory alkalosis**

Common causes of respiratory alkalosis:
- Respiratory disease
- Hypoxemia
- Pyrexia
- Hyperthermia
- Pain
- Stress



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Decreased ventilation (Hypoventilation)
→↑PCO_2→↓pH
= **Respiratory acidosis**

Common causes of respiratory acidosis:
- Pleural space disease
- Upper airway obstruction
- Neurological disease
- Anesthetic drugs
- Severe respiratory disease

METABOLIC

- As well as PCO_2, cellular catabolism produces H^+ ions as volatile acids, formed as by-products of protein and phospholipid metabolism.
- The body produces various buffers to neutralize the acids until excreted by the kidneys:
  - Bicarbonate (most important)
  - Hemoglobin
  - Plasma proteins

↑acid or ↓base↔↓bicarbonate/negative base excess = ↓ pH = **Metabolic acidosis**

↓acid or ↑base↔↑bicarbonate/positive base excess = ↑pH = **Metabolic alkalosis**

Common causes of metabolic acidosis:
- Renal failure
- Lactic acidosis
- Diabetic ketoacidosis
- Ethylene glycol toxicity
- Diarrhea/small intestinal vomiting

Common causes of metabolic alkalosis:
- Gastric vomiting
- Administration of diuretics
- Administration of sodium bicarbonate

COMPENSATION AND MIXED DISORDERS

- The body attempts to compensate for an altered blood pH.

Acid-base disturbance	Compensation mechanism	Time taken compensation
Metabolic acidosis	Increase in ventilation causing respiratory alkalosis	Minutes to hours
Metabolic alkalosis	Decrease in ventilation causing compensatory respiratory acidosis	Minutes to hours, but limited as may cause hypoxemia
Respiratory acidosis	Increased absorption of bicarbonate by kidneys causing metabolic alkalosis	Hours to days
Respiratory alkalosis	Increased excretion of bicarbonate by kidneys causing metabolic acidosis	Hours to days

Note:
- The body never over-compensates, so blood pH always moves in direction of primary disorder.
- More than one acid-base disorder may occur at the same time.
- They may partially or fully cancel each other out causing blood pH to be normal, or have an additive effect if both are acidotic or alkalotic disorders.

ASSESSING ACID-BASE RESULTS

Both arterial and venous blood may be used with appropriate reference ranges.

1) Look at the blood pH – is the patient:

a) Normal – pH 7.35-7.45?
b) Acidemic – pH <7.35?
c) Alkalemic – pH >7.45?

2) Look at the PCO_2 – is the patient:

a) Normal, between 35-45 mmHg?
b) Hyperventilating with a respiratory alkalosis – PCO_2 below 35 mmHg?
c) Hypoventilating with a respiratory acidosis – PCO_2 above 45 mmHg?

Is PCO_2 in the same direction as pH *e.g.* an alkalemia with a respiratory alkalosis?

- Yes – Together with history and clinical examination think about possible causes of primary disorder to guide further tests such as chest radiographs, but also check step 3 for a mixed disorder.
- No – This could be compensation or a mixed disorder.

3) Look at the base excess (BE) or bicarbonate - does the patient have:

a) Normal BE between -4 to +4 (bicarbonate 18-26 mmol/L)?
b) Metabolic acidosis – BE lower than -4 (bicarbonate less than 18 mmol/L)?
c) Metabolic alkalosis – BE higher than +4 (bicarbonate over 26 mmol/L)?

Are BE and bicarbonate in the same direction as pH (*e.g.* an acidemia with a metabolic acidosis)?

- Yes – Together with history and clinical examination, think about possible causes of primary disorder to guide further tests, such as measuring blood glucose and urine/plasma for ketones.
- No – This could be compensation or a mixed disorder.

TREATMENT

- Treatment will depend on the underlying cause of the acid-base disturbance.
- Metabolic acidosis is the disorder most often identified and is most commonly secondary to a lactic acidosis (shock). Appropriate treatment of the shock, commonly with fluid therapy, will lead to resolution. Administration of bicarbonate should only be considered in cases where pH is <7.2 and cannot be increased by simple means such as fluid therapy.
- Metabolic alkalosis does not need specific treatment; concurrent electrolyte disorders are common and should be assessed and treated.
- Respiratory acidosis may need to be treated with artificial ventilation or oxygen supplementation; this should be considered if arterial PCO_2 is persistently greater than 60 mmHg.
- Respiratory alkalosis does not generally need specific treatment.

"THERE WILL BE BLOOD"

For the 12'th EVECCS congress the Small Animal program will focus on the cardiovascular system in the critically ill (haemostasis and cardiology). The Equine program will focus on neonatology and the acute abdomen. These themes provide a wide range of topics and internationally renowned speakers will present what is new in their field.

Veterinary streams (small animal and equine)
Nursing stream
Workshops
Scientific abstracts
Exhibition

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