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3rd Veterinary Emergency & Critical Care International Symposium

March 14-15, 2026 TOKYO JAPAN

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大阪どうぶつ夜間急病センター

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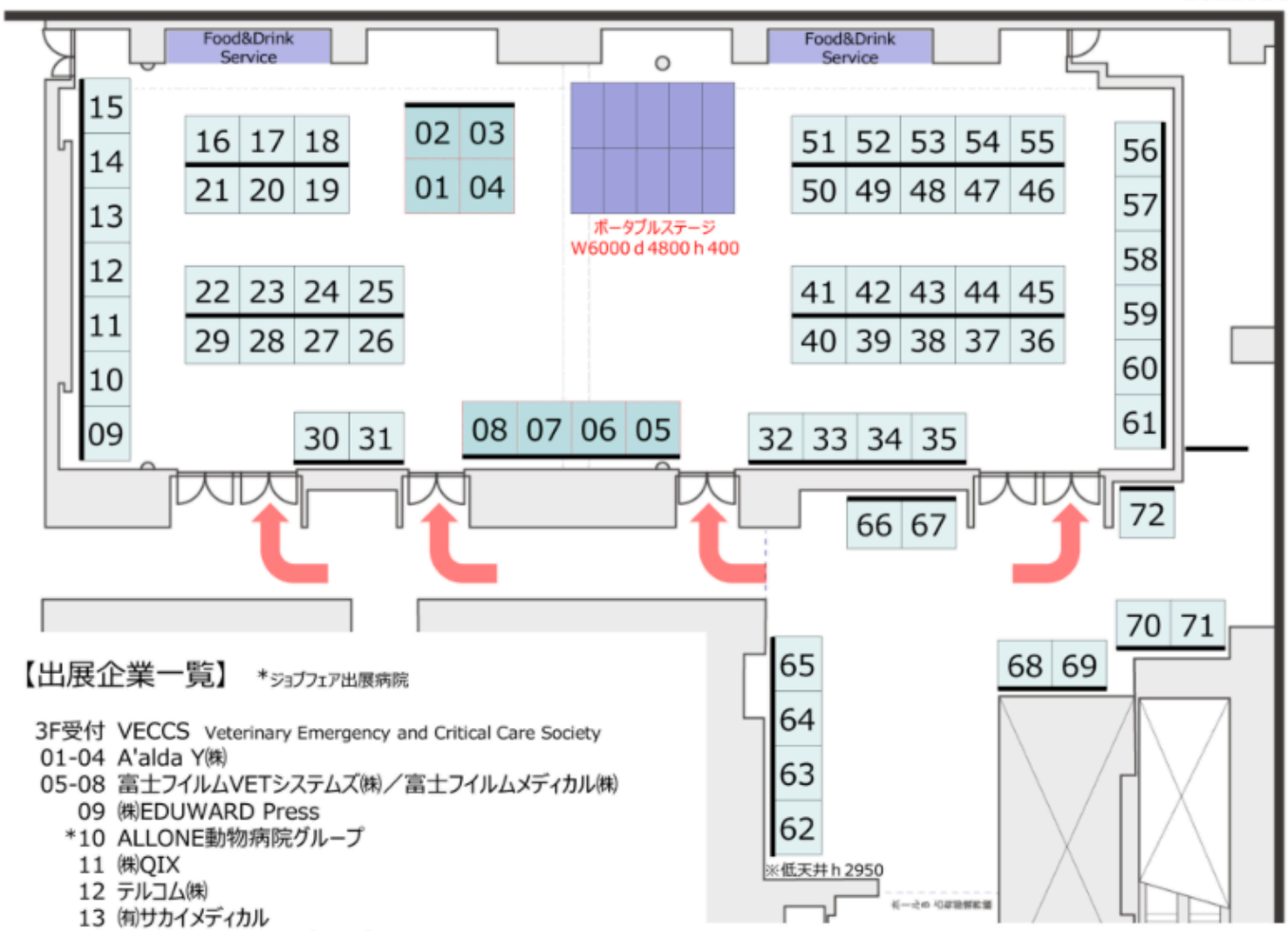
4F Exhibit Hall Layout



【出展企業一覧】 *ジョブフェア出展病院

3F受付 VECCS Veterinary Emergency and Critical Care Society
01-04 A'alda Y㈱
05-08 富士フイルムVETシステムズ㈱／富士フイルムメディカル㈱
09 ㈱EDUWARD Press
*10 ALLONE動物病院グループ
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15 合同会社どうぶつとひと出版
*16 公益財団法人 日本小動物医療センター
*17 ダクタリ動物病院 東京医療センター
18 イオンペット㈱
19 ㈱ラ・ショエット
*20 ㈱よつや動物病院
21 日本ペットフード㈱
22 ペンギンシステム㈱
*23 ルカどうぶつ二次診療クリニック
*24 岡山夜間どうぶつ救急病院
25 ベーリンガーインゲルハイムアニマルヘルスジャパン㈱
26 石原産業㈱
*27 つくば夜間動物病院
28 ㈱ウェミアス
29 いなばペットフード㈱
30-31 メディア㈱
32 物産アニマルヘルス㈱
33 ミズホ㈱
34 リーフインターナショナル㈱
35 mappin㈱
36 ゾエティス・ジャパン㈱
37 朝日インテック㈱
38 ㈱キカイヤ
*39 つくば動物高度医療センター 高度医療/整形外科/救急
40 ロイヤルカナン ジャポン合同会社
41 VEG
42 Blood Bank Registry
43 エルマ販売㈱
*44 どうぶつER救命救急センター
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*47 一般社団法人 りんくう動物救急医療協会
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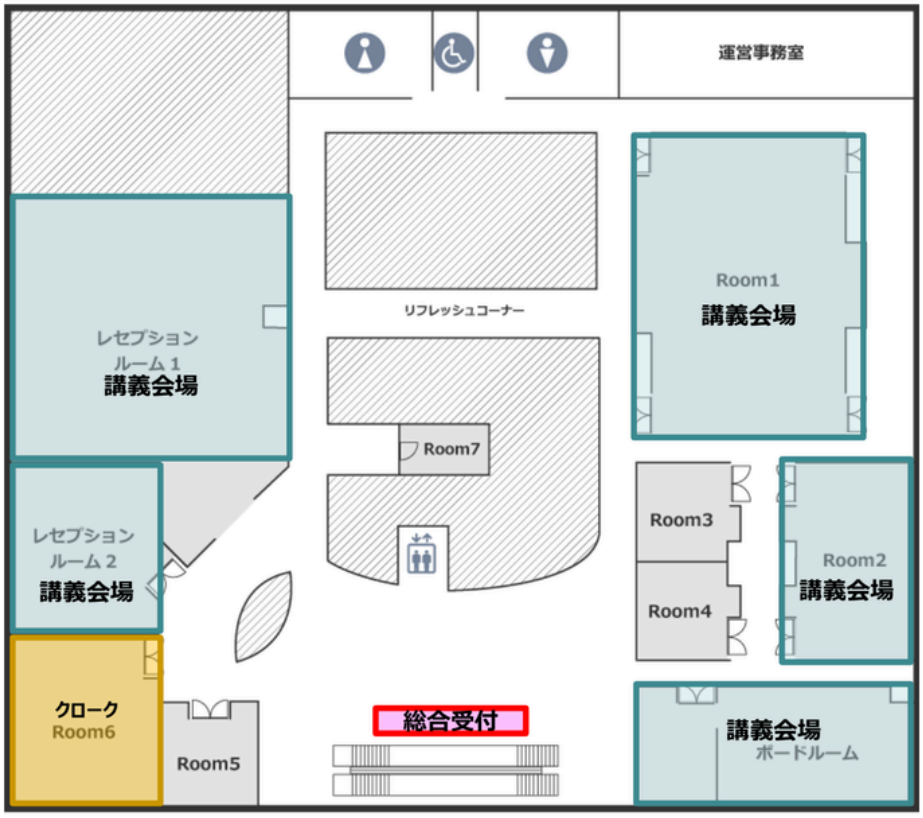
3F & 4F Floor Maps

3F



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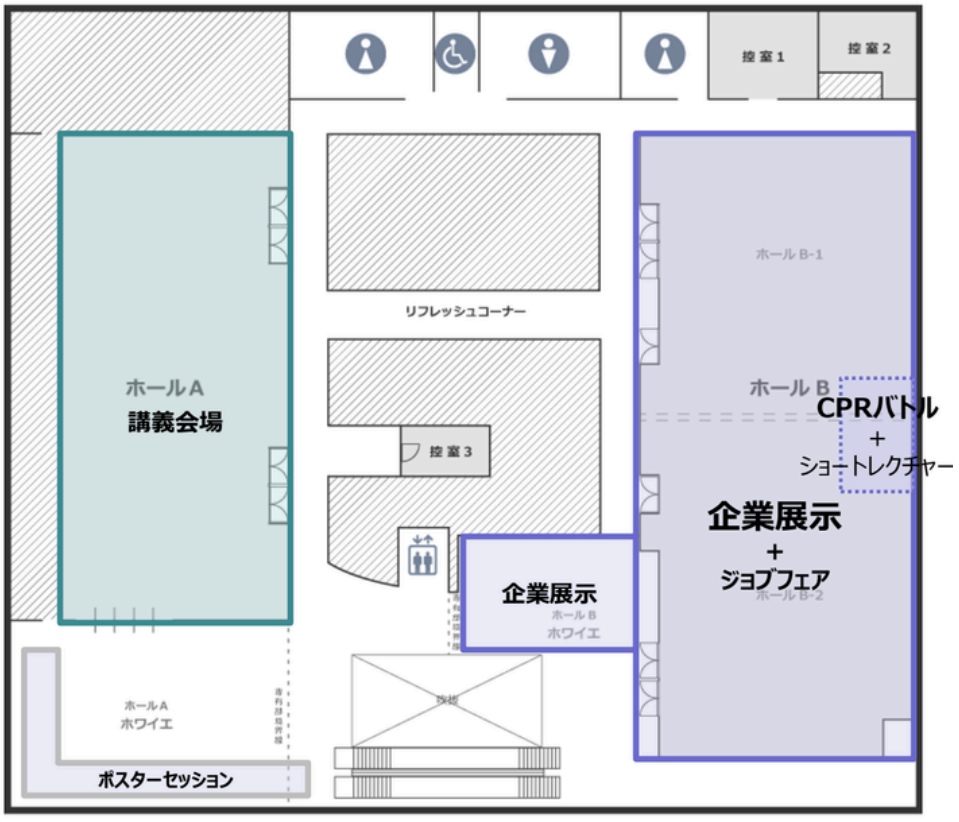

CPR Battle

CPR Battle Schedule

March 14 (Saturday)

Round 1 – 12:30 PM : <PETEMO> VS <MGL>

Round 2 – 6:30 PM : <ALOHA> VS <Annie>

March 15 (Sunday)

Finals – 11:30 AM: The winners of Round 1 and Round 2 will battle for the championship!

Room: 4th Floor, “Hall B”

Conference Schedule - March 14th

3/14 (Sat) Day 1	General Vet Track	Advanced Vet Track	General Vet Track #2	Basic Vet Nurse Track	Advanced Vet Nurse Track	Exhibit/CPR Battle
	HALL A	ROOM1	BOARDROOM	RECEPTION	ROOM2	HALL B
10:00 - 11:00	Anaphylactic shock [JP/EN] Corrin Boyd	Perioperative Anesthetic Strategies for Canine MMVD and Feline HCM [JP/EN] Hiroki Sano	Pros and cons for medical emergency [JP] Yoshiki Matsumoto Takashi Matsumoto PENGUIN SYSTEM	Discussion about VN [JP] Ken Yagi Takaomi Nuruki Atsushi Nakamura MEDIA	Neurological Examination in ER [JP/EN] Shun Nakamura	Exhibit Snacks/drinks
Break (30 min)						
11:30 - 12:30	Hypoadrenocorticism [JP/EN] Duana McBride	Assessing fluid responsiveness and tolerance [JP/EN] Xiu Ting Yiew			Common Intoxications in Daily Practice [JP/EN] Yasuhisa Kaneda	
Lunch break (90 min)	Lunch seminar A'alda Y	Lunch seminar Kikaiya	Asian ECC Society Update			CPR battle first round #1 PENGUIN SYSTEM
14:00 - 15:00	Feline Aortic Thromboembolism [JP/EN] Tommaso Rosati	Year in Review [JP/EN] Steven Epstein	General abstract [JP/EN]	Essential Blood Smear Evaluation in Emergency Practice [JP] Seigo Ogasawara zoetis	Nursing Care for Infectious Diseases [JP] Saori Shono	Exhibit Snacks/drinks
Break (30 min)						
15:30 - 16:30	Management of heatstroke [JP/EN] Claire Sharp	Urosepsis [JP/EN] Sandy Young	General abstract [JP/EN]	Blood Chemistry in Clinical Practice [JP] Koki Uemoto	Medical management of airway obstruction [JP] Takaomi Nuruki bdh 物産アニマルヘルス	
Break (30 min)						
17:00 - 18:00	Approach to intoxicated patients [JP/EN] Jiwoong Her	Interventional and Surgical Treatment of Laryngeal and Tracheal Obstruction [JP/EN] Masahiro Suematsu	Antimicrobials for ER cases [JP] Tomoki Motegi	Dystocia Management and Canine & Feline Neonatal Resuscitation [JP/EN] Kodai Kawase	CPR in practice [JP] Yuto Mori Rumi Nakajima	
Break (30 min)						Poster Session
18:30 - 20:00						Happy Hour Reception CPR Battle

3/14 Sat.	Washington Hotel	Washington Hotel
	3rd floor	3rd floor
	Hands-on Lab	Hands-on Lab
9:30 - 12:30	Blood gas worksop [EN] Tommaso Rosati Sandy Young	A Practical Workshop on Inpatient Management [JP] Go Otani Satoshi Matsukata Kaito Okada
90 min break	Lunch break 12:30~2:00	Lunch break 12:30~2:00
14:00 - 17:00	Blood gas worksop [JP] Shun Nakamura Rika Nagao	Case-based discusion of common emergency cases [EN] Corrin Boyd Xiu Ting Yiew

Time	Program	Room
12:30PM - 2:00PM	• Asian ECC Meeting	3F BoardRoom
	• Lunch Seminar (A'alda)	3F Room 1
	• Lunch Seminar (KIKAIYA)	4F Hall A
	• CPR Battle Round 1	4F Hall B
2:00PM - 4:30PM	• General Abstract / Case Report	3F Board Room
6:30PM - 8:00PM	• CPR Battle Round 2	4F Hall B
	• Reception Party	4F Hall B
9:30AM - 5:00PM	• Hands-on Training (English/Japanese)	Washington Hotel 3F

Conference Schedule - March 15th

3/15 (Sun) Day 2	General Vet Track #1	Advanced Vet Track	General Vet Track #2	Basic Vet Nurse Track	Advanced Vet Nurse Track	Exhibit/CPR Battle
	HALL A	ROOM1	BOARDROOM	RECEPTION	ROOM2	HALL B
9:00 - 10:00	Neonasal CPR/resuscitation [JP/EN] Ken Yagi	Nutrition in the Critically Ill [JP/EN] Duana McBride	Pros and cons in surgical emergency [JP] Hikaru Tezuka Hiroaki Sugiura mappin	Muscle Weakness During Hospitalization[JP/EN] Tadashi Sano	Measuring blood pressure [JP] Ryohei Suzuki	Exhibit Snacks/drinks
Break (30 min)						
10:30 - 11:30	Top 10 tips for a successful outcome in sepsis [JP/EN] Claire Sharp	Practice management [JP/EN] David Bessler Atsushi Nakamura		Pain Management in Hospitalized Patients [JP/EN] Tadashi Sano MEDIA	Pet loss and grief care [JP] Noriko Niijima	
Lunch break (90 min)		Lunch seminar VEG ER for Pets VEG ER for Pets				CPR battle FINAL
13:00 - 14:00	Hospital-acquired AKI [JP/EN] Corrin Boyd	Smoke inhalation and burn injury [JP/EN] Tommaso Rosati	Feline Cardiovascular EmergenciesHow They Differ from Dogs [JP/EN] Ryohei Suzuki	Basics of shock management [JP] Shiro Watari	Diagnostic approach to cavitary effusion [JP/EN] Seigo Ogasawara	Exhibit Snacks/drinks
Break (30 min)						
14:30 - 15:30	Ophthalmology emergency [JP/EN] Hiroki Tsujita	Sedation and Anesthesia in ICU [JP/EN] Claire Sharp	Career development for new veterinarians [JP] Hiroaki Sugiura Akira Tajima Fumiko Koshikawa	Nutritional Support for hospitalized patients [JP/EN] Chie Ishii	Career development for veterinary nurses [JP] Shun Nakamura	
Break (30 min)						
16:00 - 17:00	Surgical Drains in Veterinary Emergency and Critical Care [JP/EN] Min Su Kim	Acute respiratory distress syndrome [JP/EN] Corrin Boyd	Interdialytic Complications [JP/EN] Sandy Young	Useful techniques you can use in ER [JP] Satoshi Matsukata Yuto Mori Takaomi Nuruki INABA いなばペットフード株式会社	POCUS in ICU [JP] Go Ohtani	
Break (30 min)						
17:30 - 18:30	Management of vomiting and diarrhea in ER [JP/EN] Xiu Ting Yiew	Open cardiac surgery for congestive heart failure [JP/EN] Masashi Mizuno	Extracorporeal Therapy for Cats [JP/EN] Yu Ueda		Diabetes Ketoacidosis [JP] Ryo Kobayashi	

3/15 (日)	Washington Hotel	Washington Hotel
	3rd floor	3rd floor
	Hands-on Lab	Hands-on Lab
8:30 - 11:30	Neonatal Resuscitation Workshop — Learning from the Guidelines [JP] Koudai Kawase	Anesthesia monitoring workshop [EN] Alexander Thomson Hiroki Sano
90 min break	Lunch break 11:30~1:00	Lunch break 11:30~1:00
13:00 - 16:00	Case-based discussion of nutritional management for ICU patients [EN] Duana McBride	Anesthesia monitoring workshop [JP] Tadashi Sano Dai Nagakubo

Time	Program	Room
11:30AM - 1:00PM	• Lunch Seminar (VEG ER) • CPR Battle Final	3F Room 1 4F Hall B
8:30AM - 4:00PM	• Hands-on Training (English/Japanese)	Washington Hotel 3F

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私たちは、1次診療施設（5病院）と24時間の集中管理が可能なセンター病院、そして行動診療に特化した動物行動診療科を有する動物病院グループです。
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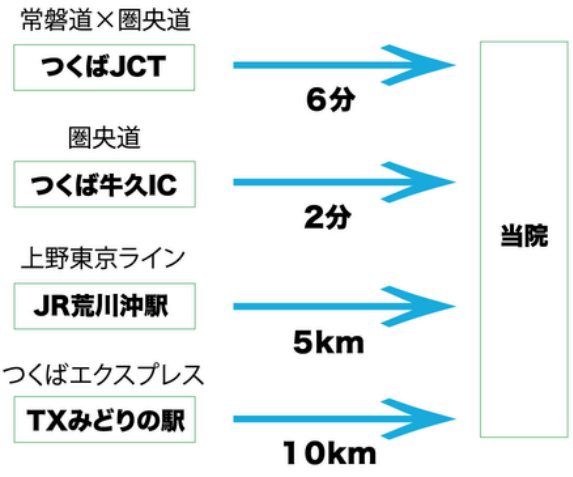

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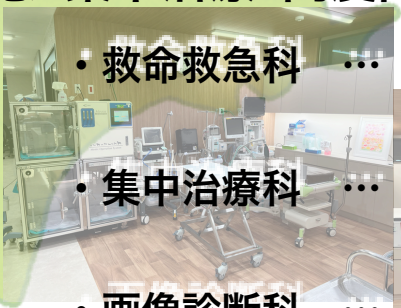
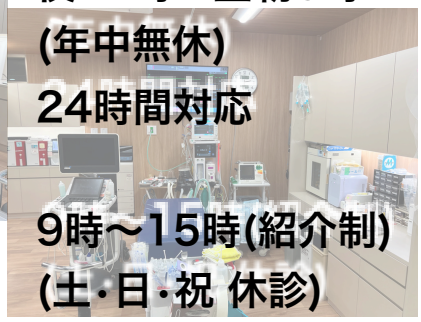


- 救命救急科 … 夜19時～翌朝6時 (年中無休)
- 集中治療科 … 24時間対応
- 画像診断科 … 9時～15時(紹介制) (土・日・祝 休診)



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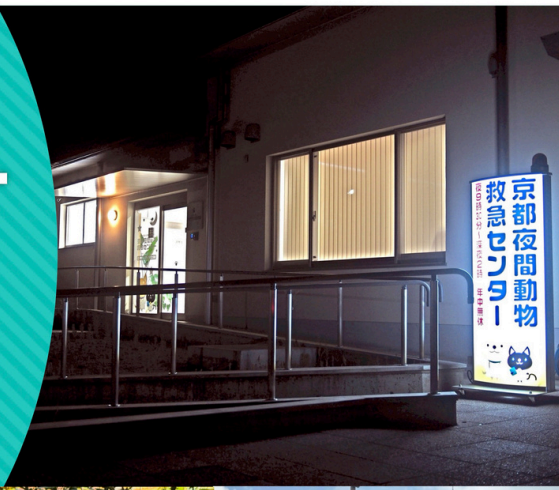

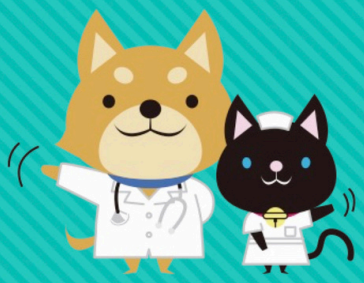

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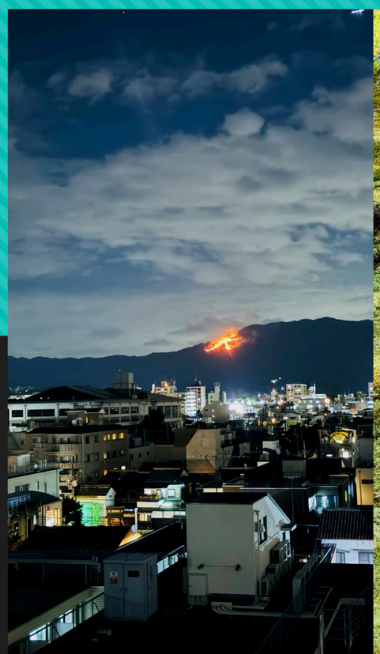

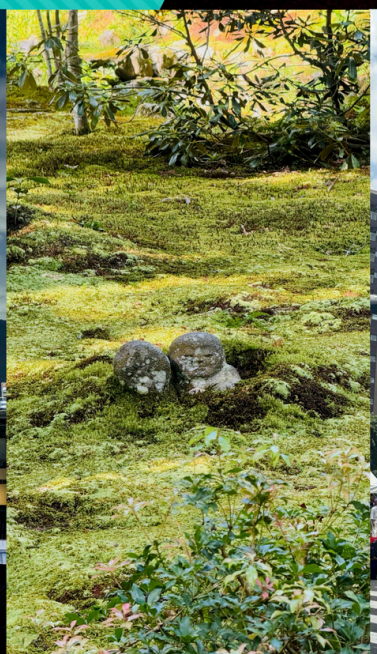

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Anaphylactic Shock
Dr. Corrin Boyd

Pathophysiology

Anaphylaxis is defined as a life-threatening acute systemic type 1 hypersensitivity reaction that results in multiple organ dysfunction. Anaphylactic shock specifically refers to cardiovascular dysfunction from anaphylaxis. Anaphylaxis develops rapidly after exposure to a causative allergen, which is not always identified. The typical pathogenesis involves the release of potent inflammatory mediators such as histamine from mast cells and basophils in response to antigen binding and cross-linking of IgE. The three main classes of effector molecules in anaphylaxis are vasoactive amines (eg. histamine), products of the arachidonic acid cascade (eg. prostaglandins and leukotrienes), and pro-inflammatory cytokines, each with a different time course. Other non-IgE immunologic pathways and non-immunologic pathways can also trigger anaphylaxis. Common allergens include hymenoptera (bee, wasp, and ant) venom, drugs, vaccines, food, environmental allergens, blood products (especially plasma products), and physical factors (such as cold and exercise). The most commonly affected organ systems are the dermatologic, cardiovascular, respiratory, and gastrointestinal systems.

