Sedation versus anaesthesia in sick patients

Matthew Gurney

I would recommend removing cost from the sedation versus anaesthesia argument by charging for your time rather than set anaesthetic fees. With a sick case, I may choose sedation, but with a similar level of monitoring and care to general anaesthesia. Ask yourself where the cost is incurred here? The answer is the time of professional colleagues and not the cost of volatile agent! Ensure owners understand this and appreciate the value to their pet.

I actively discourage the term 'light sedation' or 'light anaesthetic' as these terms are predominantly used to reassure clients, but carry a false perception. Let us be realistic and discuss risk with clients and illustrate this with numbers.

The Confidential Enquiry into Perioperative Small Animal Fatalities (CEPSAF) was conducted between 2002 and 2004 by Brodbelt and colleagues, published in 2008. The objective of this study was to estimate the risks of anaesthetic- and sedation-related mortality. Outcomes at 48 hours were recorded, with anaesthetic-/sedation-related death considered to have occurred where the pre-existing condition did not cause death. An expert panel classified cause of death. Approximately 98,000 dogs, 80,000 cats and 8000 rabbits were enrolled in the study (Figure 1).

Figures for mortality have improved since Hall and Clarke's study of 1990, but are still high compared to the risk of anaesthesia-related death in people which is

0.02–0.005%. Most veterinary postoperative deaths occur within 3 hours. For most of these the cause was classified as unknown.

Does sedation carry less risk than anaesthesia? This is confounded by health status so the reduced risk with sedation is not valid.

Assessment and stabilization of health status is far more likely to impact mortality than assuming that sedation is safer. It is often not the drugs *per se*, but rather how we use those drugs and how we monitor our patients.

KEY LEARNING OBJECTIVES

- Understand that health status is a better predictor of mortality than the choice between anaesthesia or sedation
- Apply current literature to inform clients of the best approach for their pet
- Learn the risk factors that contribute to mortality and mortality

MULTIPLE CHOICE QUESTIONS

1. Which is the riskiest period of anaesthesia?
(A) Premedication (C) Maintenance
(B) Induction (D) Recovery
2. Which of the following has been shown to increase mortality?
(A) Anaesthesia over sedation
(B) An experienced surgeon
(C) Urgent procedures
(D) Sevoflurane
3. What is the risk of anaesthetic death in sick cats?
(A) 1.4% (C) 44%
(B) 14% (D) 74%

	Overall	Healthy	Sick	Postoperative (%)
Dog	1:601 (0.17%)	1:1849 (0.05%)	1:75 (1.33%)	47
Cat	1:419 (0.24%)	1:895 (0.11%)	1:71 (1.40%)	61
Rabbit	1:72 (1.39%)	1:137 (0.73%)	1:14 (7.37%)	64

Figure 1: Risks of anaesthetic- and sedation-related mortality.

SIRS, shock and sepsis: what's the difference?

Simon Cook

In human medicine the definition of and treatment recommendations for sepsis are evolving. The diagnostic criteria for systemic inflammatory response syndrome

(SIRS) were previously used to define sepsis. However, there has been a shift towards recognizing that those SIRS criteria, in a patient with an infection, contained physiological responses, compared to the 'life-threatening organ dysfunction caused by a dysregulated host response to infection' that now defines *sepsis*. The term *septic shock* is reserved for those patients in whom there is an infection, organ dysfunction (e.g. cardiovascular, renal, respiratory, gastrointestinal, hepatic, central nervous system) and profound systemic instability.

SIRS is considered to be present in dogs and cats with two or three of the following criteria: an abnormal temperature, an abnormal heart rate, tachypnoea and a leucocytosis/leucopenia. These criteria are useful to flag concerning patients but should always be considered in conjunction with the clinical picture and physical examination findings. On identification of a patient with SIRS, the next questions are: is there an infection? And if so, where?

Trying to separate infection/inflammation (where the SIRS criteria might simply be a safe, adaptive response) from sepsis (where there is evidence of organ dysfunction, suggesting that the response has become maladaptive) is difficult but is the ultimate aim when assessing any pyrexia patient, with or without SIRS. The reason we want to separate these patients is that genuinely unstable septic patients with organ dysfunction require expedited investigations and aggressive management.

