

## Critical anaesthesia

Considerations for ECC patients

## Pain management

Analgesia for the  
ECC patient

## Critical care nursing

Triage of emergency cases















# Caring for the emergency patient



## Also in this volume:

Fluid therapy, head trauma, gastrointestinal foreign bodies,  
prognostic factors for septic peritonitis, FECAVA news,  
veterinary app & book reviews ... and more!



## Contents

- |   |   |   |       |
|---|---|---|-------|
|    |    | Analgesia for the emergency/critical care patient –<br>part 1: Pain assessment  | 4-7   |
|   |   | <i>Shalien Jasani</i>   |       |
|    |    | Analgesia for the emergency/critical care patient –<br>part 2: Analgesic agents   | 8-18  |
|   |   | <i>Shalien Jasani</i>   |       |
|  |  | Fluid therapy in dogs and cats  | 19-26 |
|   |   | <i>SLeona Raušerová-Lexmaulová</i>  |       |
|  |  | Anaesthesia considerations for critically ill patients  | 27-35 |
|   |   | <i>Ruxandra Costea</i>  |       |
|  |  | Linear versus non-linear gastrointestinal foreign bodies in 499 dogs:<br>clinical presentation, management and short-term outcome | 36-43 |
|   |   | <i>Melissa M. Hobday, Garret E. Pachtinger, Kenneth J. Drobatz<br/>and Rebecca S. Syring</i>                                      |       |
|  |  | Septic peritonitis from pyloric and non-pyloric gastrointestinal<br>perforation: prognostic factors in 44 dogs and 11 cats        | 44-50 |
|   |   | <i>Thomas Dayer, Judith Howard and David Spreng</i>   |       |
|  |  | Nursing the critical care patient – part 1: triage  | 51-55 |
|   |   | <i>Katherine Howie</i>  |       |

# Icons

Each scientific article is classified with one or more icons.

These refer to the species (in green) of animal or the veterinary discipline (in blue) relevant for the article.

	Dogs		Anaesthesia		Oncology
	Cats		Bacterial Diseases		Ophthalmology
	Dogs and Cats/ Small animals		Behaviour		Orthopaedics
	Rabbits		Cardiovascular		Practice Management
	Less common pets		Dental		Reproduction
			Dermatology		Surgery
			Diagnostic imaging		Urogenital
			Digestive System		Zoonosis
			Ear Nose Throat		
			Genetics		
			Internal Medicine		
			Neurology		





## Commissioned paper\*

# Analgesia for the emergency/critical care patient – part 1: Pain assessment

Shailen Jasani<sup>1</sup>

### SUMMARY

Pain is associated with a number of adverse physiological and psychological responses including unnecessary suffering, altered mentation (anxiety, agitation; dullness, depression), reduced food intake (which may slow recovery), prolonged recumbency, self-mutilation, impaired respiration, sensitisation to noxious stimuli, excessive sympathetic stimulation, enhanced catabolism, delayed wound healing and immune suppression. It is also difficult to monitor patients effectively due to pain-induced physiological changes. Furthermore, untreated acute severe post-operative pain increases the chances of developing chronic pain.

Adequate pain management is therefore essential, both for patient welfare and physical health. This paper will present analgesic approaches to the emergency/critical patient and an overview of commonly used analgesics in veterinary emergency settings.

\*This paper was commissioned by FECAVA for *Eur J Comp An Pract (Autumn 2016) 26(3); p4-p7*. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

### Introduction

“The inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain relieving treatment” (International Association for the Study of Pain 1994). The American Veterinary Medical Association in 2001 stated, “The AVMA believes that animal pain and suffering are clinically important conditions that adversely affect an animal’s quality of life. Drugs, techniques, or husbandry methods used to prevent and control pain must be tailored to individual animals and should be based, in part, on the species, breed, age, procedure performed, degree of tissue trauma, individual behavioural characteristics, degree of pain and health status.”

### Pain Assessment

In 2011 it was the Global Year Against Acute Pain in human medicine with the motto: “Anticipate, Assess, Alleviate”. In veterinary medicine we should amend this to “Anticipate, Assess/Assume, Alleviate”.

Pain assessment in animals involves a combination of sign recognition, patient interaction, clinical experience and anthropomorphism.

### Signs of Pain

The signs that an animal in pain may exhibit depend on various factors including the source, severity and duration (acute, chronic) of pain as well as individual patient variability – age, species, breed, tolerance, and temperament.

<sup>1</sup> Shailen Jasani MA VetMB MRCVS DipACVECC, UK. <http://www.vetecsmalltalk.com>, email [shailenjasani@gmail.com](mailto:shailenjasani@gmail.com)

**Potential signs of pain include:**

- Consistent response on palpation of painful area
- Poor appetite
- Restlessness (especially dogs); dullness, depression and lack of activity (especially cats)
- Looking, licking, biting or scratching at painful area; self-mutilation
- Salivating
- Vocalisation:  
dogs – growling, whining, groaning;  
cats – purring, growling, hissing
- Reduced grooming behaviour
- Altered gait and reluctance to walk
- Abnormal posture (especially cats who often sit with a hunched sternal posture rather than lying curled in lateral recumbency)
- Reluctance to sleep
- Uncharacteristic behaviour (aggression or affection) towards humans
- Inappropriate urination and/or defaecation
- (Unexplained tachycardia, tachypnoea/panting, hypertension, or pupillary dilation)

Cats are often much less demonstrative than painful dogs and sick animals in general may be unable to show expected behavioural signs of pain. In addition, physiological parameters such as heart rate and respiratory rate are not consistently reliable as indicators of pain. Differentiating pain from other stress (e.g. fear, anxiety, claustrophobia) as well as from dysphoria/narcotisation can be challenging. Differentiating pain from anaesthesia-related behaviours (“emergence delirium”) is usually less challenging.

If it is not clear whether an animal is in pain, the patient should always be given the benefit of the doubt, and analgesic drugs carry minimal risk as long as they are used judiciously. A low testing dose can be given and the patient monitored for a favourable response.

**Pain Scales**

Pain scales can be used as a means of trying to improve objectivity by quantifying pain for example to:

- Potentially improve reliability of (re)assessment
- Allow analgesic efficacy to be assessed and drug doses titrated
- Provide better consistency for hospitalised patients; facilitate more long-term pain management of

hospitalised patients by trying to provide some consistency and hand-over between different staff members working on consecutive shifts.

- Facilitate statistical analysis and clinical research

Pain assessment is always going to be subjective in the non-communicating patients that veterinary staff deal with but multidimensional composite pain scoring systems try to reduce the degree of subjectivity and therefore improve consistency, reliability and hopefully patient welfare.

**The Glasgow Composite Measure Pain Scale (GCMPS)**

(Holton et al, 2001) is the only pain scale validated for assessment of acute pain in dogs thus far. It is a behaviour-based system based on a structured questionnaire that includes clinical observation, assessment of spontaneous and evoked behaviour and animal-observer interaction. The questionnaire is structured around seven categories: posture, activity, vocalisation, attention to wound/painful area, demeanour, mobility and response to touch. Based on the score obtained at the end, it is possible to quantify the degree of pain the animal is experiencing.

Other composite pain scoring systems exist but GCMPS is considered most scientifically derived, validated and reliable. It was derived using psychometric principles and statistical methodologies following approach taken by Melzack (1975); this 1975 paper has been modified/revised but has stood the test of time. The original GCMPS was developed into an interval level scale (Morton et al, 2005) and then a Short Form and intervention score developed for greater clinical usability (Reid et al, 2007). It is noteworthy that while the GCMPS is the most widely used and reported system and has been applied to pain from any cause, much of what has been reported relates to acute post-operative pain and also chronic osteoarthritic pain.

Other examples include Colorado State University Canine and Feline Pain Scales. The Colorado Feline Pain Scale is probably the feline pain scoring system with which there is the greatest clinical experience. In December 2014 the Glasgow Composite Measure Pain Scale for Acute Pain in Cats: CMPS – Feline was published which is a revised version of an earlier version from 2010. More clinical experience clearly needs to be obtained with this new system and the authors themselves are intending to further develop the scale by incorporating a facial expression

component with the intention of improving its sensitivity (Holden et al, 2014).

## Pain Scales

Examples of pain scales and assessment questionnaires:

CMPS Acute pain measurement of dogs and cats (NewMetrica)

- [www.newmetrica.com/acute-pain-measurement/](http://www.newmetrica.com/acute-pain-measurement/)

CMPS – Feline

- [www.wsava.org/sites/default/files/Feline%20CMPS%20-%20SF.pdf](http://www.wsava.org/sites/default/files/Feline%20CMPS%20-%20SF.pdf)

Colorado State University Canine and Feline Pain Scales

- [csu-cvmb.colostate.edu/Documents/anesthesia-pain-management-pain-score-feline.pdf](http://csu-cvmb.colostate.edu/Documents/anesthesia-pain-management-pain-score-feline.pdf)
- [csu-cvmb.colostate.edu/Documents/anesthesia-pain-management-pain-score-canine.pdf](http://csu-cvmb.colostate.edu/Documents/anesthesia-pain-management-pain-score-canine.pdf)
- [www.vasg.org/pdfs/CSU\\_Acute\\_Pain\\_Scale\\_Kitten.pdf](http://www.vasg.org/pdfs/CSU_Acute_Pain_Scale_Kitten.pdf)

## Pain Pathways

A basic knowledge of the pain pathways is essential to understand the modes of action of available analgesics and the rationale behind multimodal therapy. The pain pathway essentially consists of peripheral and central components. Peripheral tissue receptors (nociceptors) detect the painful stimulus and the subsequent signal is then conducted via primary afferent nerve fibres to the dorsal horn of the spinal cord or to the cranial nerve nuclei. Nociceptive signals are processed (modulated) first in the spinal cord and subsequently in higher centres resulting in conscious pain perception.

Transduction is the translation of physical energy (noxious stimuli) into electrical activity at the peripheral nociceptor. Transmission is the propagation of nerve impulses through the nervous system. Afferent sensory fibres consist of myelinated A-delta fibres which conduct fast pain, and non-myelinated C fibres which conduct slower dull pain. Modulation: processing (amplification or inhibition) of stimuli within spinal cord dorsal horn cells. Perception, not considered a part of the nociceptive

process, results from integration of the noxious stimuli in the thalamocortical, reticular, and limbic regions of the brain to produce the final conscious subjective and emotional experience of pain.

Injury to peripheral tissues is often associated with inflammatory pain and injury to the central nervous system associated with neuropathic pain.

## Sensitisation

Following injury there is increased sensitivity to further noxious stimulation due to changes in nervous system processing; this is referred to as sensitisation or 'wind up'. Sensitisation may occur in either or both of the peripheral and central components of the pain pathway and may result in continued pain, hyperalgesia (exaggerated responsiveness to a given noxious stimulus) or allodynia (stimuli that are normally innocuous become painful). Inflammatory mediators are involved in central and especially peripheral sensitisation; the glutamate-activated N-methyl-D-aspartate (NMDA) receptor is very important in central sensitisation.

In practical terms, once sensitisation is underway, conscious perception of pain is greater, it is more difficult to control and higher drug doses are required. It is therefore preferable to try and prevent pain rather than to merely treat it once it is established. A significant proportion of emergency patients are already in pain at initial presentation. In order to minimise sensitisation, pain management should be instituted as soon as possible and, once the pain is under control, the patient kept on a level pain-free plateau. All animals going on to have operative procedures performed will benefit from adequate preoperative analgesia, i.e. in advance of the significant noxious stimulation resulting from surgical intervention. And it is clearly important to bear in mind the onset of action the agent you are using has to prevent noxious stimulation before analgesic efficacy has been achieved (e.g. following subcutaneous administration NSAIDs take 0.5-2.5 hours to reach maximum plasma concentration depending on the specific agent being used).

## Pain perception

More recently people are starting to consider if and how a patient's perception of pain may be affected by mood, stress or anxiety, i.e. as well as being better for patient welfare per se, improving mood may reduce perception of any pain experienced. This raises the possibility that there is a rationale for considering mood enhancing preparations such as Zylkene®, pheromones etc. to reduce pain perception.

## References

- Calvo G, Holden E, Reid J, et al. Development of a behaviour-based measurement tool with defined intervention level for assessing acute pain in cats. *J Sm Anim Pract* 2014. 55(12):622-629.
- Giordano T, Steagall PV, Ferreira TH, et al. Postoperative Holden E, Calvo G, Collins M, et al. Evaluation of facial expression in acute pain in cats. *J Sm Anim Pract* 2014. 55(12):615-621.
- Holton L, Reid J, Scott EM, et al. Development of a behaviour-based scale to measure acute pain in dogs. *Vet Rec* 2001. 148:525-531.
- Melzack R. The McGill pain questionnaire: major properties and scoring methods. *Pain* 1975. 1:277-299.
- Merola I and Mills DS. Behavioural Signs of Pain in Cats: An Expert Consensus *PLoS One*. 2016; 11(2): e0150040. Published online 2016 Feb 24. doi: 10.1371/journal.pone.0150040
- Morton CM, Reid J, Scott ME, et al. Application of a scaling model to establish and validate an interval level pain scale for assessment of acute pain in dogs. *Am J Vet Res* 2005. 66:2154–2166.
- Reid J, Nolan AM, Hughes JML, et al. Development of the short-form Glasgow Composite Measure Pain Scale (CMPS-SF) and derivation of an analgesic intervention score. *Animal Welfare* 2007. 16(S):97-104.



## Commissioned paper\*

# Analgesia for the emergency/critical care patient – part 2: Analgesic agents

Shailen Jasani<sup>1</sup>

### SUMMARY

A significant proportion of emergency patients are already in pain at initial presentation. In order to minimise sensitisation, pain management should be instituted as soon as possible and, once the pain is under control, the patient kept on a level pain-free plateau. All animals going on to have operative procedures performed will benefit from adequate preoperative analgesia, i.e. in advance of the significant noxious stimulation resulting from surgical intervention. And it is clearly important to bear in mind the onset of action the agent you are using has to prevent noxious stimulation before analgesic efficacy has been achieved.

\*This paper was commissioned by FECAVA for *Eur J Comp An Pract (Autumn 2016) 26(3)*; p8-p18. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

Balanced analgesia involves the use of one or more appropriate analgesics (multimodal therapy) in an effective dosing regimen given by the appropriate route(s). By combining multiple analgesic drug classes or techniques to target different points along the pain pathway, multimodal therapy should offer:

- Additive analgesic effects
- A reduction in the doses of individual drugs
- Reduced frequency and severity of adverse side-effects

Analgesic agents that are commonly available in emergency clinics in countries in which veterinary medicine is more developed include:

- Opioids
- Non-steroidal anti-inflammatory drugs (NSAIDs)
- Local analgesic/anaesthetic agents
- Ketamine
- Medetomidine
- Gabapentin/Amantadine

An intuitive approach to pain management is based on thorough initial evaluation followed by regular reassessment of the patient and adjustment of the treatment protocol as

indicated. This is especially relevant to the use of full agonist opioids which may be used at highly variable dose rates and dosing frequencies. As described later, non-pharmacological measures are also very important in pain management.

### Opioids

All veterinary practices that may see animals in moderate to severe pain should have a full (pure) opioid agonist available, ideally morphine or methadone. These agents are extremely affordable, highly effective, and allow dose titration to effect. Although they need to be stored and administered in accordance with regulations for controlled drugs, this should not be considered an impediment to their use.

It is a misconception that all opioids cause excitement or so-called 'morphine mania' in cats; unfortunately this fear of excitement has been one of the reasons why practitioners have historically been hesitant to use opioids in cats. Such reports were based on early literature when excessive doses, far in excess of those required to provide analgesia, were administered to typically healthy patients. Opioids (predominantly) act in the central component of the pain pathway. Most opioids used clinically are selective

<sup>1</sup> Shailen Jasani MA VetMB MRCVS DipACVECC, UK. <http://www.vetecsmalltalk.com>, email [shailenjasani@gmail.com](mailto:shailenjasani@gmail.com)



for the  $\mu$ -receptor and are full (pure) agonists, partial agonists, or agonists-antagonists. They are the mainstay of pain management in the emergency patient and block central sensitisation. Opioids are also used in low doses for sedation or anxiolysis.

High doses of pure opioids in particular may result in dysphoria (vocalisation, anxiety, restlessness) that can be difficult to differentiate from on-going pain. Naloxone (or butorphanol) can be used to reverse the effects of full opioid agonists. Both respiratory depression and bradycardia are rarely seen as side effects of concern during opioid administration in clinical veterinary patients that are not under anaesthesia. Furthermore, pain itself can cause respiratory compromise and appropriate opioid administration can normalise respiratory status in such cases. Until adequately treated, pain can also confuse assessment of other parameters such as heart rate and blood pressure.

Dose rates, routes of administration and dosing frequencies of opioids in current use in emergency clinics are summarised in table 1.

### Morphine

This full  $\mu$ -agonist is the gold standard analgesic for moderate-severe pain. The onset of action is usually 10-15 min (or less) following intravenous or intramuscular administration. It is also extremely useful as an anxiolytic in respiratory distress.

Vomiting (both dogs and cats) is more likely than with other opioids and will further exacerbate raised intracranial or intraocular pressure – avoid in patients where this is of concern. Furthermore, rapid intravenous administration may cause signs associated with histamine release (transient vasodilation and hypotension) – slow intravenous boluses or a constant rate infusion is recommended.

### Methadone

This full  $\mu$ -agonist may also have an effect as a non-competitive NMDA receptor antagonist. It has the same (or greater) potency as morphine. Its sedative and dysphoric effects are reportedly less marked than with morphine and it does not typically induce emesis. Contrary to morphine, it does not cause histamine release following intravenous administration.

It is not usually recommended to be used as a constant

*Table 1. Dose and routes of administration of opioids commonly used in emergency clinics*

Drugs	Dogs	Cats
Morphine	0.1-1.0 mg/kg slow IV, IM or SC; as needed, q 4 h  0.1-0.5 mg/kg/h CRI	0.1-0.5 mg/kg slow IV, IM or SC; as needed, q 6 h  0.05-0.2 mg/kg/h CRI
Methadone	0.1-1.0 mg/kg slow IV, IM or SC; as needed, q 4 h	0.1-0.5 mg/kg slow IV, IM or SC; as needed, q 6 h
Fentanyl	2.0-10.0 $\mu$ g/kg slow IV; as needed, q 20 min  2.0-10.0 $\mu$ g/kg/h CRI (after initial loading dose 2.0-10.0 $\mu$ g/kg)	1.0-5.0 $\mu$ g/kg slow IV; as needed, q 20 min  1.0-5.0 $\mu$ g/kg/h CRI (after initial loading dose 1.0-5.0 $\mu$ g/kg)
Buprenorphine	0.01-0.02 mg/kg IV, IM or SC; q 6-8 h	0.01-0.02 mg/kg IV, IM, SC or OTM; q 6-8 h
Butorphanol	0.1-0.6 mg/kg IV, IM or SC; as needed, q 1-4 h  0.05-0.2 mg/kg/h CRI	0.1-0.6 mg/kg IV, IM or SC; as needed, q 1-4 h  0.05-0.2 mg/kg/h CRI
Naloxone (antagonist)	0.01-0.1 mg/kg slow IV to effect	0.01-0.1 mg/kg slow IV to effect

CRI – constant rate infusion; IM – intramuscular; IV – intravenous; OTM – oral transmucosal; SC – subcutaneous

rate infusion due to variable half-life and potential to accumulate. Panting (due to effects on the thermoregulatory centre) occurs much more commonly in dogs than it does with morphine and this may complicate clinical assessment. The author has not noticed tachypnoea or open-mouth breathing in cats following methadone administration.

There is now a methadone preparation licensed in Europe for use in dogs for analgesia and premedication (Comfortan<sup>®</sup> 10 mg/ml, Eurovet Animal Health Ltd)

### Fentanyl

This full (pure)  $\mu$ -agonist is very potent (50 times more potent than morphine) and has a rapid onset (seconds-minutes) and short duration (10-20 minutes depending on dose) of action, which makes it very amenable to titration. After prolonged administration or high doses, its

duration of action is significantly prolonged at the tissues become saturated. It can be used as one-off bolus (e.g. at induction or to facilitate procedures), as intermittent boluses or as a constant rate infusion.

It should be given slowly intravenously (rapid administration can cause severe bradycardia, which will respond to atropine). Patients on fentanyl infusions must be monitored closely for bradycardia or respiratory depression (transdermal fentanyl patches are available but will not be discussed here).

### **Buprenorphine**

Buprenorphine is a partial  $\mu$ -agonist and a  $\kappa$ -antagonist. Its onset of action is slow, up to 45 min, which makes it unsuitable for rapid management of pain; full opioids are preferred in such cases.

It is indicated for mild to moderate pain. Licensed for both dogs and cats, it appears to be especially effective as an analgesic in cats – but please note comment above regarding delay in onset of action!

The mixed agonist-antagonist actions of buprenorphine mean that doses above a certain level may be progressively less effective (i.e. buprenorphine has a bell-shaped dose/response curve with a ceiling effect). This means that in theory there is limited capacity to increase the administered dose of buprenorphine in an animal that remains painful. However, the ceiling may be higher than the typical clinical dose range used and this may therefore be more of a theoretical than a real concern. Increasing doses reportedly increase the duration of effect but do not increase the analgesia achieved.

Buprenorphine is a competitive  $\mu$ -agonist that is reported to have strong receptor affinity and in theory clinical doses of alternative opioids are therefore unlikely to be effective for a variable but lengthy period of time, i.e. until the buprenorphine has detached from the receptors. However, some 'experts' question whether this is relevant at clinical doses and the author's clinical experience suggests that full agonists can be used effectively much sooner than traditionally thought possible; this may be due to surplus receptors that are free to bind to the full agonist or potentially because the full agonist displaces some of the buprenorphine from receptors.

A subcutaneous preparation of buprenorphine that

reportedly has a 24-hour duration of action and only requires once daily dosing has recently been licensed in the USA.

A sustained-release formulation of buprenorphine designed to give 72 hours analgesia after subcutaneous administration has been tested in cats having ovariohysterectomy and there is some clinical experience with it in the United States with both cats and dogs.

### **Oral transmucosal buprenorphine**

Orogastric buprenorphine preparations are available for people but have not been widely used in veterinary medicine. However, 1-2 experimental reports in a small number of healthy cats report that oral transmucosal (OTM) administration of injectable buprenorphine results in both very high bioavailability and very similar pharmacokinetics to the intravenous route. The solution is tasteless and does not typically cause salivation or nausea. Administration of buprenorphine under the tongue or into the side of the mouth may therefore be a useful option in cats requiring continued short-term analgesia at home, either in addition to a non-steroidal anti-inflammatory agent or where the use of a non-steroidal anti-inflammatory agent is inappropriate. However it is worth noting that one study (Giordano et al, 2010) of 100 female cats undergoing ovariohysterectomy concluded that "IV and IM administration of buprenorphine provided better postoperative analgesia than SC or OTM administration of the drug and these routes of administration should be preferred when buprenorphine is administered to cats." Bioavailability of oral transmucosal buprenorphine is likely to be lower in dogs (due to more alkaline oral pH), necessitating considerably higher doses than those used for intravenous, intramuscular or subcutaneous administration. The use of buprenorphine by this route using standard injectable formulations is therefore likely to be impractical in dogs.