Anaphylactic shock is typically described as vasodilatory shock in human literature. However, vasoconstrictive shock, shock with features of both vasoconstriction and vasodilation, or hypotension without evidence of either vasoconstriction or vasodilation can occur in dogs. This is due to the combination of arterial vasodilation and decreased hepatic venous drainage that occurs in this species. Furthermore, both tachycardia and bradycardia have been reported in dogs. Dysfunction of the coagulation system can also occur in dogs. In some cases this contributes to spontaneous haemorrhage, most commonly haemoperitoneum, further contributing to circulatory shock.

Diagnosis

There is no ideal diagnostic test for anaphylaxis. Thus, the diagnosis is generally based on consistent history and clinical signs. Anaphylaxis can be clinically recognised when there is an acute onset of characteristic dermatologic signs with at least one of cardiovascular, respiratory, or gastrointestinal signs. The classic dermatologic signs include angioedema (especially of the face), urticaria (wheals), erythema, and pruritis. However, not all cases exhibit these dermatologic signs. Therefore, anaphylaxis can also be diagnosed by an acute onset of severe cardiovascular or respiratory signs without dermatologic involvement, where there is known or probable exposure to an allergen. As mentioned above, a variety of cardiovascular signs are consistent with anaphylaxis. Respiratory signs can include tachypnoea, increased respiratory effort, upper airway obstruction, cough, pulmonary wheezes and/or crackles, and cyanosis. Gastrointestinal signs can include vomiting and diarrhoea, sometimes with fresh blood, abdominal pain, hypersalivation, and retching. Some diagnostic tests are useful adjuncts. Assessment of blood pressure is crucial, as hypotension may be the only sign of cardiovascular dysfunction. Routine blood tests may show elevated liver enzyme activity (especially ALT), coagulopathy, and a lactic acidosis. C-reactive protein concentration is usually normal; elevation should prompt consideration of other differential diagnoses. Point-of-care ultrasound often shows oedema of the gallbladder wall. This abnormality may aid in differentiating anaphylaxis from an uncomplicated type 1 hypersensitivity reaction. However, great care must be taken to adequately evaluate for other causes of gallbladder wall oedema. Some of these, such as acute pericardial effusion, are important differential diagnoses for an acute onset of circulatory shock. In cases of anaphylaxis, ultrasound may also show a peritoneal effusion, which may be either a modified transudate or frank haemorrhage.

Treatment

The cornerstone of anaphylaxis treatment is rapid empiric supportive care for the life-threatening cardiovascular and respiratory signs. Oxygen therapy should be provided and an IV catheter placed. Adrenaline (epinephrine) is the mainstay of acute medical management, as it both prevents further release of inflammatory mediators and directly treats both hypotension and bronchoconstriction. An initial 10 mcg/kg (0.01 mg/kg) intramuscular dose can be given. With ongoing hypotension, further adrenaline should be provided intravenously as a constant rate infusion of 0.005-1 mcg/kg/min, titrated to achieve minimally acceptable normotension. Due to the risk of extravasation injury, ensure that the IV catheter is long, atraumatically placed, and well-secured. Intravenous fluid therapy is also frequently necessary to treat shock, especially when a vasoconstrictive component is identified. Bolus therapy (10-20 mL/kg in dogs, 5-10 mL/kg in cats) over 10-15 minutes with balanced isotonic crystalloids is recommended as first line therapy, which can be repeated. Synthetic colloid fluids should be avoided due to their potential to cause worsening coagulopathy. In cases with severe haemorrhage, bolus administration of blood products may be required. Either whole blood or a combination of packed red blood cells and plasma should be administered, to replace blood volume, oxygen carrying capacity, and coagulation competence. Autotransfusion of peritoneal blood can be considered. Calcium supplementation may be necessary. Gastrointestinal supportive care may be indicated. Antihistamines and glucocorticoids may aid in resolution of dermatologic signs, but have minimal impact on the life-threatening organ dysfunction. Glucocorticoids should be avoided in cases of cardiovascular instability or severe gastrointestinal signs. Prognosis is good with sufficiently aggressive treatment, even with severe clinical signs. Follow-up allergen testing and allergen-specific immunotherapy should be recommended.

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Hypoadrenocorticism in Dogs and Cats

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Introduction

Hypoadrenocorticism is an uncommon but clinically challenging disease due to it's vague clinical signs and variability in diagnostic results. Often termed the "great pretender", hypoadrenocorticism can mimic gastrointestinal, renal, cardiac, and neurological disease, leading to diagnostic delay and inappropriate therapy. Although feline hypoadrenocorticism was not well recognized, 2 recent retrospective reviews have highlighted the need to consider this rare but important endocrinopathy in cats. Despite these challenges, once diagnosed, it carries a good prognosis.

Pathophysiology

The adrenal cortex produces mineralocorticoids (aldosterone) which is secreted by the zona glomerulosa, glucocorticoids (cortisol) secreted by the zona fasciculata, and sex hormones secreted by the zona reticularis. Cortisol secretion is regulated by the hypothalamic–pituitary–adrenal (HPA) axis via corticotropin-releasing hormone and adrenocorticotropic hormone (ACTH). Aldosterone secretion is primarily controlled by the renin–angiotensin–aldosterone system and serum potassium concentration. Cortisol is essential for maintaining vascular tone, catecholamine responsiveness, glucose homeostasis, gastrointestinal integrity, and immune modulation. Aldosterone promotes sodium and water reabsorption while facilitating potassium and hydrogen ion excretion at the renal tubules.

Primary hypoadrenocorticism most commonly results from immune-mediated destruction of the adrenal cortex, but may also occur secondary to neoplasia, haemorrhage, trauma, hypoperfusion, or iatrogenic (trilostane, ketoconazole) causes. Secondary hypoadrenocorticism arises from pituitary or hypothalamic dysfunction, or suppression following prolonged exogenous glucocorticoid administration, resulting in isolated cortisol deficiency with preserved mineralocorticoid function.

Signalment and Clinical Presentation

Hypoadrenocorticism can affect dogs of any age, from juvenile to geriatric, with reported breed predispositions including mixed-breed dogs, Portuguese Water Dogs, Standard Poodles, West Highland White Terriers, Great Danes, and Rottweilers. Although less common in cats, cases have been reported across a wide age range, with domestic shorthair and British shorthair cats potentially overrepresented.

Clinical signs are typically vague, waxing and waning, and may include lethargy, anorexia, vomiting, diarrhoea, weight loss, polyuria/polydipsia, weakness, muscle cramps, or episodic collapse. Physical examination findings range from mild dehydration to hypovolaemic shock, bradycardia, hypothermia, abdominal pain, and in severe cases, signs of cardiac dysfunction secondary to hyperkalaemia-induced myocardial depression.

Clinicopathological Findings

Classic laboratory abnormalities include hyponatraemia, hyperkalaemia, and a decreased sodium-to-potassium ratio (<28:1). However, dogs with atypical or secondary hypoadrenocorticism

may have normal electrolytes. Additional findings may include non-regenerative anaemia, absence of a stress leukogram, azotaemia, isosthenuria, hypoglycaemia, hypercalcaemia, metabolic acidosis, hypoalbuminaemia, and hypocholesterolaemia. Concurrent endocrine or immune-mediated disease may occur. In cars exocrine pancreatic insufficiency, hypocobalaminaemia and lymphoid neoplasia have been reported.

Diagnosis

The ACTH stimulation test remains the gold standard for diagnosis of hypoadrenocorticism. A post-ACTH cortisol concentration below 55 nmol/L is considered diagnostic, while a basal cortisol concentration equal to or exceeding 55 nmol/L effectively rules out the disease. If basal cortisol is 22 – 55 nmol/L, an ACTH stimulation test should be performed. Basal cortisol concentrations below 22 nmol/L are highly suggestive, however can occur in dogs with chronic gastrointestinal disease or recent glucocorticoid exposure (within last 90 days).

In dogs with normal electrolytes or equivocal results, measurement of endogenous ACTH can assist with disease localisation. Elevated ACTH concentrations are consistent with primary hypoadrenocorticism, whereas low or inappropriately normal ACTH supports secondary or iatrogenic disease. Cortisol to ACTH ratio may also be performed with results are equivocal, with low levels suggestive of hypoadrenocorticism and high levels being non-hypoadrenocorticism disease. Urine cortisol testing, including the urine cortisol-to-creatinine ratio, may be used as a highly sensitive screening test but requires confirmatory ACTH stimulation testing.

Emergency Management of Addisonian Crisis

Dogs presenting in Addisonian crisis require immediate stabilisation. Initial therapy focuses on intravenous fluid resuscitation with balanced crystalloids to correct hypovolaemia and improve renal perfusion. Hyperkalaemia should be addressed with fluid therapy, calcium gluconate for cardioprotection, insulin and dextrose to promote intracellular potassium shift, and beta-agonists in refractory cases.

Glucocorticoid replacement should be instituted promptly. Hydrocortisone is advantageous in the acute setting due to its combined glucocorticoid and mineralocorticoid activity. Alternatively, dexamethasone may be used, however has minimal mineralocorticoid effect and stabilize the cardiovascular signs. It is thought that dexamethasone does not interfere with the ACTH stimulation test, however it may affect the CRH and ACTH. Mineralocorticoid replacement with desoxycorticosterone pivalate or fludrocortisone should be initiated once the patient is stabilised.

Long-Term Management and Prognosis

Long-term management involves lifelong glucocorticoid supplementation, typically with low-dose prednisolone, and mineralocorticoid replacement in dogs with primary hypoadrenocorticism. Owners should be educated regarding stress-dose adjustments during illness or travel, and regular monitoring of electrolytes is recommended during dose titration.

The prognosis for dogs and cats with hypoadrenocorticism is excellent. Reported median survival times exceed five years, with many patients achieving a normal lifespan and quality of life. Cats demonstrate similarly favourable outcomes, with survival to discharge exceeding 95%.

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What Is New Regarding Treatment and Prognosis of Feline Aortic Thromboembolism

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Introduction

Feline aortic thromboembolism (FATE), often referred to as saddle thrombus, is a common and dramatic emergency condition in cats. It is characterized by sudden obstruction of arterial blood flow, most frequently at the terminal aorta, resulting in acute pain, paresis or paralysis, and ischemic injury of the affected limbs. Historically, FATE has been associated with a poor prognosis; however, recent advances in supportive care, antithrombotic therapy, and post-event management have improved both short- and long-term outcomes. This lecture focuses on current understanding of why FATE develops, key risk factors, and what is new regarding treatment and prognosis.

Pathophysiology and Risk Factors

FATE most commonly develops secondary to underlying heart disease, particularly cardiomyopathies. Approximately 90% of cases are cardiogenic in origin, with hypertrophic cardiomyopathy being the most frequently identified condition. In many cats, FATE is the first clinical manifestation of previously undiagnosed heart disease.

Thrombus formation typically occurs within the left atrium or left atrial auricle. Enlargement of the left atrium, reduced blood flow velocity, endothelial dysfunction, and activation of coagulation pathways create a pro-thrombotic environment. Spontaneous echocardiographic contrast, often described as “smoke,” reflects blood stasis and is a strong indicator of thrombotic risk.

Additional risk factors include male sex, increasing age, congestive heart failure, and systemic diseases such as neoplasia, hyperthyroidism, inflammatory conditions, and corticosteroid administration. Emerging evidence also suggests that individual variability in platelet responsiveness and coagulation pathways may contribute to thromboembolic risk, helping explain why some cats develop FATE despite similar degrees of cardiac disease. Once a thrombus dislodges from the heart, it travels through the arterial circulation and commonly lodges at the aortic trifurcation, abruptly interrupting blood flow to one or both pelvic limbs.

Clinical Relevance of Ischemia and Reperfusion

The sudden loss of arterial perfusion leads to severe ischemic pain, neuromuscular dysfunction, and metabolic derangements within the affected limbs. Following restoration of blood flow, either spontaneously or with treatment, reperfusion injury may occur. This process can result in systemic complications such as hyperkalemia, metabolic acidosis, myoglobin release, and acute kidney injury. Recognition of these mechanisms highlights the importance of careful monitoring during the first 24–48 hours of hospitalization. These complications remain a major contributor to early mortality, even in cats that show initial improvement in limb perfusion.

Treatment Considerations

Initial treatment of FATE remains focused on rapid stabilization, effective analgesia, and supportive care. Potent opioid analgesia is essential to control pain and reduce stress-related oxygen consumption. Oxygen supplementation and treatment of concurrent congestive heart failure, when present, are also key components of early management.

Recent advances have primarily occurred in the area of antithrombotic therapy. Clopidogrel has become the cornerstone of both acute management and secondary prevention, having demonstrated superiority over aspirin in reducing recurrence of thromboembolism. In recent years, novel oral anticoagulants such as rivaroxaban and apixaban have been increasingly used in cats. The SUPERCAT study demonstrated that clopidogrel and rivaroxaban monotherapy result in comparable survival times and recurrence rates, supporting rivaroxaban as a reasonable alternative in cats intolerant of or poorly responsive to clopidogrel.

Research led by investigators at North Carolina State University has demonstrated synergistic antithrombotic effects when clopidogrel is combined with factor Xa inhibitors such as rivaroxaban. These studies showed enhanced inhibition of platelet activation and thrombin generation, supporting the use of dual antithrombotic therapy in selected cats. Importantly, pharmacogenomic research has identified polymorphisms in platelet ADP receptors, such as P2RY1, that influence individual response to clopidogrel, providing a biological rationale for variable drug efficacy and for combination antithrombotic strategies. Clinically, combination therapy has been associated with longer survival times and lower recurrence rates compared with historical treatment protocols.

Thrombolytic therapy using tissue plasminogen activator (TPA) has also been revisited. While thrombolysis may improve limb reperfusion and functional recovery in some cats, it has not consistently demonstrated a survival benefit and carries risks of bleeding and reperfusion injury. As such, thrombolysis is generally reserved for carefully selected cases presenting very early after onset.

Prognosis

The prognosis for cats with FATE is no longer uniformly grave. Recent prospective and retrospective studies indicate that approximately 30–40% of cats survive to hospital discharge with appropriate case selection and supportive care. Cats that regain motor function, have only one limb affected, or maintain normal body temperature at presentation tend to have a more favorable short-term outcome.

Importantly, long-term prognosis has improved substantially with modern thromboprophylaxis. Median survival times exceeding one year have been reported in cats discharged on appropriate antithrombotic therapy, particularly those receiving anticoagulant therapies. The availability of multiple effective antithrombotic options allows therapy to be tailored to the individual patient, potentially improving adherence and long-term outcomes.

Client communication is essential. Owners should be counseled that while the initial hospitalization period is critical, cats that survive the acute phase may enjoy meaningful quality of life for months to years with appropriate cardiac and antithrombotic management.

Key Take-Home Messages

FATE most commonly arises from undiagnosed cardiomyopathy and left atrial thrombus formation. Early aggressive analgesia and supportive care are essential. Advances in antithrombotic therapy—such as dual therapy with clopidogrel and factor Xa inhibitors—represent a meaningful improvement in management. While prognosis remains guarded, survival and long-term outcomes are better than historically perceived, and treatment should be considered in appropriately selected patients.

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MANAGEMENT OF HEAT STROKE

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Definitions and etiologic classification

In human medicine a spectrum of heat-related illness is commonly described, with a progression from mild illness (including heat rash, heat cramps, and heat syncope), to moderate illness (heat exhaustion), and severe illness (heat stroke).[1] Heat stroke refers to a life-threatening, multisystem illness characterised by an increase in core body temperature to > 40°C (humans) or > 41°C (dogs) and central nervous system (CNS) dysfunction. Heat stroke can be classified based on the etiology as either classical / environmental heat stroke or exertional heat stroke.[2] Discussion of heat stroke is timely in this era of climate change, and increasing global temperatures.

Risk factors and epidemiology

A recent large study investigated the epidemiology of heat-related illness in dogs in the UK. This study using data from VetCompass in 2016 identified an incidence of 0.04%, and an event fatality rate of 14.18%.[3] Patient risk factors include a prior occurrence of heat stress / stroke, obesity, breed, and lack of acclimation and fitness. Brachycephalics are over-represented in dogs due to their reduced ability to dissipate heat via the upper airway. Environmental risk factors include high environmental temperature, and high humidity. The findings of a recent study suggest that the average pet dog may have greater exposure to high environmental temperatures than traditional measures indicate.[4] Additionally, data from Australia has shown a broadly linear increase risk of mortality in dogs of 0.6% (95% CI: 0.1% to 1%) for each 1°C increase in daily maximum temperature above 25°C, and a 9.5% increased mortality risk (95% CI: 4.3% to 15%) on extreme heat days compared with nonextreme heat days.[5]

Pathophysiology

In dogs, more than 70% of body heat is normally dissipated by convection and radiation.[2] Redistribution of blood flow to the skin (eg. and away from splanchnic vasculature) aids in increasing heat dissipation, but heat dissipation by convection and radiation will decrease as environmental temperature approaches skin temperature. In these circumstances evaporation via panting becomes the major heat dissipation mechanism. When environmental temperatures exceed body temperature then net heat gain will occur. Heat stroke causes multiple organ dysfunction syndrome (MODS). The pathophysiology of injury to individual organs is discussed below, concurrently with diagnostic abnormalities and treatment.

Clinical signs and physical examination findings

Collapse and neurologic signs are evident in dogs with heat stroke. Hyperthermia is classically present, although patients may be hypothermic, or normothermic by the time of presentation if they have been cooled before presentation, or associated with shock. Shock is identified by abnormalities of the 6 perfusion parameters. Tachypnea and/or panting occur in an attempt to dissipate heat. Spontaneous bleeding evidenced by petechia or ecchymoses, hematemesis, and/or hematochezia are common. Some dogs have concurrent sunburn.[6]

Diagnostic approach

Blood should be collected for point-of-care venous blood gas and PCV/TS. If hypoglcyemia is present this should be immediately treated with an IV bolus of glucose followed by a CRI. Full blood tests should also be performed including a complete blood count, with blood smear, biochemistry profile, urinalysis, and coagulation testing (generally PT/aPTT).

Body systems approach and treatment

Initial treatment should focus on rapid cooling, if still hyperthermic, and addressing life-threatening abnormalities. Cooling can be achieved by administration of room temperature IV fluids, wetting the fur down and applying a fan. It is recommended to stop cooling when the body temperature reaches 39.5°C (103°F). Addressing life-threatening abnormalities includes reversal of shock, controlling seizures, and supporting the respiratory system.

Shock: Dogs with heat stroke may develop shock through multiple mechanisms, including distributive, hypovolemic and even cardiogenic shock. First line therapy includes boluses of balanced isotonic crystalloid fluids (eg. 10mL/kg LRS over 5-10 mins), repeated as needed to normalize perfusion parameters. Fresh frozen plasma (FFP) can also be used for volume expansion in dogs with hemorrhage secondary to hemostatic dysfunction. If shock persists despite volume loading, the use of vasopressors (eg. a noradrenaline CRI) is indicated.

Cardiac arrhythmias: Myocardial injury in heat stroke is multifactorial, and can result in cardiac arrhythmias. The severity of myocardial injury is reflected in increased cardiac troponin I (cTnI). Treatment for arrhythmias is indicated if they are compromising perfusion, or there is concern for progression to ventricular fibrillation. Lidocaine is first line therapy, starting with a bolus (2mg/kg IV), followed by an IV CRI (25-75 ug/kg/min).

CNS dysfunction: Treatment focuses on optimising cerebral perfusion pressure with a focus on treating shock first (to restore mean arterial pressure), and the use of hyperosmolar agents (eg. hypertonic saline) to reduce intracranial pressure (ICP) that can occur due to cerebral edema. Seizures can be controlled starting with benzodiazepines (eg. midazolam), and the addition of levetiracetam, phenobarbitone, and/or other adjunctive agents as needed.

Lung injury / acute respiratory distress syndrome (ARDS): Oxygen supplementation is required for hypoxemic animals. More severe lung injury may require intubation and mechanical ventilation. Necrotizing pneumonia resulting in pneumothorax has recently been reported as an unusual complication of heat stroke in a dog.[7]

Gastrointestinal dysfunction: Gastrointestinal dysfunction occurs secondary to splanchnic vasoconstriction resulting in intestinal ischemia. Increased intestinal permeability results in translocation of intestinal bacteria and secondary sepsis. Treatment includes broad spectrum IV antimicrobials, antiemetics, and antacids. Prokinetics are also indicated in those with ileus and/or gastroparesis.

Acute kidney injury: Pre-renal and renal mechanisms contribute to kidney injury that can manifest as reduced urine output (oliguria, anuria), azotemia progressing to uremia, and electrolyte and acid-base derangements. Careful fluid balance to maintain euvolemia and euhydration while avoiding overhydration is vital. Close monitoring of urine output with an indwelling urinary catheter is ideal. Hemodialysis may be required for management of anuric AKI.

Hemostatic dysfunction: Heat stroke is commonly accompanied by disseminated intravascular coagulation (DIC), as a result of thermal endothelial injury initiating coagulation and microthrombosis. Dogs with heat stroke also often have thrombocytopenia, due to consumption in DIC, but also splenic sequestration, and loss in GI bleeding. The hypocoagulable state may be exacerbated by reduced clotting factor production due to liver injury, and hyperfibrinolysis.

Dogs with clinically significant hemorrhage and prolonged clotting times require treatment with FFP, with a starting dose of 20mL/kg. In one study of dogs with heat stroke, the median number of FFP units administered was ~4 (~ 40mL/kg). Antifibrinolytic drugs (eg. tranexamic acid or aminocaproic

acid) may be used empirically in patients with severe bleeding. Given that the origin of DIC is consumption of platelets and clotting factors in microthrombosis thromboprophylaxis should be considered, however the role and timing of thromboprophylaxis in heat stroke is not known.

Prognosis

Mortality for true heat stroke in dogs is ~ 40-50%. Cooling prior to presentation improves outcome and so should be performed whenever possible. Numerous studies suggest that the greatest relative risk of death is associated with the presence of AKI, and DIC. It is likely that the greater number of organ dysfunctions present, the more likely a patient is to die from heat stroke related complications. A VetCompass heat stroke grading tool has also been developed and revised that aids in outcome prediction.[8]

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Assessing Fluid Responsiveness and Tolerance

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Intravenous fluid therapy remains the cornerstone of initial resuscitation in hemodynamically unstable patients. However, only ~50% of unstable patients are truly fluid responsive. When hypotension or hypoperfusion persists after initial resuscitation, clinicians must determine whether additional fluid will meaningfully improve cardiac output (CO) or whether alternative therapies (e.g. vasopressors, inotropes) are more appropriate. Empiric fluid administration until clinical response plateaus can be harmful because up to 50% of patients may be non-fluid responders. Excess fluid contributes to fluid overload and adverse outcomes, including microcirculatory dysfunction, endothelial glycocalyx disruption, increased vascular permeability, and tissue/organ edema. Fluid overload is commonly defined as a cumulative positive fluid balance >10% of baseline body weight. While useful retrospectively, this metric is relatively crude, organ-insensitive, and often identified too late. These concerns underpin the complementary concepts of fluid responsiveness and fluid tolerance.

Fluid Responsiveness vs. Fluid Tolerance

Fluid responsiveness (FR) is defined as a ≥10-15% increase in CO or stroke volume (SV) after a preload challenge. A positive response indicates the heart is on the ascending limb of the Frank-Starling curve; a negative response suggests that additional fluid is unlikely to improve perfusion and may cause harm. Importantly, FR predicts arterial flow but does not assess venous congestion or organ tolerance to fluid.

Fluid tolerance (FT) describes how much fluid a patient can receive without developing organ dysfunction and reframes resuscitation to consider both arterial perfusion and venous congestion. Clinically, patients can be assessed for left-sided tolerance (pulmonary congestion and left heart filling pressures) and right-sided tolerance (systemic venous congestion and right heart pressures).