It is important to remember that the inflammatory pathways and maladaptive response to infection or sterile inflammation can essentially be identical. It is also the case that, once started, these processes will continue irrespective of whether the pathogen or inciting cause is removed. It is not possible to reverse the maladaptive response, but the patient can be supported through it, having removed or minimized the underlying trigger.

The cascade of cytokine production, endothelial dysfunction, fluid loss, vasoplegia and organ dysfunction creates a state of shock, where shock refers to inadequate energy production at the cellular level. Most importantly, this occurs due to reduced perfusion to tissue beds, but shock is multifactorial in septic/SIRS patients. Traditionally, septic shock is considered vasodilatory. Realistically, a septic patient probably has components of hypovolaemic, vasodilatory and cardiogenic shock. Alongside concurrent immune dysfunction, catabolism and recumbency, it becomes clear why these patients are deemed critical and have such high mortality rates.

KEY LEARNING OBJECTIVES

- Know how to interpret your physical examination findings accurately
- Characterize the type(s) of shock involved
- Appreciate the indications and complications associated with fluid therapy

MULTIPLE CHOICE QUESTIONS

1. A 3-year-old male neutered Labrador Retriever presents to you with a 3-day history of vomiting and inappetence, progressing to collapse that day, with no relevant medical history. On presentation the dog has a heart rate of 160 bpm, a respiratory rate of 40 breaths per minute, and a rectal temperature of 39.3°C with a painful abdomen. What is the most appropriate next step in the management of this patient?
 - (A) 60 ml/kg/h of saline and exploratory laparotomy
 - (B) Abdominal radiography
 - (C) Point-of-care ultrasonography, 0.1 mg/kg methadone and a 10 ml/kg fluid bolus
 - (D) Blood culture and antibiotics
2. Which of the following is considered a balanced crystalloid solution?
 - (A) 0.9% saline
 - (B) Compound sodium lactate
 - (C) 0.45% sodium chloride
 - (D) Dextrose 5% in water
3. Which of the following is considered the most reasonable first-line vasopressor for use in septic patients?
 - (A) Dobutamine
 - (B) Noradrenaline
 - (C) Adrenaline
 - (D) Ephedrine

DIC: causes, presentation and management

Simon Cook

Disseminated intravascular coagulation (DIC) is the systemic activation of coagulation, resulting in microvascular thromboses (probably with associated organ dysfunction); it can also be associated with spontaneous bleeding. It is not a disease, *per se*, but a feasible sequel of many diseases, especially those affecting major organs or organ systems. DIC exists as a spectrum; as severity increases, so does the degree of clinical manifestation of DIC, as does the associated mortality. Many more patients than we realize probably make it on to this spectrum, with widespread activation of coagulation and a hypercoagulable state. However, most do not progress to the worst-case scenario of multi-organ dysfunction, given

development of hypocoagulability, severe thrombocytopenia and spontaneous bleeding. Given the enormous variability in presentation and severity, there is no such thing as a 'standard' treatment approach.

To appreciate how DIC can be caused by many different underlying diseases, we need to recall and understand how normal coagulation is induced and regulated, and consider the downstream effects of excessive coagulation. Tissue factor (TF), expressed by subendothelial cells of the vasculature, triggers coagulation (via factor VIIa) when the endothelium is damaged, resulting in thrombin generation. In septic and inflammatory states, TF is upregulated, expressed by endothelial cells themselves, and circulates on TF-bearing microparticles. It can also be expressed by some neoplastic cells.

The most important downstream effect of excess TF is excess thrombin generation, which not only results in fibrin formation from fibrinogen, but propagates coagulation by many other means. In the cell-based model of coagulation, it is thrombin that induces the amplification phase. Thrombin propagates coagulation by direct stimulation of platelets and the activation of factors V, VIII, IX and XIII. Concurrently, thrombin can promote fibrinolysis.