### **Butorphanol**

This  $\mu$ -antagonist and a  $\kappa$ -agonist have an onset of action of 15 to 20 minutes. It is clinically more useful for its sedative and antitussive effects than as a potent analgesic. Because it is a  $\kappa$ -agonist, it may be more effective for mild-moderate visceral pain than some other opioids (i.e. as there may be more  $\kappa$  receptors in the viscera than elsewhere); this may especially be the case for gastrointestinal pain. However it is unclear if visceral

analgesia is achieved at typical clinical doses (e.g. 0.2-0.3 mg/kg) or requires higher doses (e.g. 0.8 mg/kg).

It may also be used to reverse sedation and respiratory depression associated with full opioid use (due to  $\mu$ -antagonist activity). It is licensed for use in dogs and cats

### **Tramadol**

This synthetic centrally-acting analgesic has complex interactions between opiate (primarily  $\mu$ ), serotonin and adrenergic receptor systems (it also has an effect as an NMDA receptor antagonist). At least one metabolite (*O*-desmethytramadol; M1) is significantly active and may be essential in tramadol's opioid effect. It may be indicated for long-term home use in cases in which NSAIDs are contraindicated or insufficient alone. It is probably the safest current choice in nursing dams post-caesarean section.

The main clinically relevant side effects are lethargy, sedation and nausea. The potential for cardiovascular and respiratory depression may be greater following parenteral (IV) administration but is unlikely to be clinically relevant when used judiciously.

Naloxone will partially antagonise the effects of tramadol. The currently recommended dose range for dogs is 1-5 mg/kg q 6-12 hours. A similar range is likely to be appropriate for cats but they should be started on the lower end of the dose range and/or upper end of the dosing interval, and then titrated upwards as necessary. However it is important to realise that there is still some uncertainty about the optimal dosing regime in veterinary patients. Experimental studies in healthy animals show that the pharmacokinetics of tramadol in veterinary patients is different to that in humans. In humans dosing is usually done every 4-6 hours and it may be that this frequency is appropriate in dogs and cats (i.e. more frequently than is typically currently used); moreover what doses are likely to prove effective at different intervals still remains to be clarified. Depending on geography, tramadol is reportedly available as various tablet strengths as well as other oral formulation types, an extended-release formulation, a combination preparation with paracetamol (acetaminophen)\*, and an injectable preparation. Note that the oral preparation containing paracetamol should never be used in cats.

### **Titration**

Full opioids – morphine, methadone, fentanyl – are

typically the most appropriate analgesic agents for use in moderate to severe pain. Their efficacy, rapid onset of action, linear dose/response curve, and high index of safety make them highly suitable for titration in severe pain management.

The initial dose chosen should be guided by the severity of pain identified or suspected; intuitively, the same dose of the same drug cannot be appropriate in all situations. The patient should then be reassessed once the drug has had enough time to take effect and additional analgesia administered if indicated. Mydriasis is usually a sign of adequate opioid administration in a cat.

Clinically significant side effects of full opioids are rare in companion animals that are not under general anaesthesia and these concerns should not prevent administration of adequate analgesia.

### **Constant rate infusions**

Constant rate infusions (CRI) of opioids (morphine in particular), as well as ketamine and lidocaine, are in widespread use as they offer clear advantages for pain management and most of the cases in question will have intravenous access available. Constant rate infusions avoid the peaks and troughs in plasma drug concentrations associated with intermittent boluses. They also provide more sensitive control over the degree of analgesia being provided thereby avoiding both breakthrough pain and overdosing. There is no reason why this mode of drug administration cannot be employed in the first opinion setting as long as facilities allow and adequate monitoring is available.

The author would only recommend the use of a constant rate opioid infusion if an infusion pump or syringe driver is available; without their use, it is very difficult to guarantee a constant rate of infusion and there are inherent risks of both under- and overdosing.

When a constant rate infusion is used, it is vital to ensure that all calculations are double-checked, clearly documented and legible to avoid confusion and potentially disastrous errors.

### **Morphine-lidocaine-ketamine (MLK)**

The idea behind the use of this multiagent infusion is to provide multimodal analgesia with the inherent benefits that that strategy offers.

Although some institutions use standardised prepared stock solutions of morphine, lidocaine and ketamine (MLK) for convenience, the author prefers to make up the CRI solution on an individual patient basis with the initial concentrations being guided by the patient in question. Stock solutions mean that the drug concentration is fixed and the only way to adjust the doses being delivered is by adjusting the fluid rate. MLK infusions are often administered at 1-2 ml/kg/hour to start with and there is therefore relatively little scope for dose adjustment when a stock solution is used without the potential for excessive/unnecessary fluid administration as well. Moreover the

doses of the individual agents being used cannot be adjusted independently.

Computerised calculators, including mobile phone applications, are increasingly available for working out how to prepare an MLK solution. In their absence, this is still relatively simple to do (see Fig. 1).

Although the use of MLK may make sense in terms of multimodal analgesia it is important to realise that to date, to the author's knowledge, there are no clinical studies evaluating its efficacy in painful clinical patients including

Figure 1. Example of preparation of a morphine, lidocaine and ketamine (MLK) solution for constant rate infusion

**Patient's name:**

**Patient's body weight:** 20 kg

**Initial infusion rate:** 1 ml/kg/h = 20 ml/h

**1. Select initial dose rates for analgesic agents:**

Drug: **morphine** (10 mg/ml)

Initial dose rate: 0.2 mg/kg/h = 4 mg/h

Drug: **lidocaine** (20 mg/ml i.e. 2%)

Initial dose rate: 50 µg/kg/min = 1000 µg/min = 1 mg/min = 60 mg/h

Drug: **ketamine** (100 mg/ml)

Initial dose rate: 0.5 mg/kg/h = 10 mg/h

**2. Select volume of crystalloid (normal saline or Hartmann's) to be used and calculate how long this volume will last for:**

Volume of crystalloid to be used: 500 ml bag: 500 ml bag will last for 25 h  
(i.e. 500 ml divided by 20 ml/h)

**3. Calculate volume of analgesic agents required for this period of time, i.e. in this example 25 h:**

Morphine: 4 mg/h x 25 h = 100 mg = 10 ml (i.e. 100 mg divided by 10 mg/ml solution)

Lidocaine: 60 mg/h x 25 h = 1500 mg = 75 ml (i.e. 1500 mg divided by 20 mg/ml solution)

Ketamine: 10 mg/h x 25 h = 250 mg = 2.5 ml (i.e. 250 mg divided by 100 mg/ml solution)

**4. Remove a volume of crystalloid from the bag that is equal to the total volume of analgesic agents to be added, i.e. in this example 10 + 75 + 2.5 = 87.5 ml**

**5. Add analgesic agents to the crystalloid solution**

**6. Label the MLK solution for infusion clearly with drug concentrations, date, time, name of person preparing the solution, and name of person double checking preparation**

Note: it is extremely important to ensure that two individuals check all of the above calculations and all of the volumes of analgesic agents and crystalloid removed/added. This safeguard will hopefully prevent potentially serious errors from occurring.



comparing it to other strategies. Studies published so far have evaluated effects on reducing the MAC of inhalational anaesthetics which this combination appears – unsurprisingly! – to do; however this is not the same thing as analgesic efficacy or NMDA blockade.

## Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) in general reduce the severity of the peripheral inflammatory response and minimise peripheral sensitisation; they may also reduce central sensitisation in the spinal cord. NSAIDs may be useful in moderate to severe pain and are especially effective as synergistic analgesics with opioids in multimodal therapy.

The effects of most NSAIDs are related to the impairment of prostaglandin production; there may be other important mechanisms, some not fully understood. Prostaglandins are one of the chemical mediators that sensitise nociceptors both peripherally and centrally during ‘wind up’ and this anti-prostaglandin effect of NSAIDs is why they may reduce peripheral and central sensitisation. Peripheral inflammation induces central prostaglandin production hence NSAIDs may reduce central sensitisation even if the initial stimulus is peripheral.

### COX-1 vs. COX-2

Prostaglandin inhibition involves cyclooxygenase (COX) of which there are (at least) two types and the traditional dogma has been as follows:

COX-1 (constitutive, housekeeping)

- Variably found in all tissues
- Involved in the production of prostaglandins that are essential for a variety of normal functions

COX-2 (inducible)

- Levels typically very low but increase during tissue injury
- Primarily responsible for the production of prostaglandins that mediate the inflammatory response and pain associated with tissue injury

More recently it has been suggested that the traditional description above is too simplistic and that there is some overlap in the roles of COX-1 and COX-2. For example COX-2-mediated prostaglandins are thought to be vital for normal renal physiology and may also play a role in the healing of some tissues, especially the gastrointestinal tract. Some COX-1-mediated prostaglandins may be involved in the pain pathway.

Most NSAIDs are COX inhibitors. Some agents (e.g. carprofen, meloxicam) have more selective inhibition of COX-2 versus COX-1 (i.e. COX 1-sparing, ‘preferentially selective’) and are reportedly associated with fewer side effects and a safer therapeutic index than the older NSAIDs were. Even newer agents (e.g. firocoxib, cimicoxib, robenacoxib) are said to be COX-2 specific inhibitors (coxibs). However there is disagreement in the literature with respect to the selectivity of different NSAIDs for COX-1 versus COX-2.

For the reason described above, it is likely that the significance of COX-1 versus COX-2 specificity in terms of the safety of an individual NSAID varies between tissues/organs depending on what role this enzyme plays in the health and healing of that tissue or organ. For example the gastrointestinal tract is highly COX-1 dependent for health so agents with greater COX-2 selectivity may be associated with fewer GI side effects. COX-selectivity has no effect on analgesic efficacy.

In addition to their COX enzyme profile, another consideration in terms of the safety of NSAIDs is how exposed healthy non-target tissues/organs are to them. Clearly the less they are exposed, the less likely adverse effects are. In theory NSAIDs that spend less time in the circulation (have the shortest plasma half-life) and have the greatest tendency to be taken up into inflamed tissues (i.e. target sites) offer the greatest safety.

### Side effects/contraindications

There is a greater potential for toxicity with NSAID use in cats because their limited ability to glucuronidate exogenous drugs results in a prolonged duration of effect with the potential for drug accumulation.

Cats seem to be particularly susceptible to the adverse renal effects of NSAIDs.

As a general point, lower doses of NSAIDs often retain all or most of the analgesic potency and a cautious approach may be rational in any cases where there is a potential concern of adverse effects.

The two most commonly recognised side effects and/or contraindications of NSAIDs use are gastroduodenal injury and renal injury.

**Gastroduodenal injury**

Gastroduodenal injury may occur following NSAID administration due to direct irritation of the mucosa and/or prostaglandin inhibition. Prostaglandins are important for the integrity of the gastrointestinal mucosal barrier (mucosal cytoprotection) and their impaired production therefore increases the likelihood of mucosal injury (haemorrhage, erosions, ulceration) in an already compromised tissue. Intuitively therefore NSAIDs are contraindicated in animals with gastrointestinal abnormalities.

Most serious problems reported are from administration of higher than recommended doses; but toxicity also has been reported from relatively mild doses in susceptible individuals or for example where one NSAID has been discontinued and another started without allowing for a washout period (e.g. 1 week).

However, there are patients with gastrointestinal signs in which a NSAID may be justified – examples include a dog that is heavily dependent on NSAID therapy to manage discomfort associated with chronic osteoarthritis for which opioids are less effective; although it should be said that the increasing availability of tramadol may make this scenario even less likely nowadays.

Considerations in such cases include:

- Hypovolaemia or dehydration should be treated before NSAID therapy
- An agent reported to be COX-1 sparing should be chosen
- The drug should be given at a low dose and systemically if possible (mucosal lesions are more likely to occur with oral dosing because there is a higher local concentration of the agent at the site of the tablet or liquid)
- Oral administration should be accompanied by food
- Gastroprotectants (e.g. omeprazole, ranitidine) should be used pre-emptively

**Renal injury**

Prostaglandins play an important role in the kidneys in modulating the tone of blood vessels and regulating salt and water balance. The kidney depends on COX-1 and COX-2 for prostaglandin synthesis to autoregulate water metabolism, tubular function, and renal blood flow. Prostaglandins are not critically involved in renal

haemodynamics in normal healthy animals. However, in response to a decrease in renal blood flow, locally-acting prostaglandins serve to protect renal perfusion. Their inhibition by NSAID therapy can therefore result in renal ischaemia and insufficiency. This is applicable to both older NSAIDs and newer agents with more selective COX-2 inhibition as constitutive COX-2 is produced in the kidney. Emergency patients in whom renal blood flow may be decreased include those with hypovolaemia, cardiac insufficiency and pre-existing renal disease.

NSAID administration to patients in volume deplete states (e.g. hypovolaemic shock post-trauma) poses a real risk of nephrotoxicity, acute kidney injury and possibly failure.

**Other side-effects/considerations of NSAIDs**

Any NSAID has the potential to cause hepatic injury which may be intrinsic (predictable and dose-related; e.g. acetaminophen, aspirin) or idiosyncratic (unpredictable, non-dose related – this applies to most other NSAIDs). Administration of NSAIDs to animals that have hepatic disease has been questioned because of the role of the liver in metabolising these drugs, and they should probably be used cautiously if at all with active liver disease. There is no evidence that prior hepatic disease predisposes a patient to NSAID-induced liver injury. Drug enzyme systems are remarkably well preserved in hepatic disease and pre-existing hepatic disease is not necessarily a contraindication for NSAID administration.

NSAIDs may interfere with coagulation, and should not be administered concurrently with corticosteroids

**Hypovolaemia/dehydration**

The use of NSAIDs is contraindicated in patients with hypovolaemia, hypoperfusion from other causes, or dehydration.

A large proportion of painful emergency patients fall into this category and NSAIDs should not be used in the initial management of such cases. Depending on the primary disorder, it may be appropriate to administer a NSAID following correction of hypoperfusion and dehydration, for example 12-48 hours following initial presentation, as long as the patient is stable and there are no other contraindications.

*Important information that should guide treatment:*

- All NSAIDs, regardless of COX-1/COX-2 specificity, are capable of producing gastrointestinal lesions, particularly at high doses.
- All NSAIDs (selective or non-selective) can produce other gastrointestinal signs, including vomiting, diarrhoea, and decreased appetite, without producing ulceration.
- All NSAIDs have potential for producing hepatic injury. Susceptibility seems to be [largely] idiosyncratic and unpredictable.
- All NSAIDs have the potential for producing renal injury. Previous renal disease, salt depletion, [hypovolaemia] and dehydration increase the risk.
- No NSAID is consistently more clinically effective than another.

(Papich MG, 2008)

There are currently no studies that have shown one NSAID to be a better analgesic than another; and given they have similar adverse effect profiles, other considerations such as palatability, licensed routes and costs may affect which agent is chosen.

**Paracetamol (acetaminophen)**

Paracetamol (acetaminophen) is not commonly used therapeutically in veterinary medicine due to the widespread availability of licensed veterinary products that are both more effective and have been subjected to extensive clinical trials. However it may be an underused treatment option and is gaining more traction especially in referral centres both for treating fever and pain. Paracetamol is also widely available from numerous outlets and frequently present within the home of many pet carers. It is therefore common in emergency practice to either receive enquiries about administering paracetamol to dogs or to see animals to which this agent has been administered.

Paracetamol may be a useful analgesic in dogs but it should never be administered to cats at any dose due to the reduced capacity for glucuronidation in this species and therefore significantly increased risk of toxicity. Paracetamol is a NSAID and interferes with prostaglandin synthesis, but it is unclear whether COX inhibition is involved and/or the only mechanism of action. It is

analgesic but not anti-inflammatory at clinical doses; it also has antipyretic activity. However it is not associated with either renal or gastrointestinal injury at clinical doses. Paracetamol can therefore be used either in addition to conventional NSAIDs or alone in dogs in which these agents are contraindicated or poorly tolerated. As paracetamol interferes with prostaglandin production, it should be avoided in hypoperfused or dehydrated patients. Hepatotoxicity is a potential side effect of this drug but it has a high safety index in dogs.

The recommended dose range in dogs is 10-15 mg/kg PO q 8-12 hours.

A combined preparation of paracetamol and codeine is commercially available for dogs but the author has no experience with its use.

**Local anaesthetic agents**

Local anaesthetic agents work by blocking sensory input via afferent nerve fibres to the central nervous system and prevent central sensitisation.

Longer-acting agents in particular (e.g. bupivacaine, ropivacaine) are used extensively in referral centres in a number of ways including:

- Epidural administration (often combined with morphine)
- Intrapleural administration via thoracostomy tubes following thoracotomy
- Local and regional nerve blocks
- Constant rate infusion of 2% lidocaine solution without adrenaline for analgesic purposes in dogs:
  - o There are some studies showing efficacy of this route in dogs (and humans)
  - o Traditionally it has been said that lidocaine infusion may result in significant cardiovascular depression in cats and is not recommended unless essential for controlling ventricular dysrhythmia. The evidence base for this is minimal however and to the author's knowledge is all based on the haemodynamic effects of lidocaine infusion in healthy cats under general anaesthesia. Some clinicians have used lidocaine infusion for analgesia purposes in cats for some time without noting more adverse effects than in dogs. Nevertheless until and unless the recommendation not to use lidocaine infusions in cats for analgesia changes generally, it is probably best to avoid this practice.

- o The analgesic mechanism of action when administered intravenously is not known but may involve both peripheral and central sites of action; systemic lidocaine may block propagation of ectopic discharge from the site of neuronal injury as well as within the dorsal root ganglion.

While these uses may not be applicable to most non-referral emergency patients, local anaesthetic agents can be employed to good effect in this setting. Local and regional anaesthesia is underused probably due to a combination of lack of training for small animal (versus equine) vets but also because it is overlooked because many of our patients undergoing procedures receive full general anaesthesia. However physiological principles mean that there is still a benefit from using local anaesthesia regardless of general anaesthesia as part of a multimodal approach to block the nociceptive impulses close to their source. Furthermore this may have a dose-sparing effect on the general anaesthetic agents.

“Regional anaesthesia always works – provided you put the right dose of the right drug in the right place” (Denny and Harrop-Griffiths, 2005).

## Topical analgesia

Lidocaine has a shorter duration of action (1-2 hours) than other agents but a rapid onset of action (can be as quick as 3-5 minutes). It is the most widely available local anaesthetic agent and may be used for emergency cases in a number of ways. Examples include:

### 1. Topical analgesia for wounds

While an emergency patient is undergoing initial stabilisation, sterile gauze swabs can be soaked in a solution of 2% lidocaine mixed with normal saline (0.9% sodium chloride) and applied to wounds which are then wrapped in cling film pending management at the appropriate time. Lidocaine can also be mixed with the gel that is applied to the wound prior to clipping. The maximum recommended dose is 12 mg/kg in dogs [6 mg/kg in cats]. A 5% lidocaine patch (Lidoderm®, Endo Pharmaceuticals) is available and has been used for dogs (and cats) with severe skin abrasions, severe bruising and surgical wounds. However the author has no experience with its use. In human medicine, there is also experience with the use of lidocaine putty.

### 2. Lidocaine-prilocaine cream

A 2.5% lidocaine, 2.5% prilocaine cream (EMLA® cream 5%, AstraZeneca) can be applied to the skin prior to intravenous catheter placement or venepuncture. Depending on the site, a light occlusive dressing may be appropriate while anaesthesia takes effect. EMLA® cream is especially useful for example in puppies (and kittens, rabbits etc.) and in stressed animals. It can also be applied to wound edges to reduce irritation and thereby patient interference. Clinical experience suggests that onset of action in some cases is significantly shorter than the commonly reported 30-60 minutes. It may be that unless you wait for at least 30 minutes, the desensitisation achieved will not be as full as possible. However clinical experience does suggest that the effect is sufficient in less than 30 minutes (even if this is just due to a soothing placebo effect of the cream preparation!). Minimal if any systemic drug absorption has been reported following topical application.

### 3. Lidocaine spray

In addition to facilitating endotracheal intubation of cats, proprietary lidocaine spray (e.g. Intubeaze®, Dechra Veterinary Products) may have diverse uses for mucosal desensitisation, for example to facilitate nasal catheter placement or to examine the oropharyngeal region. Some people have also used Intubeaze® on clipped skin prior to venepuncture/catheterisation in lieu of EMLA® cream, and also for minor stitch-ups/stapling procedures, although there is debate about how well and quickly this preparation would be absorbed through the skin to achieve nerve anaesthesia (it lacks the ‘vehicle’ in the EMLA® cream base that is meant to allow effective absorption across the skin).

### 4. Lidocaine lubricant

A 2% lidocaine lubricant is commercially available; otherwise it can be constituted by mixing 2% lidocaine solution (without adrenaline) for injection with a sterile lubricant. This is then used topically, for example prior to insertion of nasal catheters or urinary catheterisation.

Solutions of other local anaesthetics (e.g. tetracaine hydrochloride, proxymetacaine) have also been used topically to facilitate conscious small stitch-ups or ‘staple-ups’ although the same comments about how effectively they are absorbed across the skin apply to those under ‘Lidocaine spray’ above.



## Ketamine

There are said to be four stages of ketamine brain continuum – analgesia, sedation, partial dissociation, dissociation; they have overlapping dose ranges that can vary between patients. Ketamine has been used mostly as a dissociative anaesthetic agent or sedative but it also has a role in pain relief. This is predominantly an anti-hyperalgesia and anti-allodynic preventative analgesic effect by blocking (and potentially reversing) central sensitisation via its action as an N-methyl-D-aspartate (NMDA) receptor antagonist; the NMDA receptor is a key receptor in the dorsal horn of the spinal cord. It is especially effective in this respect when combined with a full opioid agonist and may be used as a constant rate infusion.

The recommended dose for analgesic purposes is 0.1-1 mg/kg IV as needed (often every 30 minutes) or a single loading dose followed by a constant rate infusion of 0.1-1 mg/kg/hour. Following a single bolus, ketamine's effect on blocking central sensitisation likely persists for much longer than the direct analgesic effect. Ketamine is sympathomimetic and may therefore cause increases in heart rate and blood pressure; it is relatively contraindicated therefore for example in cats with hypertrophic cardiomyopathy. Ketamine was previously said to be contraindicated in raised intracranial pressure (e.g. after head trauma) but this is no longer considered the case.

It should NOT be used alone, especially in dogs, as severe hyperexcitability/mania/other horrible behavioural effects may ensue!

## Medetomidine

Medetomidine is an alpha2-adrenergic agonist that is used most commonly for its sedative properties. This agent does however also possess potent analgesic properties, acting at similar sites in the central nervous system as opioids as well as at primary afferent fibres, and can be a useful adjunctive analgesic in some cases.