Growing evidence linking fluid overload to increased morbidity and mortality suggests that hemodynamic evaluation should move beyond FR alone and incorporate FT. Coordinated assessment of both variables has the potential to prevent or mitigate fluid overload and promote fluid stewardship/responsibility. Considering both FR and FT yields four hemodynamic profiles, each with a different management approach:

1. Fluid responsive + fluid tolerant □ volume expansion
2. Non-fluid responsive + fluid tolerant □ conservative fluids
3. Fluid responsive + fluid intolerant □ early use of vasopressors
4. Non-fluid responsive + fluid intolerant □ de-escalation (diuretics, ultrafiltration)

Assessing Fluid Responsiveness

The gold standard remains a formal fluid challenge with measurement of CO or SV before and after a defined bolus. However, predicting FR before administering additional fluid is preferable. Two main predictive strategies are used:

1. Preload challenge: external (mini fluid bolus) or internal (passive leg raising, end-expiratory occlusion test)
2. Heart-lung interaction during mechanical ventilation: evaluation of respiratory variation in SV, pulse pressure, or caudal vena cava dimensions

Static vs. Dynamic Hemodynamic Measurements

Static Indices – Volume Status; Poor Predictors of FR!	**Dynamic Indices** – Better Predictors of FR
Physical examination findings†	Mini fluid challenge†
Macrohemodynamic parameters (HR, MAP)†	Passive leg raising (PLR)
Blood lactate, mixed venous oxygen saturation†	End-expiratory occlusion test (EEOT)
Central venous pressure (CVP) †	Stroke volume variation (SVV)
Pulmonary artery occlusion pressure (PAOP)	Pulse pressure variation (PPV)†
Pulmonary capillary wedge pressure (PCWP)	Systolic pressure variation (SPV)†
Global end-diastolic volume (GEDV)	Plethysmographic variability index (PVI)†
End-tidal carbon dioxide ($ETCO_2$)	Caudal vena cava collapsibility index (CVC-CI)†
Caudal vena cava diameter (CVCd) †	Tidal volume challenge (VTC)
Point-of-care ultrasound (POCUS, e.g. LA:Ao) †	

Static indices reflect a single measure of cardiac preload and are generally poor predictors of FR, although they can still provide information about volume status. In contrast, dynamic indices assess changes in SV after a transient preload challenge and are generally superior for predicting FR. These tests require real-time monitoring of CO or SV using techniques that vary in invasiveness, cost, technical skill, and time burden, which can limit their practicality in some clinical settings.

In mechanically ventilated patients, marked respiratory variation in SV, pulse pressure, or perfusion index suggests likely fluid responsiveness, but only in the absence of arrhythmias or spontaneous breathing. In dogs, PPV generally performs best, followed by PVI and SVV. However, these indices can be altered by drugs (e.g. dexmedetomidine, acepromazine), ventilatory settings (e.g. high PEEP), hypothermia, vasoconstriction, low SV, and reduced chest wall or lung compliance. Because PPV and SVV primarily reflect left ventricular preload responsiveness, their accuracy may be reduced with significant right ventricular dysfunction (i.e. fluid administration may not increase left ventricular preload). PLR maneuver is among the most reliable predictors of fluid responsiveness for both spontaneously breathing and mechanically ventilated human patients, but not as easy to perform in veterinary patients.

POCUS for Volume Status, Fluid Responsiveness, and Fluid Tolerance

Point-of-care ultrasound (POCUS) has become a practical bedside tool for evaluating cardiac function, volume status, fluid responsiveness, and both left- and right-sided fluid tolerance, while also detecting early complications of fluid therapy. Used serially and in clinical context, POCUS supports fluid therapy decisions from initial resuscitation through de-escalation.

Cardiac POCUS provides rapid assessment of cardiac function and volume status. Subjective evaluation of cardiac contractility, including wall motion and chamber size, helps distinguish hypovolemia from systolic/diastolic dysfunction. In severe hypovolemia, a functional left ventricular “pseudohypertrophy” (small left ventricular chamber and apparent myocardial thickening) and “kissing sign” (end-systolic obliteration) may mimic hypertrophic cardiomyopathy (an uncommon disease in dogs), thereby suggesting critical underfilling or hypovolemia in dogs. The left atrial-to-aortic root ratio (LA:Ao) adds important context: a decreased ratio may support hypovolemia and fluid tolerance, whereas a markedly increased ratio (>2:1) raises concern for volume overload or underlying cardiac disease. When technical expertise is available, aortic velocity-time integral using Doppler echocardiography can estimate SV and track response to a fluid challenge, aiding in the assessment of FR.

Thoracic and abdominal POCUS further refine volume status and fluid tolerance. Increased lung ultrasound B-lines indicate alveolar-interstitial syndrome and may signal developing pulmonary

edema in high-risk or aggressively resuscitated patients. New cavitary effusion or organ edema (e.g. gallbladder halo sign) suggests third spacing from high hydrostatic pressure, low oncotic pressure, systemic venous congestion, and possibly fluid overload.

In spontaneously breathing dogs, a CVC-CI ≥27% predicts FR with high sensitivity and specificity; however, interpretation must consider patient positioning, intra-abdominal pressure, and respiratory effort. Respiratory swings in intrathoracic pressure are transmitted to the right atrium and compliant vena cava; when the vena cava is underfilled, these changes produce marked diameter variation, supporting intravascular volume depletion and likely FR. A distended CVC with minimal variation suggests venous congestion. In humans, venous excess ultrasound score (VExUS) evaluates right fluid tolerance by grading venous congestion via inferior vena cava diameter and Doppler venous flow pattern in portal, intrarenal, and hepatic veins. Estimation of extravascular lung water using lung ultrasound and the evaluation of left ventricular filling pressures (E/A wave, E/E' wave) help in the evaluation of left fluid tolerance.

Integrating All the Pieces

Traditional clinical variables (mentation, HR, CRT, pulse quality, mucous membrane color, extremity temperature) provide a global snapshot of perfusion and are readily available to all clinicians. Modern, responsible fluid therapy should integrate serial assessment of vital signs, laboratory trends (e.g. lactate), POCUS findings, and dynamic surrogates of CO to evaluate both FR and FT and guide management according to the four hemodynamic profiles. Fluid boluses should be viewed as diagnostic and therapeutic tests of circulation rather than reflexive fluid-loading protocols. Response to fluid administration should be reassessed frequently using the same bedside strategies alongside other objective measures (e.g. body weight, urine output), with early transition to vasoactive therapy or fluid de-escalation when indicated.

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Urosepsis

Sandy Young

a critical clinical syndrome, is defined as sepsis caused by an infection originating in the urogenital tract. While well-established in human medicine, its characterization in veterinary medicine is less standardized. Emergency clinicians often encounter septic patients with urinary origins, ranging from pyometra to pyelonephritis. Understanding evolving sepsis definitions, the unique urogenital tract pathophysiology, and the specific therapeutic challenges of blood-prostate barriers is essential.

Historically, veterinary medicine relied on the Sepsis-1 (1991) and Sepsis-2 (2001) definitions, along with the Systemic Inflammatory Response Syndrome (SIRS) criteria to identify sepsis. However, these criteria were found to be too sensitive and lacked specificity. Many hospitalized patients meet SIRS criteria without infection or adverse outcomes, while one in eight critically ill patients with infection and organ failure failed to meet SIRS criteria. Sepsis-3 (2016) redefined sepsis as "life-threatening organ dysfunction caused by a dysregulated host response to infection.**"**

Sepsis-3 emphasizes organ dysfunction, quantified by the Sequential Organ Failure Assessment (SOFA) score. A SOFA score increase ≥ 2 indicates a significantly higher mortality risk. The "qSOFA" (quick SOFA) relies on three criteria: altered mental status, respiratory rate ≥ 22/min, and systolic blood pressure ≤100 mmHg. Validation of these human-centric scores in veterinary patients has shown mixed results.

Cats: qSOFA alone is often insufficient to diagnose sepsis, but it improves accuracy when combined with biochemical markers like bilirubin and creatinine.

Dogs: qSOFA has predictive value in specific conditions, such as pyometra, where mortality odds are higher for patients with a qSOFA ≥2. In surgically treated sepsis, high qSOFA groups have significantly lower survival rates compared to the overall population. Despite the shift in human medicine, there's currently no agreed-upon veterinary definition of sepsis that supersedes Sepsis-1 and 2, though expert committees are working on it. Veterinary-specific scoring systems like the Acute Patient Physiologic and Laboratory Evaluation (APPLE) score correlate with increased mortality.

Pathogenesis and the Urobiome: Contrary to belief, the bladder is not sterile. Healthy dogs have a diverse urobiome, and alterations in it are linked to urogenital disease. The origin of these microbes in dogs is likely similar to humans, involving migration from the gut and vagina. Urosepsis occurs when pathogens overwhelm host defenses, including mechanical flushing, anatomic barriers, mucosal defenses, and prostatic fluid in males. Risk factors include anatomic abnormalities, obstruction, indwelling catheters, and systemic immunosuppression.

Gram-negative bacteria, particularly *Escherichia coli* (Uropathogenic or ExPEC strains), are the most common pathogens in urosepsis. Other common isolates include *Klebsiella, Proteus, Enterobacter,* and *Pseudomonas*. Gram-positive organisms like *Staphylococcus, Streptococcus,* and *Enterococcus* are also seen.

Diagnosing urosepsis involves demonstrating infection within the urogenital tract concurrent with systemic sepsis. Biomarkers like Procalcitonin (PCT) and C-Reactive Protein (CRP) are used in humans, but their utility in veterinary urosepsis is limited. PCT overlaps between septic and non-infectious SIRS dogs, and CRP increases in sepsis but lacks specificity. Therefore, diagnosis relies heavily on clinical signs, imaging, and culture. Three specific clinical presentations are examined: Bacterial Prostatitis, Pyometra, and Pyelonephritis.

1. Bacterial Prostatitis

Prostatitis is most common in intact or recently neutered male dogs with benign prostatic hyperplasia (BPH).

Diagnosis: Clinical signs include caudal abdominal pain, stiff gait, dysuria, and scrotal or prepuce edema. Ultrasound reveals a heterogeneously enlarged prostate with abscess pockets. Prostatic fluid is in the third ejaculate fraction, but prostatic and urine cultures are concordant in only 50% of cases, making direct prostatic sampling valuable despite infection risk. *Other Biomarker:* Canine Prostate Specific Esterase (CPSE) is elevated in prostatic disorders but doesn't differentiate well between BPH, prostatitis, and carcinoma.

Therapeutics and the Blood-Prostate Barrier: Acute inflammation disrupts the blood-prostate barrier, allowing antibiotic penetration. However, as the barrier restores, antibiotics with poor penetration may fail, leading to chronic infection. *Antibiotic Selection:* Empiric choices are lipophilic, weak bases not highly protein-bound. Fluoroquinolones, trimethoprim-sulfamethoxazole (TMS), and macrolides are preferred. Treatment duration is 4–6 weeks, with re-culture recommended 2–3 weeks post-treatment. *Surgical Management:* Large abscesses require drainage (omentalization, marsupialization, or suction). Castration is definitive for BPH-related predisposition, but ideally performed after the acute septic phase to reduce complications. Anti-androgens can shrink the prostate in the interim.

2. Pyometra: Typically occurs 2–4 months post-estrus.

Diagnosis: Ultrasound or radiography reveals a fluid-filled, distended uterus. Dogs may not have a fever. Lethargy, depression, dehydration, and inappetence are common signs. Elevated AST (indicating tissue injury) and low cholesterol are common due to disrupted lipid metabolism from endotoxemia are common clinicopathologic changes. While pyometra lacks specific biomarkers, diagnostics images are the mainstay method for diagnosis. *Stump pyometra* must be considered in spayed females presenting with sepsis. It appears as a heterogeneous mass dorsal to the bladder.

Therapeutics: Ovariohysterectomy is curative, along with antibiotics. Preoperative antibiotics are standard, but the necessity of postoperative antibiotics is debated. *E. coli* remains the primary pathogen, but resistance patterns vary. Amoxicillin-clavulanate resistance may be as high as 38%. Ampicillin or amoxicillin remains a common first-line choice, often yielding good outcomes. Fluoroquinolones are common alternatives. A 2023 study found that surgical site infections occurred exclusively in dogs receiving no antibiotics. Postoperative courses may be minimized if the source is removed and no spillage occurs.

3. Pyelonephritis:

Diagnosis: Definitive diagnosis is challenging. Clinical signs (fever, PU/PD, renal pain) and ultrasound findings (pyelectasia, hyperechogenicity) are supportive but not pathognomonic. In a study of 47 dogs with pyelonephritis, only 72% showed ultrasonographic signs, and pyelectasia was present in only two-thirds of cases. In cats, dilation may be absent in up to 15% of cases. Culture of pyelocentesis is the gold standard, but it carries an 18% risk of minor complications. A study found no animals had a positive renal pelvic culture with a negative bladder culture, suggesting cystocentesis may suffice unless there's ureteral obstruction.

Therapeutics: *E. coli* infection is common in cases (over 50%). The International Society for Companion Animal Infectious Diseases recommends 10-14 days of antimicrobial therapy for acute pyelonephritis, a reduction from previous 4-6 week protocols. Initial therapy often includes fluoroquinolones or third-generation cephalosporins. Clinical response should be seen within 72 hours; lack of improvement warrants investigation for obstruction, abscess, or resistant organisms.

Prognosis: Mortality in human and veterinary urosepsis is lower than sepsis of other origins. In dogs, pyometra mortality is around 12-15%, rising if MODS develops. Septic peritonitis of urogenital origin has a mortality rate of about 50%, comparable to GI-origin septic peritonitis.

Sepsis-Associated Delirium (SAD) is an emerging area of interest. In humans, up to 70% of septic patients experience delirium, linked to neuroinflammation and blood-brain barrier dysfunction. While difficult to quantify in animals, signs of anxiety, restlessness, and vocalization in septic pets may represent SAD. A small study in dogs confirmed neuropathologic changes associated with systemic bacterial infection, suggesting further investigation in veterinary critical care.

Urosepsis, a prevalent and potentially life-threatening condition in small animal practice, requires prompt identification of the septic source, targeted diagnostics (ultrasound, culture), strategic antibiotic stewardship, and timely source control (surgery or medical management). By integrating these principles, emergency clinicians can improve outcomes for these complex patients.

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Pros and cons for medical emergency — How Should We Act in Clinical Practice?

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Introduction

In emergency medicine, clinicians constantly face two major enemies: *incomplete information* and *limited time*. Although evidence-based guidelines provide important direction, real-world cases do not always fit neatly within these frameworks.

This session focuses on two particularly challenging internal medicine emergencies: sepsis and feline arterial thromboembolism (ATE). When translating the latest knowledge into clinical practice, how should we address the dilemmas that cannot be fully resolved by existing evidence? Two frontline emergency veterinarians will present and debate their clinical reasoning and philosophies, while the moderator will further explore the discussion from a practical emergency medicine perspective.

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1. Sepsis: Speed or Certainty?

Early recognition and early intervention are the cornerstones of sepsis treatment; however, the optimal approach remains controversial.

Early Antibiotic Administration vs. Evidence Collection

In septic shock, delays in antibiotic administration are associated with increased mortality. However, initiating treatment before a definitive diagnosis can have drawbacks, including antimicrobial resistance and reduced diagnostic accuracy. Where is the appropriate boundary between a speed-prioritized, life-saving decision and an evidence-prioritized approach?

Sepsis-2 vs. Sepsis-3: Confusion and Evolution in Definitions

In human medicine, the definition of sepsis was significantly revised in 2016, yet the older definition is still widely used in veterinary practice.

- Sepsis-2: Based on the criteria for systemic inflammatory response syndrome (SIRS), emphasizing the *extent of inflammation*. While highly sensitive, it lacks specificity.

- Sepsis-3: Focuses on *organ dysfunction* caused by a dysregulated host response to infection and emphasizes severity assessment using tools such as the qSOFA score.

This session will discuss whether and how these two definitions should be applied in clinical practice and their real-world practicality in emergency settings.

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2. Feline Arterial Thromboembolism (ATE): Aggressive Thrombolysis or Conservative Management?

Feline ATE remains a disease with a poor prognosis, and treatment decisions directly affect the quality of life of both patients and owners.

Thrombolytic Therapy (t-PA, etc.): To Use or Not to Use

Aggressive thrombolytic therapy offers hope for vascular recanalization but carries the serious risk of fatal reperfusion injury. Does aggressive intervention truly increase survival, or does supportive care offer better outcomes? Emergency clinicians will debate both perspectives.

Furosemide: Bolus Administration vs. Continuous Rate Infusion (CRI)

In cardiogenic ATE, managing pulmonary edema is critical for survival. Should clinicians prioritize rapid diuresis with bolus administration or aim for hemodynamic stability with continuous infusion (CRI)? The session will examine how treatment should be tailored based on disease severity and patient background.

Conclusion

In emergency medicine, the “correct answer” lies at the intersection of the patient’s pathophysiology, the owner’s values, and the veterinarian’s clinical judgment. In this pro-con debate, the speakers will openly share their successes and failures, while the moderator organizes the discussion and explores solutions beyond textbook recommendations. Through this debate, we hope to provide clinicians with practical insights that will support their next encounter with emergency cases.

Antimicrobials for ER cases

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Many veterinarians report that they have struggled with the treatment of bacterial infections, yet only a minority report that they have struggled with antimicrobial administration. Ideally, the number of veterinarians answering these two questions should be roughly the same, and herein lies the essence of the problem. In other words, this discrepancy is caused by an insufficient understanding of how bacterial infections should be diagnosed and treated. I have advocated this point for many years, and more than 90% of problems can be resolved by following the principles of infectious disease practice. These consist of five steps: **1. understanding the patient background, 2. identifying the infected organ, 3. identifying the causative microorganism, 4. selecting the antimicrobial agent, and 5. conducting appropriate follow-up.** It is essential to proceed through these steps without reversing the order[1].

What, then, is meant by "judicious use"? It refers to selecting the diseases in which antimicrobials should be used, choosing antimicrobials that are beneficial for those diseases, and determining the treatment duration and evaluation. To gain confidence in this approach, it is necessary to understand the evidence. How many of the following cases represent evidence-based antimicrobial administration?

Metronidazole was administered after *Escherichia coli* was detected in a dog with diarrhea lasting one week.
Amoxicillin/clavulanate was administered to prevent worsening in a dog with acute hemorrhagic diarrhea syndrome.
Cephalexin was administered after *Enterococcus* was detected in the bladder of a dog with spinal cord injury.
Nasal discharge from a cat with rhinitis that began last week was submitted for susceptibility testing, and doxycycline was administered.
Cephalexin was prescribed as postoperative infection prophylaxis after a skin biopsy in a cat.
One week of enrofloxacin was prescribed for infection prophylaxis in a dog with multicentric lymphoma treated with CCNU.

Unfortunately, none of the above represents evidence-based antimicrobial administration. In this lecture, I will explain why these practices lack evidence, how clinicians should think about antimicrobial therapy, and present the speaker's real-world examples in which judicious antimicrobial use led to a reduction in antimicrobial-resistant bacteria [2].

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Essential Blood Smear Evaluation in Emergency Practice — The Role of Veterinary Nurses in Balancing Speed and Accuracy

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Background and Objectives

In emergency medicine, hematologic data serve as a critical “compass” for understanding the patient’s condition, and rapid availability of these data is directly linked to survival outcomes. Although automated hematology analyzers (CBC) are widely used in veterinary hospitals and provide rapid quantitative results, crucial diagnostic information often lies within cellular morphology that cannot be fully captured by automated systems alone. In particular, early indicators of life-threatening conditions such as immune-mediated hemolytic anemia (IMHA), sepsis, and disseminated intravascular coagulation (DIC) can often be detected through blood smear evaluation.

However, in the time-critical environment of emergency practice, prioritizing speed may compromise accuracy, while excessive focus on accuracy may delay critical decisions. This lecture discusses practical strategies by which veterinary nurses (VNs) can balance speed and accuracy throughout the entire process—from smear preparation and microscopic evaluation to timely clinical alerts to veterinarians—and highlights key points that improve diagnostic efficiency in emergency settings.

Improving Speed: Optimization of In-Hospital Protocols

Enhancing efficiency begins with optimizing pre-analytical protocols. Appropriate anticoagulant selection, rapid preparation of high-quality smears, and standardized staining procedures are essential for minimizing turnaround time. In emergency cases, cellular morphology can deteriorate quickly due to artifacts if smear preparation is delayed after sample collection. Therefore, immediate smear preparation is critical.

In addition, rushed preparation can lead to poor-quality smears that necessitate repeat testing and cause significant time loss. Standardizing the technique for reliably producing a proper monolayer region suitable for evaluation ensures consistent information quality and ultimately allows faster and more accurate interpretation.

Improving Accuracy: Establishing Systematic Morphologic Evaluation

To enhance diagnostic accuracy, VNs must have clear guidelines regarding what morphological changes should be prioritized during microscopic examination. This lecture focuses on identifying qualitative “panic values” that may indicate life-threatening conditions within three major cell categories.

1. Red Blood Cell (RBC) Evaluation

RBC morphology often reflects the severity of underlying pathology. Spherocytes, a key indicator suggesting IMHA, should be carefully evaluated. Fragmented erythrocytes such as schistocytes and keratocytes may indicate intravascular hemolysis or microangiopathic processes, including DIC and hemangiosarcoma. Additionally, the presence of Heinz bodies and eccentrocytes, commonly associated with oxidative injury such as onion toxicity, can provide valuable diagnostic clues that complement clinical history.

2. Platelet Evaluation

An important role of VNs is determining whether thrombocytopenia reported by the analyzer represents true thrombocytopenia or pseudothrombocytopenia. Examination of the feathered edge of the smear helps identify platelet clumping or the presence of large platelets, both of which can cause falsely low platelet counts.

3. White Blood Cell (WBC) Evaluation

Indicators of severe inflammation or sepsis, such as toxic changes in neutrophils, must not be overlooked. Cytoplasmic vacuolation, basophilia, and toxic granulation reflect the intensity of the inflammatory response. Accurate evaluation of a left shift—an increase in band neutrophils—and differentiation from segmented neutrophils is also essential. Furthermore, the presence of nucleated red blood cells or circulating neoplastic cells in peripheral blood can signal significant disease progression and should prompt immediate clinical attention.

Conclusion

The role of veterinary nurses in emergency medicine extends beyond performing laboratory procedures. VNs are responsible for extracting critical signals indicating life-threatening conditions from laboratory findings and accelerating clinical triage. By systematically evaluating blood smears and promptly reporting qualitative panic values to veterinarians, VNs can significantly shorten the time to diagnosis and intervention. Mastery of blood smear evaluation enhances the professional contribution of VNs as essential members of the emergency care team. This lecture aims to support faster clinical decision-making and ultimately improve patient survival in emergency veterinary practice.

Blood Chemistry in Clinical Practice

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Blood testing serves as a fundamental diagnostic tool that supports every stage of medical care—from assessing the patient's condition, establishing a diagnosis, determining a treatment plan, and evaluating therapeutic response, to estimating prognosis. Among these tests, blood biochemistry is particularly important, as it enables broad assessment of organ function and metabolic abnormalities by measuring serum enzymes, proteins, glucose, lipids, electrolytes, and other analytes.

In actual clinical practice, however, clinicians must interpret a vast amount of information simultaneously. This includes not only biochemical profiles with numerous parameters, but also hematology, blood gas analysis, physiological tests such as ECG and blood pressure measurement, and various imaging modalities. In emergency site, where many patients are in danger of life, rapid and accurate decision-making is essential. In such situations, awareness of "panic values" becomes critically important.

Panic values—also referred to as critical values or emergency alert values—are abnormal laboratory results that indicate a condition so dangerous that it may immediately threaten the patient's life. Because these abnormalities can lead to fatal outcomes within a short time, prompt notification and intervention are mandatory when they are detected. Each medical facility must determine which parameters should have panic value thresholds and define appropriate cut-off levels based on its clinical environment and patient population.

In emergency medicine, where quick judgment is required, it is not practical to review all laboratory parameters sequentially. Instead, it is more efficient to prioritize interpretation in the following order:

1. Parameters directly related to life-threatening conditions
2. Parameters necessary for assessing overall systemic status
3. Parameters required for differential diagnosis

This lecture focuses on four life-critical biochemical parameters frequently encountered in emergency settings—electrolytes (particularly potassium), blood glucose, calcium (ionized calcium), and lactate—and discusses their clinical significance and practical interpretation.