At the microdoses recommended here, cardiovascular (marked peripheral vasoconstriction, vagal baroreceptors-mediated bradycardia, decreased cardiac output) and respiratory (depression) effects seen should be minimal and can be reversed by atipamezole if necessary. The sedative effects are likely to be advantageous for patient

management in the types of cases in which this agent may be employed as an analgesic.

Recommended doses for medetomidine when used for analgesic purposes are 1-5 µg/kg slow IV or IM as needed (often q 30-90 minutes but potentially much longer if an opioid is used concurrently); alternatively a constant rate infusion of 1-3 µg/kg/hour may be used and is likely more effective; an initial loading dose of 1-5 µg/kg is usually given. Note: The above comments also apply to dexmedetomidine although lower doses are typically used. Alpha-2-adrenergic agonists can be given via the oral transmucosal/buccal route in fractious cats.

## Gabapentin/Amantadine

**Gabapentin** is an analogue of the neurotransmitter GABA. It originated as an anti-convulsant drug in people; its successor is pregabalin. It has been used widely for analgesic purposes in small animals (e.g. chronic neuropathic pain, chronic cancer pain, peri-operative pain) but the mechanism(s) of action remain unclear; it may work (in part) by increasing synaptic levels of GABA in the CNS. To the author's knowledge there are no published clinical trials in dogs or cats.

Dosing recommendations were extrapolated from humans but significant species differences are likely to exist in pharmacokinetics; clinical experience suggests to start with 3-10 mg/kg PO q 8-12 h, then adjusting to effect without causing sedation or ataxia.

**Amantadine** is an agent that is starting to gain some traction although as with gabapentin not really in the emergency setting. It has antiviral properties but as an NMDA receptor-antagonist it is also being used in small animals for analgesia. This is typically in the adjunctive treatment of chronic pain in combination with one or more other agents such as an NSAID or tramadol. It is also used as an adjunct drug for treating neuropathic pain. Oral and intravenous formulations exist although the author is not sure how readily the injectable preparation is available.

## Sedation

Although sedation is not a substitute for pain relief, it can be invaluable in improving the welfare of animals that are especially anxious or distressed, allowing them to rest

and to utilise their metabolic reserves for recovery rather than coping behaviour. In cases in which opioids used for analgesia do not provide sufficient sedation, other agents such as acepromazine or microdoses of medetomidine are options.

## Non-Pharmacological Measures

Although analgesic agents are the mainstay of pain management, it is essential not to overlook the important contribution of good nursing care and other non-pharmacological measures. These measures reduce stress, improve patient wellbeing, and contribute significantly to pain management. Some of these measures are listed below:

- TLC, gentle handling (independent of interventions) and regular grooming
- Providing a suitable environment (some animals prefer a dimly lit quiet environment, others like to be distracted)
- Tempting to eat if appropriate
- Ensuring bedding is dry, clean and comfortable
- Providing a familiar blanket or toy from home
- Allowing or preventing visits by the owner depending on patient response
- Providing cats with a box or carrier to sleep in
- Helping dogs out to urinate/defaecate
- Bladder management as necessary
- Co-ordinating interventions (e.g. venepuncture) to minimise the number of painful procedures performed
- Grouping treatments to allow undisturbed rest time
- Removing unnecessary catheters, tubing and bandages as soon as possible
- Using warm compresses or cold packs if appropriate
- Outside for fresh air and hopefully some sunshine!

External stabilisation of orthopaedic injuries may offer considerable pain relief and reduce the likelihood of further patient-induced damage.

Cats are more sensitive and more susceptible to stress than dogs, and the importance of non-pharmacological measures cannot be overstated in this species.

## Pain perception

More recently people are starting to consider if and how a patient's perception of pain may be affected by mood, stress or anxiety, i.e. as well as being better for patient welfare per se, improving mood may reduce perception of any pain experienced. This raises the possibility that there is a rational for considering mood enhancing preparations such as Zylkene®, pheromones etc. to reduce pain perception.

## References

- Giordano T, Steagall PV, Ferreira TH, et al. Postoperative analgesic effects of intravenous, intramuscular, subcutaneous or oral transmucosal buprenorphine administered to cats undergoing ovariohysterectomy. *Vet Anaesth Analg* 2010. 37(4):357-366.
- Papich MG. An Update on Nonsteroidal Anti-Inflammatory Drugs (NSAIDs) in Small Animals. *Vet Clin Small Anim* 2008. 38:1243-1266
- Denny NM, Harrop-Griffiths W. Location, location, location! Ultrasound imaging in regional anaesthesia (Editorial). *Br J Anaesth* (2005) 94 (1): 1-3. doi: 10.1093/bja/aei001



## Commissioned paper\*

# Fluid therapy in dogs and cats

Leona Raušerová-Lexmaulová<sup>1</sup>

## SUMMARY

Critically ill dogs and cats are commonly hypovolaemic and/or dehydrated and fluid therapy is necessary for the correction of these abnormalities. The fluid deficit in the intravascular and/or extravascular space is estimated based on clinical examination. Many types of fluids exist. The results of blood gas and acid-base analysis, and serum biochemistry are essential in selecting the best solution for the correction of the presenting abnormalities in the body. However, should blood gases/acid base analysis not be available, biochemical profile findings can be a good alternative for assessing the acid-base status of the patient. Following selection of the appropriate fluid, careful monitoring is important for optimizing on-going fluid requirements and recovery of critically ill patients.

\*This paper was commissioned by FECAVA for *Eur J Comp An Pract (Autumn 2016) 26(3)*; p19-p26. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

## Introduction

The adult body is composed of approximately 60% water, 80% in neonates and weaners. The blood volume of dogs is approximately 80 ml/kg and for cats 65 ml/kg. The water component creates the solution in which electrolytes and all metabolic substrates are dissolved and is also an important transport medium. The water easily passes through membranes by diffusion and osmosis and its movement depends on osmotic and oncotic pressures. The water distribution is dependent on sodium, which, along with chloride and bicarbonate, belongs to the main extracellular electrolytes. The basic intracellular electrolytes are potassium, magnesium and organic phosphates (Wellman et al 2006).

The maintenance of fluid, electrolyte and acid-base balance in the body is a complicated process, where the kidneys play the most important role (DiBartola 2006).

Indications for fluid therapy include: hypovolaemia, shock, dehydration (e.g. due to diarrhoea, vomiting), inability to intake water and food (e.g. vomiting, anorexia, unconsciousness, serious orofacial trauma...), the correction of acid-base and electrolyte abnormalities (Mensack 2008).

The main goal of fluid therapy is the correction of water, electrolyte and acid-base abnormalities in ill dogs and cats (Mathews 2006).

Fluids can be administered via several routes. Intravenous administration is the most common and effective way to provide fluid therapy in all hospitalized patients and is essential in seriously ill dogs and cats. However, when intravenous catheterization is impossible (e.g. in puppies and kittens), intraosseous access is recommended (Mathews 2006, Boag and Hughes 2007).

The subcutaneous route of fluid administration is effective only in normovolaemic and stable patients with maximum volume restricted to 20 ml/kg to one site and total volume dependent upon patient comfort. Only Lactated Ringer's or normal saline should be administered subcutaneously, acetate and dextrose products are painful (Mathews 2006).

<sup>1</sup> Department of Surgery and Orthopaedics, Small Animal Clinic, University of Veterinary and Pharmaceutical Sciences Brno, Czech Republic  
E-mail: [rauseroval@vfu.cz](mailto:rauseroval@vfu.cz)

## Fluid selection

Many types of infusion solutions exist. Three main categories of fluids are described: crystalloids, colloids, and additional fluids.

Crystalloids are a mixture of water and electrolytes and are categorized as hypertonic, isotonic and hypotonic based on osmolarity (number of solute particles per litre).

Isotonic solutions have an osmolarity similar to blood plasma (e.g. normal saline, Lactated Ringer's, Plasmalyte). These solutions are the basic fluids for treatment of dogs and cats because they quickly cross the extravascular space when treating dehydration, and restoring intravascular volume and homeostasis (Griffel and Kaufman 1992, Boag and Huges 2007).

Isotonic crystalloids are also categorized based on their acidifying or alkalizing influence on acid-base balance. The acidifying solutions may contain only sodium and chloride (normal saline 0.9%) or the main electrolytes (sodium, chloride, potassium, calcium, magnesium). The administration of large volumes of acidifying solutions can result in metabolic acidosis. On the other hand, these solutions are the fluid of choice in patients with metabolic alkalosis (e.g. in cases of acute vomiting) (Mathews 1998).

Alkalizing solutions are composed of basic electrolytes in a similar concentration to that of plasma and a buffer (e.g. lactate, acetate, gluconate or malate). Based on this composition, these solutions are also referred to as buffered balanced solutions. The administration of large volumes of these solutions rarely negatively impacts on the acid-base balance unless the patient is alkalaemic. Due to a similar electrolyte composition to plasma, and the positive effect on acid-base balance in acidaemic patients, which, to some degree are the majority of all ill patients, these are used as basic fluids for emergency and intensive care (ICU) patients (Mathews 1998).

The hypertonic solutions have a higher osmolarity than plasma and their administration leads to a water shift from the extravascular to the intravascular space, but their effect is very short lived, 15–20 minutes (Rudloff and Kirby 1998). Due to this effect they are used for rapidly increasing circulating volume, or removing oedema from the brain when an increase in intracranial pressure is present.

The hypotonic solutions have a lower osmolarity than plasma and deliver more water than electrolytes to the patient. These solutions can be prepared in a mixture of isotonic crystalloid with an equal, or smaller, volume of sterile water for injection or 5% dextrose solution. These solutions are usually used for supplementation of basal fluid requirements (Mensack 2008).

The colloids are solutions of high molecular weight and persist in the intravascular space for a longer period of time compared to crystalloids. Colloids increase oncotic pressure, which results in water shift from the extravascular space, and its retention in plasma (Rudloff and Kirby 1998). Colloids are very effective in increasing circulatory volume, but the risk of adverse effects with overdose is higher than with crystalloids.

The colloids are divided into natural (blood, blood products, human serum albumin) and synthetics (e.g. hydroxyethyl starch, dextrans and stroma-free haemoglobin-based oxygen-carrying solutions). Hydroxyethyl starch is the most commonly used synthetic colloid. Its effect depends on molecular weight and place of molecular substitution. The recommended daily dose is 20–30 ml/kg (Rudloff and Kirby 1998, Silverstein et al 2005, Sigrist 2011). The negative effects of colloids are precipitating or worsening, hypo-coagulopathy and a risk of kidney damage. Present recommendation are not to use them in animals with renal or hepatic diseases (Falco et al 2012, Adamik et al 2015), hypocoagulopathy and sepsis.

Blood and blood products play an important role in therapy for dogs and cats with severe anaemia, thrombocytopenia, coagulation disorders and hypoproteinaemia (Mathews 2006).

## Phases of fluid therapy

1. Correction of hypovolaemia
2. Correction of dehydration
3. Maintenance phase

### 1. Correction of hypovolaemia

Hypovolaemia refers to a fluid deficit within the intravascular space. Loss may be the water component or haemorrhage, ranging from mild to severe, with correction based on patient assessment. Where hypotension and shock is identified, rapid resuscitation is required with a reduction in fluid therapy once normovolaemia and



normotension are reached. A maintenance and rehydration phase follows. However, where haemorrhage is not controlled permissive hypotension, SBP 100mmHg, MAP 60mmHg is maintained until haemorrhage is controlled. The fluids are administered intravenously or via the intraosseous route when IV access cannot be obtained such as in some kittens and puppies (Mathews 2006, Silverstein 2014).

Based on the patient's problem and co-morbidities, there are a few approaches to increasing circulatory volume in hypovolaemic, non-bleeding patients. The approach to bleeding patients is quite different (see below).

## No haemorrhage

### ***Isotonic crystalloids, buffered balanced solutions–***

Lactated Ringers, Plasmalyte or Ringerfundin are usually preferred, calculated as 15 minute boluses of an approximate hourly dose of 40–90 ml/kg/h for dogs and 20–60 ml/kg/h for cats. This volume is based on the clinical problem, patient assessment, and response to therapy. No definitive recommendation with respect to volume can be given but usually a ¼ of an hourly dose is given over 15 minutes) and we repeat this boluses until stabilization of circulation (restoration of normal blood pressure, capillary refill time and heart rate). Monitoring response every 5 minutes is essential (Driessen and Brainard 2006, Sigrist 2011).

***A combination of synthetic colloid*** (e.g. Voluven® Tetraspan®, Volulyte®, Vetastarch®) and buffered balanced isotonic crystalloids are preferred in patients with moderate or severe shock. A recommended volume is 1-5 ml/kg/5 minutes in dogs and 1 ml/kg/5 minutes in cats initially to improve circulation, then continue with isotonic crystalloids at 10–20 ml/kg/ 15 minutes, with assessment every 5 minutes to complete stabilization of the circulation (Driessen and Brainard 2006, Sigrist 2011, Silverstein et al 2005, Silverstein et al 2012).

***A combination of isotonic crystalloids (buffered balanced solutions) and hypertonic saline (HS)***, 4-7% sodium chloride solution, 4–5 ml/kg over 15 minutes. A recent study identified no difference between 3.5-5% HS and 7%; however, there were more adverse effects with 7% (Bulger et al 2008, Forsyth et al 2012, Wurlod et al 2015). The HS cannot be administered to dehydrated patients because administration leads to hyperosmolarity of blood

plasma. The infusion of isotonic crystalloids has to follow hypertonic saline administration to restore homeostasis. Hypertonic saline at a dose 1–2 ml/kg over 10 minutes is a good choice in cases of intracranial trauma with signs of high intracranial pressure (neurological signs, bradycardia and normal or increased blood pressure) (Sande and West 2010).

The goal of therapy in non-bleeding, hypovolaemic patients is to achieve optimal physiological circulatory parameters (Table 1); however, excessive volumes of crystalloids, and HES, can result in adverse effects (Palmer and Martin 2014).

*Table 1: The goal of hypovolaemia resuscitation in dogs and cats.*

Parameter	Dogs	Cats
Heart rate	80–120 beats/min	160–200 beats/min
Temperature	38–39.5°C	38–39.5°C
Mucous membranes	Pink	Pink
CRT	1-2s	1-2s
Blood pressure	100–120 mmHg	100–120 mmHg
PCV	>25%	>25%
Total solids	>45 g/l	>45 g/l
Urine output	1-2 ml/kg/h	1-2 ml/kg/h

## Bleeding patients

***Blood or blood products*** are essential in the event of severe-life-threatening blood loss. Caution with fluid and/or synthetic colloid administration is essential in patients with uncontrolled bleeding, where the rapid increase in blood pressure can disrupt a delicate, formed clot and contribute to worsening haemorrhage and patient decompensation. Also, crystalloids and synthetic colloids will contribute to coagulopathy. A small volume of buffered balanced isotonic crystalloid (1–2 ml/kg/h) is recommended in mild or moderate haemorrhage (blood loss 10–25% of blood volume) (Hammond and Holt 2009). Until haemorrhage is controlled, the patient should be reassessed every 15 minutes to address further losses and an increase in fluid requirements. Blood donation should not exceed 25% of the donor's blood volume.

In severe haemorrhage (blood loss 25-35%) buffered balanced isotonic crystalloids in boluses of 5-10 ml/kg/15 minutes are recommended, synthetic colloids 3-5

ml/kg/15 minutes may be of value. However, for patients with an estimated blood loss >25%, blood must also be administered to achieve systolic blood pressure 90 mmHg, MAP 60mmHg (low volume hypotensive resuscitation) until haemorrhage is controlled (Adamantos and Hughes 2008, Hammond and Holt 2009).

Patients with life-threatening haemorrhage (blood loss 35–50%) may receive 5% HS (dogs 6–10 ml/kg and cats 1.5 – 2.5 ml/kg), or a synthetic colloid, combined with a crystalloid initially, while preparing a blood transfusion or/and haemoglobin-based oxygen carrying solutions (HBOCS) combined with crystalloids. The total dose of HBOCS should not exceed 30 ml/kg in dogs and 14 ml/kg in cats. With this degree of blood loss, administration of blood or haemoglobin-containing solutions is necessary (Mathews 2006). The goal of therapy is a systolic blood pressure of 90 mmHg and MAP 60mmHg until bleeding is controlled (Hammond and Holt 2009).

## 2. Correction of dehydration

Dehydration is a fluid deficit in the extravascular space and should be corrected slowly, over 12–24 hours, with an isotonic crystalloid solution.

The fluid deficit is estimated based on clinical examination (Table 2), then calculated using the formula in Fig. 1. In addition, the on-going maintenance fluid requirement, due to fluids lost in the urine and faeces (sensible losses) and during respiration and evaporation (insensible losses), are added to the deficit volume. These losses are normally supplemented by food and water intake, but ill animals are usually anorexic, or unable to eat and drink to meet fluid requirements. The basal fluid requirement for 24 hours is calculated from formula (30 x BW (Kg) + 70) (Boag and Hughes 2007). (Table 3)

Table 2: Estimation of dehydration

Dehydration (%)	Clinical signs
< 5 %	Without abnormalities
5–6 %	Sticky mucous membranes
6–8 %	Dry mucous membranes, elongated CRT, ↓ skin elasticity
10–12 %	Dry mucous membranes, significantly longer CRT, ↓ skin elasticity (skin tenting), ↑ heart rate, weak pulse, dull eyes
12–15 %	Shock

Figure 1: Formula for calculation of fluid deficit (Rudloff and Kirby 1998)

$$\text{Body weight (kg)} \times \text{dehydration (\%)} \times 10 = \text{mL}$$

Table 3: Basal fluid requirements

BW (kg)	Total Water (ml/day)	ml/kg/day	ml/h
1	100	100	4
2	130	65	5
3	160	53	7
4	190	48	8
5	220	44	9
6	250	42	10
7	280	40	12
8	310	39	13
9	340	38	14
10	370	37	15
11	400	37	17
12	430	36	18
13	460	35	19
14	490	35	18
15	520	35	22
16	550	34	23
17	580	34	24
18	610	34	25
19	640	34	27
20	670	34	28
25	820	33	34
30	970	32	40
35	1120	32	47
40	1270	32	53
45	1420	32	59
50	1570	31	65
60	1870	31	78
70	2170	31	90
80	2470	31	103
90	2770	31	115

The estimated fluid volume to correct the deficit is divided by 12 or 24 based on the hourly rate selected. The hourly maintenance is added to the replacement volume. Balanced crystalloid solutions are usually a better choice than normal saline. The fluid selected, acidifying or alkalinizing solutions, depends on the acid-base status of the patient (Davis et al 2013). Approximately 95% of the time patients are normal or acidaemic and the alkalinizing solution is prescribed for these patients. The remaining 5% that are alkalaemic should receive 0.9% saline. In addition, electrolytes such as potassium or magnesium may be required.

Occasionally, a hypotonic solution for basal fluid requirements will have to be prepared for some critical patients (e.g. puppies with parvovirus). These special solutions are prepared by mixing an equal volume of 5 % dextrose with a balanced alkalinizing solution and added potassium 20–25 mmol/l of the final solution, unless contraindicated (Mathews 2006).

### Example case

#### Replacement Phase:

Dog: 10 kg, estimated to be 8% dehydrated.

Calculation of fluid deficit:

$$\text{Body weight (kg)} \times \text{dehydration (\%)} \times 10 \text{ (ml)}$$

For our 10 kg dog that is 8% dehydrated:

$$10 \times 8 \times 10 = 800 \text{ ml}$$

Calculation of basal fluid requirement per 24 hours  $[30 \times \text{BW (kg)} + 70]$ , in this case:

$$[30 \times 10(\text{kg}) + 70] = 370 \text{ ml}$$

Total fluid requirement: deficit correction + basal fluid requirements:

$$800 + 370 = 1170 \text{ ml}$$

Calculation of hourly dose:

$$1170 : 24 = 48.75 \text{ ml}$$

### 3. Maintenance phase

During this phase, both the intravascular and extravascular deficits are corrected, but there is insensible loss and potentially other on-going losses (vomiting, diarrhoea, polyuria ...). The formula for basal fluid requirement (insensible losses) is used plus on-going losses. The on-

going losses are estimated during a particular time period (e.g. during a 4 hour period). This volume is calculated for 24 hours and added to basal fluid requirements.

During this phase monitoring of on-going losses is important as a decrease in the added fluid volume for on-going losses should be reduced as the patient improves and begins to drink and eat. The hourly flow rate should be reduced slowly based on the patient's fluid losses and volume of water and food intake (Mathews 2006).

### Correction of the most common electrolyte disorders and acid-base disorders

The aim of fluid therapy is not only the correction of fluid deficit, but also correction of electrolyte and acid-base disorders (hypokalaemia, hyperkalaemia, hyponatraemia, hypernatraemia, hypomagnesaemia, hypocalcaemia, hypercalcaemia, hypochloraemia, metabolic acidosis or alkalosis) (Davis et al 2013).

#### Potassium

Potassium is the most commonly affected electrolyte. The physiological range is 3.5–5 mmol/l for dogs and cats. Hypokalaemia is more common in dogs and cats than hyperkalaemia. Hypokalaemia usually results from anorexia and excessive potassium losses in urine, vomit or diarrhoea.

The approaches for correction depend on the severity of potassium loss and associated clinical signs:

1. For patients with **serum potassium below 3 mmol/l**, calculate the deficit using the following calculation:

$$\text{Potassium deficit (mmol/l)} = (4 - \text{patient potassium blood level}) \times \text{body weight} \times 0.6$$

The calculated volume of potassium is given by infusion as quickly as possible, but at a maximum flow rate of 0.5 mmol/kg/h.

2. Continuous potassium supplementation, as an addition to the fluid infusion, based on the serum level (Table 4) can be used for patients with **mild hypokalaemia (potassium level 3–3.5 mmol/l)**. This continual supplementation can also be used after rapid hypokalaemia correction (based on the above formula) in anorexic

patients with on-going losses to maintain appropriate plasma potassium level

Table 4. Potassium supplementation

Potassium level in plasma/serum (mmol/L)	mmol/kg/h
Normal (3.5–5)	0.05–0.1
Mild hypokalaemia (3–3.5)	0.15–0.2

## Caution

Patients with kidney disease or urinary tract obstruction (rupture) may be hyperkalaemic but if not, have reduced ability to excrete potassium and will become hyperkalaemic with continuous supplementation. Serum potassium should be reassessed.

Once potassium is added to the fluid bag, and set at the maintenance rate, any increase in rate as a flush for IV medications, or in an emergency, will lead to potassium overdose with risk of cardiac arrhythmias developing and potential death.

The calculation of the amount of potassium in all fluids in relation to its flow rate is important especially during quick potassium correction, to avoid potassium overdose and cardiac arrhythmias.

Correction of other electrolyte and acid-base abnormalities  
Correction of other electrolyte abnormalities and acid-base deficits is usually achieved using solutions according to their composition (e.g. in the case of hypochloraemia, a solution with higher chloride concentration, such as Ringer or Ringerfundin is selected), see Table 4.

For metabolic alkalosis, normal saline or Ringer's solution can be used. Ringer's solution is the better choice because in addition to sodium it also contains potassium and calcium (Table 4).

Patients with metabolic acidosis or mixed metabolic disorders usually require a balanced alkalinizing solution such as Lactated Ringer's, Plasmalyte or Ringerfundin (Table 5) which, in addition to correcting electrolyte deficits, also contributes buffers which help to correct metabolic abnormalities.

## Monitoring fluid therapy

Careful monitoring of the patient is essential to ensure the appropriate fluid volume is administered and fluid overload is avoided.

The heart rate, mucous membrane colour, capillary refill time (CRT) and blood pressure should be monitored every 5–15 minutes, based on severity, during fluid resuscitation and after stabilization of circulation (Table 1). Mental status should also be assessed as moderate to severely hypovolaemic patients are depressed due to reduced cerebral blood flow. The mental status should improve with resolution of hypovolaemia.

Restoration of normal urine production (~1ml/kg/h) based on lean body weight and normalization of urine specific gravity are also very important parameters for assessment of circulation (Mathews 2006).

Table 5: Composition of fluids used for fluid therapy compared to plasma in dogs and cats.

Fluid/ composition	pH	Na <sup>+</sup> (mmol/L)	K <sup>+</sup> (mmol/L)	Cl <sup>-</sup> (mmol/L)	Ca <sup>2+</sup> (mmol/L)	Mg <sup>2+</sup> (mmol/L)	lactate (mmol/L)	malate (mmol/L)	acetate (mmol/L)	gluco- nate (mmol/L)
Blood plasma Cats	7.28–7.4	145–157	3.5–5.2	95–120	1.8–2.8	0.8–0.9	2.5			
Blood plasma Dogs	7.30–7.42	135–155	3.5–5	95–125	2.2–3	0.8–1.2	2.5			
Normal saline	5	154	0	154	0	0	0	0	0	0
Ringer	6	147	4	155.5	2.3	0	0	0	0	0
Lactate Ringer's	6.5	130	5.4	109	3	0	28	0	0	0
Ringerfundin	5.1–5.9	145	4	127	2.5	1	0	5	24	0
Plasma-Lyte A	7.4	140	5	98	0	1.5	0	0	27	23



Lactate level can be a marker of anaerobic metabolism and changes during fluid resuscitation can be used for assessment of perfusion, or a possible worsening of a patient's condition with on-going monitoring (Allen and Holm 2008).

During the second and third phase of fluid therapy, assessment of the patient's heart rate, mucous membranes, CRT, skin elasticity and blood pressure should be carried out every 4 hours with a recalculation of the fluid plan where appropriate. The change in body weight during an 8-12 hour period is due to either excessive fluid losses or excessive fluid administration and retention. The urine production, assessed on ideal body weight, associated with urine specific gravity, and patient hydration status are also so important factors to include when calculating on-going hourly fluid rate (Mathews 2006).

Repeated serum biochemistry and haematology are advised to identify, and monitor on-going correction of acid-base and electrolyte abnormalities in patients, especially when adding electrolytes to the fluids.

## Paediatric patients

Paediatric patients (puppies, kittens under 12 weeks of age) must be considered separately from adults due to their many anatomical and physiological differences. The most important difference regarding fluid therapy is the high water content of their bodies, 80% in neonates and 75% in paediatrics (with 40% in the extravascular space), large surface area, and immaturity of function of many organs (heart, kidneys, liver) (Mathews 2006). Dehydration is difficult to detect by skin turgor over the dorsum in patients younger than 6 weeks of age. However, with advanced dehydration, the skin will lose turgor and remain tented or spontaneously wrinkle; this is best examined on the ventral abdomen as there is less subcutaneous fat here. Dry and darker oral mucous membranes indicate dehydration. Urine colour is a good indicator of hydration as it is normally pale in neonates; even slight colouring is suggestive of dehydration in dogs less than 4 weeks; however, there may be a slight yellow colour due to bilirubin metabolism in kittens. Bilirubin metabolism occurs at ~3-4 weeks in puppies. More accurate assessment should be made using urine specific gravity. Urine specific gravity > 1.020, also indicates dehydration as does constipation. In extreme circumstances, fluid

volume status can also be estimated by changes in body weight and, where there are concerns for over-hydration, changes in lung opacity on X-rays. However, auscultation should be used to ensure this does not occur. Lack or reduction of pulmonary vascular image is an indication of hypovolaemia. Neonatal and infant puppies and kittens rely on heart rate to maintain stroke volume and have a dominant parasympathomimetic and reduced sympathetic drive; therefore, they cannot increase their heart rate very much to maintain stroke volume during hypovolaemia. They are also poorly responsive to parasympatholytic drugs like atropine and glycopyrrolate. Due to these unique physiological aspects of the very young, paediatric patients are very sensitive to hypovolaemia and fluid overload and also, due to a low energy pool, are predisposed to hypoglycaemia.

The optimal fluid is Lactated Ringer's at 120–220 ml/kg/day with the addition of dextrose to make a 5% dextrose solution (Mathews 2006). All solutions must be warmed (McMichael 2005). This must not be given subcutaneously (SC), therefore an IV or IO catheter must be placed or, Lactated Ringer's given SC and glucose administered per os. However, this volume is too large for SC administration. Where vomiting is not a concern, this may also be administered by careful gavage.

## Conclusion

Fluid therapy is an important aspect of managing critically ill patients. The fluid plan has to be designed for the individual patient's needs initially and flexible according to the patient's on-going requirements and response (clinical status and laboratory results) to optimize patient recovery.

## Acknowledgement

The author thanks Dr. Karol A. Mathews, DVSc, DACVECC for her help and support.

## References

1. Adamantos S, Hughes D (2008): Fluid therapy in patients with pulmonary disease. *Vet Clin North Am Small Anim Pract* 38(3):719-25.
2. Adamik KN, Yozova ID, Regenscheit N (2015): Controversies in the using of hydroxyethyl starch solutions in small animal emergency and critical care. *J Vet Emerg Crit Care* 25(1):20-47.

3. Allen SE, Holm JE (2008): Lactate: physiology and clinical utility. *J Vet Emerg Crit Care* 18(2):123-132.
4. Boag A, Hughes D: Fluid Therapy. In: King LG, Boag A: BSAVA Manual of Canine and Feline Emergency and Critical Care. Second edition. 2007, BSAVA Quedgeley, pp: 30-45.
5. Bulger EM, Jurkovich GJ, Nathens AB, Copass MK, Hanson S, Cooper C, Liu PY, Neff M, Awan AB, Warner K, Maier RV (2008): Hypertonic resuscitation of hypovolemic shock after blunt trauma: a randomized controlled trial. *Arch Surg*, 143(2): 139-148.
6. Davis H, Jensen T, Johnson A, Knowles P, Meyer R, Rucinsky R, Shafford H (2013): Fluid Therapy Guidelines for Dogs and Cats. *J Am Anim Hosp Association*. 49:149-159.
7. DiBartola SP (2006): Applied renal physiology. In: DiBartola SP, editor: Fluid, electrolyte and acid-base disorders in small animal practice. 3rd edition. St. Louis (MO): Saunders Elsevier 2006:26-44.
8. Driessen B, Brainard B (2006): Fluid therapy for the traumatized patient. *J Vet Emerg Crit Care* 16(4):276-299.
9. Falco S, Bruno B, Maurella C, Bellino C, D'Angelo A, Gianella P, Tarducci A, Zanatta R, Borrelli A (2012): In vitro evaluation of canine hemostasis following dilution with hydroxyethyl starch (130/0.4) via tromboelastometry. *J Vet Emerg Crit Care* 22(6):640-645.
10. Forsyth LL, Liu-DeRyke X, Parker D, Rhoney DH (2012): Role of Hypertonic Saline for the Management of Intracranial Hypertension After Stroke and Traumatic Brain Injury. *Pharmacotherapy: The Journal of Human Pharmacology and Drug Therapy* 28(4):469-484.
11. Griffel MI, Kaufman BS (1992): Pharmacology of colloids and crystalloids. *Crit Care Clin* 8(2):235-53.
12. Hammond TN, Holt JL (2009): Limited fluid volume resuscitation. *Comp Cont Vet Edu* 31(7):309-321.13. Mathews KA (1998): The various types of parenteral fluids and their indications. *Vet Clin North Am Small Anim Pract* 28(3):483-513.
14. Mathews KA (2006): Fluid therapy: Non-Hemorrhage. In: Mathews KA: Veterinary Emergency and Critical Care Manual 2006, Lifelearn Inc. Guelph, pp: 347-372.
15. Mathews KA (2006): Hemorrhage. In: Mathews KA: Veterinary Emergency and Critical Care Manual 2006, Lifelearn Inc. Guelph, pp: 619-629.
16. McMichael M (2005): Pediatric Emergencies. *Vet Clin North Am Small Anim Pract* 35(2):421-434.
17. Mensack S (2008): Fluid Therapy: Options and Rational Administration. *Vet Clin North Am Small Anim Pract* 38(3):575-586.
18. Palmer L, Martin L (2014): Traumatic coagulopathy- Part 1: Pathophysiology and diagnosis. *J Vet Emerg Crit Care* 24(1):63-74.
19. Rudloff E, Kirby R (1998): Fluid therapy. Crystalloids and Colloids. *Vet Clin North Am Small Anim Pract* 28(2):297-328.
20. Sande A, West Ch (2010): Traumatic brain injury: a review of pathophysiology and management. *J Vet Emerg Crit Care* 20(2):177-190.
21. Sigrist N (2011): Stabilization of the emergency patient part II: Circulation. *EJCAP* 21 (2): 133-141.
22. Silverstein DC, Aldrich J, Haskins S, Drobatz KJ, Cowgill LD (2005): Assessment of changes in blood volume in response to resuscitative fluid administration in dogs. *J Vet Emerg Crit Care* 15(3):185-192.
23. Silverstein DC, Kleiner J, Drobatz KJ (2012): Effectiveness of intravenous fluid resuscitation in the emergency room for treatment of hypotension in dogs: 35 cases (2000-2010). *J Vet Emerg Crit Care* 22(6):666-673.
24. Wellman ML, DiBartola SP, Kohn CW (2006): Applied physiology of body fluids in dogs and cats. In: DiBartola SP, editor: Fluid, electrolyte and acid-base disorders in small animal practice. 3rd edition. St. Louis (MO): Saunders Elsevier 2006:3-26.
25. Wurlod VA, Howard J, Francey T, Schweighauser A, Adamik KN (2015): Comparison of the in vitro effects of saline, hypertonic hydroxyethyl starch, hypertonic saline, and two forms of hydroxyethyl starch on whole blood coagulation and platelet function in dogs. *J Vet Emerg Crit Care* 25(4):474-487.



## Commissioned paper\*

# Anaesthesia considerations for critically ill patients

*Ruxandra Costea<sup>1</sup>*

## SUMMARY

While critically ill patients often need surgery for their illness or for the complications of their illness, anaesthesia based on advanced planning is crucial to understand the goals and priorities and to avoid complications. In order for incidents not to become anaesthetic accidents, knowledge and attention to detail are essential. Critically ill patients are prone to have a low ability to maintain homeostasis and tissue oxygenation. Anaesthesia contributes additional physiological stresses that may worsen patient status. Pre-anaesthetic physical evaluation for the emergency or critically ill patient may be difficult and should cover first the cardiovascular, respiratory and neurological status of the patient and after stabilization, a secondary evaluation should be performed. Time for diagnosis and planning can be very limited in these patients, which increases the patient's anaesthetic risk. This review discusses the various aspects to be taken into consideration related to the anaesthetized critically ill patient as well as the most commonly expected complications.

**Key words:** anaesthesia, emergencies, critical, patients

\*This paper was commissioned by FECAVA for *Eur J Comp An Pract* (Autumn 2016) 26(3); p27-p35. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

## Introduction

The aim of this review is to present a general approach to anaesthesia of critically ill patients and the major complications that can occur during procedures. It covers pre-anesthetic evaluation, selection of drugs for premedication, induction and maintenance, monitoring, complications and recovery. Critical illness can result in significant potential life-threatening health problems. In most patients, critical illness is preceded by a period of physiological deterioration; but evidence suggests that the early signs of this are frequently missed<sup>[1]</sup>. Critically ill patients are likely to be unstable and complex, requiring intense, vigilant care and monitoring. Assessment of the critically ill patient should be undertaken by an

appropriately trained clinician and follow a structured ABCD (airway, breathing, circulation, disability) format. This prioritizes the correction of life-threatening problems and provides a standardized approach between professionals. Good outcomes rely on rapid identification, diagnosis and definitive treatment and all veterinarians should possess the skills to recognize the critically ill patient and initiate appropriate management. Supportive care with individualised plans should be developed for every patient based on the recognition of individual needs and risks in order to make anaesthesia a safe and reversible procedure for the critically ill patient.

## Pre-anaesthetic evaluation

Pre-anaesthetic evaluation will help identify individual physiological challenges and risk factors and clarifies the anaesthetic risks and concerns, prior to performing a procedure involving anaesthesia. A thorough history and physical examination should be performed to assess the animal's overall health status.

<sup>1</sup> Clinical Science Department, Faculty of Veterinary Medicine Bucharest, Romania. Email: [costea.ruxandra@yahoo.com](mailto:costea.ruxandra@yahoo.com)

The following factors should be evaluated: history (including known medical conditions, previous anaesthesia, age, breed, behaviour), physical examination, type of procedure (risk of pain, haemorrhage, limited monitoring access, risk of hypothermia), type of anaesthesia (sedation, general anaesthesia, loco-regional anaesthesia). Assessment of the critically ill patient should be undertaken following a structured ABCD (airway, breathing, circulation, disability) format. Critically ill patients may have multiple organ dysfunctions. Based on the findings during the clinical and diagnostic work-up, patients may be classified according to American Society of Anesthesiologists (ASA) physical status classification system modified from the American Society of Anesthesiologists<sup>[2]</sup>. This is used to grade patients' anaesthesia risk (Table 1).

Withholding food before anaesthesia is recommended to reduce the risk of regurgitation and aspiration. Gastric empty times vary widely among individual patients and with the type of ingesta. For emergency procedures fasting is often not possible and the airway should always be protected as if the stomach is full! Administration of metoclopramide in a bolus loading dose of 1.0 mg/kg, IV, followed by continuous infusion at a rate of 1.0 mg/kg/h, will reduce the incidence but not totally prevent gastroesophageal reflux in anaesthetized dogs<sup>[3]</sup>.

Create an individual and flexible anaesthesia plan based on the risks identified, the physical status category and the resources available (staff, equipment for anaesthesia, monitoring). Prioritize the problems, support and stabilize the patient's clinical condition before commencing

anaesthesia. Address the underlying disease as much as possible before anaesthesia then continue according to the individual plan.

A complete plan should address: drug and protocol selection (pre-anaesthetic, induction and maintenance medication), patient support, monitoring, medication and equipment prepared for adverse events or complications. Pre-oxygenation may be considered during patient preparation prior to induction as this will help to improve oxygen saturation for critically ill patients. Placing an IV line is mandatory before anaesthetic procedures because it helps with induction medication, intravascular volume support or a rapid administration of emergency drugs. Connect the monitoring equipment prepared according to the patient's risk and procedure type and duration.

## Premedication

Premedication alleviates anxiety and reduces stress, calms excited patients, allowing reductions in the amount of anaesthetic drugs and allowing a smooth recovery from anaesthesia. Critically ill patients may need lower doses of the same drug compared to healthy ones, for an elective procedure or may not require any premedication if they are depressed by their condition. Common pre-anaesthetic dosages should always be checked carefully and utilised in combination, adjusted to the patient's clinical condition. The medication used for anaesthesia and analgesia protocols in critically ill patients has to be clearly labelled, without shortcuts, writing very clearly the concentrations when they are prepared. Drug administration has to be performed only

Table 1. ASA physical status categories<sup>[2]</sup>

ASA category		Description
ASA 1	Normal healthy patients	no discernable significant disease; general anesthesia with minimal risk
ASA 2	Patients with mild systemic disease	localised infection, compensated disease
ASA 3	Patients with severe systemic disease	severe systemic disease (ex. fever, anemia or moderate hypovolaemia, co-morbidity influencing anaesthesia)
ASA 4	Patients with severe systemic disease that is a threat to life	e.g. uraemia, toxemia, severe dehydration and hypovolaemia, anaemia, cardiac decompensation or high fever
ASA 5	Moribund patients (life threatening disease, patient not expected to survive 24 hours with or without procedure)	e.g. extreme shock, terminal malignancy or infection, severe trauma
E	Emergency	Emergency

under strict surveillance of a veterinary surgeon. Dose and administration time of the premedication must be written on the anaesthesia sheet.

**Opioids** were primarily used for pain management and some sedative qualities. Opioids are psychoactive chemicals like morphine that interact with opioid receptors, they can be utilised in the pre-, intra- and post-anaesthetic phases. Opioids may be antagonized by administration of naloxone. The majority of the opioids are metabolized in the liver and produce their effects depending on the type of receptor that is stimulated<sup>[4]</sup>. The analgesic effects are the result of  $\mu$ -receptor stimulation (for supraspinal analgesia) or  $\kappa$ -receptor stimulation (for spinal analgesia). Central nervous system effects are: analgesia, sedation, euphoria, myosis, nausea and vomiting and cough suppression. Cardiovascular effects are hypotension and bradycardia (increased vagal tone). Opioids cause respiratory depression, gastric stasis, ileus, constipation and an increased biliary pressure. Other side effects: pruritus, urinary retention, muscle stiffness.

**Benzodiazepines** (diazepam, midazolam, zolazepam) are a group of substances causing sedation, anxiolysis, muscle relaxation and anticonvulsive effects. They are used alone or in combination with a variety of drugs, including ketamine, medetomidine, dexmedetomidine and opioids. Flumazenil is a competitive benzodiazepine antagonist used to antagonize sedation with benzodiazepines. Benzodiazepine tranquilizers have minimal cardiorespiratory side effects and are considered safe for critically ill patients, but may cause paroxysmic reactions for patients ASA I-II and dysphoric manifestation in cats.

Diazepam is an insoluble emulsion in water, metabolized by the liver while midazolam is water soluble, metabolized by the liver faster than diazepam. Zolazepam it is used in combination with Ketamine.

**$\alpha$ -2 agonists** (medetomidine, dexmedetomidine) are sedative drugs with analgesic, muscle relaxing and anxiolytic effects, that may be used alone or in combination with other drugs (e.g. ketamine, propofol, midazolam) to provide anaesthesia<sup>[6]</sup>. Medetomidine is a very effective  $\alpha$ -2 agonist. Dexmedetomidine is a highly selective  $\alpha$ -2 agonist, it is protein bound, with a predominantly hepatic metabolism. Common adverse effects are bradycardia, cardiac output reduction, hypertension followed by hypotension, arrhythmias, minimal respiratory depression. Other important effects: reduced intraocular pressure, decreased

intracranial pressure, reduction in gastrointestinal tract motility, hyperglycaemia, platelet aggregation, increased uterine activity (ecbolic effect). To reverse the effects of the  $\alpha$ -2 agonist, atipamezole ( $\alpha$ -2 antagonist) may be administered in doses varying from 1-5 times the medetomidine/dexmedetomidine dose.

**Phenothiazine derivates** (acepromazine, chlorpromazine) sedative and muscle relaxant drugs used to complement the anaesthetic medication. Side effects include peripheral vasodilation due to their alpha-adrenergic blockage, which can lead to hypotension and hypothermia, vagal syncope and collapse<sup>[5]</sup>. Effects on respiratory rate are minimal. They have to be used on critically ill patients with caution and careful monitoring.

## Induction

Induction is usually performed with injectable anaesthetics for rapid airway control and then maintained with inhalational anaesthetics or by injectable anaesthetics. Injectable anaesthetic techniques permit less control over depth and duration of the anaesthesia and various undesired side effects, especially in critically ill patients, with longer recovery times compared to inhalant anaesthetics. It is advisable to reserve the use of injectable anaesthetics for short procedures. A mask or chamber can be used for induction, but neither are recommended for critically ill patients because of the associated stress, lack of airway control and environmental contamination risk. These methods are reserved for excitable or dangerous animals. Commonly used drugs for induction and maintenance of anaesthesia:

- o **Propofol** is a non-barbiturate alkyl phenol drug that produces a rapid deep sedation of short duration. It does not have any analgesic properties. Patients can often be aroused easily by nociceptive stimulation. When administered in higher doses propofol can cause apnoea upon rapid injection and severe respiratory depression<sup>[7]</sup>. A single bolus can be given IV for induction. Due to its short-lived effect (~5 min), recovery after propofol sedation is usually smooth and rapid. Propofol does not accumulate, so repeated administration and/or continuous rate infusion is possible.
- o **Etomidate** is a propylene glycol based imidazol derivate, used for anaesthesia induction. Is a good choice for neurological patients because it can decrease cerebral blood flow and cerebral oxygen consumption.



It has some anticonvulsant effects.

- o **Ketamine** is the most widely used dissociative agent, it has a rapid IV onset. It may be used as a single drug for induction or in combination with other drugs. When administered as a single drug, it has a sympathomimetic effect, resulting in increased heart rate, cardiac output and blood pressure. It can be administered as a bolus or CRI for adjunctive analgesia at 2-10 µg/kg/min.
- o **Tiletamine** is a dissociative drug available in combination 1:1 with zolazepam. It has a duration of action of 1-3 hours and more analgesic effect compared with ketamine.
- o **Thiopental** is a short-acting barbiturate which does not provide analgesia. Beneficial for patients with neurological disease, occasionally used during the induction phase to facilitate endotracheal intubation.
- o **Alphaxalone** is a potent neuroactive steroid injectable anaesthetic agent that provides good muscle relaxation, but has poor analgesic properties, minimal cardiovascular and respiratory depressant effects. It produces a light to medium depth of anaesthesia over a short period of time. It can be administered for induction by bolus or CRI to effect for the desired plane of anaesthesia.

## Endotracheal intubation

Choose the correct endotracheal tube for the patient in correlation with its size and individual anatomy. Insert the endotracheal until the distal tip lies midway between the larynx and the thoracic inlet. Inflate the cuff being aware that over inflation may cause tracheal injury<sup>[8]</sup>. Pay attention to the brachycephalic breeds (Fig.1), frequently they have an elongated soft palate, everted laryngeal sacculles and need endotracheal tubes with a smaller than anticipated diameter (endotracheal tube size is often overestimated). Remember that obese animals do not have a bigger trachea. When changing the patient's position after intubation, do not rotate the endotracheal tube in the trachea, because that can induce tracheal tears. Supraglottic airway devices, also referred to as laryngeal masks can be used for cats and rabbits. They were initially used in human medicine, especially in paediatrics as an alternative to endotracheal intubation. The supraglottic airway device is located on top of the larynx and after ensuring proper positioning (e.g. using capnography), is secured into place using a strap or tie<sup>[8]</sup>.



FIG 1. Endotracheal intubation in a 2-year-old English Bulldog (24 kg) with an elongated soft palate.

## Maintenance

Maintenance of general anaesthesia can be achieved using inhalant or injectable anaesthetics (continuous infusion or intermittent boluses).