Potassium

Abnormal potassium levels—whether increased or decreased—represent a critical electrolyte disturbance that can endanger life. Both hyperkalemia and hypokalemia affect the muscles, nervous system, and heart, and arrhythmias in particular may become fatal.

Treatment of hyperkalemia can be broadly divided into three categories:

- Cardioprotection, typically achieved through administration of calcium gluconate
- Shifting potassium into cells, using therapies such as glucose–insulin infusion, β_2-agonists, or sodium bicarbonate
- Eliminating potassium from the body, through diuretics or dialysis

Blood Glucose (Glucose)

Neurons, including those in the brain, rely almost exclusively on glucose as their primary energy source. Therefore, hypoglycemia impairs normal neuronal function and can lead to severe brain disorders such as seizures, stupor, and coma, which may become life-threatening.

In such cases, rapid vascular access and intravenous administration of glucose are essential. However, if the underlying cause of hypoglycemia persists, blood glucose may drop again, making repeat measurement after a short interval crucial.

Calcium (Ionized Calcium)

Approximately 40% of serum calcium is bound to proteins, about 10% forms complexes with anions, and the remaining 50% exists in a free, biologically active form known as ionized calcium. Because only ionized calcium has physiological activity, accurate assessment of calcium status requires measurement of ionized calcium rather than total serum calcium.

A characteristic manifestation of hypocalcemia is tetany, a state of neuromuscular hyperexcitability that causes limb stiffness, muscle spasms, and paresthesia. Hypocalcemia may also induce ECG changes such as QT interval prolongation, and in severe cases can progress to cardiac arrest.

Lactate

Lactate is a metabolic byproduct generated during glycolysis when cells experience oxygen deficiency and shift to anaerobic metabolism. Under normal conditions, lactate is rapidly metabolized by the liver and kidneys, keeping blood concentrations low.

However, in circulatory failure caused by shock or cardiovascular and respiratory dysfunction, lactate production increases. If hepatic or renal impairment is present, lactate clearance decreases, leading to marked elevation of blood lactate and the development of lactic acidosis.

Lactate concentration is widely used in clinical practice as a sensitive indicator of tissue hypoperfusion. It plays a crucial role in assessing the severity and prognosis of shock, sepsis, and critically ill patients. Persistent hyperlactatemia is strongly associated with increased mortality, making lactate one of the most important biomarkers in emergency and ICU medicine.

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Dystocia Management and Canine & Feline Neonatal Resuscitation

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Obstetrical emergencies, particularly dystocia, represent a critical area in veterinary emergency medicine requiring rapid decision-making and intervention. Dystocia is one of the most common emergencies encountered during parturition, and early diagnosis and appropriate treatment strongly influence the prognosis of both the dam and neonates. The incidence of dystocia is reported to be approximately 5% in dogs and 3–6% in cats, with increased risk in older primiparous bitches, small breeds, and brachycephalic breeds, and cesarean section rates exceeding 80% in breeds such as Bulldogs and French Bulldogs (1–4). This lecture outlines the diagnostic approach to dystocia and the fundamental principles of neonatal resuscitation.

The gestation period is approximately 63 days from ovulation in dogs and 65 days in cats. Accurate knowledge of the estrous cycle and ovulation timing is essential for predicting parturition and preventing dystocia. Parturition is generally divided into three stages: stage I (latent phase), stage II (expulsion phase), and stage III (placental expulsion). Stage II typically lasts 3–12 hours in dogs and 4–16 hours in cats, and dystocia should be suspected when strong contractions persist for more than 60 minutes without fetal delivery or when more than 3 hours elapse between deliveries (3,4).

Causes of dystocia are categorized as maternal (approximately 75%) or fetal (approximately 25%). Uterine inertia is the most common maternal cause and is associated with primiparity, advanced age, hypocalcemia, and hypoglycemia. Primary uterine inertia occurs at the onset of labor, whereas secondary uterine inertia develops after partial delivery due to uterine fatigue or hypocalcemia. Other maternal causes include pelvic narrowing and uterine torsion. Fetal causes include malpresentation, fetal oversize, congenital anomalies, and fetal death (3–5).

Early recognition of clinical signs of stalled labor is essential. Immediate intervention is required when strong contractions persist for more than 30 minutes without delivery or when dark green vaginal discharge appears without fetal expulsion. Diagnostic evaluation includes physical and vaginal examination, radiography to assess fetal number and positioning, and ultrasonography to evaluate fetal heart rate. A fetal heart rate below 180 bpm indicates fetal distress, and rates below 160 bpm warrant emergency intervention (3).

Treatment depends on maternal and fetal status. When obstruction is absent and fetal heart rates are adequate, medical management including fluid therapy, glucose, calcium, and oxytocin may be attempted; however, oxytocin must be used cautiously to avoid uterine rupture. When fetal heart rate declines or mechanical obstruction is present, early cesarean section is recommended, and surgery is required in approximately 60–80% of dystocia cases (3–5).

Newborns delivered after dystocia or cesarean section frequently experience apnea and bradycardia due to failure of cardiopulmonary transition, making resuscitation critical for survival. Resuscitation following RECOVER guidelines is recommended. The core principles include establishing ventilation, maintaining body temperature, and supporting circulation. Neonates should be warmed to 35–37 °C, stimulated with towel rubbing, and gently suctioned if needed. Positive pressure ventilation should be initiated immediately in cases of respiratory failure or heart rate <120 bpm. Chest compressions are indicated when heart rate is <60 bpm, with a recommended compression-to-ventilation ratio of 4:1. Post-resuscitation care includes prevention of hypothermia, hypoglycemia, and dehydration, continued warming, glucose supplementation, and consideration of reversal agents when anesthetics have been used (6).

Veterinary obstetrical emergencies are unique in requiring simultaneous care for the dam and multiple neonates, demanding knowledge that integrates both obstetrics and emergency medicine. Appropriate diagnosis, timely intervention, and effective neonatal resuscitation markedly improve survival outcomes. A coordinated team approach involving veterinarians and veterinary nurses is essential for delivering high-quality emergency care (3,6).

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Neurological Examination in ER

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Introduction
A complete neurological examination requires extensive knowledge and technical skill and is often difficult to quantify, making it a highly specialized assessment. How much of this examination should veterinary nurses working in emergency and critical care understand? Ideally, a broad and deep understanding is beneficial. Emergency departments frequently encounter patients presenting with neurological signs such as status epilepticus, increased intracranial pressure, and vestibular disorders. In the intensive care setting, severe non-neurological diseases may also manifest neurological symptoms. Therefore, it is important for veterinary nurses to understand the fundamentals of neurological examination, particularly those elements related to assessing urgency. This lecture highlights key components of the neurological examination sheet developed by the Japanese Society of Veterinary Neurology that are especially relevant for emergency veterinary nurses.

Observation
A neurological examination begins with careful observation of the patient. Many neurological abnormalities can be detected visually, and in critically ill patients it is essential to obtain as much information as possible while minimizing stress and handling. This section focuses primarily on assessing the level of consciousness, as well as selected involuntary movements and vestibular signs.

Postural Reactions
Determining whether abnormalities observed in emergency or ICU patients are due to neurological dysfunction can be deceptively challenging. As noted above, many neurological abnormalities are identified through observation, and these findings contribute to the assessment of neurological involvement. Among neurological tests, postural reactions—such as proprioceptive positioning—serve as screening tools to evaluate the presence of neurological dysfunction. However, caution is required: severely compromised systemic conditions may falsely appear as abnormal postural reactions, and lesions in areas such as the olfactory bulb may not produce detectable abnormalities in these tests.

Pupillary Assessment
Pupillary abnormalities are critical indicators when assessing urgency. However, evaluating urgency based solely on pupillary findings is insufficient. Abnormal pupils may result not only from neurological disorders but also from primary ophthalmic disease. Therefore, pupillary assessment must be interpreted alongside additional neurological and clinical findings. This lecture will discuss the key points necessary for accurate interpretation of pupillary abnormalities in emergency settings.

Common Intoxications in Daily Practice

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Objectives

1. To review common toxicities frequently encountered in clinical practice
2. To understand triage and monitoring in toxicology cases
3. To learn about advanced treatment options for poisoning

Introduction
Poisoning cases are among the most frequently encountered emergencies in veterinary practice. According to data from our hospital, toxicities account for approximately 15.7% of nighttime emergency visits. Depending on the toxin, clinical signs may develop rapidly after ingestion, and previously healthy animals can deteriorate quickly. However, prognosis can improve significantly when the causative agent is identified promptly and appropriate initial treatment is initiated. This lecture summarizes the fundamental principles and practical management of commonly encountered toxicities in daily practice.

Common Toxicities in Clinical Practice
Many toxic exposures in dogs and cats involve household foods, medications, and plants. Frequently encountered toxins with the potential to cause severe illness include chocolate, xylitol, onions and garlic, NSAIDs, acetaminophen, lilies, and grapes/raisins. This lecture reviews the clinical signs, toxic doses, and essential monitoring parameters that veterinary nurses should understand. Knowledge of toxin-specific characteristics is essential for early management and treatment planning.

Triage and Monitoring in Toxicology
Toxicology management requires simultaneous identification of the toxin and assessment of patient urgency. Triage is performed using the ABCDE approach combined with the MATTERS history framework.
The ABCDE approach evaluates Airway, Breathing, Circulation, Dysfunction of the central nervous system, and Exposure/Environment to determine whether the patient is at immediate risk of death. The MATTERS history includes: Medication/Toxin, Amount, Time of ingestion, Therapy already given, Emesis, Route/Reason, and Symptoms/Signs. Gathering accurate information using this framework is critical. Monitoring should be tailored to the specific toxin and may include assessment of mentation, hemodynamics, blood glucose, renal and hepatic function, and urine output to detect early clinical deterioration.

Advanced Treatment Options in Toxicology
The foundation of poisoning treatment is decontamination and supportive care; however, some cases require advanced therapies. Antidotes, intravenous lipid emulsion therapy, and extracorporeal blood purification (hemodialysis) may be indicated depending on the toxin. When an antidote is available, early consideration can significantly expand treatment options. Intravenous lipid emulsion therapy is effective for lipophilic toxins such as ivermectin, NSAIDs, and local anesthetics. Hemodialysis may be considered in cases of life-threatening ingestion or poor response to treatment. Appropriate patient selection is essential, but these therapies can markedly improve outcomes when used correctly.

Conclusion

Toxicology is a race against time, and early triage and appropriate initial management strongly influence prognosis. Veterinary nurses who can apply the ABCDE approach and MATTERS history, recognize toxin-specific clinical signs, and perform appropriate monitoring play a vital role in saving animal lives. It is hoped that this lecture will provide practical knowledge that can be immediately applied in clinical practice.

Conflict of Interest Statement

The author declares no conflicts of interest related to this presentation.

Nursing Care for Infectious Diseases

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Infection control is based on standard precautions and transmission-based precautions, and this principle applies equally in emergency medicine. In emergency settings, decisions often must be made rapidly with limited time and personnel, frequently before diagnostic results are available. Therefore, infection control is not an optional task performed only when time permits; rather, it is a core element that directly influences the quality of emergency care. When a potential infectious disease is first suspected, infection control measures should already be in progress. All animals presented to a hospital should be managed under the assumption that their infectious status is unknown, and standard precautions should be consistently and instinctively applied.

Effective infection control requires four essential components: knowledge, practical application, simulation, and teamwork. In emergency practice, these elements must be integrated and applied instantly. For example, leptospirosis is a bacterial zoonosis that can infect many mammals, including humans, and in veterinary hospitals canine urine is a major source of exposure. Transmission may occur through skin or mucous membranes and potentially via aerosols. The pathogen survives for long periods in moist environments but is susceptible to common disinfectants. While such knowledge is fundamental, the most critical skill in clinical settings is the ability to immediately determine what actions must be avoided when managing a suspected case. Although the level of infection control may be limited by facility resources and staffing, certain practices must always be avoided to prevent nosocomial infection. Establishing shared protocols and conducting regular team simulations enable confident and coordinated responses during emergencies.

In recent years, cases of Severe Fever with Thrombocytopenia Syndrome (SFTS) in animals have been reported across Japan, and infections among veterinary personnel have occurred. Many cases are believed to have resulted from inadequate adherence to standard precautions during initial management prior to definitive diagnosis, including insufficient use of personal protective equipment (PPE) and direct exposure to blood or bodily fluids. These infections often occurred not due to lack of knowledge, but due to delayed suspicion and insufficient implementation of standard precautions. When SFTS is suspected, animals must never be handled without gloves, and procedures such as venipuncture or sample handling must not be performed without appropriate PPE. Rapid decisions regarding isolation, required PPE levels, and designation of contaminated areas must be made and communicated clearly to the entire team. The ability to lead these decisions and actions is critical to preventing hospital-acquired infection.

Veterinary nurses play a central role in infection control by protecting animals, clients, and veterinary staff. This lecture presents practical strategies that can be implemented in clinical settings, including decision-making criteria and team preparedness. Using leptospirosis and SFTS as representative examples of infections encountered in emergency practice, the lecture discusses what veterinary nurses can do during initial management, which actions must be strictly avoided, and how effective infection control can be implemented even with limited personnel and resources.

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Medical management of airway obstruction

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Respiratory distress is a common presenting complaint in emergency veterinary practice. Among these cases, upper airway obstruction represents a particularly high-risk condition that can rapidly progress from severe hypoxemia to cardiopulmonary arrest. Because even a brief delay may be life-threatening, airway assessment and stabilization (“A: Airway”) is the highest priority within the ABCD emergency approach.

What is expected of veterinary nurses when faced with such patients? Simply waiting for instructions from a veterinarian may result in the loss of lives that could otherwise be saved. Veterinary nurses play a critical role in early recognition, preparation, and team-based emergency response.

This seminar reviews common diseases that cause upper airway obstruction, including brachycephalic airway syndrome, laryngeal paralysis, and tracheal collapse, as well as associated complications. Clinical indicators used to assess urgency and severity will be discussed. In addition, practical emergency interventions—such as oxygen therapy, endotracheal intubation, and temporary tracheostomy—will be introduced as essential components of team-based care.

By connecting knowledge of diseases and pathophysiology into a practical clinical framework, this lecture aims to help veterinary nurses approach airway emergencies with confidence. The goal is to transform uncertainty and anxiety into logical, evidence-based decision-making, enabling veterinary teams to respond effectively to life-threatening airway emergencies.

CPR in practice

Yuto Mori, Rumi Nakajima

In cardiopulmonary resuscitation (CPR), attention is often focused on direct interventions such as chest compressions and drug administration. In reality, however, CPR is a highly coordinated team effort involving multiple professionals working simultaneously within a limited timeframe, and non-procedural elements greatly influence both the quality and safety of resuscitation. The role of overseeing the entire situation, organizing information, and supporting the flow of care is essential. Veterinary nurses are not merely assistants who follow instructions; they are key team members who recognize subtle changes in the patient, communicate these findings, and, when necessary, prompt the initiation of CPR. Daily clinical observation skills and preparation for potential emergencies contribute to rapid initial response and significantly affect patient outcomes.

A shared understanding of the RECOVER guidelines is fundamental for the entire team. Without familiarity with the recommended workflow and priorities, decision-making may be delayed and confusion may arise during resuscitation. Veterinary nurses who understand the RECOVER guidelines can anticipate upcoming procedures, prepare necessary equipment and medications in advance, and support veterinarians' decision-making, thereby improving the overall quality of CPR. Such proactive involvement also strengthens team cohesion.

This lecture discusses practical strategies to ensure safe and effective CPR from the perspective of veterinary nurses. Particular emphasis is placed on the role of the timekeeper, who maintains situational awareness, tracks elapsed time, and manages transitions between interventions. The importance of accurately recording events—such as medications administered, timing of interventions, and return of spontaneous circulation (ROSC)—is also highlighted. These responsibilities are not merely supportive tasks but central nursing roles that sustain the resuscitation process and support clinical decision-making.

Documentation during CPR serves not only for real-time information sharing but also as an essential resource for post-resuscitation debriefing. Objective records enable teams to identify challenges regardless of whether ROSC is achieved, allowing lessons learned to be applied to future cases. Continuous reflection based on accurate documentation enhances institutional CPR performance and strengthens educational systems beyond individual experience.

Through this lecture, we aim to reaffirm the importance of preparation, shared understanding of RECOVER guidelines, early recognition and decision-making, and systematic documentation and debriefing. These perspectives highlight the proactive role veterinary nurses play within CPR teams and provide practical insights that can be applied in daily clinical practice.

TOP 10 TIPS FOR A SUCCESSFUL OUTCOME IN SEPSIS

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Sepsis is a potentially devastating disease syndrome, associated with high morbidity and mortality in both human and small animal veterinary medicine. There is growing evidence based medicine regarding best practice for management of people with sepsis and that outcome can be improved by implementing treatment recommendations.[1] The tips presented here are based on recommendations from the Surviving Sepsis Campaign and personal experience.[1]

1. Have a high index of suspicion for sepsis. Sepsis in veterinary medicine is classically recognized / diagnosed on the basis of fulfillment of criteria for the systemic inflammatory response syndrome (SIRS) in the presence of a suspected or confirmed infection. SIRS is essentially diagnosed on the basis of meeting 2 or 3 out of 4 of the following clinical criteria; notably an abnormal temperature, heart rate, respiratory rate or a change in the leukon (leukocytosis, leucopenia, or significant left shift).[2, 3] Identification of SIRS criteria in any dog or cat should encourage a proactive diagnostic approach to rule in or out infection. In some cases the source of infection is immediately obvious, while in others it is not. In addition to a thorough physical examination, the preliminary search for infection may include point-of-care (POC) infectious disease testing (FIV/FeLV in cats, 4Dx in dogs), thoracic radiographs (looking for evidence of pneumonia, especially if lower respiratory signs), abdominal ultrasound (looking for free fluid – septic peritonitis, hepatobiliary sepsis etc.), urine sediment examination and/or evaluation of any abnormal fluid accumulations. Infection can be definitively diagnosed on the basis of cytology (intracellular bacteria), culture or less commonly histopathology. The sepsis 3.0 definitions in human medicine no longer use SIRS criteria but instead define asepsis as “Life-threatening organ dysfunction caused by a dysregulated host response to infection” as use the sequential organ failure assessment (SOFA) score as part of the clinical criteria.[4]

2. Obtain samples for culture prior to (but don’t delay) initiation of antibiotics. This may include abdominal fluid, airway lavage fluid, joint fluid, pleural fluid, urine and/or blood, depending on the case. Even if you are not sure if you are going to submit samples, it is best to obtain them upfront. If you are submitting, submit promptly so as to ensure results ASAP. Although blood cultures are not used as commonly in veterinary medicine, it is recommend to collect them prior to antimicrobial therapy, particularly when the sources of sepsis is not obvious. Recent literature reports blood culture findings in dogs and cats.[5-7]

3. Give appropriate IV antibiotics early. Perhaps the single most important factor in stabilizing the septic patient (following early recognition of sepsis) is early appropriate use of parenteral antibiotics. This cannot be overemphasized, especially in the emergency room setting. Evidence in people with severe sepsis and septic shock suggests that every hour delay on administration of appropriate antibiotics increases mortality quite significantly.[8, 9] It is not just about giving an antibiotic, rather you need to give an appropriate antibiotic.[10] This means understanding the origin of sepsis and the likely pathogens involved. There is evidence to suggest that using a combination of antimicrobials to optimize both gram negative and gram positive coverage is ideal.[11] Some recent literature also suggests benefit of early antimicrobials in sepsis in dogs,[12] which had not been previously documented[13]. It is also vital to be aware of local resistance patterns, to ensure that the empiric antibiotics that you choose are likely to be effective against the suspected pathogen. For animals that develop hospital acquired sepsis empiric antibiotic choice is usually based on the assumption of multidrug resistant (MDR) organisms, including methicillin resistant Staphylococcus species, as well as resistant *E.coli* and Enterococcus species.

4. Identify and treat shock early and aggressively. The surviving sepsis campaign describes a 1 hour bundle for the initial resuscitation of patients with sepsis and septic-shock. After obtaining

blood cultures and giving antibiotics it recommends IV fluid resuscitation and administration of vasopressors based on the patient's conditions. It is important to identify clinical signs of shock including abnormal perfusion parameters (eg. tachycardia, weak pulses, abnormal CT, depressed level of consciousness), as well as measuring blood pressure and blood lactate to aid in documentation of hypotension and hypoperfusion. Remember that cats in shock look different to dogs. If signs of shock are present then resuscitation should be to end-points based on serial monitoring of the aforementioned parameters. IV fluid resuscitation starts with a balanced isotonic crystalloid. Then don't be afraid to administer vasopressor agents following restoration of adequate intravascular volume. Noradrenaline is generally used as the first line vasopressor, with a target MAP of ≥ 65 mmHg.

5. Instrument early. Sepsis and septic shock require multimodal pharmacologic intervention, and management of organ dysfunctions is challenging and intensive. For the hemodynamically unstable patient you will need multiple sites of vascular access, so why wait. Get lines in early, before the edema starts, since it only gets progressively more challenging. It is also imperative to be prepared if going into the operating room (OR). Having an aseptically placed multiple lumen sampling catheter is particularly valuable since you will be rechecking blood work frequently. Arterial catheterization is also recommended if giving vasopressors for septic shock or during anesthesia. Additionally placing a urinary catheter can be very helpful in septic patients to monitor urine output (UOP) given the risk of acute kidney injury (AKI). It is also imperative to de-instrument appropriately. That is, once you patient has 'turned the corner' start to progressively remove 'lines' and tubes that are no longer needed.

6. Ensure early source control. Following identification of sepsis, sample collection, administration of IV antibiotics and resuscitation from shock, the next step of management of sepsis is source control. Without effective source control you are unlikely to have treatment success in situations of an enclosed infection (e.g. septic peritonitis, septic arthritis, pyothorax etc.). This too must be pursued as soon as is reasonably possible following initial stabilization of the patient. This often means emergency surgery; the septic focus cannot wait! Since source control often requires surgery (e.g. exploratory celiotomy or thoracotomy), provision of analgesia becomes an important part of management, so consider your options here in regards to efficacy of analgesics, effects on the immune system, and side effects.

7. Don't trust a post-op GI surgery. Dehiscence following enterotomy, or intestinal resection and anastomosis are some of the more common causes of hospital acquired sepsis (R&A > enterotomy > gastrotomy). Early detection of dehiscence is vital to maximize chances of successful management, however sometimes this is hard. We don't exactly know the best indicator of dehiscence but worrisome signs in the post-operative period include new onset vomiting, ongoing anorexia or new anorexia when they had been eating, development of a fever when they had been afebrile and a dramatic increase in drain output when there had been relatively constant or decreasing drain output (from closed suction abdominal drains). Obviously identifying septic peritonitis requires identification of septic (intracellular bacteria) suppurative abdominal fluid and then dehiscence is confirmed on re-explore. Remember that risk factors for dehiscence (albeit inconsistently reported) include hypoalbuminemia, per-operative peritonitis, intestinal foreign body, hypotension and corticosteroid administration.[14]

8. Be vigilant for hospital acquired sepsis. In addition to hospital acquired abdominal sepsis secondary to dehiscence, other sources of HA sepsis include catheter associated blood stream infections, urinary tract infections, incisional infections and pneumonia (including aspiration and ventilator associated pneumonia). Sources of vascular access should checked daily for evidence of infection. Additionally they should be inspected / interrogated if a hospitalized pet develops a new onset fever. Central venous catheters must be placed with strict aseptic technique. They should be bandaged and the bandages changed daily. Unused catheters should be removed. The risk of

incisional infections is minimized by having incisions covered with sterile adhesive dressings while the animal is in the ICU. If an incisional infection develops, the site should be explored in an OR, and you should be prepared to go into the body cavity (for abdominal and thoracic surgeries). Ultrasound can also be used to guide fluid collection for cytology and assess deep tissue involvement.[15] Sometimes the skin is just the tip of the iceberg.

9. Don't underestimate the 2nd hit. Our septic patients live on the edge; while they may present to you more or less stable, it is likely that their body is only just coping with the pathology it is experiencing. Anything that we do to place further physiologic stress on the animal has the potential to result in destabilization. Perhaps the most common 2nd hit scenario is general anesthesia and the potential for associated vasodilation, hypotension, hypothermia and hemorrhage. While we generally can't avoid the second hit, minimizing its severity (i.e. minimizing hypotension, maintaining body temperature and transfusing aggressively in the event of significant intraoperative blood loss) is imperative. Additionally monitoring for the development of complications in the early post-operative period is critical.

10. Be prepared for multiple organ dysfunction syndrome (MODS). While there is not a consensus definition for what denotes an organ dysfunction in veterinary medicine, things to consider include the list below modified from Kenney et al.[16]:

- Renal dysfunction (rise in [Creatinine] > 0.5mg/dL, in the absence of pre-renal or post-renal causes)
- Cardiovascular dysfunction (shock, myocardial dysfunction)
- Respiratory dysfunction (requirement of supplemental oxygen or mechanical ventilation)
- Hepatic dysfunction (Tbili >0.5mg/dL)
- Coagulation dysfunction, DIC (thrombocytopenia, prolonged PT, PTT or ACT, abnormal viscoelastic test results)
- Gastrointestinal dysfunction (vomiting, regurgitation, ileus, constipation, diarrhea)
- Septic encephalopathy; and
- Endothelial dysfunction (vascular leak).

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Hospital-Acquired AKI
Dr. Corrin Boyd

Acute kidney injury (AKI) is associated with substantial morbidity and mortality.
Hospital-acquired AKI refers to the development of AKI as a complication during hospitalization for treatment of another illness. Rather than having a sole causative agent, it is usually multifactorial in etiology. It has the potential to complicate the management of the original underlying disease and increase both morbidity and mortality. Proactive surveillance is necessary for recognition, given the subtle signs and insensitivity of current diagnostic modalities. Management involves optimizing physiology to provide an ideal environment for nephron healing.

Prevention
Prevention of hospital-acquired AKI relies upon recognizing risk factors and eliminating or reducing their effects wherever possible. It has long been recognized that hypovolemia has the potential to cause AKI. However, the potential devastating impact of hypovolemia on the kidney has led to a somewhat oversimplified perception that 'intravenous fluid therapy (IVFT) is good for the kidney'. Hypervolemia and fluid overload are also detrimental to the kidney, which is exceptionally susceptible to the adverse effects of oedema due to its rigid capsule. Thus, hospital-acquired AKI is minimized when IVFT aims to achieve and maintain normal *blood volume* (normovolemia/euvolemia) and normal *interstitial hydration* (normohydration/euhydration). In human critical care this has led to the formation of a conceptual model for IVFT that features four distinct phases: the ROSE model. ROSE is an acronym for the distinct phases of Rescue, Optimization, Stabilization, and de-Escalation (or Evacuation). Choice of fluid type may also impact the development of hospital-acquired AKI. Two factors that have gained substantial interest are the use of synthetic colloid fluids and high-chloride crystalloids such as 0.9% NaCl. Nephrotoxic medications should be avoided where a suitable alternative exists, but individual risk/benefit analysis remains important. The underlying disease still must be adequately treated. Limiting to the lowest effective dose for the shortest effective duration can reduce the risk. The contribution of the underlying disease is complex. Systemic inflammatory diseases, notably sepsis, have the strongest association. Whilst previously thought to primarily be due to ischemia, growing evidence suggests the mechanism is more complex. No specific treatments directed at prevention of sepsis-associated AKI have proven successful. At this point, prompt attention should be given to the core principles of sepsis management: intravenous antimicrobial therapy, source control, and hemodynamic optimization.

Recognition
Effective management of hospital-acquired AKI is facilitated by early recognition. This is aided by close attention to subtle indications of renal dysfunction. Electrolyte imbalances may indicate decreased tubular function. The urinalysis may show evidence of proteinuria, glucosuria, or casts. Urine output may trend downwards despite normovolemia. These findings should prompt the consideration of hospital-acquired AKI and evaluation of a marker of GFR: serum creatinine and/or symmetric dimethylarginine (SDMA) concentration. Creatinine is an insensitive marker of GFR, but this sensitivity is increased by use of scoring systems that emphasize small changes, even if they are within the reference interval. Veterinary scoring systems have been developed, which allow diagnosis with a serum creatinine increase of only 26.4 µmol/L (0.3 mg/dL). There has been substantial research into biomarkers that could allow earlier detection of hospital-acquired AKI, such as neutrophil gelatinase-associated lipocalin (NGAL) and cystatins B and C.

Management
The treatment of hospital-acquired AKI centers on optimizing the conditions for renal recovery. Any suspected causative agent should be withdrawn or treated. IVFT should aim to maintain euvolemia and euhydration, and careful attention must be paid to avoiding fluid overload in patients that are

oliguric or anuric. Nephrotoxic medications should be avoided where possible. Electrolyte disturbances should be treated and monitored closely. Clinical signs such as vomiting may require symptomatic management. Indications for renal replacement therapy may include severe azotemia, fluid overload with oliguria or anuria, and refractory hyperkalemia. An understanding of the time course of AKI is important for assessing response to therapy and long-term outcome. Improvement in GFR, and therefore serum creatinine concentration, should not be expected until the recovery phase at least a week after the initial insult. Despite the potential for nephron regeneration, it is unlikely that any animal that has sustained clinically significant AKI returns to having a completely normal number of functional nephrons. Staging for chronic kidney disease is recommended in all animals that have sustained a hospital-acquired AKI. Even if they return to being non-azotemic, these animals should be managed according to the recommendations for IRIS stage 1 (non-azotemic) chronic kidney disease.

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Ophthalmology emergency

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Abstract
In daily ophthalmic practice, emergencies typically present with three major categories of signs: acute pain, acute vision loss, and changes in ocular appearance. These conditions often reduce not only quality of vision (QOV) but also the patient's quality of life (QOL). When confronted with acute ocular pain or vision loss, the most critical initial step is to determine whether the cause is a primary ocular disease—such as primary glaucoma or rhegmatogenous retinal detachment—or secondary to systemic or neurological disease, including encephalitis, diabetic cataracts, or lymphoma, or due to external factors such as trauma, animal fights, or ocular infections.

A key principle in the diagnosis and treatment of ophthalmic emergencies is the use of logical clinical reasoning rather than a "one diagnosis–one treatment" approach. Even with the same diagnosis, treatment and prognosis should be determined by evaluating the underlying cause, clinical findings, disease progression, laterality (unilateral vs. bilateral), ocular versus systemic involvement, and epidemiological factors. These considerations must be integrated into a treatment plan and clearly communicated to the owner, including expectations for prognosis.

For example, bilateral uveitis may indicate life-threatening systemic disease such as lymphoma or severe infection, and ocular findings may lead to the diagnosis of systemic illness requiring long-term treatment. In a study of 18 dogs with sports ball–related ocular trauma, globe rupture and hyphema were associated with enucleation rates of 83% and 71%, respectively, both indicating poor visual prognosis.[1] Corneal ulcers also vary widely in severity: some heal within days with simple topical therapy, while melting ulcers can progress rapidly and become vision-threatening. In cases of glaucoma or retinal detachment, delayed or inappropriate initial management may lead to permanent blindness and significantly impact the veterinarian–client relationship, potentially resulting in legal disputes.

Common misconceptions in primary practice also affect outcomes. For instance, cataracts are sometimes described as a benign condition in older animals that can be left untreated. However, secondary complications such as uveitis, lens luxation, and secondary glaucoma may develop, and early surgical intervention could have prevented pain and blindness in some cases referred to specialty ophthalmology services.

Veterinarians must cultivate a systematic diagnostic approach and maintain a broad perspective to identify underlying causes. This lecture presents practical strategies for recognizing and managing ophthalmic emergencies using real clinical cases, with the goal of improving emergency ophthalmic care in everyday practice.

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Conflict of Interest Statement

The author declares no conflicts of interest related to this presentation.

Surgical Drains in Veterinary Emergency and Critical Care: Options, Placement, and Management

Min Su Kim

Abstract

Surgical drains are essential adjuncts in veterinary emergency and critical care (ECC), facilitating the removal of fluid, air, or contaminants from surgical or traumatic wounds while reducing dead space and local complications.

In emergency settings, where patients frequently present with contaminated wounds, extensive soft tissue damage, postoperative bleeding, or septic processes, appropriate drain selection, placement, and management can significantly influence patient outcomes.

However, inappropriate use of drains may increase the risk of infection, prolong hospitalization, and delay wound healing. This lecture provides a comprehensive, clinically oriented review of surgical drains with specific emphasis on their application in veterinary ECC. Drain types are categorized into passive and active systems, including Penrose drains, closed-suction drains, and thoracic drainage systems. The advantages, disadvantages, and specific indications for each drain type are discussed with respect to wound contamination level, anatomic location, expected fluid characteristics, and patient stability.

To make a drain work properly and avoid complications, placement is key. We will review how to position it correctly, handle tissues gently, and secure it firmly to prevent infections. We'll also focus on how to manage high-risk patients (like those with bleeding or immune issues) and cover common mistakes you can avoid in your daily practice. Post placement management plans are examined, focusing on monitoring drain fluid (both volume and appearance), maintaining closed systems, and ensuring clean handling. Key topics include pain management and clear standards for when to safely remove the drain. These guidelines are combined with some emergency cases to provide practical decision making tools. Common complications, such as infections, tissue damage, clogs, accidental removal, and patient interference, are also discussed.

The lecture provides practical prevention and troubleshooting tips for these everyday clinical challenges. By combining current research with hands on emergency experience, it offers a structured, step-by-step approach to using surgical drains effectively while keeping patients safe.

Key Words

Surgical drains; Veterinary emergency and critical care; Wound; Postoperative complications; Drain placement; Management

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Management of Vomiting and Diarrhea in ER

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Vomiting and diarrhea are common presenting complaints in small animal emergency and general practice. An intact gastrointestinal (GI) mucosal lining is essential for normal digestion and absorption. Because the GI tract absorbs ~99% of ingested fluid, even mild mucosal inflammation or injury can cause clinically important disturbances in fluid balance, electrolytes, and acid-base status. The most significant consequences of vomiting and diarrhea are dehydration and/or hypovolemia with associated electrolyte-acid-base derangements from ongoing GI losses and reduced intake. Hypochloremic metabolic alkalosis is classically associated with upper GI losses (e.g. protracted vomiting or obstruction), whereas hyperchloremic metabolic acidosis or mixed disorders are more common with lower GI losses (e.g. diarrhea). Hypokalemia is frequent in patients with significant GI disease. Aspiration pneumopathy is less common than with regurgitation but remains a risk in laterally recumbent patients or those with impaired laryngeal function. Intestinal barrier disruption may permit bacterial translocation, increasing the risk of bacteremia or sepsis.

Initial Clinical Approach: History, Examination, and Triage

Vomiting and diarrhea are nonspecific signs with broad differentials, including primary GI and extra-GI (systemic) causes. The clinical approach begins with differentiating vomiting from regurgitation, small-bowel from large-bowel diarrhea, and true vomiting from respiratory signs (e.g., coughing or retching). A thorough history should characterize onset (acute vs. chronic), pattern (intermittent, recurrent, or progressive), and severity (mild/self-limiting vs. moderate/severe). Physical examination may reveal dehydration, nausea, abdominal pain, and diarrhea. Systemic signs should prompt consideration of an extra-GI cause. Signs of hypovolemia or shock (e.g. tachycardia in dogs, bradycardia in cats, weak pulses, prolonged capillary refill time, cool extremities, or hypotension) warrant immediate stabilization prior to further diagnostics. Patient stability, duration of signs, and clinical severity guide the intensity of diagnostics vs. empirical therapy.

Minimum Database

At minimum, every vomiting or diarrheic patient should have PCV/TS and blood glucose assessed. Basic electrolytes (Na^+, K^+, Cl^-), acid-base status (pH, PCO_2, HCO_3^-, BE), lactate, and BUN further help identify derangements requiring immediate intervention, guide fluid selection, and assess patient acuity.

Point-of-Care Ultrasound (POCUS)

POCUS is a rapid, non-invasive extension of the physical examination. Any free abdominal fluid should be sampled and evaluated in-house to rule out surgical emergencies (e.g. septic peritonitis, hemoabdomen, bile peritonitis). POCUS also assists in assessing volume status and guiding fluid therapy and further diagnostics.

Survey Radiographs

Abdominal radiographs are a reasonable first-line test to rule out obstruction, dilatation, torsion, or volvulus, particularly in persistently vomiting or moderately to severely affected patients. Findings are often nonspecific in medical disease aside from fluid-filled intestines or mass effect. Thoracic radiographs are indicated if esophageal disease, aspiration pneumopathy, or metastatic disease is suspected.

Gross Assessment & Point-of-Care Fecal Testing

Assessment of fecal consistency, colour, and evidence of melena or hematochezia can guide further diagnostics and therapy. Direct smear for protozoa and ELISA testing for parvovirus or Giardia antigen are appropriate in many cases. Canine parvovirus ELISA can also be used to screen for feline panleukopenia in cats with compatible signs.

When to Pursue Further Testing

Moderately to severely affected or persistently vomiting patients warrant a complete database (CBC, biochemistry, urinalysis). Normal results tend to support a primary GI cause, whereas abnormalities often suggest extra-GI disease. Additional diagnostic considerations should be made based on the patient's history, signalment, geographic location, results of prior diagnostic testing, and response to treatment. Species-specific pancreatic lipase (PLI) may be considered if pancreatitis is suspected. Baseline cortisol is useful in dogs with waxing-waning GI signs and absent stress leukogram (with/without characteristic Na:K changes). Advanced diagnostics may include abdominal ultrasound, contrast radiography, CT imaging, comprehensive fecal testing, specific GI testing (cobalamin, folate), GI endoscopy with biopsies, or surgical exploration depending on clinical suspicion.

Treatment Principles

Most cases of uncomplicated vomiting and diarrhea respond well to supportive care and therapy tailored to the underlying cause. Animals with moderate to severe signs (e.g. hypovolemia, significant dehydration, frequent vomiting, hypoglycemia, or major electrolyte abnormalities) should be hospitalized.

Fluid Therapy

Hypovolemia requires rapid intravenous resuscitation with isotonic crystalloids administered in small boluses titrated to effect. Dehydration should be corrected more gradually over 12–24 hours while accounting for maintenance needs and ongoing losses. Balanced isotonic crystalloids are first line in most patients; however, 0.9% NaCl is particularly useful in cases of hypochloremic metabolic alkalosis to restore chloride. Hypoglycemic patients should receive rescue dextrose (0.5-1 mL/kg IV, diluted at least 1:1) or corn syrup if no IV access, followed by a 2.5-5% dextrose CRI after fluid resuscitation. Enteral rehydration is an effective and underutilized option for mildly to moderately dehydrated patients with intact GI function. Subcutaneous fluids may be appropriate for stable outpatient cases.

Antiemetics & Antinausea Therapy

Antiemetics are indicated for the first 24-72 hours when vomiting is prominent. Options include maropitant (1 mg/kg IV or SC), ondansetron (0.5 mg/kg IV TID) or dolasetron, and metoclopramide (0.5 mg/kg IV TID or 2 mg/kg/day CRI). Effective control of nausea facilitates earlier refeeding and reduces aspiration risk.

Gastroprotectants

Routine use of acid suppressants (proton pump inhibitors, H_2-blockers) and coating agents (sucralfate) is not recommended in uncomplicated gastroenteritis. Gastroprotectants should be reserved for patients with evidence of GI bleeding (hematemesis or melena) or suspected reflux esophagitis.

Antibiotics: When Are They Indicated?

Current evidence does not support routine empiric antimicrobial use in stable animals with acute diarrhea. International guidelines recommend reserving antibiotics for patients with severe disease and signs of sepsis, including fever, hypotension, leukopenia or marked leukocytosis with toxic changes, hypoglycemia, or immunosuppression.

Analgesia

Pure μ-opioids are appropriate for moderate to severe pain; buprenorphine (0.01-0.03 mg/kg IV q6-8h) is often preferred for mild to moderate discomfort due to lower risk of ileus. NSAIDs should be avoided in dehydrated, inappetent, or vomiting patients because of the risk of GI ulceration and renal injury.

Probiotics and Fecal Microbiota Transplantation
Evidence for routine probiotic use remains mixed, but probiotics are reasonable adjuncts, particularly after antibiotic use or in suspected dysbiosis. Fecal microbiota transplantation in parvoviral puppies has been associated with faster diarrhea resolution and shorter hospitalization without affecting mortality.

Nutrition
Food may be withheld 12–24 hours while vomiting is controlled; early refeeding is strongly encouraged once vomiting subsides. A highly digestible, low-fat gastrointestinal diet is preferred. Persistently anorexic patients should receive assisted enteral nutrition via nasogastric or nasoesophageal tube within 48–72 hours. Early feeding is especially important in cats to prevent hepatic lipidosis.

Other Supportive Care
Deworming with a broad-spectrum anthelmintic can start once stable and tolerating oral medication. Prokinetics (e.g. metoclopramide, erythromycin, cisapride) may help with ileus or regurgitation. Antidiarrheals (e.g. loperamide) are rarely used and not recommended in ABCB1-mutant breeds due to risk of severe neurologic toxicity. Treatment of the primary disease process provides the best means for eliminating diarrhea.

Prognosis
The prognosis for primary acute gastroenteritis is generally excellent with appropriate supportive care. Outcomes for secondary disease depend on underlying pathology and timeliness of intervention. Early assessment of hydration and volume status, prompt stabilization, and rational use of diagnostics are key to successful management of vomiting and diarrhea in the ER.

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Nutrition in Critically Ill Dogs and Cats

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Content

1. NASOGASTRIC TUBE PLACEMENT

Indications & Rationale: Critically ill animals often nauseated and anorexic. Lack of intake, in addition to the metabolic demands of illness result in energy and protein depletion that can then hinder recovery. In these animals, the function of the gastrointestinal tract itself can also be compromised, however the enterocytes receive nutrition only from the GI lumen, thus providing enteral nutrition is important. With gastrointestinal dysfunction, gut stasis can occur with fluid buildup in the stomach putting patients at risk of nausea, pain, vomiting, regurgitation and aspiration pneumonia. An NG tube can also be used to suction the stomach in patients with gastric ileus and regurgitation. NG tubes are used for short term hospitlised feeding (approximately up to 7 days)

Contraindications:

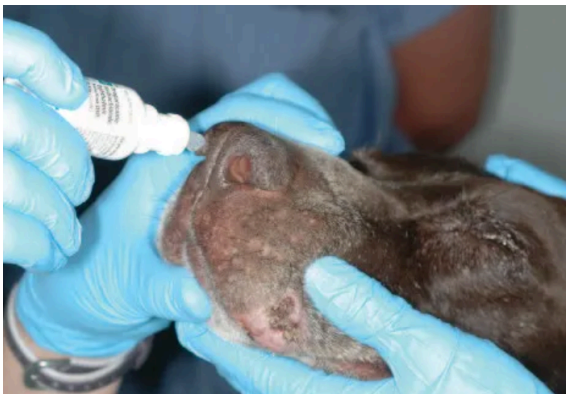
- Facial trauma
    - Particularly trauma to the nose, nasal passages
- Head trauma
    - since sneezing associated with placement or an indwelling nasal tube may increase ICP
- Dyspnea
    - as animals with respiratory difficulty are more likely to be intolerant of tube placement, and are also at higher risk of aspiration
- Hemostatic disorders
    - Animals with a bleeding tendency (especially primary hemostatic disorders) are likely to have epistaxis (that can be protracted) associated with nasal tube placement
- Vomiting or regurgitation
    - these are not absolute contraindications, but it is likely that these patients will continue to vomit / regurgitate whatever food is put into their stomach. Feeding tubes can be used to empty the stomach in patients that are accumulating fluid in associated with ileus; this may be beneficial in reducing nausea. There is also some evidence to suggest that feeding despite vomiting / regurgitation (assuming it is mild) is beneficial (eg. Dogs with parvovirus).
- Impaired consciousness / reduced gag reflex
    - These are no absolute contraindication, but care must be taken to minimize risk of aspiration pneumonia. Provide NG tube suction every 4 hours, and if there is large amount of suctioned content consider reducing the volume of nutrition provided through the NG tube.

Materials:

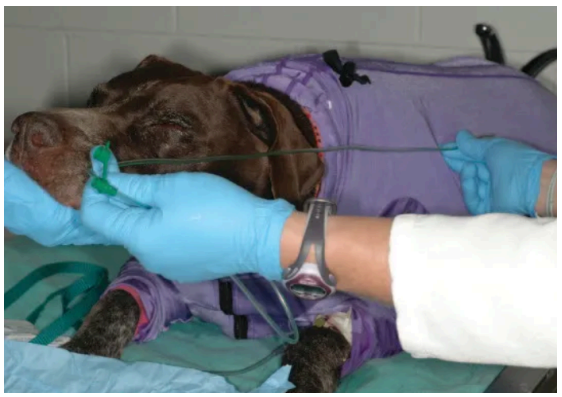
- Feeding Tube
    - Usually 8 or 10Fr for dogs; 5 or 6 Fr tends to be more comfortable for cats
    - Human infant nasogastric feeding tubes are preferred
    - Can use red-rubber catheters
- Gloves (sterile or non-sterile)
- Sharpie / permanent marker
- Topical 2% lignocaine drops (drawn up from a bottle) or lignocaine jelly
    - to provide local anesthesia in the nose
- 0.5% proparacaine ophthalmic solution
    - also to provide local anesthesia in the nose /oro-naso-pharynx
- KY jelly or lignocaine jelly
    - to lubricate the feeding tube, and minimize trauma during tube passage
- Nonabsorbable monofilament suture:
    - Eg. 2-0 ethilon / nylon to secure the tube to the nose
- Elizabethan collar
- Assistant for placement

Procedure:

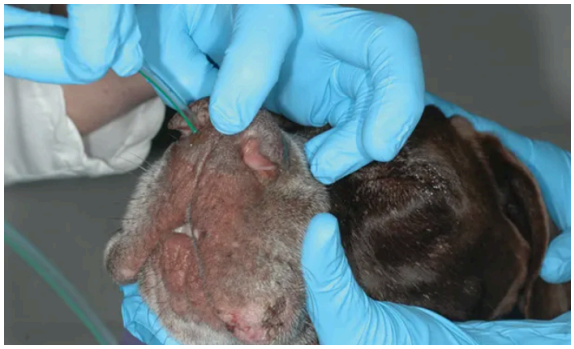
1. Lightly sedate the patient if necessary
    1. Note that while very sick dogs and cats tolerate this procedure awake, many require or benefit from conscious sedation or an analgesic. The aim is to reduce their anxiety associated with tube placement without precluding their ability to swallow (as a conscious swallow helps ensure that the tube goes into their esophagus and not the trachea)
2. Instill local anesthetic (lignocaine or proparacaine) into the selected nostril. 1 or 2 drops is adequate.
    1. Hold the head up as you do this and for a few seconds afterwards.
    2. Ideally allow 3-5 minutes for this to work before placing the tube.



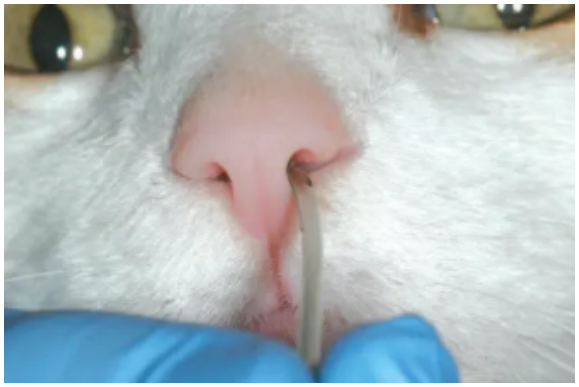
3. Measure the feeding tube to ensure appropriate length, and mark the tube with a permanent marker or piece of tape at the designated length, aiming for the stomach (beyond the last rib = NG tube)



4. Preplace a suture at the edge of the nostril (so that you can secure the tube as soon as it is in). We may also preplace sutures on the nasal planum and the top of the head, or along the side of face.
5. Coat the tip of the catheter with lubricant (sterile KY jelly or lignocaine jelly).
6. Inserted the tip of the tube into the nostril. Direct the tube in a caudo-ventro-medial direction. Plasing your finger against the nose dorsally like a 'Pig nose' can helps facilitate placement.
    1. As soon as the tip hits the medial septum of (in dogs), push the external nares dorsally which opens up the ventral meatus. If you reach a "dead end" you are likely encountering the ethmoid turbinates; withdraw the tube and redirect. A little resistance is normal, but don't keep pushing if you get a lot of resistance otherwise you can cause trauma and bleeding.