- Oxygen enriched mixture with an inhalant anaesthetic (sevoflurane, isoflurane)
- Continuous infusion or intermittent doses of injectable anaesthetic
- Combination of injectable drugs (anaesthetic, opioids) with inhalant anaesthetics

Inhalant anaesthesia provides excellent control over anaesthetic depth and duration. Induction and recovery are rapid, which is particularly beneficial for critically ill patients. The selected inhalant anaesthetic is delivered to the patient in a process that utilizes specific equipment (vaporizer, anaesthetic machine). Isoflurane and sevoflurane are minimally metabolized in the liver, which renders them ideal for use in critically ill patients with impaired liver and/or renal function. Inhalation anaesthetic agents are myocardial depressants, peripheral vasodilators, hypotensive agents and respiratory depressants. Blood pressure is lowered through a vasodilator effect and decreased myocardial contractility. The hypotensive effect is stronger in cats. The minimum alveolar concentration (MAC) to prevent muscular movement in response to strong surgical stimulation in 50% of patients provides a guide for the minimum inhalational dose. The lower the MAC is, the lower the dose required to induce and maintain anaesthesia. MAC depends

on medication, age or physical changes in the patient's clinical status. Adjunctive injectable drugs (anaesthetic, opioids) can be used during maintenance to decrease the amount of inhalant anaesthetic necessary to maintain anaesthesia [9]. Once intubated and connected to the anaesthetic machine, the critically ill patient may breathe spontaneously or be mechanically/manually ventilated.

## Analgesics

Systemic and local analgesia drugs and techniques are used in critically ill patients post operatively and/or given pre-emptively, in order to lower the amount of anaesthetic required during the procedure. Analgesics can be divided in two groups, each exerting their action on the peripheral and central nervous systems.

Systemic analgesics include opioids, nonsteroidal anti-inflammatory drugs,  $\alpha$ -2 agonists (medetomidine, dexmedetomidine) and adjunctive analgesics (drugs with synergistic activity or that potentiate the primary analgesic medication) such as lidocaine, ketamine, amantadine, gabapentin or acetaminophen.

Drugs and techniques should be chosen according to individual risks, pain intensity and procedure duration. In a multimodal approach for critically ill patients, local anaesthesia and analgesia will decrease pain and the use of systemic drugs. Local anaesthetics provide regional anaesthesia by reversibly blocking the transmission of nociceptive signals from nerve endings to the central nervous system. Drug selection is an important step in the local anaesthesia protocol, considering different side effects, onset of action and duration of action.

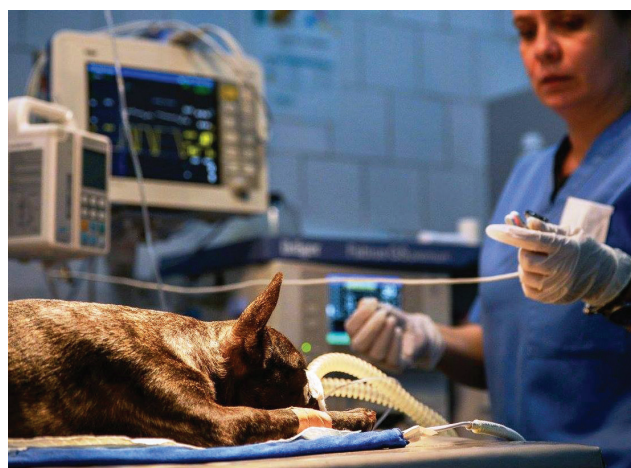
- o Lidocaine is a local amino-amide anaesthetic, in the form of an injectable solution 0.5%, 5% (with or without adrenaline), dermal patches, oral gels, topical gels and nasal sprays. Onset of action is 5-10 minutes, duration of action is 60-120 minutes. Dosage is 1-2 mg/kg for local infiltration, intrapleural block, intraperitoneal block and epidural. Intravenous administration of lidocaine should not exceed 12 mg/kg for dogs and 6 mg/kg for cats, because there is a risk for convulsions<sup>[10]</sup>.
- o Bupivacaine hydrochloride is a local amino-amide anaesthetic, injectable solution 0.25%, 0.5%, 0.75% (with or without adrenaline). Onset of action is 20-30 minutes, duration of action is 180-480 minutes. Dosage is 1-2 mg/kg for local infiltration, intra-pleural

block, intraperitoneal block and epidural use. It is contraindicated for IV regional anaesthesia, because of the potential risk of systemic absorption. Accidental IV injection is cardio toxic and may lead to death.

- o  $\alpha$ -2 agonists (medetomidine, dexmedetomidine) are sedative drugs with analgesic and anxiolytic effects which reduce the use of opioids. Intrathecal and epidural administration can be effective due to the presence of  $\alpha$ -2 receptors in the spinal cord that are crucial in the pain pathways. Adding them to brachial plexus blocks, dental blocks or intercostal blocks enhances nerve blockade. They should be used with caution in combination with opioids or local amino-amide anaesthetics, optimizing local technique, in doses from 0.001 to 0.005 mg/kg, due to possible systemic effects<sup>[11]</sup>.
- o Preservative free opioids (morphine, fentanyl, buprenorphine) can be used alone or combined with local amino-amide anaesthetic or  $\alpha$ -2 agonists (medetomidine, dexmedetomidine)<sup>[12]</sup>.

## Anaesthetic monitoring

Anaesthesia monitoring should include information from the clinical assessment and monitoring equipment. Continued monitoring of the patient during anaesthesia is vital and accurate record keeping is essential. The use of non-invasive and/or invasive monitoring devices can measure the maintenance of tissue oxygen and help in the detection of organ dysfunction (Fig.2).



*FIG 2. Continuous monitoring during anaesthesia is essential in critically ill patients.*

Basic monitoring during anaesthesia includes assessing the depth of anaesthesia, heart rate and rhythm, mucous membrane colour (pale, pink, red, brick red, blue), capillary refill time, pulse (rate, quality), rate and pattern

of respiration, pulse oximetry, temperature (oesophageal probe or periodic rectal temperature).

Advanced monitoring through continuous electrocardiogram (ECG) assesses cardiac rhythm and detects conduction disorders. Direct blood pressure monitoring via arterial catheterization is the most accurate but technically difficult way to measure blood pressure. Indirect blood pressure monitoring through oscillometry or Doppler is easy to perform, but can be inaccurate. Trends should be evaluated in conjunction with other parameters. Central venous pressure (CPV) measurement of the hydrostatic pressure in the intrathoracic vena cava is beneficial in any patient that presents with hypovolaemia to avoid the risk of overload. Capnography and capnometry measure and display carbon dioxide concentrations in expired air as a function of time and report them as inspired and end-expired (end-tidal). End-tidal carbon dioxide tension is used to provide a non-invasive estimate of the arterial partial pressure of carbon dioxide ( $\text{PaCO}_2$ )<sup>[13]</sup>. Arterial blood gas analysis during a critically ill patient's anaesthesia assesses the adequacy of patient ventilation, oxygenation and acid-base status. Glycaemia should be monitored for diabetics and neonatal patients in the peri-anaesthetic period. Urinary output quantity, via a closed collection system is a marker of renal perfusion and should be monitored during the procedure and after it.

## Complications

Dealing with complications during a critically ill patient's anaesthesia and recovery time, requires proper anaesthetic management, accurate and continued monitoring. Using inhalant anaesthesia reduces the risk of an emergency situation arising but does not eliminate it. The most commonly expected problems are hypoxaemia, hypoventilation, hypotension, cardiac arrhythmias, acidosis and hypothermia.

### Respiratory emergencies

Respiratory emergencies occur when there is an inability to maintain the correct gas exchanges. Either transport of oxygen (bound to haemoglobin  $\text{SpO}_2$  or soluble  $\text{PaO}_2$ ) from air to the tissue cells (oxygenation) or of carbon dioxide from cells to outside (ventilation). Low  $\text{SpO}_2$  levels ( $<95\%$ ) may result in low  $\text{PaO}_2$ . Hypoxaemia can be due to a low fraction of inspired oxygen, ventilation problems or diffusion problems. Respiratory minute volume represents the amount of gas exhaled by the lungs over 1 minute

(respiratory rate x tidal volume). A decreased respiratory minute volume arises from a decrease in respiratory rate or/and tidal volume, leading to hypoventilation. The most common respiratory complication in anaesthetized patients is hypoventilation (hypercapnia) associated with anaesthetic drug administration<sup>[14]</sup>. Hypoventilation leads to hypercapnia (increased dissolved  $\text{CO}_2$  in blood, high  $\text{PaCO}_2$ ), which leads to respiratory acidosis (low blood pH) and its consequent effects. End-tidal carbon dioxide tension ( $\text{ETCO}_2$ ) provides data related to ventilation, perfusion, respiratory rate, endotracheal intubation. An  $\text{ETCO}_2$  greater than 55mmHg is an indication of hypoventilation possibly due to a decreased tidal volume (pneumothorax, intubation problems, anaesthetic machine problems, central nervous system disturbances, deep sedation or anaesthesia). To correct hypoventilation the respiratory minute volume should be increased through assisted ventilation (increased respiratory rate, tidal volume). Lighten the level of anaesthesia, check the breathing system for obstruction or leaks and check if the soda lime has expired.

All anaesthetic drugs can depress respiration and cause bradypnoea (slow, regular respiratory rate). Other direct causes for bradypnoea are overdosage, direct depression of the central respiratory centre, hypothermia. Central nervous system depression during anaesthesia can lead to apnoea. Apnoea or respiratory arrest is an emergency situation during anaesthesia, caused by drug depression, obstruction or increased intracranial pressure. Immediate actions must be initiated: turn off anaesthetic gas or reverse injectable anaesthesia, provide 100% oxygen, support ventilation until the patient can maintain adequate tidal volume, breath and gas exchange (mechanical/manual) and administer doxapram. Upper airway obstruction is an emergency that can be associated with laryngeal oedema, laryngospasm or endotracheal tube blockage. Turn off anaesthetic gas and provide 100% oxygen. Verify endotracheal tube's position, use suction if necessary, ensure correct positioning of the patient's head to prevent kinking of the tube, remove or replace the tube if it is defective, deliver oxygen. In case of laryngeal oedema administer corticosteroids or consider delivering oxygen through a tracheostomy. Pre-oxygenation with 100% oxygen (3-4 minutes before induction) can markedly prolong the time to haemoglobin desaturation, hypoxaemia and cyanosis. High respiratory minute volume patients are hyperventilating, which decreases  $\text{PaCO}_2$  (hypocapnia). Hyperventilation is associated with low  $\text{ETCO}_2$  in cases of hypoxia, low levels of anaesthesia, pain, central nervous system problems,



endotracheal tube obstruction, hyperthermia, cardiac arrest. Hyperventilation may be accompanied by tachycardia and hypertension. Hyperventilation cases require a decrease in the respiratory minute volume (rate or tidal volume). Stabilization of the patient implies oxygen supplementation, increasing anaesthesia and analgesia levels, reducing body temperature. If  $\text{ETCO}_2$  drops suddenly a possible circulatory collapse or a pulmonary thromboembolism may be present.

### Cardiovascular emergencies

Cardiovascular emergencies are cases which result in an inability to maintain adequate blood flow, tissue perfusion and oxygenation to vital organs. Cardiovascular monitoring during anaesthesia of critically ill patients should cover haemodynamic parameters relating to tissue oxygenation: mucous membranes, capillary refill time, heart rate/rhythm/contractility, pulse, blood pressure, central venous pressure and urinary output, lactate, central venous oxygen saturation, base excess.

Mucous membrane colour is an indicator of perfusion of peripheral tissues, except in anaemia cases. The normal appearance is pink, moist, with a capillary refill time between 1-2 seconds (Fig.3). Red coloured mucous membranes indicate peripheral vasodilatation (SIRS, sepsis, early stages of shock), a pale colour indicates anaemia or severe vasoconstriction, grey a massive vasoconstriction, late decompensated shock, yellow icterus, cyanosis-severe hypoxemia. Pulse quality indicates the heart rate and the difference between systolic and diastolic pressure. A weak pulse can suggest increased peripheral vascular resistance or a low stroke volume (hypovolaemia, decompensated shock). In cases of hypovolaemia consider volume replacement



FIG 3. Normal, pink mucous membranes.

with isotonic fluids, colloids or blood. A bounding pulse is related to decreased peripheral vascular resistance and strong stroke volume (sepsis, fever, compensated shock). Heart rate, rhythm and contractility are measured using a stethoscope, oesophageal stethoscope, a Doppler flow probe or by continuous ECG.

Common causes of cardiovascular emergencies are respiratory failure, acid-base and electrolyte imbalance, hypothermia, air embolism, medication and cardiac disease. Common problems related to the heart rate, rhythm and contractility are tachycardia, bradycardia and arrhythmias.

Tachycardia during anaesthesia can be related to a light level of anaesthesia, pain, fever, SIRS, sepsis, hypovolaemia. Heart efficiency is decreasing and oxygen needs are increasing. Anaesthetic dosage and analgesia level should be increased, the patient must be ventilated and oxygenated and any acid-base and electrolytes abnormalities corrected. Bradycardia during anaesthesia can be related to medication ( $\alpha$ -2 agonists, opioids), acid-base abnormalities, decompensated shock, hypoxia, hypothermia, high levels of  $\text{K}^+$ ,  $\text{Ca}^{2+}$ . Increased vagal tone due to difficult intubation, abdominal surgery, eye surgery or direct vagal stimulation can cause bradycardia during anaesthesia. In these cases anaesthesia levels should be decreased or injectable anaesthetics reversed. Assure ventilation and oxygenate the patient, correct any acid-base and electrolytes abnormalities. Identify and treat the main cause. Administer anticholinergic drugs for bradycardia. Consider adrenaline and CPR for cardiac arrest. Arrhythmias occur following tissue hypoxia, hypercapnia, acidosis, inflammatory mediators, myocarditis or sympathomimetic drugs. The decision to treat arrhythmias should be based on the physiologic effect and the potential to degenerate into a lethal rhythm.

Arterial blood pressure has three components: systolic arterial pressure, diastolic arterial pressure and mean arterial blood pressure. Mean arterial blood pressure (MAP) is the force generated by systolic arterial pressure (SAP) and diastolic arterial pressure (DAP). It can be calculated from the formula  $\text{MAP} = 1/3 \text{ SAP} + 2/3 \text{ DAP}$ .

Hypotension ( $\text{MAP} < 60 \text{ mmHg}$ ) is the most commonly reported anaesthetic complication<sup>[14]</sup>. It is related to medication during anaesthesia ( $\alpha$ -2 agonists, acepromazine, opioids, isoflurane, sevoflurane), hypovolaemia, hypothermia, vasodilatation (vasoplegic

shock), heart disease (cardiac dilatation). Any severely critically ill patient should be considered a potential hypotensive patient. Actions should target lightening of the anaesthetic level and address the cause of the hypotension (IV bolus of fluids for hypovolaemic patients, transfusion or positive inotropic drugs for dilated cardiomyopathy). In cases of extreme vasodilatation, pressors are indicated for vasoconstriction (dopamine, noradrenaline, vasopressin, phenylephrine).

The causes of hypertension (SAP>180 mmHg) are related to fever, pain, kidney disease, obesity, endocrine pathology (diabetes, hyperthyroidism, hyperadrenocorticism), medication, arterial thrombus. Hypertension may have consequences such as retinal detachment or cerebral haemorrhage.

### Hypothermia and hyperthermia

The temperature of an anaesthetized critically ill patient affects cardiovascular, respiratory and metabolic functions. Anaesthetics suppress normal thermoregulatory mechanisms<sup>[15]</sup>. It is challenging to maintain normal body temperature during anaesthesia.

Hypothermia (<37°C) is a common complication during anaesthesia and causes peripheral vasoconstriction, decreased cerebral circulation, arrhythmias, decreased ventilation and inhalant anaesthetic elimination, lowers the effect of analgesics. For all critically ill patients undergoing anaesthesia try to minimize the duration of anaesthesia and decrease the dosages. Measures must be taken as soon as

possible for warming the patient: administration of warm IV fluids, forced air warming units, warm water circulating blankets, radiant heat sources (Fig.4).

Hyperthermia (>40.5°C) during or after anaesthesia may cause complications. It is due to thermoregulation problems, medication (opioids in cats) or iatrogenic (extreme warming of the patient). Hyperthermia can be associated with vasodilation, tachycardia, arrhythmias, tachypnoea and coagulopathy. Treatment may be initiated by administering cold IV fluids, increasing the oxygen flow, applying cold water or alcohol to the footpads.

### Prolonged recovery

Prolonged recovery from anaesthesia is always a challenging task for critically ill patients and can be caused by medication over dosage (antagonize if possible), inadequate metabolism and excretion or hypothermia. Previous neurological injuries, ischaemia, embolism, myocardial disturbance or severe hypoxia can all lead to a prolonged recovery from anaesthesia. Continue monitoring until complete awakening (Fig.5). Record the parameters at intervals of 5-10 minutes or more frequently if changes in the clinical status are developing. Cardio-respiratory arrest can occur at any time during anaesthesia or in the



FIG 4. Warming a patient during the recovery period.



Fig 5. Patient monitoring at the end of the surgery, to be continued during the recovery period.



recovery period. Provide supplementary oxygen and IV fluids in the recovery period. Be prepared to act quickly, to start cardio-pulmonary resuscitation if it is necessary<sup>[16]</sup>. Post anaesthetic complications are in direct relation to a lack of analgesia, when fluids and electrolytes are administered inappropriately, lack of metabolic support or patient abandonment (no monitoring or support during recovery time). Greater patient care in the postoperative period may reduce fatalities<sup>[16]</sup>.

## Conclusion

Anaesthesia in the critically ill patient can be challenging due to the multiple complications and the possibility of decompensation at any time. A comprehensive pre-anaesthetic examination along with an appropriate protocol selection, regular attention and monitoring will lead to an early assessment of complications and a better outcome for the critically ill patient. A multi-modal approach is recommended as this helps minimize the side effects that may occur. While small animal anaesthesia is increasingly safe, good outcomes are based on knowledge, preparation, rapid diagnosis and treatment of any emergency situation.

## References

- [1] Robertson LC, et al. Recognizing the critically ill patient. *Anaesthesia & Intensive Care Medicine*. 2013; 14 (1): 11-14
- [2] Mohamed D. American Society of Anesthesiologists physical status classification. *Indian J Anaesth*. 2011; 55(2): 111-115
- [3] Wilson D, Evans T, Mauer WA. Influence of metoclopramide on gastroesophageal reflux in anesthetized dogs. *American Journal of Veterinary Research*. 2006; 67 (1): 26-31
- [4] Branson KR, Gross ME, Booth NH. Opioid agonists and antagonists. *Veterinary pharmacology and therapeutics*. 2001; 8: 268-298
- [5] Gross ME, Marjorie E, Booth NH. Tranquilizers,  $\alpha$ 2-adrenergic agonists, and related agents. *Veterinary pharmacology and therapeutics*, 2001, 8: 299-342
- [6] Costea R et al. Contributions to the peri-operative supportive care and anesthesia for urogenital surgeries in small animals. *Scientific Works. Series C. Veterinary Medicine*, 2013; 59(3): 72-73
- [7] Costea R, Vitalaru BA. Propofol induction anesthesia for central venous catheterization in dogs with renal failure. *Journal of Biotechnology*. 2015; 208: S42-S43
- [8] Hardie EM, Spodnick GJ, Gilson SD, et al. Tracheal rupture in cats: 16 cases (1983-1998). *J Am Vet Assoc*. 1999; 214 (4): 508-512
- [8] Crotaz IR. Initial feasibility investigation of the v-gel airway: an anatomically designed supraglottic airway device for use in companion animal veterinary anaesthesia. *Veterinary Anaesthesia and Analgesia*. 2010; 37 (6):579-580
- [9] Shaughnessy MR, Hofmeister EH. A systematic review of sevoflurane and isoflurane minimum alveolar concentration in domestic cats. *Vet Anaesth Analg*. 2014 Jan;41(1):1-13. doi: 10.1111/vaa.12083. Epub 2013 Sep 10
- [10] Lemke KA, Dawson SD. Local and regional anesthesia. *Vet Clin North Am Small Anim Pract*. 2000 Jul;30(4):839-57
- [11] Lamont, L. A.; Mathews, K. A. Opioids, nonsteroidal anti-inflammatories, and analgesic adjuvants. In: Lumb and Jones' Veterinary Anesthesia and Analgesia, 4th edn (2007). Eds WJ Tranquilli, JC Thurmon, KA Grimm. Wiley-Blackwell, USA
- [12] Pascal M, Costea R, Țogoe D, Vitalaru BA, Diaconescu A, Birtoiu IA. Epidural delivery of lidocaine and tramadol to control pain during ovariohysterectomy in the bitch. *Scientific Works. Series C. Veterinary Medicine*. Vol. LXI (1): 168-171
- [13] Rich GE, Sullivan MR, Adams JM (1990). Is distal sampling of endtidal CO2 necessary in small subjects? *Anesthesiol* 73, 265-268
- [14] Dyson DH, Maxie MG, Schnurr D. Morbidity and mortality associated with anesthetic management in small animal veterinary practice in Ontario. *J Am Anim Hosp Assoc* 1998;34:325-335
- [15] Hipotemie- Kurz A (2008). Thermal care in the perioperative period. *Best Pract Res Clin Anesthesiol* 22, 39-62
- [16] <http://www.acvecc-recover.org>
- [17] Brodbelt DC, Blissitt KJ, Hammond RA, Neath PJ, Young LE, Pfeiffer DU, Wood JL. The risk of death: the confidential enquiry into perioperative small animal fatalities. *Vet Anaesth Analg*. 2008, 35(5):365-73



## Reprint paper\*

# Linear versus non-linear gastrointestinal foreign bodies in 499 dogs: clinical presentation, management and short-term outcome

Melissa M. Hobday<sup>1</sup>, Garret E. Pachtinger, Kenneth J. Drobatz and Rebecca S. Syring

## SUMMARY

**Objectives:** To compare clinical signs, clinicopathological abnormalities, imaging findings and outcome of dogs with linear and non-linear foreign bodies in the gastrointestinal tract.

**Methods:** Retrospective review of case records of dogs with a confirmed diagnosis of gastrointestinal foreign body. Signalment, history, clinical signs, clinicopathological data, diagnostic imaging studies, surgical and endoscopic procedures, hospital stay, costs and outcome were compared between groups.

**Results:** A total of 176 dogs had linear and 323 had non-linear foreign bodies. Dogs with a linear foreign body were more likely to have a history of vomiting, anorexia, lethargy and pain on abdominal palpation. They were also more likely to have the foreign body anchored in the stomach and continuing into the small intestine, experience intestinal necrosis, perforation and peritonitis, and require intestinal resection and anastomosis. The duration of hospitalisation was longer for dogs with linear foreign body (3 versus 2 days), and the cost of treatment was 10% higher. However, in both groups, 96% of dogs survived to hospital discharge.