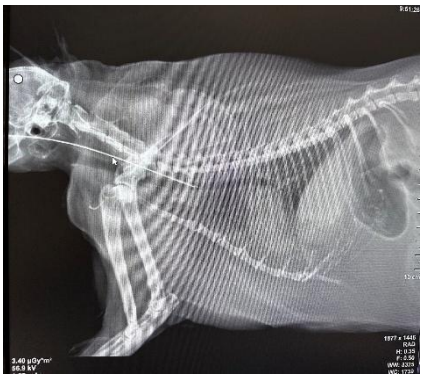


For cats, insert the tube into the desired, anesthetized nares, and direct it in the oral direction.

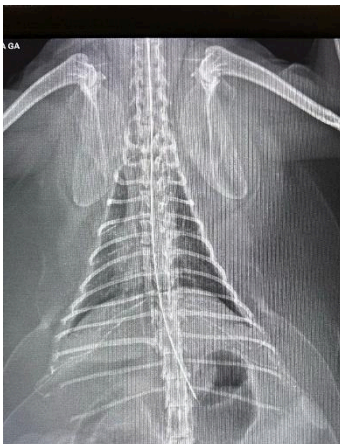


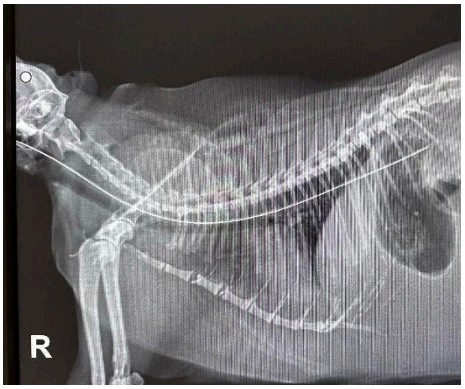
7. Keep the patient's head in a neutral position to facilitate passage into the esophagus.
    1. Brace the introducing hand against the animal and introduce the tube in short, well-controlled insertions
    2. When the tube reaches the pharynx, the animal will usually swallow,

3. Once the tube is in the cervical region, take 1 radiograph to ensure you are in the oesophagus rather than the tracheal. Taking this first survey radiograph minimizes the risk of pulmonary tube placement and pneumothorax.

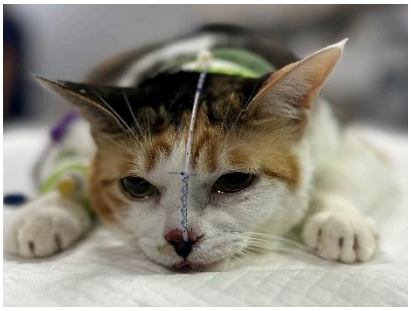


4. Once you have confirmed the placement of the tube in the oesophagus, the tube can be advanced to the predetermined distance and a lateral and DV thoracic radiograph can be taken.





8. Tie one of the pre-placed sutures around the tube to secure it while you make sure it is in the right place.



9. Tie the remaining preplaced sutures around the tube
10. Place an Elizabethan collar on the patient.

Potential Complications:

- Inadvertent tracheal intubation

    - If food is then administered through the tube this can then result in pneumonia, severe respiratory distress and even death. Thus, ensuring proper placement in the GI tract is vital.
    - Pneumothorax has been reported
- Epistaxis (even in the absence of a bleeding disorder)
- Lack of tolerance of the procedure (requiring sedation)
- Lack of tolerance of the tube – sneezing, pawing at the nose etc.
    - It is generally recommended that the animal wear an Elizabethan collar to help prevent inadvertent removal.
- Note that sneezing and/or vomiting may dislodge the tube.
    - In some cases the tube comes completely out, but in others it may not be visible and the tip of the tube will sit in the back of the oropharynx or larynx, increasing the risk of aspiration if not detected and feeding is continued
- Rhinitis
- Sinusitis
- Clogging
    - Avoid by ensuring a liquid only diet is fed
    - DO NOT attempt to put crushed tablets down NO / NG tubes
    - If clogging does occur, a mixture of pancreatic enzymes and bicarbonate is likely most effective at resolving the tube obstruction or normal coca cola (NOT diet)

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- Odunayo, A., Hoen, M., Wolf, J., Marshall, K., Osterbur, K., & Maxwell, K. (2023). Outcomes, including death, in dogs with pneumothorax following nasogastric feeding tube misplacement in the tracheobronchial tree: 13 cases (2017-2022).. Journal of the American Veterinary Medical Association, 1-7 . https://doi.org/10.2460/javma.22.12.0585.

2. OESOPHAGOSTOMY TUBE PLACEMENT

Indications: Oesophagostomy tubes are used for longer term feeding plans, e.g. if the patient may remain in hospital for over a week. Other indication include maxillofacial or oral disease, neurological impairment affecting prehension or swallowing. It can help transition patients for care at home as oesophagostomy tube feeding can be provided by pet owners. It also allows for soft food (rather than liquid) diet, administration of medications and water.

Contraindications:

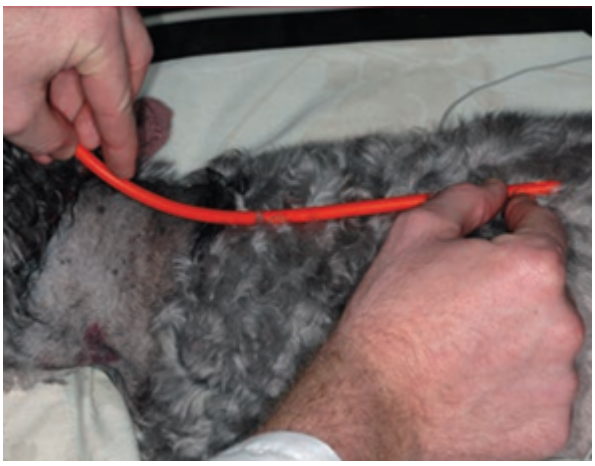
- Severe oesophageal disease (e.g. oesophagitis, strictures, or perforation)
- Coagulopathies
- Situations with high anaesthetic risks
- If the plan is to discharge the patient with an oesophageal tube, ensure careful client discussions regarding home care have been made

Equipment:

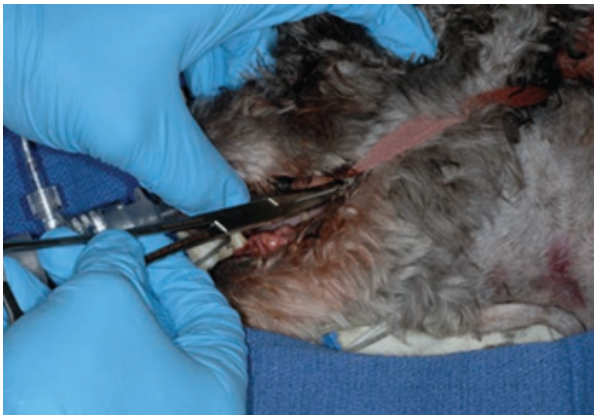
- 8–14 Fr tube in cats and small dogs
- 14–20 Fr tube in medium to large dogs
- Mouth gag
- Mayo scissors
- Right-angled/curved forceps (Carmalt)
- #10 or 11 scalpel blade
- Nylon/polypropylene suture material
- Marker
- Luer slip catheter plug
- Conforming bandage / light bandage material

Procedure:

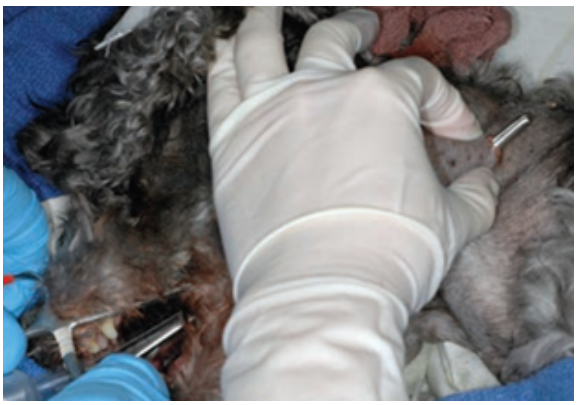
- All patients are anaesthetized and intubated during oesophageal tube placement.
- Place the anesthetized patient in right lateral recumbency and clip and aseptically prepare the lateral cervical region from the back of the ear to the point of the shoulder.
- Premeasure the tube from the mid-cervical esophagus to the level of the 8th to 9th intercostal space.

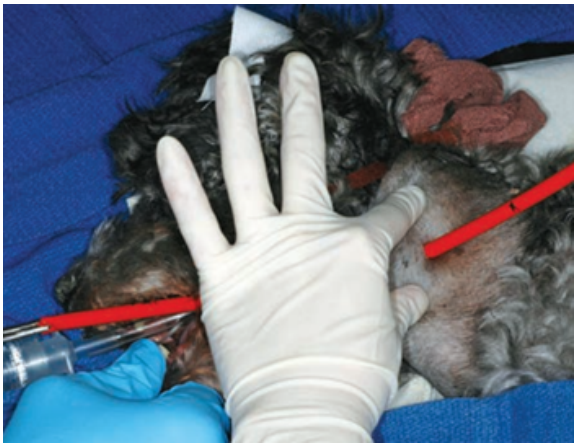


- Insert Carmalt (medium and large dogs) or Kelly (cats and small dogs) forceps through the oral cavity in the oral direction and into the mid-cervical esophagus with the tip facing outward. Avoid the region of the jugular furrow.
- Tilt the forceps so that the tip of the Carmalt forceps are tenting the skin overlying the esophagus laterally, again making sure to identify and avoid the jugular vein.

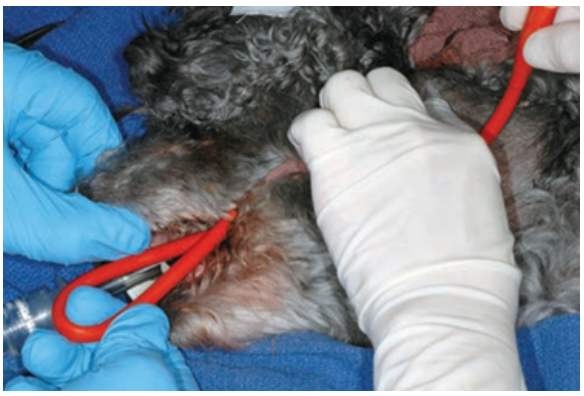


- Placing a sandbag or towel under the neck may facilitate passing and tilting forceps and aid in isolation of the esophagus against the skin.
- As the Carmalt is inside the esophagus and tenting this skin overlying this region, the opposite hand will make a small incision a #11 scalpel blade, incising the skin over the forceps tips with constant tension on the skin. The tip of the forceps is pushed through the small incision. Gently open the tips just enough to stretch the incision and grasp the distal tip of the esophagostomy tube. Clamp the tube along its long axis and pull the tube cranially towards the oropharynx.





- Redirect the distal tip of the tube through the oropharynx and gently advance the tube manually in the aboral direction, into place. Once the tube has been advanced to the appropriate, premeasured location, take 2 orthogonal radiographs to ensure the O-tube is in the esophagus at the correct anatomical location (distal third of the esophagus approximately 7-9 th rib space).



- After positional confirmation then secure the tube to the skin with 2-0 Ethilon (or other non-absorbable) suture material in a Roman sandal pattern. Apply a soft bandage or stockinette with sterile gauze or dressing the neck to cover the stoma site. The bandage should be changed daily for the first week and as needed thereafter.

- Once the oesphageal tube is no longer needed, the tube should be flushed, the anchoring sutures removed, and the tube clamped/kinked then smoothly taken out. The site should be cleaned, and a loose-fitting bandage applied. Wound closure occurs via second intention, which usually occurs within a week of tube removal.

Potential complications:

- Local stoma infection or cellulitis
- Tube dislodgement or obstruction
- Oesophagitis
- Regurgitation
- Aspiration pneumonia
- Less commonly, oesophageal stricture formation

References:

1. Dumont, R., Lemetayer, J., Desquilbet, L., & Darnis, E. (2023). Tolerability of naso-esophageal feeding tubes in dogs and cats at home: Retrospective review of 119 cases. Journal of Veterinary Internal Medicine, 37, 2315 - 2321. https://doi.org/10.1111/jvim.16732.
2. Breheny C, Alisfair Boa, Alice Le Gal, et al. Esophageal feeding tube placement and the associated complications in 248 cats. JVIM 2018 DOI: 10.1111/jvim15496
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3. DRUM LINE PLACEMENT

Indication: This procedure establishes secure, long-term venous access for critical care including delivery of prolonged fluid therapy, administration of various hyperosmolarity medications (e.g. potassium), administration of medications which extravasation can be detrimental (e.g. noradrenaline), parenteral nutrition, and blood withdrawal (e.g. when multiple blood sampling is required). It is a single lumen catheter (so less versatile than a central venous catheter), however can be placed with minimal or no sedation.

Contraindications:

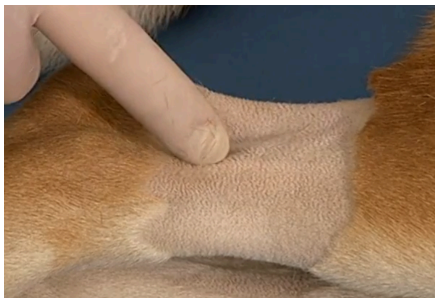
- Significant bleeding disorders – this is a relative contraindication, as careful haemostasis during placement ban minimize bleeding
- Prothrombotic disease – this is a relative contraindication. In prothrombotic conditions, clot formation can occur at the tip of the catheter.

Equipment:

1. Mila Drum Long Line Catheter Kit: Contains drum catheter device, introducer needle and catheter, sterile gauze/swab
2. #11 scalpel blade
3. Clippers
4. Surgical scrub (chlorhexidine gluconate) + 70% isopropyl alcohol
5. Sterile gloves
6. Sterile gauze
7. Sterile drape
8. Securement & Dressing: Tissue glue, sterile transparent semi-permeable dressing (e.g., Tegaderm), 1" white cloth tape or equivalent, bandage material

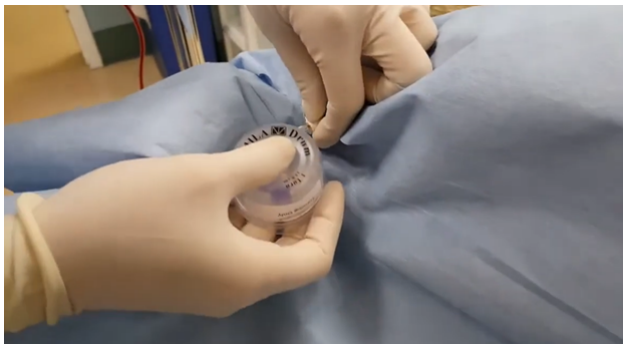
Procedure: Preparation

1. Position Patient. Place patient in lateral recumbency with the "down" hind limb extended. The target medial saphenous vein runs cranio-medial to the tibia. Equally, lateral saphenous can also be used, however, medial site is preferred.
    1. The dependent position aids in venous distension, making the vessel more prominent

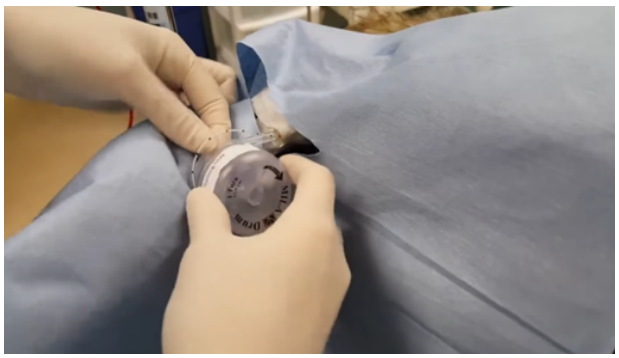


2. Clip & Aseptically Prepare. Clip a wide area over the medial tibia, extending from the tarsus to the mid-femur. Perform a sterile surgical scrub
    1. This is a sterile procedure. The clip area must be extensive to allow for a large sterile field and secure bandaging.
3. Wear sterile gloves. Apply a sterile drape, isolating the prepared site
    1. A full sterile barrier technique significantly reduces the risk of infection
4. Puncture Vessel with Introducer. Using the introducer catheter from the kit, puncture the medial saphenous vein. Confirm flashback of blood in the chamber.
    1. Ensure a clean, single-wall puncture.
5. Advance & Remove Needle. Advance the entire introducer catheter assembly 2-3 mm further into the vessel. Stabilize the plastic catheter hub and completely remove the needle. Dispose of the needle in a sharps container.
    1. The plastic introducer catheter is now sitting in the vein.

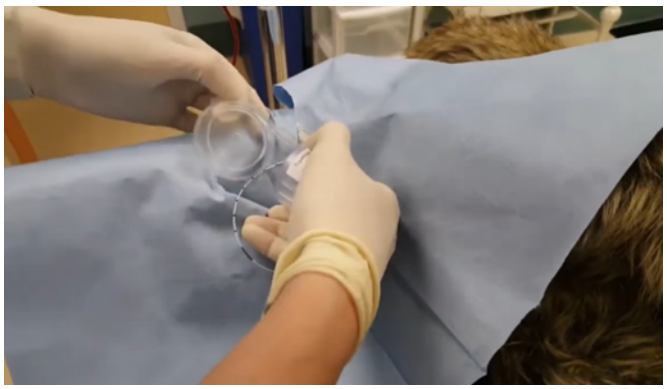
6. Connect Drum to Introducer. Insert the tip of the drum into the hub of the indwelling introducer catheter.
    1. This creates a closed system from the drum directly into the vessel

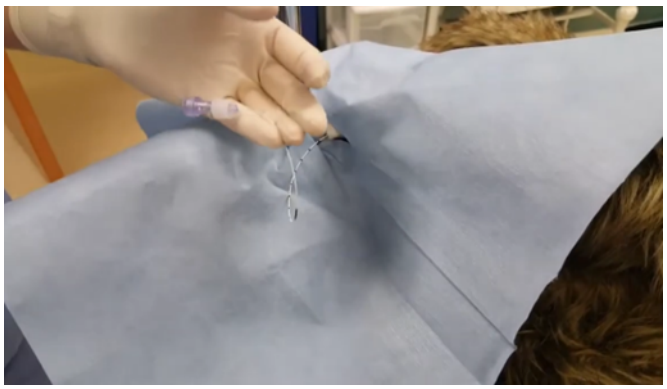


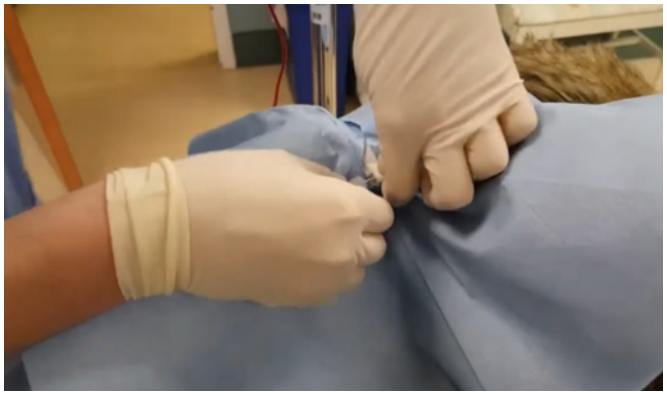
7. Advance Catheter via Wheel. Rotate the wheel clockwise to advance the long catheter through the introducer and into the vessel. Continue until firm resistance is met OR the long black mark on the catheter appears at the drum's exit port.
    1. The wheel provides controlled, sterile advancement. The black mark indicates the pre-measured correct depth for a central location.



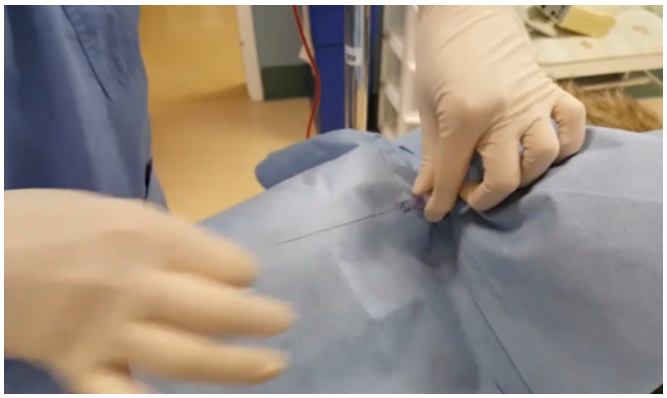
8. Disengage & Lock. Further advance the long catheter manually the last 1-2 cm until the hub of the long catheter seats firmly into the hub of the introducer. Luer-lock the two hubs together.
    1. This step finalizes the connection and secures the system.







9. Remove Stylet & Open Drum. Remove the stiffening stylet from the long catheter. Fully open and remove the empty drum housing. Dispose of the drum.
    1. The stylet is only for pre-placement stability and must be removed.



10. Support the Hub. Place a folded sterile gauze sponge underneath the catheter hub to absorb pressure and prevent skin tenting.
    1. This improves patient comfort and prevents pressure necrosis at the site.
11. Confirm Placement. Aspirate for blood to confirm intravascular placement. Attach a stasis valve or extension set and flush the catheter with saline.
    1. Easy aspiration and smooth flushing confirm patency and placement.

12. Apply Final Dressing & Bandage. Cover with a sterile transparent dressing or equivalent and secure catheter with the current hospital standard technique. Apply a soft padded bandage over the entire limb.

Complication:

1. Bleeding
2. Obstruction
3. Phlebitis / catheter related infection

References

1. Mila International. (2022, June 15). Drum Long Line Catheter / Mila International [Video]. YouTube. https://youtu.be/BggUzPfX6F4?si=74PiHn81HiYqMnqp

2. Mila International. (n.d.). Drum long line catheter. Retrieved November 5, 2024, from https://www.milainternational.com/drum-long-line-catheter.html

4. PARENTERAL NUTRITION

Indications: Parenteral nutrition (PN) is a method of feeding that bypasses the gastrointestinal tract, delivering essential nutrients directly into the bloodstream via a catheter, typically a central line, peripherally inserted intravenous catheter or drum line. It is used when enteral nutrition cannot provide adequate resting energy requirement (RER).

Contraindications:

- Relative contraindications (check with veterinarian if these are present): Neutropenia, hypervolaemia (fluid overload or at risk of fluid overload), azotaemia and lipemia.
- Contraindications in placing a long stay catheter (central venous catheter, drum catheter) may be present.

Equipment and Materials

- SmofKabiven 550 kcal 493mL bag
- Fluid administration set
- Extension set
- Fluid administration pump
- Medical tape
- Permanent marker

- Central venous catheter or drum line placed for administration

Procedure

- Aseptically place a central venous catheter in the jugular or saphenous vein or a drum catheter in the saphenous vein. If a central venous catheter is already in place, a sterilely protected port which has not been used can be utilized.
    - The catheter must not have been used for other purposes.
    - Using a previously used port or catheter will result in risk of bacteraemia
- Activate the bag of parenteral nutrition by compressing/rolling the bag and mixing adequately.
    - Recommend using a commercially prepared bag such as Kabiven
- Aseptically (wash hands and wear gloves) connect a giving set +/- extension line to the bag of PN and flush the line with the PN ensuring no air remains in the line.
- Label date and time of when the PN bag has been activated.
- Connect Parenteral nutrition to a dedicated line which has NOT been previously used.
- Place medical tape around the catheter port connection and connections between extension lines and label 'DO NOT DISCONNECT'.