**Clinical Significance:** Dogs with a linear foreign body had more severe clinical signs and gastrointestinal pathology, and an increased duration of hospitalisation and cost of care. However, overall survival rates were not different in dogs with linear and non-linear foreign bodies.

\*This paper originally appeared in the *Journal of Small Animal Practice* (2014) 55, 560–565 (DOI: 10.1111/jsap.12271) and was submitted by the British Small Animal Veterinary Association *Eur J Comp An Pract* (Autumn 2016) 26(3); p36–p43. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

## Introduction

Gastrointestinal foreign bodies are a common diagnosis amongst dogs presenting for emergency veterinary care and yet they often represent a diagnostic challenge (Clark 1968, Guilford 1996, Aronson et al. 2000, Tyrrell & Beck 2006, Hayes 2009, Sharma et al. 2011). After ingestion, the onset of clinical signs can vary from hours to weeks (Capak et al. 2001, Gianella et al. 2009, Hayes 2009). Clinical signs commonly associated with gastrointestinal disease such as

anorexia, vomiting, diarrhoea, abdominal discomfort and lethargy are non-specific and are variably present in dogs with gastrointestinal foreign bodies (Mishra et al. 1974, Capak et al. 2001, Hayes 2009). Moreover, while radiography is commonly used to evaluate patients with suspected gastrointestinal foreign bodies, plain abdominal radiographs may be unremarkable in many cases and abdominal ultrasonography may be required to confirm the diagnosis (Root & Lord 1971, Tidwell & Pennick 1992, Graham et al. 1998, Tyrrell & Beck 2006, Sharma et al. 2011).

<sup>1</sup> (Formerly:) Department of Clinical, School of Veterinary Medicine, University of Pennsylvania, Philadelphia, PA 19104-6010, USA

Previous studies suggest that dogs with linear foreign bodies (LFB) have a higher frequency of postoperative complications and a worse prognosis compared with non-linear foreign bodies (NLFB) (Evans et al. 1994, Hayes 2009). The purpose of the current study was to compare the clinical signs, clinicopathological abnormalities, diagnostic imaging findings, treatment and outcome between a group of dogs with LFB and a group with NLFB to determine if the groups would have a similar prognosis.

## Materials and methods

Using a retrospective case-ascertainment design, the computerised medical record database of the Mathew J. Ryan Veterinary Hospital at the University of Pennsylvania was searched to identify dogs that were presented to the emergency service between 1997 and 2008 and diagnosed with a gastrointestinal foreign body. The records were then grouped into those with LFB and NLFB. To maximise case identification, the computerised medical record database was cross-referenced with both the complete surgical case log and abdominal fluid analysis submissions.

Dogs were included in the study if a foreign body was the cause of any of the presenting clinical signs of vomiting, anorexia, lethargy or diarrhoea. Dogs with objects in the oral cavity or colon were included only if those objects extended to or from another portion of the gastrointestinal tract. Finally, the foreign material had to be confirmed by surgical or endoscopic removal, or necropsy. Dogs were excluded from the study if the clinical signs were caused by aetiologies other than a foreign body. Incomplete medical records were not grounds for exclusion from the study.

Clinical variables extracted from the medical record included: history, signalment, body weight, age at presentation, clinical signs (anorexia, lethargy, vomiting, diarrhoea), physical examination findings (abdominal pain, ability to palpate a foreign body), clinicopathological data (venous blood gas, complete blood count, biochemistry, lactate concentration), diagnostic procedures (radiography, ultrasonography, contrast radiography), surgical procedures (gastrotomy, enterotomy, intestinal resection and anastomosis), endoscopic evaluation, surgical time, anaesthesia time, intraoperative findings (perforation, peritonitis, bowel necrosis), location of the foreign body, outcome (survival, euthanasia, death), necropsy results, cost for hospitalisation and treatment and length

of hospitalisation. Dogs were categorised as having peritonitis based solely on the surgical report, and a distinction between septic and aseptic peritonitis was not made.

Abdominal radiograph reports, which were completed by board certified radiologists, were reviewed. Additional diagnostic imaging tools, such as repeated radiographs, contrast radiography or ultrasonography were also recorded.

Flexible gastrointestinal endoscopy was used for foreign body retrieval, and was performed by internal medicine specialists and house officers directly supervised by internists. The endoscopy reports and patient discharge summaries were reviewed for foreign body location and gross pathological changes resulting from the foreign body ingestion.

In dogs that underwent an exploratory coeliotomy, a ventral midline incision was made, and a routine examination of the gastrointestinal tract was carried out. Foreign body location, surgical method and anatomical pathology as a result of the foreign body were recorded from the surgical report. Surgical specialists or house officers under the guidance of the surgical specialists performed all surgeries.

Dogs were categorised as having either LFB or NLFB on the basis of the attending veterinarian's description of the gastrointestinal abnormalities from the surgical or endoscopic procedures, or necropsy. Linear foreign bodies were classified as compliant objects anchored at one anatomic site, inducing plication through one or more aboral sites in the gastrointestinal tract. Non-linear foreign bodies were defined as ingested, discrete objects causing clinical signs of vomiting, anorexia, lethargy, abdominal pain and/or diarrhoea.

## Statistical analysis

T-test for independent groups was used to examine the differences in the mean values of continuous dependent variable measures (days hospitalised, hospital costs, age, surgery time and related factors) and the independent categorical measures (linear foreign bodies, non-linear foreign bodies). Chi-squared test ( $\chi^2$ ) was used to compare differences in categorical factors (e.g. linear

foreign bodies/non-linear foreign bodies with intervention categories, presenting signs and the remaining signalment parameters). All reported P-values are two-sided with P-values lower than 0.05 indicating statistical significance. Descriptive statistics utilised proportions by group and or variable. The calculations were completed using Epi Info (2013) and SPSS (2011).

## Results

Between January 1997 and January 2008, there were 499 dogs that met the criteria for inclusion into the study. The LFB group included 176 dogs (35.3%) and the NLFB included 323 dogs (64.7%). There were 87 different breed classifications. The majority of the dogs were pedigree, with the most common breeds being the Labrador retriever (67/499 13.4%; 23/176 LFB 13.1%, 44/323 NLFB 13.6%;  $P=0.86$ ), golden retriever (28/499 5.6%; 12/176 LFB 6.8%, 17/323 NLFB 5.3%;  $P=0.39$ ) and the American pit bull terrier (21/499 4.2%; 9/176 LFB 5.1%, 12/323 NLFB 3.7%;  $P=0.46$ ). Additionally, there were 69 mixed breed dogs (69/499 13.8%; 34/176 LFB 19.3%, 35/324 NLFB 10.8%). Of the 499 dogs, 337 were male (67.5%; 116 LFB 23.2%, 221 NLFB 44.3%;  $P=0.31$ ) and 162 were female (32.5%; 60 LFB 12.0%, 102 NLFB 20.4%;  $P=0.63$ ). Overall, 354 of patients were neutered (70.9%; 42 spayed female LFB 8.4%, 78 castrated male LFB 15.6% and 75 spayed female NLFB 15.0%, 159 castrated male NLFB 31.8%). The mean  $\pm$ sd age of LFB dogs at the time of admission was  $4.06 \pm 3.32$  years (range 0.21-14.0 years). The mean  $\pm$ sd age of NLFB dogs at the time of admission was  $4.27 \pm 3.51$  years (range 0.085-14.5 years), which was not statistically significantly different ( $P=0.51$ ).

Several clinical signs and examination findings were evaluated in the dogs with LFB and NLFB (Table 1). As per the inclusion criteria, all dogs had at least one clinical sign

associated with the gastrointestinal foreign body, and with the exception of diarrhoea, each of these clinical signs was more frequently present in dogs with LFBs. Dogs in the LFB group were also more likely to have pain on abdominal palpation. The ability to palpate the foreign object on initial physical examination was uncommon and similar in both groups (LFB 27/176, 15.3%, NLFB 43/323, 13.3%;  $P=0.53$ ).

The results of the most common laboratory studies are summarised in Table 2. Dogs with LFB had significantly lower sodium, potassium and chloride, and higher bicarbonate, haematocrit and blood urea nitrogen as compared to those with NLFB. Percentages of blood work parameters below and above reference ranges were calculated for each group.

Survey abdominal radiography was performed in 483 of 499 dogs. In the 16 patients for which radiographs were not performed, 10 dogs had ultrasound as the sole diagnostic imaging study, 2 dogs had a string foreign body under the tongue and were taken directly to surgery, 2 dogs were euthanased prior to diagnostics, with the foreign body confirmed on necropsy, and 2 case records lacked documentation of imaging studies. The initial set of radiographs was considered sufficient to provide a diagnosis of foreign body in 337 dogs (LFB 110/166; 62.5%, NLFB 227/318; 71.8%). There was no statistically significant difference in the ability to diagnose LFB or NLFB based on the initial radiographic evaluation ( $P=0.271$ ). Radiographs were repeated in 10 dogs (five LFB, five NLFB), where the initial set had not yielded a diagnosis. This confirmed the presence of a foreign body in seven dogs (two LFB, five NLFB). Barium contrast radiography was utilised in 30 dogs and it confirmed a diagnosis of foreign body in 26 of 30 dogs (LFB 12/14; 86%, NLFB 14/16; 88%). For the four dogs where contrast

*Table 1. Frequency of presenting clinical signs and examination findings at admission for dogs with LFB (n=176) compared to NLFB (n=323)*

Variable	LFB (n=176)	LFB %	NLFB (n=323)	NLFB %	P-value
Vomiting	173	98.3	286	88.6	0.0001
Anorexia	164	93.1	261	80.8	0.0002
Lethargy	162	92.0	254	78.6	0.0001
Abdominal pain	96	54.5	122	37.8	0.0003
Diarrhoea	35	16.5	77	23.8	0.2798
Ability to palpate foreign body	27	15.3	43	13.3	0.53

Table 2. The mean values for the most common laboratory tests ordered on admission and between-group comparisons for dogs with LFB (n=176) and NLFB (n=323)

Variable	Reference intervals	LFB			NLFB			P-value
		Mean (sd)	% *		Mean (sd)	% *		
			+	-		+	-	
Significant								
pH	7.35-7.45	7.44 (0.06)	44.1	8.2	7.42 (0.05)	27.4	8.3	0.02
Na (mmol/L)	139-154	143.70 (6.00)	1.1	16.0	145.53 (6.07)	3.4	9.9	0.001
K (mmol/L)	3.6-5.5	3.76 (0.49)	0.0	33.1	3.92 (0.49)	0.6	24.7	0.001
Cl (mmol/L)	102-120	104.55 (9.59)	0.6	33.5	108.30 (8.52)	2.2	12.9	<0.001
HCO3 (mmol/L)	21-24	28.70 (5.36)	82.4	2.4	26.57 (6.97)	64.6	11.5	0.001
HCT (L/L)	0.36-0.60	0.49 (0.093)	11.0	13.3	0.47 (0.08)	6.9	3.7	0.006
BUN (mmol/L)	2.14-11.1	7.90 (7.26)	0.19	0.6	6.12 (6.57)	6.1	1.3	0.009
Non-Significant								
Lactate (mmol/L)	0.5-2.5	2.56 (1.55) 4	1.1	1.2	2.38 (1.46)	36.5	1.6	0.21
Glucose (mmol/L)	3.85-7.59	6.26 (1.98)	14.9	1.1	5.97 (1.02)	5.9	0.9	0.07
ALT (U/L)	12-118	69.83 (69.03)	11.8	1.3	68.76 (114.47)	8.3	0.4	0.92
AST (U/L)	15-66	69.20 (67.27)	33.6	0.0	63.64 (70.55)	24.2	0.0	0.43
Albumin (g/L)	24-42	31.9 (8.87)	9.0	17.4	32.1 (7.12)	4.5	8.2	0.88
WBC (×10 <sup>9</sup> /L)	4.1-15.5	16.24 (6.35)	47.5	0.6	15.60 (7.54)	45.9	1.8	0.37
Creatinine (μmol/L)	44.2-141.4	86.63 (44.2)	6.7	3.0	91.94 (76.91)	5.3	1.4	0.37

n Number of dogs, LFB Linear foreign bodies, NLFB Non-linear foreign bodies, sd Standard deviation, Cl Chloride, K Potassium, Na Sodium, BUN Blood urea nitrogen, WBC White blood cells, HCO<sub>3</sub> Bicarbonate, HCT Haematocrit

\*Percentage of dogs above (+) and below (-) reference range

radiography was considered insufficient for definitive diagnosis, ultrasound was ultimately used. Abdominal ultrasonography was performed on 123 dogs in this study and confirmed the diagnosis of foreign body in all cases, based on ultrasound reports. This was true if ultrasound was the sole imaging study, or if it was performed when survey or contrast radiography failed to yield a diagnosis. Of the 499 dogs evaluated in the study, 494 underwent

general anaesthesia for a surgical or endoscopic procedure. Five dogs were euthanased before any treatment with a diagnosis of foreign body later confirmed at necropsy (one LFB, four NLFB). The 324 dogs with NLFB had a total of 374 foreign bodies removed, while 176 were removed from the 176 dogs with LFB. The locations of the LFB and NLFB retrieved from the gastrointestinal tract are reported in Fig 1. Dogs with LFB most commonly had foreign material

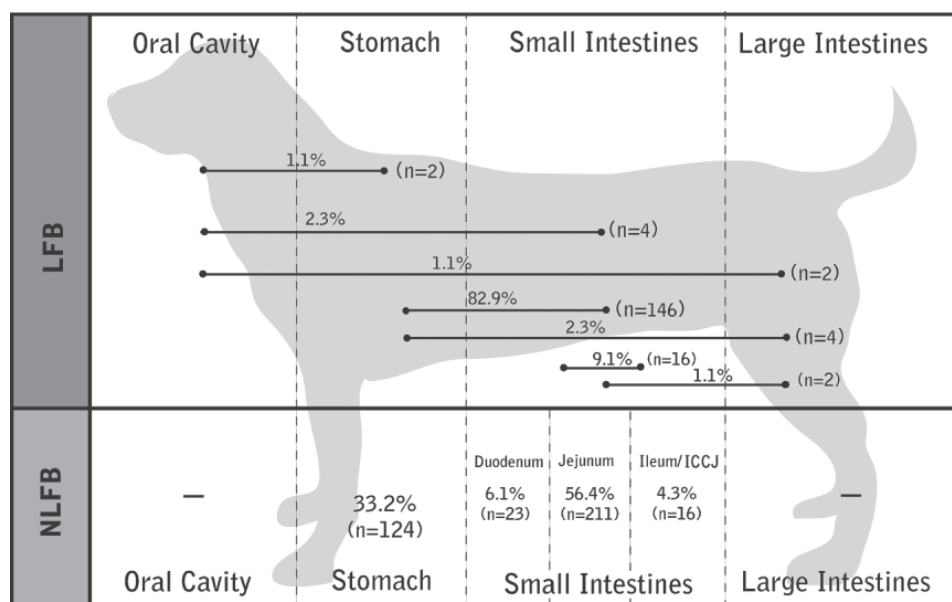


FIG 1. Location of 550 foreign bodies (176 linear, 374 non-linear) in 499 dogs



anchored in their stomach (150 dogs, 85.2%), whereas the foreign material was most commonly found in the jejunum in dogs with NLFB (211 dogs, 65.3%).

Endoscopic foreign body retrieval was attempted in 57 dogs (5 LFB, 52 NLFB), and was successful in 34 dogs (LFB 1/5, 20.0%; NLFB 33/52, 63.5%). The remaining dogs required exploratory coeliotomy following the unsuccessful endoscopic procedure. A gastrotomy was performed in 248 dogs (157 LFB, 91 NLFB) for the removal of foreign material. Six more gastrotomies were performed (1 LFB, 5 NLFB) to assess the gastric lumen for further foreign material or to inspect the pylorus, but foreign material was not found.

Exploratory coeliotomy was performed in 460 dogs (460/499, 92%; LFB 174/176, 99%; NLFB 286/323, 88.5%). Dogs in the LFB group were more likely to have a gastrotomy, at least one enterotomy, and intestinal resection and anastomosis, as well as intestinal perforation, peritonitis and intestinal necrosis. Surgery and anaesthesia time were significantly longer in dogs with LFB. These results are summarised in Table 3.

## Mortality risk

Four hundred and seventy-nine dogs (96%) survived to discharge, while 19 dogs were euthanased (5 preoperatively, 2 intraoperatively, 12 postoperatively) and 1 died postoperatively. Both groups had a 96% survival rate to time of discharge from hospital. The reasons for death or euthanasia are summarised in Table 4.

## Duration of hospitalisation and cost of care

The median length of hospitalisation for LFB dogs was 3 days (range 1-18 days) compared with 2 days (range 1-22 days) for dogs in the NLFB group, which was significantly different ( $P<0.0001$ ). Similarly, the LFB group median total costs were \$2479.25 (range \$486-\$18,307), which was significantly greater than costs for dogs with NLFB, median \$2263.50 (range \$215-\$7490). The difference in mean costs was significant with the LFB group having increased costs ( $P<0.001$ ).

Table 3. Summary of surgical techniques, intraoperative findings, and procedure times between the LFB and NLFB groups

Dogs having surgery	LFB n=174	LFB 174/176 (99%)	NLFB n=286	NLFB 286/324 (88%)	P-value
Gastrotomy	159	90.3	96	29.7	<0.001
Enterotomy (one or more)	130	73.9	167	52.0	<0.001
Resection and anastomosis	57	32.4	51	15.8	<0.001
Intestinal perforation	34	19.3	16	4.9	<0.001
Peritonitis	31	17.6	22	6.8	<0.001
Intestinal necrosis	48	27.3	46	14.2	<0.001
Mean surgical time (minutes) $\pm$ sd	122.7 $\pm$ sd 50.84	–	94.2 $\pm$ sd 37.26	–	<0.0001
Mean anaesthesia time (minutes) $\pm$ sd	176.5 $\pm$ sd 53.27	–	144.8 $\pm$ sd 51.97	–	<0.0001

Total number of dogs having surgery 460/500 (92%). LFB Linear foreign bodies, NLFB Non-linear foreign bodies, sd Standard deviation

Table 4. Summary of mortality for the dogs with LFB and NLFB

Dogs having surgery	LFB 7/176 (4%)	NLFB 13/323 (4%)
Dogs euthanased preoperatively	1 – financial reasons	2 – financial reasons
		2 – undocumented reasons
Dogs euthanased intraoperatively	0	2 – significant intestinal necrosis, perforation and sepsis
Dogs euthanased postoperatively	1 – recurrent septic abdomen 1 – ARDS 1 – financial reasons 3 – undocumented reasons	1 – intestinal infarct 2 – ARDS 1 – financial reasons 1 – sepsis 1 – undocumented reason
Dogs that died	0	1

LFB Linear foreign bodies, NLFB Non-linear foreign bodies, ARDS Acute respiratory distress syndrome

## Discussion

This is the largest retrospective study to date evaluating the clinical course of LFB and NLFB in dogs. It reports excellent rates of survival, following diagnosis of a gastrointestinal foreign body (96%) with no difference in outcome when comparing dogs with LFB to those with NLFB, despite a higher frequency of intestinal necrosis, intestinal perforation, peritonitis, longer surgical times, longer hospital stay and increased costs in dogs with LFB. Previous reports suggest that LFBs cause partial obstruction, and animals may not present with clinical signs as severe as those with a discrete, complete obstruction (Aronson et al. 2000, Brown 2012). In the current study, however, this was not the case, as dogs with LFBs had more frequent reports of anorexia, vomiting, lethargy and pain on abdominal palpation compared to dogs presenting with NLFBs. These results are comparable to another study where 94% of dogs had vomiting, 66% had anorexia and 63% had signs of lethargy, with LFB obstructions (Evans et al. 1994). Diarrhoea and clinician ability to palpate the foreign body was an uncommon finding in dogs in either group. The increased prevalence of abdominal pain in dogs with LFB may have been related to the increased occurrence of intestinal necrosis, perforation and peritonitis in that study population.

In this study, survey radiographs were adequate to confirm a diagnosis of gastrointestinal foreign body in the majority of cases (LFB 62.5%, NLFB 70.1%). While radiographs may fail to demonstrate radiolucent foreign bodies, they can reveal segmental dilation of intestines with fluid and or gas or disparate bowel populations; however, this is not pathognomonic for foreign body obstruction (Clark 1968, Root & Lord 1971, Gibbs & Pearson 1973, Graham et al. 1998, McNeel & Riedesel 1998, Tyrell & Beck 2006, Sharma et al. 2011, Ciasca et al. 2013). It is important to note that this study was not designed to assess the accuracy of radiography for detection of gastrointestinal foreign bodies, as all dogs included in this study had confirmed foreign bodies. When survey radiographs alone were not diagnostic, either repeated radiographs or other diagnostic modalities, such as barium contrast studies or abdominal ultrasonography were used to confirm the diagnosis. Of the 483 dogs that had survey radiographs, repeat survey radiographs, or barium contrast studies, an abdominal ultrasound examination was still required to confirm the diagnosis of foreign body in 23% of the cases.

Hypochloraemia, metabolic alkalosis, hypokalaemia and hyponatraemia have been reported in dogs with various gastrointestinal foreign bodies, with LFB being more likely associated with hyponatraemia (Boag et al. 2005). Similarly, the present study demonstrated that dogs with LFB were more likely to have lower sodium, potassium and chloride concentrations, but also demonstrated haemoconcentration, an increase in blood urea nitrogen concentrations, and higher pH than dogs with NLFB. While these findings were statistically different between dogs with LFB and NLFB, these clinicopathological parameters are unlikely to be useful in distinguishing LFB from NLFB in individual cases, as many values remained within their respective reference intervals. There were no statistical differences between the two groups of dogs when comparing admission values for white blood cell count, serum lactate, glucose, albumin or creatinine concentrations and alanine aminotransferase activity.

Dogs with LFB required significantly more gastrotomies, enterotomies, and intestinal resection and anastomosis, than dogs with NLFB. This finding is consistent with the linear nature of the foreign body and the difficulty in their removing through one gastrointestinal incision. A high frequency of gastrotomy in dogs with LFB is expected as the majority of cases had linear material anchored in the stomach, which is similar to other studies (Evans et al. 1994, Hayes 2009). In contrast, 33 of 52 (63.5%) of dogs with NLFBs were able to have the material removed by endoscopy, while only 1 dog had a LFB removed by endoscopy. This is likely because the linear material was anchored further along the gastrointestinal tract. This study had one successful case of endoscopic removal of a LFB (a needle and thread from the stomach into the duodenum); however, this is unique and the current authors do not advocate endoscopy as a means to remove LFBs. Surgical times were nearly 30 minutes longer in the LFB group, likely related to the greater complexity of the surgical procedures. Similar findings were reported in a study comparing large and small bowel surgery, where increased gastrointestinal surgical time did not adversely affect mortality (Wylie & Hosgood 1994).