Prescription

1. Calculate daily resting energy requirements (RER):
    - RER= 70X(BW)^0.75
- Determine daily RER required:
    - Day 1: ¼ - 1/3 RER
    - Day 2: 2/3 RER
    - Day 3: Full RER
    - > 1 x RER may be required in some patients
- Calculate infusion rate (ml/hr):
- Kabiven contains 1.1 Kcal/mL
    - Infusion rate (per hour) = RER ÷ 1.1 / 24
- Ensure infusion rate is below maximum rate of 2.5 mL/kg/hr
    - Higher than 2.5 ml/kg/hr can result in hyperkalaemia

Maintenance of infusion and catheter:

- DO NOT disconnect any of the lines for PN from the patient. When patients are walked, the PN and line must be walked together with the dog.
- Check the catheter site q.12hrs using aseptic technique. If there are any concerns about the integrity of the catheter site, please check with a veterinarian.
- Every 24 hours: Check PCV and TP to check for lipaemia in serum, electrolytes, blood glucose, body temperature.
- **Change the parenteral nutrition bag, giving set and extension line every 48 hours**. Use aseptic technique when changing bags.

Complications:

- Thrombophlebitis
- Fluid overload
- Hyperglycaemia

- Hyponatraemia
- Hyperkalaemia
- Refeeding syndrome (hypophosphataemia, hypokalaemia, hypomagnesemia)
- Breach of the extension and giving set (disconnecting from patient) can lead to risk of bacteremia.
- Fluid overload
- Lipidaemia

References:

Advanced Monitoring and Procedures for Small Animal Emergency and Critical Care - JM Burkitt Creedon and H Davis

5. NUTRITION CALCULATION

When performing nutritional requirements consider the following:

- Route of feeding:
  - Voluntary (per os)
  - Assisted feeding (via tube)
- Diet to be fed:
  - Type of diet: Dry / Wet / Liquid / Other
- % of RER per day:
  - Start with 25 – 30%
  - Increase daily to achieve 100% RER by day 3 of hospitalization
- Number of feeds per day
  - Divide 4 – 6 feeds a day depending on practicality, volume to be fed during each feed (should not exceed > 10ml/kg).
  - When performing bolus feeding, bolus over 10 – 15 minutes (can use a syringe driver), and monitor the patient for any vomiting or regurgitation.
  - CRI feeding over 24 hour can be performed, especially in patients who's bolus feeding would be > 10ml/kg. Risk of CRI feeding is that the patient isn't observed if vomiting, regurgitation or tube dislodgement occurs
- Special considerations:
  - Age: paediatrics/neonates require2 – 3 X RER
  - Risk of refeeding syndrome
  - Organ dysfunction (cardiac, renal, hepatic)
  - Electrolyte imbalances
  - Other co-morbidities.

CALCULATING DAILY ENERGY REQUIREMENTS AND FOOD INTAKE

RER calculation:
2–30 kg: RER = 30 x BW(kg) + 70
<2 kg or >30 kg: RER = 70 x BW(kg)^0.75

- Calculate RER (kcals/day)
- Calculate % of RER required per day
- Identify kcal per gram/ml of diet
- Calculate grams/ml per day
- Calculate grams/ml per feed

Case 1:

Jack, a 2 month old male entire Staffordshire Bull Terrier (3.5 kg) presents to you with a 2 day history of vomiting, diarrhoea and anorexia. He has not been vaccinated or wormed. On physical examination he is cardiovascularly stable, breathing is normal, 8% dehydrated, hypersalivating and has frequent vomiting. Inhouse parvovirus antigen test is positive.

Using the WSAVA feeding guide, develop a feeding plan for Jack. Please specify type of tube and size.

Daily BW gain:

- **wk 1: 8% of BW**
- **wk 2: 6% of BW**
- **wk 3: 4% of BW**
- **wk 4: 3.5% of BW**
- **1 – 2 months of age; 3g/kg**
- **2 – 5 months of age; 2 – 4g/kg**

Recommended energy intake of growing dogs (50% BW @ 4-5 months of age):

- **Weaning – 50% adult BW: 3 x RER**
- **50 – 80% BW: 2.5 x RER**
- **>80% BW: 1.8 – 2.0 x RER**

Table 21–1 **Recommended Daily Caloric Requirements for Maintenance of Average Growing Puppies of Different Body Weights and Ages**

BODY WEIGHT		DAILY KILOCALORIE REQUIREMENTS*	
Kilograms	Pounds	Weaning to 3 Mo†	3–6 Mo‡
1	2.2	268	214
2	4.4	464	373
3	6.6	649	520
4	8.8	808	646
5	11.0	915	732
7	15.4	1167	934
9	19.8	1394	1115
11	24.3	1670	1336
13	28.7	1929	1543
15	33.1	2179	1743
17	37.5	2415	1932
19	41.9	2640	2112
21	46.3	2856	2285
23	50.7	3062	2450
25	55.1		2618
27	59.5		2785
29	63.9		2945
31	68.3		3104
33	72.8		3250
35	77.2		3422
37	81.6		3551

*These values only approximate the daily energy needs. The reduction in kilocalories required per unit of body weight occurs gradually as the puppy approaches maturity. Requirements vary with environmental conditions, activity, and temperament. The amount fed should be adjusted to maintain optimal body weight and condition. This may require substantially more or less food than the amount indicated.

†Values represent two times the maintenance energy requirement of the adult per unit of body weight.

‡Values represent 1.6 times the maintenance energy requirement of the adult per unit of body weight.

Case 2:

Maggie, a 5-year-old female neutered domestic short hair cat has been missing for 6 days. Prior to this, she has been a healthy in-door/out-door cat. On examination she is 12% dehydrated, in hypovolaemic shock, abdominal palpation is unremarkable, body temperature is 36.0 °C, body weight is 4.2 kg with a body condition score of 2/9, and remaining examination is unremarkable. Her blood results are as follows:

	Patient	**Reference range**
Haematology		
RBC	8.1 x 10^{-6}/µl	6.56 – 11.2
Hb	13.2 g/dl	10.6 – 15.6
Hct	35%	31.7 – 48
PCV	30%	25 – 45
Plt	220 x 10^{-3}/ml	175 – 500
WBC	28 x 10^{-3}/ml*	4.04 – 18.70
Neutrophils Segmented	18 x 10^{-3}/ml*	2.3 – 14
Neutrophils Band	3.1 x 10^{-3}/ml*	0.0

Lymphocytes	0.6 x 10^{-3}/ml*	0.8 – 6.1
Monocytes	0.0 x 10^{-3}/ml	0.0 – 0.7
Eosinophils	0.0 x 10^{-3}/ml	0.0 – 1.5
Basophils	0.0 x 10^{-3}/ml	0.0 – 0.1
Biochemistry		
Albumin	20 g/L*	24 – 38
Globulin	35 g/L	28 – 51
ALKP	540 U/L*	14 – 111
ALT	220 U/L*	12 – 130
AST	60 U/L*	0 – 48
TBil	22 µmol/L*	0 – 15
Amyl	2200 U/L*	500 – 1500
Lipase	1800 U/L*	100 – 1400
Urea	14 µmol/L*	5.7 – 12.9
Crea	310 µmol/L*	71 – 212
CK	660 U/L*	0 – 314
Chol	4.8 mmol/L	1.68 – 5.81
Calcium	2.2 mmol/L	1.95 – 2.83
Glucose	4.1 mmol/L	4.1 – 8.83
Phos	0.8 mmol/L*	1.00 – 2.42
Na^+	164 mmol/L	150 – 165
K^+	2.8 mmol/L*	3.5 – 5.8
Cl^-	128 mmol/L	112 – 129

What concerns should you address prior to initiating a nutrition plan?

Using the WSAVA feeding guide, develop a feeding plan for Maggie. Please specify type of tube and size.

On day 3

Her vital parameters are normal, she remains recumbent and anorexic. Her body weight is 4.0kg. Her owners are getting cost concerned. You repeat her biochemistry as she is still not eating:

Biochemistry		
Albumin	18 g/L*	24 – 38
Globulin	35 g/L	28 – 51
ALKP	2577 U/L*	14 – 111
ALT	880 U/L*	12 – 130
AST	66 U/L*	0 – 48
TBil	20 µmol/L*	0 – 15
Amyl	1100 U/L*	500 – 1500
Lipase	1400 U/L*	100 – 1400
Urea	3.3 µmol/L*	5.7 – 12.9
Crea	101 µmol/L*	71 – 212
Chol	2.5 mmol/L	1.68 – 5.81
Calcium	2.2 mmol/L	1.95 – 2.83
Glucose	9.4 mmol/L	4.1 – 8.83
Phos	1.1 mmol/L*	1.00 – 2.42
Na^+	155 mmol/L	150 – 165
K^+	5.4 mmol/L*	3.5 – 5.8
Cl^-	128 mmol/L	112 – 129

What are your major concerns? Create an ongoing nutrition plan:

Case 3:

Newey a 4-year-old male neutered cross breed dog (35 kg with a body condition score of 5/9) presents to you with a 36 hour history of risus sardonicous (facial muscle contraction), trismus (locked jaw), stiff gait in all 4 limbs, and regurgitation once a day. He was 8% dehydrated, but other clinical parameters were normal. Haematology, biochemistry, and electrolytes were within normal limits.

You have diagnosed him with tetanus, started IVF to correct his dehydration, and placed a nasogastric feeding tube, feeding EnteralCare liquid diet with an RER of 100%, with no vomiting, regurgitation or diarrhoea. However, 4 days after hospitalisation his tetanus is not improving, and you have noticed his body weight is now 31 kg and has a body condition score is 3/9. His hydration status is normal.

Using the WSAVA feeding guide, develop a new feeding plan which will address the weight loss and poor body condition score. Please specify type of tube and size.

Calculate daily fluid requirement to determine if IVF is required, and if so, calculate the rate (note: maintenance daily fluid requirement calculation is the same as RER).

Creating a Mind-Blowing Employee Experience in the ER

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What is the veterinary emergency room experience like in the United States for clients, patients, and the people who work there? Emergency medicine is fast, unpredictable, emotionally intense, and often exhausting. Across veterinary medicine, burnout, compassion fatigue, staffing shortages, and disengagement are persistent challenges. Many ER professionals accept stress and dissatisfaction as inevitable features of the job.

The most successful and fastest growing companies challenge that assumption. They understand Employee Experience (EX) is just as important as Customer Experience (CX). VEG ER for Pets has approached business growth in the same way. Our goal is not merely to create a functional workplace, but to create a "*mind-blowing"* employee experience–one so compelling that employees *want* to come to work every day and more importantly for business growth, tell their friends to join them.

This session explores, through real examples, stories, videos, and images, how applying the science of customer experience to employee experience can transform emergency medicine culture, engagement, and organizational performance.

Why Employee Experience Matters
Employee experience is not a soft metric. It's a leading indicator of organizational success. Research consistently demonstrates employee engagement directly affects productivity, commitment, retention, and revenue generation. If we accept that customers drive business outcomes, then we must also accept a parallel truth: employees are the internal customers of an organization. Just as customer experience determines whether someone returns and refers friends, employee experience determines whether a team member stays, thrives, and recruits others.

Understanding Experience: From Customers to Employees
As customers, service is what is done for us; experience is how it makes us feel. As customers, we intuitively understand that satisfaction alone does not create loyalty. Frameworks such as the Kano model and the REDU (Required, Expected, Desired, Unexpected) model demonstrate that while "required" elements prevent dissatisfaction and "expected" elements create competence, it is the "unexpected"—the mind blowing 🤯—that drives return behavior *and* advocacy. An experience becomes memorable and worth recommending when it exceeds baseline expectations in meaningful ways.

The same principles apply to employees. If we view employees as internal customers, we can design their experience using the same intentional structure. By treating employees as internal customers, organizations can transform emergency medicine from a place people endure into a place they are proud to belong.

Delivering the Unexpected in the ER
When employees are invited to build – not just operate – the organization, engagement shifts from compliance to ownership.

Participation extends beyond the hospital floor. Employees contribute ideas, influence innovation, and engage in broader company initiatives. This decentralization reinforces agency and investment.

Measuring the Results
If employee experience is a leading indicator, outcomes should follow. Key performance metrics include:

- Referral rates and organic hiring growth
- Employee Net Promoter Score (eNPS)
- Retention rates
- Internal mobility and promotion rates
- Client NPS

When employees become advocates, recruitment becomes cultural rather than transactional. Growth becomes fueled by belief rather than necessity.

Conclusion
Emergency medicine does not have to be synonymous with burnout. By intentionally designing the employee experience using principles traditionally reserved for customer experience, we can create ER workplaces where professionals feel safe, valued, challenged, and inspired.

When we deliver not just what is required or expected–but what is unexpected–we create cultures where employees want to come to work, stay, and invite others to join them.

A mind-blowing employee experience is not accidental. It is designed.

Smoke Inhalation and Burn Injury

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Introduction

Smoke inhalation injury is a complex and potentially life-threatening condition encountered in dogs and cats exposed to fire in confined or poorly ventilated environments. Injury results from the combined effects of inhaled toxic gases, superheated particulate matter, and chemical irritants, frequently occurring with concurrent thermal burn injury. Although animals affected by fires often suffer high prehospital mortality, those that survive to hospital presentation may initially appear only mildly affected, despite the potential for significant delayed respiratory and neurological deterioration. Smoke inhalation injury is an independent risk factor for morbidity and mortality and substantially worsens outcomes in patients with concurrent burns. Early recognition, immediate oxygen supplementation, and vigilant monitoring for both early and delayed complications are essential components of successful management.

Etiology and Pathophysiology

Smoke inhalation injury results from incomplete combustion of organic and synthetic materials, producing a heterogeneous mixture of toxic gases, chemicals, and particulate matter. The composition of smoke varies depending on the material burned, temperature achieved, and oxygen availability, but commonly includes carbon monoxide, hydrogen cyanide, aldehydes, nitrogen oxides, sulfur compounds, and superheated soot particles.

The pathophysiology of smoke inhalation injury involves both systemic toxicity and local respiratory tract injury, often compounded by concurrent thermal burn injury. Carbon monoxide binds hemoglobin with an affinity approximately 200–250 times that of oxygen, forming carboxyhemoglobin and impairing oxygen delivery and mitochondrial respiration. Hydrogen cyanide inhibits cytochrome-c oxidase, leading to cellular hypoxia despite normal oxygen tension. These mechanisms primarily contribute to early and delayed neurological dysfunction, which may occur independently of measurable hypoxemia.

Local respiratory injury is caused by thermal damage, chemical irritation, and particulate deposition. Thermal injury primarily affects the upper airways, while chemical and particulate injury predominates in the lower airways and lung parenchyma. Resulting airway edema, bronchoconstriction, pulmonary edema, surfactant dysfunction, and ventilation–perfusion mismatch may present acutely or evolve over hours to days, explaining why respiratory signs can be absent or mild at presentation yet progress significantly during hospitalization.

Thermal burn injury adds a significant systemic inflammatory burden and increases insensible fluid losses, metabolic demand, and risk of shock. Burns involving more than approximately 20% of total body surface area are associated with burn shock and markedly increase morbidity and mortality, particularly when combined with smoke inhalation injury.

Clinical Presentation

Clinical manifestations vary widely and may be early or delayed, involving both respiratory and neurological systems. Early clinical signs commonly reflect systemic hypoxia, airway irritation, and pain, and include tachypnea, tachycardia, hypotension, arrhythmias, altered mentation, and seizures in severe toxicity. Physical examination findings raising concern include singed hair, soot deposition, facial burns, and upper airway edema manifested as stertor or stridor.

Importantly, absence of severe signs at presentation does not exclude injury. Respiratory deterioration may be delayed as inflammatory lung injury, chemical pneumonitis, airway cast formation, or secondary infection develop. Delayed respiratory signs include increasing oxygen requirements, progressive dyspnea, pulmonary edema, bronchoconstriction, and pneumonia, often occurring 12–72 hours after exposure.

Neurological signs may also be delayed and are particularly associated with carbon monoxide exposure. These may include obtundation, ataxia, behavioral changes, seizures, blindness, or deafness, developing hours to days after the initial event. Delayed neurological dysfunction may occur even in patients whose respiratory signs appear mild or improving.
Burn injuries should be systematically assessed once the patient is stabilized. Burns involving the face, paws, perineum, or circumferential limbs are of particular concern due to airway compromise, severe pain, vascular compromise, and functional impairment.

Diagnosis and Monitoring
Diagnosis is based on compatible history and physical examination findings, with recognition that clinical signs may lag behind injury severity. Conventional pulse oximetry is unreliable in the presence of carboxyhemoglobin and should not be used to exclude hypoxia. Co-oximetry is required to confirm carbon monoxide exposure when available. Suspicion of cyanide toxicity is based on exposure history, severe neurological signs, and marked hyperlactatemia.
Thoracic radiography may be normal initially and should be repeated if respiratory signs progress, as radiographic changes frequently develop after clinical deterioration. Serial neurological examinations are equally important to identify delayed central nervous system injury.
Burn extent should be assessed using estimation of total body surface area affected by partial- and full-thickness burns. In dogs, regional estimation assigns approximately 9% to the head and neck, 9% to each forelimb, 18% to each hindlimb, and 18% each to the thorax and abdomen. In cats, the head represents proportionally more surface area and limbs slightly less. As a practical method, the patient's paw (including digits) approximates 1% TBSA in both species.

Treatment
Immediate oxygen supplementation is the cornerstone of treatment and should never be delayed, even in patients without obvious hypoxemia. High inspired oxygen concentrations accelerate elimination of carbon monoxide and improve tissue oxygen delivery. Airway protection should be considered early in patients with facial burns, progressive airway edema, or neurological impairment, as airway compromise may worsen over time. Mechanical ventilation may be required for refractory hypoxemia or hypercapnia.
Fluid therapy must be carefully titrated. Patients with significant burns may require higher volumes due to increased losses, while isolated smoke inhalation patients are at risk of pulmonary edema with over-resuscitation. Adjunctive therapies include bronchodilators, nebulization, chest physiotherapy, analgesia, and anxiolysis. Prophylactic antibiotics and corticosteroids are not routinely recommended. Hydroxocobalamin should be considered when cyanide toxicity is suspected.

Prognosis
Prognosis depends on severity of inhalation injury, extent of concurrent burns, and development of delayed respiratory or neurological complications. Mildly affected patients that respond rapidly to oxygen therapy often have a favorable outcome. Negative prognostic indicators include burns exceeding 20% TBSA, progressive respiratory failure, development of ARDS, and severe or persistent neurological dysfunction. Owners should be counseled regarding the risk of delayed complications even after apparent clinical improvement.

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Sedation and anesthesia in the ICU

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Sedation and anesthesia are commonly required for patients in the ICU, yet there are few veterinary resources to guide clinicians in this area. Resources are limited to textbook chapters,[1-3] and occasional original literature[4, 5]. This lecture will review indications for sedation and anesthesia in the ICU, different drug options (including doses, advantages, and disadvantages), and review relevant literature.

Indications for sedation and anesthesia in the ICU

Indications are broad and include procedural sedation (eg. wound management, instrumentation), intubation and mechanical ventilation,[1] as well as seizure control for status epilepticus or cluster seizures[6]. In all cases particular consideration must be given for patient requirements for analgesia and anxiolysis, and analgesia first sedation should be a priority in veterinary medicine as it is in human medicine.[7]

In human medicine, procedures associated with greatest pain intensity include arterial catheter insertion, chest tube removal, wound drain removal, turning and repositioning, and tracheal suctioning.[8] As such the need for excellent analgesia, as part of sedation protocols for such procedures, is imperative.

While anesthesia for seizure control is somewhat of a unique indication, you may be surprised that some of the drugs recommended are very similar to those we would use in other ICU patients such as animals receiving mechanical ventilation. Further information about seizure control can be found in recent consensus guidelines.[6]

Choice for drugs for sedation and anesthesia

There is no perfect sedation protocol, indeed I often say when it comes to sedation and anesthesia you must “pick your poison” recognizing that all drugs have adverse effects that must be considered. Choice of agents should be based on features including drug characteristics (safety and effectiveness), availability, patient disease processes, predicted duration of sedation/anesthesia, expense, as well as the clinician’s experience and comfort level. Careful consideration of drug choices is perhaps even more important in an ICU setting since “In critically ill patients, unpredictable pharmacokinetics and pharmacodynamics secondary to drug interactions, organ dysfunction, inconsistent absorption and protein binding, hemodynamic instability, and drug accumulation can lead to adverse events.”[9]

Inhalant anesthesia

While inhalants such as isoflurane and sevoflurane remain the mainstay of general anesthesia outside of the ICU they are not commonly used for patients in the ICU and hence will not be covered here. They have the advantage of effectiveness and rapid onset, but require a scavenging system, and have adverse effects including profound peripheral vasodilation and impairment of hypoxic pulmonary vasoconstriction which is problematic for patients with lung disease requiring ventilation.

Multimodal agents used as part of total intravenous anesthesia (TIVA)

Drugs commonly used for sedation and anesthesia in ICU include opioids, alpha-2 agonists, benzodiazepines, ketamine, and propofol. Examples of drugs within these categories, advantages, disadvantages, as well as drug doses are included below.

Opioids are a mainstay of sedation and anesthesia in the ICU since they provide both analgesia and sedation. Indeed, sedation guidelines in human medicine emphasize good pain control since there is evidence that critically ill adults experience moderate-to-severe pain at rest and during standard care procedures.[9] Advantages of opioids include their ability to provide excellent analgesia, short duration of action of some (eg. fentanyl, remifentanil) that facilitates easy titration, their cardiovascular sparing, and reversibility with naloxone (at least for pure mu agonists). Disadvantages include panting, which may worsen ventilator dyssynchrony, reduced gastrointestinal motility, hyperthermia in some cases, and potentially also immunosuppression.[10] Sedation is dose dependent and so doses used vary based on drug and intended level of sedation.

Alpha-2 agonists such as medetomidine and dexmedetomidine have experienced a bit of a resurgence in the ICU setting in the last decade with evidence in human medicine demonstrating benefit when used as part of multimodal protocols, particularly for ventilator patients. Alpha-2 agonists are anxiolytic and have some analgesic properties, a short duration of action facilitating titration, and are reversible with atipamezole. Additionally, they cause vasoconstriction, at least in the early stage after administration, which may be beneficial in ICU patients with inappropriate vasodilation and/or hypotension. They can however also cause cardiovascular instability including bradycardia, and ultimately hypotension. Studies in human ICU patients have shown that dexmedetomidine may be associated with shorter time to extubation,[11] increased days free from coma or delirium,[12] reduced incidence of agitated delirium,[13] and lower mortality than other sedative agents in certain populations[14, 15].

There is less research on alpha-2-agonists in dogs in an ICU setting than people, but one study of demonstrated that dexmedetomidine (1ug/kg/h CRI) resulted in lower noradrenaline requirements than saline placebo in septic dogs undergoing surgery.[16] The median (Min-Max) noradrenaline dose was 0.12 ug/kg/min (0-0.86) in the dexmedetomidine group vs. 0.8 ug/kg/min (0.4–2) in the saline group.[16] Commonly used doses of alpha-2-agonists in an ICU setting are 0.5-2 ug/kg as an IV bolus, and 0.5-2ug/kg/h CRI. Another veterinary study has compared two different TIVA protocols for sedation in mechanically ventilated dogs (n = 12, 6 dogs per group) with the use of medetomidine being one of the differences between groups. Dogs in both protocol groups received diazepam or midazolam at 0.5mg/kg/h. Protocols were different in the opioid they received where protocol 1 dogs received morphine (0.6 mg/kg/h) and protocol 2 dogs received fentanyl (18 ug/kg/h). The other difference was the protocol 1 dogs received medetomidine (1 ug/kg/h), while protocol 2 dogs received propofol (2.5 mg/kg/h = 0.04 mg/kg/min). While this was an experimental study in healthy dogs, routine care of ventilator patients was performed during the 24h study. Protocol 1 (ie. the protocol using medetomidine instead of propofol) was associated with decreased heart rate, oxygen consumption (VO2), and oxygen extraction ratio (O_2ER).

Medetomidine is a 1:1 racemic mixture of the D-isomer dexmedetomidine and the L-isomer levomedetomidine. There is evidence that only the D-isomer is pharmacologically active at clinically relevant doses and that levomedetomidine has no biological activity such that dexmedetomidine can be used at half the dose of medetomidine. However, few clinical studies have compared their use. One study in cats that used twice the dose of medetomidine than dexmedetomidine in combination with ketamine, buprenorphine, and midazolam focused on recovery time rather than effectiveness.[17]

Benzodiazepines have long been used in an ICU setting for sedation and as part of multimodal anesthetic protocols. They are predominantly sedative and anxiolytic, as opposed to analgesic, and have anti-epileptic properties. Again, advantages include their short duration of action and reversibility with flumazenil. Midazolam tends to be used in preference to diazepam since the propylene glycol carrier in diazepam is problematic when it accumulates and phlebitis concerns that

necessitate CRI administration via a central catheter. Midazolam is generally used as a 0.2mg/kg IV bolus and 0.2 mg/kg IV CRI.