Reported survival rates of dogs undergoing surgery of the gastrointestinal tract vary from 80 to 99% (Evans et al. 1994, Boag et al. 2005, Shales et al. 2005, Hayes 2009), with the present study results being 96% for both groups. However, it should be noted that the current study was

unable to provide follow-up past the time of discharge for all patients, and it is therefore difficult to directly compare survival rates with other studies. Overall, it appears that surgical management is more complex, costly and time consuming in dogs with LFBs, and variable rates of dehiscence and septic peritonitis are reported (Allen et al. 1992, Wylie & Hosgood 1994, Ralphs et al. 2003, Shales et al. 2005). Despite these findings, treatment for a linear foreign body was not associated with a worse outcome in the present study, as it was in previous studies that investigated LFBs or one that compared both groups (Evans et al. 1994, Hayes 2009). Reasons for improved outcome in the dogs with LFB in this study may be the result of the improved resources at a specialty centre. This study occurred in a large specialty care facility, where there are many practitioners of varying experience. This may cause variations in surgical technique, efficiency in surgery and postoperative care. For this reason it was hard to objectively compare cases. The retrospective nature of the study made it difficult to standardise results to fit a specific category, as the information retrieved was based on the documentation of the supervising clinician. Additionally, even though several steps were taken to ensure all dogs meeting the inclusion criteria were enrolled in the study, it is not possible to determine if any records were omitted. This is particularly true of septic peritonitis, which has been associated with an unfavourable prognosis. The current search criteria may have failed to identify dogs presenting with septic peritonitis, whereby inclusion of such cases may have resulted in fewer surviving to discharge. Finally, any complication occurring past time of discharge may not be known if the clients elected follow-up care through their first opinion practice. This is particularly important when considering the high success rate in the current study, as some dogs could have had intestinal dehiscence and septic peritonitis, without the authors' knowledge, if follow-up was performed elsewhere. Despite a higher frequency of clinical signs, intestinal necrosis, perforation, peritonitis, surgical procedures, longer hospitalisation and a greater cost in dogs with LFB, the overall short-term outcome was excellent for both groups.

## Acknowledgements

The authors thank Dr. Peter Chapman for his and guidance in manuscript preparation, Kathleen Carr for graphics and Dr. Gary Leonardson for statistical assistance.

## Conflict of interest

The authors have no conflicts to declare.

## References

- Allen, D. A., Smeak, D. D. & Schertel, E. R. (1992) Prevalence of small intestinal dehiscence and associated clinical factors: a retrospective study of 121 dogs. *Journal of the American Animal Hospital Association* 28, 70-76
- Aronson, L. R., Brockman, D. J. & Brown, D. C. (2000) Gastrointestinal emergencies. *The Veterinary Clinics of North America: Small Animal Practice* 30, 555-579
- Boag, A. K., Coe, R. J., Martinez, T. A., et al. (2005) Acid-Base and electrolyte abnormalities in dogs with gastrointestinal foreign bodies. *Journal of Veterinary Internal Medicine* 19, 816-821
- Brown, D. C. (2012) Small intestine. In: *Veterinary Surgery: Small Animal*. Eds K. Tobias and S. Johnson. Saunders, Philadelphia, PA, USA. pp 1533-1536
- Capak, D., Brkic, A., Harapin, I., et al. (2001) Treatment of the foreign body induced occlusive ileus in dogs. *Veterinarski Arhiv* 71, 345-359
- Ciasca, T. C., David, F. H. & Lamb, C. R. (2013) Does measurement of small intestinal diameter increase diagnostic accuracy of radiography in dogs with suspected intestinal obstruction? *Veterinary Radiology and Ultrasound* 54, 207-211
- Clark, W. T. (1968) Foreign bodies in the small intestine of the dog. *Veterinary Record* 83, 115-119
- Evans, K. L., Smeak, D. D. & Biller, D. S. (1994) Gastrointestinal linear foreign bodies in 32 dogs: a retrospective evaluation and feline comparison. *Journal of the American Animal Hospital Association* 30, 445-450
- Gianella, P., Pfammatter, N. S. & Burgener I. A. (2009) Oesophageal and gastric endoscopic foreign body removal: complications and follow-up of 102 dogs. *Journal of Small Animal Practice* 50, 649-654
- Gibbs, C. & Pearson, H. (1973) The radiographical diagnosis of gastrointestinal obstruction in the dog. *Journal of Small Animal Practice* 14, 61-82
- Graham, J. P., Lord, P. F. & Harrison, J. M. (1998) Quantitative estimation of intestinal dilation as a predictor of obstruction in the dog. *Journal of Small Animal Practice* 39, 521-524
- Hayes, G. (2009) Gastrointestinal foreign bodies in dogs and cats: a retrospective study of 208 cases. *Journal of Small Animal Practice* 50, 576-583.

- McNeel, S. V. & Riedesel, E. A. (1998) Small bowel. In: *Textbook of Veterinary Diagnostic Radiology*. 4th edn. Eds D. E. Thrall. Saunders, Philadelphia, PA, USA. pp 540-556
- Mishra, N. K., Appert, H. E. & Howard, J. M. (1974) The effects of distention and obstruction on the accumulation of fluid in the lumen of small bowel of dogs. *Annals of Surgery* 180, 791-795
- Ralphs, S. C., Jessen, C. R. & Lipowitz, A. J. (2003) Risk factors for leakage following intestinal anastomosis in dogs and cats: 115 cases (1991-2000). *Journal of the American Veterinary Medical Association* 223, 73-77
- Root, C. R. & Lord, P. F. (1971) Linear radiolucent gastrointestinal foreign bodies in cats and dogs: Their radiographic appearance. *Journal of American Veterinary Radiology Society* 12, 45-53
- Shales, C. J., Warren, J., Anderson, D. M., et al. (2005) Complications following full thickness small intestinal biopsy in 66 dogs: a retrospective study. *Journal of Small Animal Practice* 46, 317-321
- Sharma, A., Thompson, M. S., Scrivani, N. L., et al. (2011) Comparison of radiography and ultrasonography for diagnosing small-intestinal mechanical obstruction in vomiting dogs. *Veterinary Radiology and Ultrasound* 52, 248-255
- Tidwell, A. S., Pennick, D. G. (1992) Ultrasonography of gastrointestinal foreign bodies. *Veterinary Radiology and Ultrasound* 33, 160-169
- Tyrell, D. & Beck, C. (2006) Survey of the use of radiology vs. ultrasonography in the investigation of gastrointestinal foreign bodies in small animals. *Veterinary Radiology and Ultrasound* 47, 404-408
- Wylie, K. B. & Hosgood, G. (1994) Mortality and morbidity of small and large intestinal surgery in dogs and cats: 74 cases (1980-1992). *Journal of the American Animal Hospital Association* 30, 469-474



## Reprint paper\*

# Septic peritonitis from pyloric and non-pyloric gastrointestinal perforation: prognostic factors in 44 dogs and 11 cats

Thomas Dayer<sup>1</sup>, Judith Howard and David Spreng

## SUMMARY

**Objectives:** To identify potential prognostic factors affecting outcome in septic peritonitis caused by gastrointestinal perforation in dogs and cats.

**Methods:** A retrospective study. Animals operated on for septic peritonitis because of gastrointestinal perforation were evaluated. Risk factors assessed included age, duration of clinical signs, recent prior abdominal surgery, recent prior anti-inflammatory drug administration, placement of a closed-suction drain and location of perforation.

**Results:** Fifty-five animals (44 dogs and 11 cats) were included. The overall mortality was 63.6%. No association was found between age, duration of clinical signs or prior abdominal surgery and outcome. Animals with a history of prior anti-inflammatory drugs were significantly ( $P=0.0011$ ) more likely to have perforation of the pylorus (73.3%). No significant difference in outcome was found between animals treated with closed-suction drains and those treated with primary closure or between pyloric perforation and perforation at other gastrointestinal sites.

**Clinical Significance:** Administration of anti-inflammatory drugs in dogs and cats is a significant risk factor for pyloric perforation. Pyloric perforation was not associated with a poorer outcome than perforation at other gastrointestinal sites. Placement of a closed suction drain did not improve outcome compared to primary closure.

\*This paper originally appeared in the *Journal of Small Animal Practice* (2013) 54, 625–629  
DOI: 10.1111/jsap.12151  
*Eur J Comp An Pract* (Autumn 2016) 26(3); p44-p50. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

## Introduction

Generalised peritonitis is a medical and surgical emergency, which often requires intensive and costly treatment. Both septic and non-septic causes of peritonitis are

recognised, the former generally being characterised by inflammation of the peritoneum secondary to bacterial contamination and infection. The most frequent cause of septic peritonitis in small animals is gastrointestinal (GI) leakage causing intraperitoneal infection by commensal intestinal bacteria. Mortality in dogs and cats with septic peritonitis is reported with wide variation between 20 and 80% (Woolfson & Dulisch 1986, Hosgood & Salisbury 1988, Allen et al. 1992, King 1994, Swann & Hughes 2000, Lanz et al. 2001, Mueller et al. 2001, Staatz et al. 2002, Bonczynski et al. 2003, Levin et al. 2004, Shales et al. 2005). Rapid diagnosis, perioperative treatment to stabilise hypovolaemia, acid-base and electrolyte

<sup>1</sup> Department of Small Animal Surgery, Vetsuisse Faculty, University of Bern, Bern, Switzerland



disturbances, as well as surgical correction of the source of peritoneal contamination are key to successful outcome.

Factors reported to influence outcome in a negative manner include preoperative activities of serum alanine aminotransferase (ALT) and  $\gamma$ -glutamyl transferase (GGT), hypotension that is not correctable with treatment by intravenous (iv) fluids, development of respiratory dysfunction, development of disseminated intravascular coagulation (King 1994, Winkler & Greenfield 2000, Grimes et al. 2011) and development of multiple organ dysfunction syndrome (defined as dysfunction of at least two organ systems) (Kenney et al. 2010). The location of GI perforation has thus far not been found to influence prognosis. Indeed, previous studies investigating outcome following perforation of the colon compared to small bowel perforation found no difference in mortality despite a potentially greater degree of bacterial contamination in the former (Christou et al. 1993, Wylie & Hosgood 1994, Mueller et al. 2001). Perforation of the pylorus is technically more challenging to repair and may therefore carry a higher risk of complication, but to the authors' knowledge, no previous study has investigated whether pyloric perforation carries a worse prognosis than perforation at other GI sites. In addition, some reports suggest an association between the administration of non-steroidal anti-inflammatory drugs (NSAIDs) and pyloric perforation (Stanton & Bright 1989, Hinton et al. 2002), including studies based on pharmacovigilance data, toxicity studies and case series of dogs treated with NSAIDs or dogs presenting with gastric ulceration (Stanton & Bright 1989, Poortinga & Hungerford 1998, Lascelles et al. 2005, Case et al. 2010). However, the frequency with which NSAID or steroidal anti-inflammatory drug administration is associated with pyloric and other GI-site perforation is largely unknown. Finally, some studies have investigated outcome in dogs with open peritoneal drainage, closed suction drains or with primary abdominal closure following surgical correction of GI perforation and septic peritonitis (Lanz et al. 2001, Mueller et al. 2001, Staatz et al. 2002, Szabo et al. 2011), but few studies have compared outcome between treatment groups and, to the authors' knowledge, no study has directly compared outcome between primary closure and closed suction drains.

The main aim of this study was to identify the potential association between the site of GI perforation and outcome by retrospectively examining cases treated over a 9-year period. It was hypothesised that the location of

GI perforation is a significant risk factor and that pyloric perforation carries a worse prognosis than perforation at other sites. The secondary aims of the study were to assess the association between a history of steroid or NSAID administration and pyloric perforation as well as the placement of closed-suction drains and outcome.

## Materials and methods

The medical records of all cats and dogs that were treated surgically in Vetsuisse Faculty veterinary teaching hospital for septic peritonitis resulting from GI perforation between January 2002 and June 2011 were reviewed. Animals were admitted on an emergency basis both as primary care cases and by referral from private veterinarians during daytime and out-of-hours services.

Cases were included if a complete surgical record was available. The septic character of peritonitis was based on intraoperative findings and, in some cases, preoperative cytology of abdominal fluid. The surgical team was composed of a board-certified or third-year resident surgeon and an assistant (resident, intern or student). All cases of perforation of the pylorus were treated with primary closure of the perforation by pyloroplasty. All cases of small or large bowel perforation were treated with enterectomy and end-to-end anastomosis. All cases of gastric perforation, other than perforation of the pylorus, were treated with partial gastrectomy and primary closure. In general, postoperative treatments included iv fluids, broad-spectrum antibiotics and opiate-derived analgesics. The choice of anaesthetic protocol and perioperative medications and supportive care were at the discretion of the surgeon and anaesthesia team. The decision to place an intraoperative closed-suction drain was at the discretion of the surgeon.

Final outcome was considered good if the animal survived until hospital discharge and poor if the animal died or was euthanased before discharge. Euthanasia was only performed at the request of the owners, based on both financial and prognostic considerations, following recommendations and advice from the surgeon.

Data collected included the age, breed, gender and bodyweight of the animal, the duration of clinical signs before surgery, a history of recent abdominal surgery, recent prior administration of anti-inflammatory drugs

(steroids or NSAIDs), intraoperative placement of a closed-suction intraperitoneal drain (Jackson-Pratt type), survival 24 hours after surgery and survival to hospital discharge (outcome). For statistical analyses, the location of GI perforation was categorised as pylorus (not including antrum and proximal duodenum) or other GI site.

## Statistical analyses

Continuous variables (age at presentation, duration of clinical signs) were evaluated for far outliers using Grubbs' double-sided (alpha-level 0.05) and Tukey (1977) tests and summary statistics were performed. A single far outlier was excluded for duration of clinical signs. Evaluation of a difference between survivors and non-survivors for mean age and for mean duration of clinical signs was performed using independent samples t tests (duration of clinical signs was log transformed to achieve normality). Evaluation of an association between perforation site and survival, recent prior treatment with anti-inflammatory drugs and perforation site, recent prior treatment with anti-inflammatory drugs and survival, recent prior abdominal surgery and survival, and between placement of an intraperitoneal drain and survival was performed using Fisher's exact test. All statistical analyses were performed using commercial software (MedCalc version 12.4, Medcalc Software bvba). The level of significance was set at  $P < 0.05$ .

## Results

A total of 72 animals underwent laparotomy for peritonitis during the study period. Of these, 55 of 72 (76%) had septic peritonitis resulting from GI perforation and were included in the study. The animals included 11 cats (8 domestic shorthair, 1 Persian, 1 Siamese and 1 Burmese) and 44 dogs (3 mixed-breed dogs and 41 dogs representing a total of 27 different pure breeds). Cats weighed a median of 4.3 (range: 2.1 to 6.5) kg and dogs weighed a median of 24.8 (range: 1.2 to 70) kg. The cats included five males (four neutered) and six females (five neutered). The dogs included 29 males (13 neutered) and 15 females (eight neutered). The mean age of animals at presentation was  $5.9 \pm 3.7$  (median: 5.5, range: 0.5 to 16.6) years. The mean duration of hospitalisation was 4.2 (median: 3 days, range: 0.5 to 20) days. The overall mortality was 35 of 55 (63.6%).

There was no significant difference in mean age between animals that survived 24 hours postoperatively and those that did not or between animals that survived to hospital

discharge and those that did not.

Gastrointestinal perforation was found in the pylorus in 21 of 55 (38%) cases, in other areas of the stomach in 6 of 55 (11%) cases, and in the intestine in 28 of 55 (51%) cases (24 in the small intestine and 4 in the colon). Only one case had two perforations (both in the small intestine). No statistical association was found between the location of perforation and survival although more animals with perforation of the pylorus (15 of 21, 71.4%) died than did those with perforation at other sites (20 of 34, 58.8%) (Fig 1). Of the 15 animals that died following pyloric perforation, 5 were euthanased intraoperatively and 10 were euthanased postoperatively. Of the 20 animals that died following perforation at other GI sites, 6 were euthanased intraoperatively, 4 died postoperatively and 10 were euthanased postoperatively.

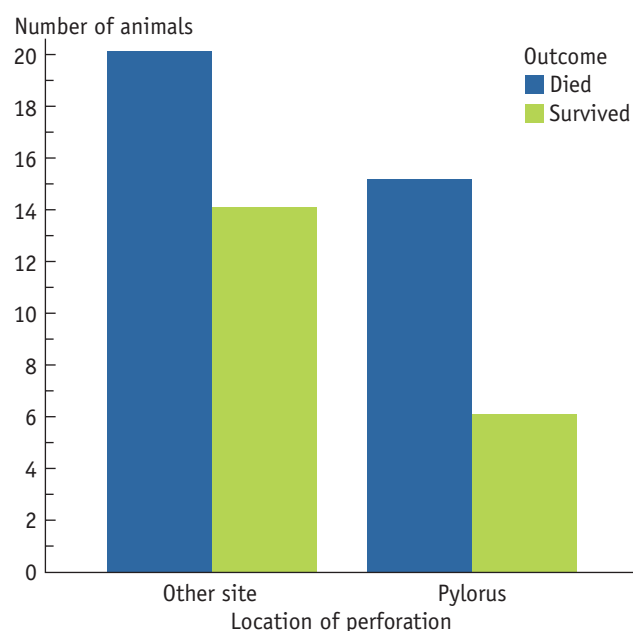


FIG 1. Frequency bar chart showing the location of gastrointestinal perforation and outcome in 55 dogs and cats

There was no significant association between duration of clinical signs before surgery and outcome or between the duration of clinical signs and survival at 24 hours postoperatively.

Nineteen animals had a recent prior abdominal surgery. There was no significant association between prior abdominal surgery and outcome or survival at 24 hours.

A total of 15 animals had a recent history of steroid or NSAID administration. In four cases, information regarding preoperative drug administration was unavailable.

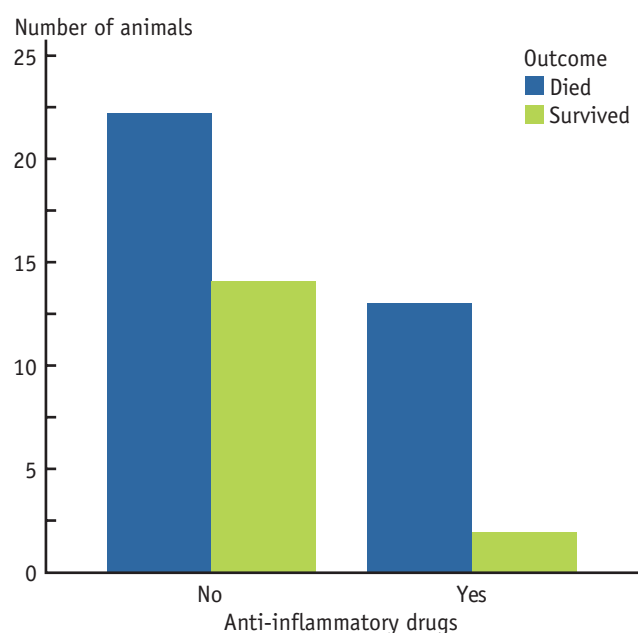


FIG 2. Frequency bar chart showing a prior history of anti-inflammatory drug administration and outcome in 55 dogs and cats

Although no significant association was found between prior drug history and outcome or survival at 24 hours (Fig 2), only 2 of 15 (13.3%) animals with a history of anti-inflammatory drugs survived compared to 14 of 36 (38.8%) animals not receiving anti-inflammatory drugs.

Prior treatment with anti-inflammatory drugs was, however, significantly ( $P=0.0011$ ) associated with perforation of the pylorus (11 of 19 (57.9%)) compared to perforation at other GI sites (4 of 32 (12.5%)) (Fig 3).

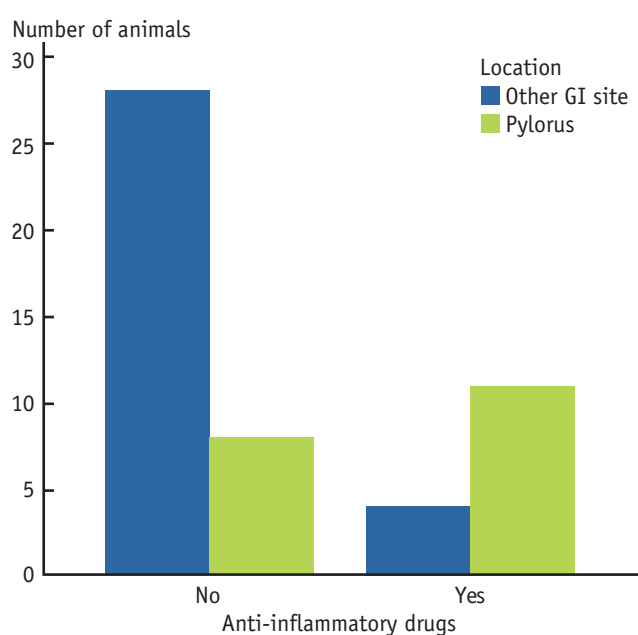


FIG 3. Frequency bar chart showing a prior history of anti-inflammatory drug administration and location of gastrointestinal perforation in 55 dogs and cats

Of the 35 non-survivors, 21 of 35 (60%) died or were euthanased within 24 hours of surgery. Of the 34 animals that survived the first 24 hours after surgery, 24 of 34 (70.6%) had a closed-suction drain placed during surgery. The mortality was 41.7% (10 of 24) for the animals treated with a close suction drain and 40% (4 of 10) for animals treated with primary closure. There was no significant association between presence of a drain and survival to hospital discharge.

## Discussion

The wide range in mortality from 20 to 80% reported in previous publications on septic peritonitis resulting from GI perforation in small animals is likely due in part to the diversity of clinical cases and differences in intra- and perioperative procedures and care (Woolfson & Dulisch 1986, Hosgood & Salisbury 1988, Allen et al. 1992, King 1994, Swann & Hughes 2000, Lanz et al. 2001, Mueller et al. 2001, Staatz et al. 2002, Bonczynski et al. 2003, Levin et al. 2004, Shales et al. 2005). These differences also render direct comparison between results of this study and previous studies difficult. Moreover, due to the retrospective nature of this and many previous reports, a large number of confounding factors that may influence findings were undoubtedly present. These include differences in surgeon skill, pre- and postoperative supportive care and treatment, and concurrent illness affecting outcome, age and concurrent illness affecting prior treatment with anti-inflammatory medications, and surgeon identity, the animal's general clinical condition and intraoperative findings affecting the decision to place intraoperative drains. Results of this study must be interpreted with these limitations in mind and prospective studies are necessary to confirm the present findings.

Nevertheless, the mortality of 63.6% observed in this study lies within the range previously reported. However, this study included a relatively large number of cases with gastric and particularly pyloric perforation, which is rarely reported in previous studies except those investigating NSAID administration (Enberg et al. 2006). Similar to findings in other studies, the duration of clinical signs, prior surgery and age were not found to be associated with outcome in this study (Winkler & Greenfield 2000, Smeltoys et al. 2004, Eisele et al. 2010). However, only those animals treated surgically were included. It may therefore be argued that older animals or those subjected

to recent previous surgeries are more likely to die or be euthanased before surgery.

Despite the high mortality in animals with pyloric perforation (71.4%) observed in this study, this was not found to be significantly different to mortality in animals with perforation at other GI sites, refuting the initial hypothesis that the technically more challenging surgery may impact on outcome. In a previous study of 16 dogs and cats with gastroduodenal perforation that were treated with a variety of techniques (open peritoneal drainage, primary ulcer closure, resection and anastomosis), mortality was found to be 56% (Hinton et al. 2002). However, direct comparison with this study is difficult as all cases included in this study were treated with pyloroplasty. Whether surgical technique may play a role in outcome cannot therefore be assessed. Moreover, analyses on larger numbers of cases may result in statistical significance where none was found in this study.

Changes in perioperative care, surgeon identity and surgical technique may have changed in the study period from 2002 and 2011, affecting outcome in animals in this study. However, a previous study reported no significant difference in survival among dogs treated surgically for septic peritonitis between 1988 and 1993 (21 of 33, 64%) and 1999 to 2003 (29 of 51, 57%) (Bentley et al. 2007). As differences due to progress in surgical and intensive care are likely greater in that study than during the current study period, it is unlikely that any such difference greatly affected survival in animals in this study.

Common predisposing factors reported for gastroduodenal ulcers include NSAID or corticosteroid administration, hepatic disease, major surgery, periods of high stress, shock, renal disease, other causes of decreased gastric circulation, gastric hyperacidity, GI neoplasia and idiopathic inflammatory bowel disease (Jarfens et al. 1992, Sullivan & Yool 1998, Simpson 2010). The concern regarding gastroduodenal ulceration and subsequent gastroduodenal perforation following NSAID or corticosteroid administration is based on pharmacovigilance data, toxicity studies or case series of dogs treated with NSAIDs or presenting with gastric ulceration (Stanton & Bright 1989, Poortinga & Hungerford 1998, Hinton et al. 2002, Liptak et al. 2002, Lascelles et al. 2005, Cariou et al. 2010, Case et al. 2010). Previous studies have shown that the pyloric region is most susceptible to ulceration due to NSAID administration

(Stanton & Bright 1989, Boston et al. 2003). In this study, prior treatment with anti-inflammatory drugs was significantly associated with perforation of the pylorus compared to other GI sites, corroborating previous findings (Stanton & Bright 1989). As data from the medical records was often incomplete regarding the details of the duration and dose of prior medications, and some animals had a prior history of both steroid and NSAID administration, any possible effect of type of drug, dose or duration of administration on GI perforation could not be analysed in this study.

Only a few studies investigating the type of drainage used in small animals treated with septic peritonitis have been published and the advantage of drains remains controversial. Previous reports include studies in dogs treated with open peritoneal lavage (Orsher & Rosin 1984, Woolfson & Dulisch 1986, Greenfield & Walshaw 1987), with primary closure and no drainage (Lanz et al. 2001), with active suction drains (Mueller et al. 2001) and comparison of outcome in dogs treated with drains and open peritoneal lavage (Staatz et al. 2002). However, no previous study has directly compared outcome between animals treated with drains and those treated with primary closure at the same institution over the same time period. In this study, the mortality of 41.7% in animals treated with drains was similar to 30% observed in a previous study (Mueller et al. 2001) and no difference in mortality was found between animals treated with or without drains (41.6 versus 40%) in this study, suggesting that no advantage in treatment is attained through the use of drains compared to primary closure. However, the decision to place drains was at the discretion of the surgeon and was likely influenced by the surgeon's perception of prognosis, the severity of peritonitis, and the general condition of the animal. The extent to which drainage improves outcome following correction of the leakage site therefore remains unclear and prospective studies are needed to make conclusions in this regard.

In conclusion, the results of this study corroborate previous findings of an association between the administration of anti-inflammatory drugs and pyloric perforation in dogs and cats. Pyloric perforation was not associated with a worse outcome compared to perforation at other GI sites, but further studies are required to confirm this finding. No difference in survival was found between animals treated with closed suction drains and those treated with primary abdominal closure and prospective studies are warranted to

establish the advantage, if any, of drains in animals with GI perforation after surgical correction of the leakage site.

## Conflict of interest

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

## References

- Allen, D. A., Smeak, D. D. & Schertel, E. R. (1992) Prevalence of small intestinal dehiscence and associated clinical factors: a retrospective study of 121 dogs. *Journal of the American Animal Hospital Association* 28, 69-76
- Bentley, A. M., Otto, C. M. & Shofer, F. S. (2007) Comparison of dogs with septic peritonitis: 1988-1993 versus 1999-2003. *Journal of Veterinary Emergency and Critical Care* 17, 391-398
- Bonczynski, J. J., Ludwig, L. L., Barton, L. J., et al. (2003) Comparison of peritoneal fluid and peripheral blood pH, bicarbonate, glucose, and lactate concentration as a diagnostic tool for septic peritonitis in dogs and cats. *Veterinary Surgery* 32, 161-166
- Boston, S. E., Moens, N. M., Kruth, S. T., et al. (2003) Endoscopic evaluation of the gastroduodenal mucosa to determine the safety of short-term concurrent administration of meloxicam and dexamethasone in healthy dogs. *American Journal of Veterinary Research* 64, 1369-1375
- Cariou, M. P. L., Haldacree, Z. J., Lee, K. C., et al. (2010) Successful surgical management of spontaneous gastric perforations in three cats. *Journal of Feline Medicine and Surgery* 12, 36-41
- Case, J. B., Fick, J. L. & Rooney, M. B. (2010) Proximal duodenal perforation in three dogs following deracoxib administration. *Journal of the American Animal Hospital Association* 46, 255-258
- Eisele, J., McClaran, J. K., Runge, J. J., et al. (2010) Evaluation of risk factors for morbidity and mortality after pylorotomy and gastroduodenostomy in dogs. *Veterinary Surgery* 39, 261-267
- Enberg, T. B., Braun, L. D. & Kuzma, A. B. (2006) Gastrointestinal perforation in five dogs associated with the administration of meloxicam. *Journal of Veterinary Emergency and Critical Care* 16, 34-43
- Greenfield, C. L. & Walshaw, R. (1987) Open peritoneal drainage for treatment of contaminated peritoneal cavity and septic peritonitis in dogs and cats: 24 cases (1980-1986). *Journal of the American Veterinary Medical Association* 191, 100-105
- Grimes, J. A., Schmiedt, C. W., Cornell, K. K., et al. (2011) Identification of risk factors for septic peritonitis and failure to survive following gastrointestinal surgery in dogs. *Journal of the American Veterinary Medical Association* 238, 486-494
- Hinton, L. E., McLoughlin, M. A., Johnson, S. E., et al. (2002) Spontaneous gastroduodenal perforation in 16 dogs and seven cats (1982-1999). *Journal of the American Animal Hospital Association* 38, 176-187
- Hosgood, G. & Salisbury, K. (1988) Generalized peritonitis in dogs: 50 cases (1975-1986). *Journal of the American Veterinary Association* 193, 1448-1450
- Jerfens, A. E., Moore, F. M., March, P., et al. (1992) Idiopathic inflammatory bowel disease associated with gastroduodenal ulceration-erosion: a report of nine cases in the dog and cat. *Journal of American Animal Hospital Association* 28, 21-26
- Kenney, E. M., Rozanski, E. A., Rush, J. E., et al. (2010) Association between outcome and organ system dysfunction in dogs with sepsis: 114 cases (2003-2007). *Journal of the American Veterinary Medical Association* 236, 83-87
- King, L. G. (1994): Postoperative complications and prognostic indicators in dogs with septic peritonitis: 23 cases (1989-1992). *Journal of the American Veterinary Medical Association* 204, 407-414
- Lanz, O. I., Ellison, G. W., Bellah, J. R., et al. (2001) Surgical treatment of septic peritonitis without abdominal drainage in 28 dogs. *Journal of the American Animal Hospital Association* 37, 87-92
- Lascelles, B. D., Blikslager, A. T., Fox, S. M., et al. (2005) Gastrointestinal tract perforation in dogs treated with selective cyclooxygenase-2 inhibitor: 29 cases (2002-2003). *Journal of the American Veterinary Medical Association* 227, 1112-1117
- Levin, G. M., Bonczynski, J. J., Barton, L. J., et al. (2004) Lactate as a diagnostic test for septic peritoneal effusions in dogs and cats. *Journal of the American Animal Hospital Association* 40, 364-371
- Liptak, J. M., Hunt, G. B., Barrs, V. R., et al. (2002) Gastroduodenal ulceration in cats: eight cases and a review of the literature. *Journal of Feline Medicine and Surgery* 4, 27-42



- Mueller, M. G., Ludwig, L. L. & Barton, L. J. (2001) Use of closed-suction drains to treat generalized peritonitis in dogs and cats: 40 cases (1997-1999). *Journal of the American Veterinary Medical Association* 219, 719-724
- Orsher, R. J. & Rosin, E. (1984) Open peritoneal drainage in experimental peritonitis in dogs. *Veterinary Surgery* 13, 222-226
- Poortinga, E. W. & Hungerford, L. L. (1998) A case-control study of acute ibuprofen toxicity in dogs. *Preventive Veterinary Medicine* 35, 115-124
- Shales, C. J., Warren, J., Anderson, D. M., et al. (2005) Complications following full-thickness small intestinal biopsy in 66 dogs: a retrospective study. *Journal of Small Animal Practice* 46, 317-321
- Simpson, K. W. (2010) Diseases of the stomach. In: Textbook of Veterinary Internal Medicine. 7th edn. Eds S. J. Ettinger and E. C. Feldman. WB Saunders, Philadelphia, PA, USA. pp 1504-1526
- Staatz, A. J., Monnet, E. & Seim, H.B. (2003) Open peritoneal drainage versus primary closure for the treatment of septic peritonitis in dogs and cats: 42 cases (1993-1999). *Veterinary Surgery* 31, 174-180
- Stanton, M. E. & Bright, R. M. (1989) Gastroduodenal ulceration in dogs. Retrospective study of 43 cases and literature review. *Journal of Veterinary Internal Medicine* 3, 238-244
- Sullivan, M. & Yool, D. A. (1998) Gastric disease in the dog and cat. *Veterinary Journal* 156, 91-106
- Swann, H. & Hughes, D. (2000) Diagnosis and management of peritonitis. *The Veterinary Clinics of North America: Small Animal Practice* 30, 603-615
- Szabo, S. D., Kieri, J., Neel, J., et al. (2011) Evaluation of postceliotomy peritoneal drain fluid volume, cytology, and blood-to-peritoneal drain fluid lactate and glucose differences in normal dogs. *Veterinary Surgery* 40, 444-449
- Winkler, K. P. & Greenfield, C. L. (2000) Potential prognostic indicators in diffuse peritonitis treated with open peritoneal drainage in canine patient. *Journal of Veterinary Emergency and Critical Care* 10, 259-265
- Woolfson, J. M. & Dulisch M. L. (1986) Open abdominal drainage in the treatment of generalized peritonitis in 25 dogs and cats. *Veterinary Surgery* 15, 27-32
- Wylie, K. B. & Hosgood, G. (1994) Mortality and morbidity of small and large intestinal surgery in dogs and cats: 74 cases (1980-1992). *Journal of the American Animal Hospital Association* 74, 677-692



## Commissioned paper\*

# Nursing the critical care patient – part 1: triage

Katherine Howie<sup>1</sup>

## SUMMARY

Good nursing of the emergency and critical care patient is essential. From triage to assessment, fluid therapy and monitoring, the veterinary nurse has a vital role to play. From telephone triage checklists, initial and follow-up assessment of the cardiovascular, respiratory and central nervous system to patient comfort, this paper gives an overview how the veterinary nurse can assist in the care for the emergency and critical care patient.

\*This paper was commissioned by FECAVA for the *Eur J Comp An Pract* (Autumn 2016) 26(3); p51-p55. Go to <http://www.ejcap.org> for the interactive online presentation of this paper

## Triage of the emergency patient

Triage is essential in any veterinary environment. It allows veterinary surgeons and veterinary nurses to identify those patients need immediate interventions and those whose clinical status will not be compromised by waiting a short period of time.

## Telephone Triage

In many cases, the first point of contact for an emergency patient is the client calling the practice and speaking to either a receptionist or veterinary nurse. Appropriate telephone triage is a skill that is relevant to every member of the practice team.

There are several compromising factors that affect the ability to triage a patient effectively over the telephone and the following things should be considered:

- Clients are likely to be upset and distressed
- They may not be able to relay important information accurately
- The veterinary nurse cannot “see” what the animal is actually doing, which makes it very difficult to say with certainty if it is an emergency case or not
- Clients may have different perceptions of what is an emergency case

It is therefore sensible to have a checklist of questions for clients when they contact the clinic. This ensures gathering all relevant information to prepare for the clients arrival (e.g. oxygen for a dyspnoeic patient), allows effective prioritisation and ensures the veterinary team is aware that an emergency is on its way.

Questions to ask should include the following:

- A contact telephone number and the client's name in case the phone gets cut off
- The pet's name, age, breed and other pertinent details
- A brief overview of what the client perceives to be the problem and how the patient is behaving at that time, focusing on the 3 major body systems
- Does the client have transport?
- Estimated time of arrival

There are several examples of true emergency patients, which all veterinary nurses should be aware of and in these cases it is imperative that the patients are brought in as soon as possible. The veterinary surgeon on duty should also be made aware of all telephone calls concerning any patients...

- having breathing difficulties
- involved in road traffic accidents (confirmed or suspected)
- showing signs of difficulty urinating
- that are bleeding
- that have collapsed
- in status epilepticus / seizing
- with known or suspected intoxications

<sup>1</sup> Katherine Howie RVN VTS(ECC), Principal Nurse Manager, Vets-Now Farnham, 51 Hale Road, Farnham, Surrey GU9 9RB, UK. Katherine.Howie@vets-now.com

- non-productively vomiting/ retching with or without abdominal swelling that may indicate a gastric dilation volvulus/ acute abdomen
- whelping and straining continuously for longer than 30 minutes or that have developed a green discharge

Please note that this is not an exhaustive list.

Once triage has been carried out over the telephone, offer advice on safe transportation of their pet to the surgery. Animals that have been in a traumatic incident or are in pain may become aggressive and clients may be concerned about moving them. Simple advice such as how to make a tape muzzle or how to use a stretcher to get the patient into the car and gentle reassurance may need to be given.

### Carrying out a triage assessment

A very basic triage assessment should be able to be carried out within 90 seconds, focusing upon the three major body systems: the respiratory system, cardiovascular system and central nervous system.

The veterinary nurse may be required to carry out a triage examination upon arrival of the patient arriving in the clinic if the veterinary surgeon is not (yet) available.

#### Initial triage:

Is the patient breathing? Is there dyspnoea present, tachypnoea, orthopnoea or an increased respiratory effort or noise?

Does the patient have a heart beat? Can a femoral pulse and a peripheral pulse be felt?

Is the patient conscious?

If so, is the level of consciousness normal, obtunded (reduced alertness), stuporous (greatly reduced sensibility or consciousness), depressed or altered in any other way?

This very rapid primary survey will help determine which patients require immediate intervention, which patients can wait a very short period of time or even longer. Triage should focus in particular on conditions such as hypovolaemia, hypoxia, hypotension which can cause rapid decline and death. Although other injuries such as fractures may be present, these are generally not

immediately life threatening and would be a secondary concern.

## Secondary Assessment

Once established that the patient has a patent airway, functioning circulation and is or is not neurologically altered, a second assessment can take place, this time recording parameters and obtaining further information about the patient's status. It is important to record parameters accurately, particularly in the emergency patient whose status can change very rapidly. Although veterinary nurses will not make a diagnosis, having a good knowledge of what is normal compared to what is abnormal will allow alerting the veterinary surgeon to potentially life-threatening conditions.

### Assessment of the cardiovascular, respiratory and central nervous system

A systematic assessment of the major body systems will give essential information on the interventions that need to be carried out. It does not necessarily require a lot of equipment to carry out major body system assessments; observations, palpation and auscultation are just as important.

### Cardiovascular System

The cardiovascular system is comprised of the cardiac muscle, venous system and arterial systems. They all have different roles and any change to the circulating volume in a patient can lead to serious detrimental effects on our patients.

The results of reduced cardiac output or decreased circulatory function leading to poor tissue perfusion can be assessed using perfusion parameters.

Assessment of the cardiovascular system gives us essential information about the status of a patient, the degree of compromise and how aggressively the veterinary surgeon needs to be with their interventions. There are six main perfusion parameters that should be assessed and recorded for the presenting emergency patient.

- Mucous membrane colour
- Capillary refill time
- Heart rate (auscultated)
- Pulse quality and synchrony with the heart rate
- Temperature of the extremities – in hypovolaemic patients or those with poor cardiac output, extremities

Table 1. The six main perfusion parameters allowing assessment of the cardiovascular system in emergency patients.

Parameters	Normal values		Abnormal values	
	Dogs	Cats	Dogs	Cats
Mucous membrane colour	Pink	Pink	Pale, Cyanotic, Red, Grey	Pale, Grey, Cyanotic
Capillary refill time	1-2 seconds	1-2 seconds	<1s or >2s	<1s or >2s
Heart rate	80-140 bpm	150-200 bpm	< 60bpm or > 140bpm	< 140bpm or > 200bpm
Pulse quality	strong and regular	strong and regular	Bounding, snappy, weak, absent	Bounding, snappy, weak, absent
Temperature of extremities	warm to touch	warm to touch	Noticeably cold or hot	Noticeably cold or hot
Rectal temperature	38.3-38.7 °C	38.1 -38.7 °C	< 37°C or > 39.5 °C	< 37°C or > 39.5 °C

will be significantly colder than expected due to vasoconstriction

- Rectal temperature – This should be recorded last, as some patients may be less tolerant of this, leading to a false increase in heart rate

All initial parameters should be recorded accurately on a hospitalisation chart including the initials of the person taking these parameters and the time they have been recorded.

Any prolonged reduction in cardiac output or compromise of the cardiovascular system will lead to starvation of cells and tissues of oxygen and nutrition with the potential for the major body systems to begin to fail as the patient decompensates.

### “Shock”

Although this term is often used as a diagnostic term in veterinary practice, it is not. It is an combination of clinical signs causing a patient becoming compromised

either by loss of circulating volume, trauma, serious compromise of a body system or any condition leading to poor oxygenation or cardiac output. Some of the typical signs seen in a shock patient include:

- Tachycardia (bradycardia often occurs in cats)
- Tachypnoea
- Pale mucous membranes
- Cold extremities
- Poor peripheral pulses

### Respiratory System

Disruption in any part of the respiratory system, may lead to respiratory distress, abnormal breathing patterns or occasionally respiratory arrest. Note that patients in pain may also often have an increased respiratory rate and may show signs of dyspnoea.

Animals that are truly dyspnoeic are extremely fragile and a “hands-off “ approach in an oxygen-enriched environment and observation is required.

Table 2. The main parameters for the assessment of the respiratory system in emergency patients.

Parameter	Normal findings		Abnormal findings	
	Dogs	Cats	Dogs	Cats
Respiratory rate	20-40 bpm	20-40 bpm	> 40 bpm	> 40 bpm
Respiratory effort and noise	None	None	Increased, changed postures, abducted elbows, open mouth breathing, stertor, stridor, wheezes, crackles, cough, gagging, retching	Increased, changed postures, abducted elbows, open mouth breathing, wheezes, crackles, coughing, gagging, retching
Mucous membranes	Pink	Pink	Pale/grey/congested	Pale/grey/congested
Thoracic wall movement	Minimal	Minimal	May be overly exaggerated or absent.  Paradoxical abdominal breathing may occur	May be overly exaggerated or absent.  Paradoxical breathing may occur

Breathing is an unconscious activity and takes place regardless of the level of consciousness. It is controlled by respiratory centres in the brain which may be affected by increases in carbon dioxide and decreased oxygen levels in the blood. There should be minimal effort involved for the normal patient.

Any patient presenting with signs of respiratory dysfunction should immediately be placed in an oxygen-enriched environment while a veterinary surgeon is alerted. These critical patients decompensate rapidly and require immediate but stress-free interventions.

## Central Nervous System

An emergency patient's level of consciousness (LOC) can be extremely variable. It is therefore important to recognise what is normal. Many factors can affect a patient's LOC, including analgesics, fluid therapy, movement, pain, hypoxia, poor cardiac output and hypoglycaemia.

Some patients will naturally be more withdrawn in a different environment whereas others will show outward signs of stress and anxiety. These should all be recorded, as any change in a patient's behaviour in the emergency clinic can indicate either an improvement or a deterioration of its status.

Some patients with e.g. intoxications will be hyperaesthetic, hyperactive and over-stimulated until the intoxication has been resolved. It is an important part of the veterinary nurse's role to determine, with the help of the clients, the patient's normal demeanour in a clinic environment and with unknown people. This will help determine if the behaviour is normal or not and allow to develop nursing plans during hospitalisation.

Patients with head trauma in particular require extremely close monitoring and accurate record keeping – the use of the adapted Glasgow coma scale is recommended.

*Table 3. Main parameters for the assessment of the central nervous system in emergency patients.*

- Alertness and responsiveness to the environment, people and voice and touch
- Pupillary light reflex
- Pupil size (bilateral comparison)
- Gait and spinal reflexes

## Abnormal Findings

Levels of consciousness should be monitored extremely closely in any emergency patient from admission to resolution of the problem. Patients with disorders of the central nervous system can deteriorate rapidly and catastrophically.

### Mild intracranial lesions

Patients with mild intracranial lesions may show depressed or inappropriate mentation (although this can also be due to drugs or the patient's temperament) and a decreased pupillary light reflex. The pupil size is normal but the patient may show some form of paresis

### Moderate intracranial lesions

Patients with moderate intracranial lesions are stuporous, and will be less responsive to noxious stimuli such as pain. The pupillary light reflex is absent and pupils are miotic (pinpoint). Patients are recumbent and may show rigidity of the forelimbs.

### Severe intracranial lesions

These patients are usually comatose or unresponsive to painful stimuli. There is a loss of muscle tone (relaxed limbs). Pupils are dilated and the pupillary light reflex is absent.

## Seizure Activity

Often, patients presenting to an emergency clinic will show some kind of seizure activity.

These patients may already be diagnosed as epileptics or be newly presenting epileptics. Certain intoxications may cause the seizures, including hypoglycaemia and hypocalcaemia. It is important to be able to give the client the correct advice for managing seizure activity and safe transportation to the clinic, e.g.:

- Turn off the lights and any loud noises (television, radio)
- Move anything away from the dog/cat could cause harm
- Do NOT try and hold the dog's or cat's tongue or even place your hands anywhere near their mouth. Seizure activity is unconscious and these animals may bite
- Keep calm and record the time
- If the seizure lasts longer than 5 minutes or if the patient has a known intoxication then an emergency appointment is required
- If the patient is cluster-seizuring or is in status epilepticus and not recovering fully from the previous



seizure before going into the next one then they should be seen as an emergency

- Advise clients on safe transport of the seizing patient

Assessment of the three major body systems can give us essential information to ensure that patient's needs are met and to enable the veterinary surgeon to develop a suitable medical and care plan.

## **Acknowledgements**

This paper was written with the help of staff of Vets Now.