Ketamine is a dissociative anesthetic and NMDA receptor antagonist. Ketamine has the advantage of providing good adjunctive analgesia with a generally favourable safety profile with regard to the cardiovascular and respiratory systems. Nonetheless ketamine increases sympathetic tone resulting in increased myocardial work and oxygen consumption, such that it is not recommended in animals with restrictive or hypertrophic cardiomyopathy, or where increased sympathetic tone is undesirable (eg. hyperthyroidism, phaeochromocytoma). It also has anti-inflammatory and immunomodulatory properties and so may be particularly useful in sepsis.[18] Disadvantages of ketamine include behavioural adverse effects such as excitement on emergence from anesthesia, myoclonus, and increased motor activity, but these tend not be a problem when used with other drugs such as benzodiazepines or alpha-2 agonists. Ketamine is excreted by the kidneys and so particular care should be taken in those with reduced GFR. In an ICU setting ketamine is commonly used at a CRI dose of 0.2-0.5 mg/kg/h.

Propofol produces reliable anesthesia and its short duration of action facilitates titration. It also has the benefit (although not always desired) of providing calories due to its lipid content. Disadvantages are numerous and warrant efforts to minimise CRI doses. It is commonly associated with respiratory depression and hypotension / CV depression. It also has some unique adverse effects in that it can induce oxidative injury to erythrocytes (Heinz body formation, eccentrocytes), which may lead to anemia in dogs and cats.[19] Doses of propofol required for a brief procedure in a sick patient may be as low as 0.5-1mg/kg after premedication, while CRI doses may be 0.1-0.6 mg/kg/h titrated to effect.

Barbiturates such as phenobarbital and pentobarbital are only used as anesthetic agents in the context of status epilepticus or cluster seizures in modern ICUs. Phenobarbital loading (eg. 20-24mg/kg on the first day) is part of 2nd line stabilisation in patients with status epilepticus, while inducing a barbiturate coma with pentobarbital (Bolus 6-15mg/kg, CRI 0.5-2.5mg/kg/h) is part of third line treatment.

Monitoring and Patient comfort

Monitoring of anesthesia in the ICU is similar to what is done for anesthesia in general including assessing palpebral reflexes, eye position, jaw tone, response to stimulation, and ventilation. Concurrently vital signs should be monitored with continuous ECG, SpO2, blood pressure, and temperature monitoring.

Patient comfort should also be considered as a comfortable patient whose physiologic needs are met will be more physiologically stable and ultimately then require less pharmacologic intervention. This includes avoiding hypoxemia and hypercapnia, performing bladder care, using padded bedding and comfortable positioning, eye care, maintaining a quiet environment and/or using ear plugs, mimicking day/night cycles and other considerations to maximise sleep, as well as heat support / temperature management.

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Acute Respiratory Distress Syndrome
Dr. Corrin Boyd

The acute respiratory distress syndrome (ARDS) is a severe respiratory complication of critical illness. It requires intensive management and leads to high morbidity and mortality.

Pathophysiology
The most current conceptual model of ARDS (Matthay *et al*, 2024) summarises the important pathophysiology. It states that:

"ARDS is an acute, diffuse, inflammatory lung injury precipitated by a predisposing risk factor, such as pneumonia, nonpulmonary infection, trauma, transfusion, burn, aspiration, or shock. The resulting injury leads to increased pulmonary vascular and epithelial permeability, lung edema, and gravity-dependent atelectasis, all of which contribute to loss of aerated lung tissue. The clinical hallmarks are arterial hypoxemia and diffuse radiographic opacities associated with increased shunting, increased alveolar dead space, and decreased lung compliance. The clinical presentation is influenced by medical management (position, sedation, paralysis, positive end-expiratory airway pressure, and fluid balance). Histological findings vary and may include intraalveolar edema, inflammation, hyaline membrane formation, and alveolar hemorrhage."

The underlying pathophysiology occurs in acute exudative and subacute fibroproliferative phases. Mediators involved in this process are summarised in Ware and Matthay, 2000.

Clinical Diagnosis
Development of ARDS is suspected when there is new or progressive respiratory dysfunction in a critically ill animal. There is no single definitive diagnostic test, as ARDS is a syndrome rather than a single disease.

The clinical diagnosis of ARDS is based on the ARDSVet consensus definitions (Balakrishnan *et al*, 2025). There are five criteria that apply to all ARDSVet categories: four essential and one optional. The essential criteria are:

- Risk factors: precipitated by a known or suspected acute predisposing risk factor or clinical insult. Common risk factors in small animals include aspiration pneumonia, sepsis, non-infectious systemic inflammation, and shock.
- Origin of edema: left-sided congestive heart failure (L-CHF) and fluid overload as the cause of pulmonary infiltrates should be ruled out when diagnosing ARDS. Ultrasound (echocardiography, POCUS) is useful; however, thoracic radiography, history, and physical examination findings may be used to support ruling out L-CHF and fluid overload.
- Timing: new or worsening respiratory distress within 1 week of known or suspected clinical insult.
- Thoracic imaging: thoracic imaging demonstrating diffuse pulmonary infiltrates using CT, radiography, or thoracic ultrasound is required. Thoracic radiographs or CT are preferred; however, ultrasound can be considered if these are not available and the operator is sufficiently trained.

The optional supporting criterion is:

- Neutrophilic inflammation and high protein levels in airway fluid collected through tracheal wash or bronchoalveolar lavage.

Additionally, there are criteria that vary based on the means of oxygen supplementation at the time of diagnosis. For animals that are intubated and mechanically ventilated (IMV-ARDS), there must be positive end-expiratory pressure (PEEP) of 5 cmH_2O or higher and one of the below criteria:

- Mild/moderate: PaO_2 :FiO_2 ratio >100 and ≤300 OR SpO_2 /FiO_2 ratio >150 and ≤315 (with SpO_2 ≤97%).
- Severe: PaO_2 :FiO_2 ratio ≤100 OR SpO_2 /FiO_2 ratio ≤150 (with SpO_2 ≤97%)

For nonintubated animals, the criteria for most situations are that there must be provision of high-flow nasal oxygen with a flow rate >1 L/kg/min or total >30 L/min, or supplemental oxygen with known FiO_2, in addition to the mild/moderate or severe oxygenation criteria above. There are separate criteria for equine nonintubated ARDS (Equines >24-h old: PaO_2 ≤60 mm Hg, equines <24-h old: PaO_2 ≤45 mm Hg).

Management

There is no specific medication that definitively improves outcome in ARDS. Management is primarily supportive. Effective treatment of the underlying risk factor/s is important, if these can be identified and treated.

The key principle of ARDS treatment is maintenance of adequate oxygenation for tissue oxygen delivery, whilst avoiding further iatrogenic injury. Some cases of mild ARDS may be treated with carefully titrated conventional oxygen therapy, but this is often inadequate. High-flow nasal oxygen has recently emerged as a promising intermediate modality that can effectively treat some moderate cases that fail conventional oxygen support, avoiding intubation. However, most cases of ARDS require intubation and mechanical ventilation. There is little evidence in veterinary species to support any specific ventilatory strategy. In human medicine, there is support for the concept of lung-protective ventilation. Compared to conventional mechanical ventilation, lung-protective ventilation involves using smaller tidal volumes to limit peak and driving pressure, which requires permissive hypercapnia. Oxygenation targets are lower than conventional (such as SpO_2 of 88-90%) and higher PEEP is used, to allow the use of lower FiO_2. This technique minimises ventilator-induced lung injury by limiting alveolar overdistension, cyclic atelectasis, and oxygen toxicity.

Careful attention to fluid balance is paramount, as the increased endothelial permeability in ARDS may magnify fluid transudation in response to increased capillary hydrostatic pressure. Fluid therapy should aim to maintain adequate perfusion whilst avoiding excess volume administration.

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Open cardiac surgery for congestive heart failure

Masashi Mizuno

Cardiogenic pulmonary edema (CPE) is a well-recognized acute decompensating event in dogs with myxomatous mitral valve disease (MMVD) and severe mitral regurgitation. The reported incidence of pulmonary edema in dogs with MMVD is approximately 20%, and its onset significantly worsens prognosis.[1] In our study, the median survival time after the first episode of pulmonary edema was 334 days, with a one-year survival rate of 46%.[2] The discharge rate for dogs and cats hospitalized with acute congestive heart failure, including pulmonary edema, is reported to be approximately 80%,[3] although this rate may decrease in more severe cases requiring positive pressure ventilation.[4]

The mainstays of treatment for pulmonary edema consist of oxygen supplementation, diuretics, and positive inotropic agents. Diuretic therapy may require not only dose escalation but also adjustment of the administration route. Pimobendan dosage may be increased, and in hospitalized patients, dobutamine may be administered. In cases where urine output and respiratory improvement remain inadequate despite these measures, carperitide may be considered. When repeated therapeutic adjustments fail to achieve adequate clinical response, early consideration of surgical intervention is warranted.

Surgical treatment for mitral valve disease can be broadly categorized into valve replacement and valve repair (commonly referred to as mitral valvuloplasty). Valve replacement using mechanical or bioprosthetic valves has been reported; however, no prosthetic valves are specifically manufactured for canine use. Although there is a report describing mitral valve replacement using a reversed human mechanical aortic valve,[5] valve replacement is not currently a standard procedure in dogs. Limitations in valve availability and the requirement for lifelong antithrombotic therapy represent major disadvantages of valve replacement. In contrast, mitral valve repair preserves the native valve by replacing ruptured or elongated chordae tendineae with artificial sutures and reducing annular dilation to restore proper coaptation and control regurgitation. At our center, postoperative antithrombotic therapy is typically required for only three months.

Mitral valve repair is not without risk, and postoperative complications may occur. However, in recent years, our center has achieved a discharge rate exceeding 97%, with one-year survival rates in the low 90% range and three-year survival rates exceeding 70%. These outcomes are substantially superior to those reported with medical management alone following pulmonary edema and suggest that surgery represents a viable treatment option. Appropriate stabilization after pulmonary edema is critical for surgical safety. Although based on limited experience, cases requiring mechanical ventilation for pulmonary edema prior to surgery appear to have a poorer prognosis. While early surgical intervention may be considered in cases refractory to medical therapy, successfully overcoming the acute phase of pulmonary edema and proceeding to surgery after clinical stabilization are key factors for optimal outcomes.

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Pros and cons in surgical emergency

Panelists: Hikaru Tezuka (TRVA Animal Medical Center), **Hiroaki Sugiura** (Yokohama Animal Emergency Medical Center)
Moderator: Atsushi Morita (Okayama Night Animal Emergency Hospital)

In emergency veterinary medicine, clinicians are frequently challenged by determining the appropriate level of surgical intervention for patients presenting unexpectedly with a wide range of conditions. One of the primary roles of emergency animal hospitals is to stabilize patients and provide time until definitive treatment can be performed. However, deciding whether surgery should be performed immediately in the emergency setting is often complex. For example, in cases such as pyometra, while surgery is ultimately required, it may be difficult to determine whether immediate emergency surgery is essential for survival or whether it significantly influences prognosis. Decisions regarding surgical intervention depend on the surgeon's expertise, hospital resources, and the time required for cardiovascular stabilization. Additional considerations include whether referral to a primary care veterinarian or a specialty referral center may provide more appropriate care.

Nevertheless, excessive hesitation may result in missed opportunities for life-saving intervention. Delayed surgical decisions can lead to rapid deterioration and a point at which intervention is no longer feasible. Numerous emergency conditions may require surgical consideration. This session focuses on intra-abdominal hemorrhage and ureteral obstruction as representative examples. Experienced emergency veterinarians will discuss real-world decision-making, including criteria for proceeding with emergency surgery, key precautions when performing surgery in the emergency setting, and factors supporting a decision to prioritize medical stabilization rather than immediate surgical intervention. Through interactive discussion, this session aims to provide practical guidance for balancing the risks and benefits of emergency surgical care.

Feline cardiac emergency

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Introduction

Acute respiratory distress is the most common presentation in feline cardiovascular emergencies, and rapid decision-making strongly influences survival. Unlike dogs, heart disease in cats is often difficult to detect clinically, as heart murmurs and cardiomegaly may be absent. Many cats with cardiomyopathy remain asymptomatic until decompensation occurs, frequently presenting in severe respiratory distress. Cardiogenic pulmonary edema (CPE) and pleural effusion, either alone or in combination, commonly lead to life-threatening ventilatory failure. This lecture outlines a practical emergency protocol to achieve rapid diagnosis and treatment while minimizing stress.

Initial Assessment and the Hands-Off Approach

The highest priority in emergency feline management is avoiding iatrogenic stress-related death. Forced restraint, extensive physical examination, or immediate radiography in severely dyspneic cats can be dangerous. Oxygen therapy should be initiated first, and remote observation (“hands-off” assessment) is recommended. CPE typically presents with tachypnea, shallow breathing, and increased respiratory effort, while pleural effusion often causes inspiratory effort and abdominal breathing. Open-mouth breathing, commonly seen in dogs, is a late and critical sign in cats. Subtle changes such as mild nasal flaring or orthopnea may indicate severe disease and must be recognized early.

Rapid Diagnosis Using POCUS

Auscultation of heart sounds is often difficult in dyspneic cats. Point-of-care ultrasound (POCUS), including TFAST (thoracic focused assessment with sonography), lung ultrasound, and focused cardiac ultrasound (FCU), is therefore highly valuable. Cats more frequently develop cardiogenic pleural effusion than dogs, and detection of anechoic pleural fluid by TFAST allows rapid identification of pathology. Lung ultrasound detection of B-lines is key for diagnosing CPE. While thoracic radiography remains central for diagnosing CPE in dogs, in cats it is limited by stress, pleural effusion, and variable radiographic findings. Noninvasive, real-time POCUS offers significant advantages. FCU assessment of left atrial enlargement (subjectively or via LA/Ao ratio) further supports a cardiogenic cause of dyspnea.

Emergency Treatment Protocol

Once a cardiogenic cause is suspected, decongestive therapy should begin immediately. When significant pleural effusion is present, thoracocentesis should be prioritized over diuretic therapy. In cats, removal of pleural fluid rapidly improves ventilation. Ultrasound guidance helps identify a safe puncture site, and fluid should be drained using a three-way stopcock while collecting samples for cytology. When CPE is suspected, early administration of furosemide is indicated. Respiratory rate and effort should be reassessed every 1–2 hours, with additional dosing or continuous infusion considered if improvement is inadequate. In cases with reduced cardiac output or hypotension, inotropic support may be required.

Monitoring and Secondary Evaluation

Thoracic radiography and comprehensive echocardiography should be performed only after respiratory stabilization. These assessments allow identification of underlying cardiac diseases such as hypertrophic or restrictive cardiomyopathy and facilitate long-term management planning.

Conclusion

Successful management of feline cardiogenic respiratory distress relies less on performing advanced diagnostics immediately and more on recognizing the patient's tolerance and minimizing stress. Rapid, minimally invasive assessment using POCUS, early oxygen therapy, and prompt, condition-specific decongestive treatment are key to improving survival in feline cardiovascular emergencies.

Career development for new veterinarians

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Emergency and critical care has become widely recognized as a core discipline in veterinary medicine, alongside internal medicine and surgery. As the final line of defense in regional veterinary care and as a rescue team supporting other specialties, veterinarians and veterinary nurses in this field work on the front lines every day. Foundational knowledge of emergency and critical care is increasingly valuable for all veterinary professionals pursuing long-term clinical careers. However, many essential skills—such as experience-based judgment, intuition, and decision-making—can only be developed through real-world clinical practice rather than textbooks or lectures.

Despite its importance, emergency and critical care is a demanding career path. Clinicians frequently face emotional challenges, including the loss of patients and exposure to high-pressure situations. Many professionals recognize the importance of this field and are interested in it, yet hesitate to pursue it due to concerns about whether they are suited to the lifestyle and responsibilities. What motivates those who choose this path? What experiences shape their careers, and what realities await them in this field?

In this session, three veterinarians representing diverse backgrounds—including early-career and experienced clinicians, practice owners and employed veterinarians, and both male and female perspectives—share their career journeys, motivations, and current reflections candidly. This unique discussion, characteristic of JaVECCS, explores emergency and critical care not only from an academic perspective but also from the viewpoints of workplace environment and practice management.

Interdialytic Complications

Sandy Young

Interdialytic complications are a significant concern in managing renal failure via intermittent hemodialysis (IHD) or other extracorporeal therapies. The term "interdialytic" refers to the time frame between dialysis sessions. The Dialysis Symptom Index (DSI) tracks 30 specific symptoms associated with this period, indicating that human patients on maintenance hemodialysis experience a median of nine concurrent symptoms, including fatigue, pruritus, muscle cramps, sleep disturbances, and gastrointestinal distress. Veterinary data on interdialytic complications is limited, as most studies have focused on technical efficacy and intradialytic safety rather than patient experience. Consequently, clinicians often rely on extrapolation from human data or owner-driven observations. Understanding interdialytic complications requires a conceptual framework that categorizes issues by timing, pathology, and origin. A systems-based approach, examining neurologic, gastrointestinal, musculoskeletal, mineral, and hematologic systems, can help clinicians better anticipate and mitigate adverse events.

Neurologic complications

Dialysis Disequilibrium Syndrome (DDS)

DDS, a severe neurologic complication, causes rapid removal of small solutes from the plasma during dialysis, while brain clearance is delayed. This creates an osmotic gradient, driving water into the brain and causing cerebral edema and potential herniation. Research in rat models shows the "reverse urea effect," where a 53% decrease in plasma urea leads to only a 13% decrease in brain urea, causing a 6% increase in brain water content. This highlights the risk of aggressive solute removal in naive patients. DDS typically occurs during dialysis or up to 24 hours afterward. Clinical signs are non-specific and may include restlessness, nausea, seizures, and in severe cases, death. Diagnosis is exclusionary, ruling out other causes of encephalopathy.

Prevention involves reducing dialysis efficiency during the first session in highly uremic patients, lowering blood flow rates, reducing dialysate flow, or using concurrent mannitol therapy to increase plasma osmolality. Treatment for active DDS involves stopping dialysis, administering mannitol, and managing seizures with anticonvulsants.

Uremic encephalopathy, on the other hand, is often a subacute or chronic condition resulting from the accumulation of uremic toxins in under-dialyzed patients.

Clinical Features: CKD onset is insidious, developing over weeks to months, while AKI presents within days of GFR decline. Symptoms include lethargy, irritability, stupor, and coma. Advanced imaging, like MRI, may reveal the "lentiform fork sign"—a bilateral, symmetrical hyperintensity in the basal ganglia surrounded by a hyperintense rim—associated with metabolic acidosis and uremia.

The management of uremic encephalopathy includes dialysis intensification to clear uremic toxins, but standard hemodialysis primarily removes small, water-soluble solutes. Some toxins, bound to proteins or middle-molecules, are not efficiently cleared, explaining persistent cognitive impairment. Hemoperfusion, using sorbents to adsorb larger toxins, improves sleep quality and melatonin levels, suggesting standard dialysis may be insufficient for all neurotoxins.

Uremic Neuropathy and Aluminum Toxicity: Uremic neuropathy manifests as a distal, mixed motor and sensory polyneuropathy, often affecting the legs more than the arms. Symptoms include ataxia, weakness, and "Restless Leg Syndrome." Aluminum toxicity is less common but critical. Suspect in

patients with well-controlled uremia but persistent neurologic deficits. Sources include phosphate binders and inadequate dialysate water treatment. A case report of two dogs with renal failure showed inconsistent menace response, tetraparesis, and reduced withdrawal reflexes, accompanied by microcytic anemia. Diagnosis is confirmed via serum aluminum levels, and treatment involves deferoxamine chelation.

Gastrointestinal and Nutritional Complications

Renal failure profoundly affects the gastrointestinal system, with up to 80% of ESRD patients experiencing GI symptoms. Common interdialytic signs include nausea, vomiting, anorexia, and dyspepsia, caused by gastroparesis, mucosal irritation, and autonomic neuropathy. ESRD patients have a five-fold increased risk of GI bleeding due to platelet dysfunction, anticoagulant use, and angioectasia. Pancreatitis is common concurrent condition seen in ESRD patients. Diagnosing pancreatitis in dialysis patients is challenging. Human ESRD patients are at higher risk for severe pancreatitis due to uremia and secondary hyperparathyroidism, but in veterinary patients, elevated amylase and lipase are common due to reduced renal clearance, not active inflammation. This “signal noise” necessitates clinical context and imaging for diagnosis.

Uremic sarcopenia

Sarcopenia, a progressive loss of muscle mass and strength, affects 11-30% of CKD patients. “Uremic sarcopenia” differs from simple starvation and is driven by metabolic acidosis, chronic inflammation, and hormonal resistance. Hemodialysis itself is catabolic, causing amino acid loss. Patients undergoing hemodialysis can lose 4 grams of amino acids daily, exceeding the basal daily loss in healthy controls. Aggressive nutritional support is crucial for preventing wasting. The “Tuft’s recipe” for AKI is a widely used high-protein formula. Specific micronutrient deficiencies must be addressed. For example, carnitine deficiency, found in 90% of IHD patients, correlates with sarcopenia and should be supplemented with L-carnitine (50-100 mg/kg q8h). Taurine deficiency is associated with fatigue and may be improved with supplementation (500-1000 mg/day for animals <25 kg and 1-2 grams/day for 25-40 kg).

Chronic Kidney Disease-Mineral and Bone Disorder (CKD-MBD) is a complex systemic disorder characterized by abnormalities in calcium, phosphorus, PTH, and vitamin D metabolism. Management of CKD-MBD centers on addressing phosphorus and calcium abnormalities. Patients with hyperphosphatemia require dietary phosphate restriction and phosphate binders such as aluminum hydroxide, sevelamer, lanthanum, or ferric citrate. In contrast, patients with hypophosphatemia may experience a rapid rebound after dialysis, necessitating intra-dialytic phosphate supplementation (e.g., Fleet enema) in the latter half of treatment. Hypercalcemia, often caused by neoplasia in dogs or idiopathic in cats, necessitates a comprehensive workup. Calcium and vitamin D supplements should be discontinued, and a low-calcium dialysate should be used. Hypocalcemia should be treated with intravenous calcium gluconate therapy for symptomatic patients (tremors, weakness) or severe hypocalcemia (iCa < 0.75 mmol/L).

Cardiovascular Complications:

Cardiovascular disease (CvRD) is the leading cause of death in human dialysis patients. Arrhythmias are prevalent, affecting up to two-thirds of patients, often due to rapid fluctuations in potassium and volume during or after dialysis. Historically, uremic pericarditis was a complication of severe uremia, but it is now less common with modern, timely dialysis intervention.

Respiratory Complications:

Respiratory compromise is a significant marker of poor prognosis in veterinary AKI. A study found respiratory signs in 54% of AKI and 26% of CKD cases, with alveolar patterns (suggestive of edema or

hemorrhage) being the most common. However, pulmonary abnormalities didn't change survival in the study. Fluid overload is the primary driver, but other causes include aspiration pneumonia and pulmonary hemorrhage associated with leptospirosis.

Hematologic complications are common in dialysis populations. Anemia is nearly universal, with 50% of dogs and 87% of cats requiring blood transfusions. Causes include blood loss, frequent phlebotomy, GI bleeding, EPO resistance, uremic thrombocytopahy, anticoagulant use, and pulmonary hemorrhage.

Thrombosis is another common complication. The hemodialysis catheter is thrombogenic, leading to clot formation within or outside the lumen. Outside the catheter, complications include caval syndrome, pleural effusion, and facial edema. Pulmonary thromboembolism is the most common non-catheter-related thrombosis.

Heparin-Induced Thrombocytopenia (HIT) is a serious immune-mediated adverse reaction to heparin exposure. It's caused by IgG antibodies binding to complexes of Platelet Factor 4 (PF4) and heparin, which paradoxically activates platelets and leads to thrombosis instead of bleeding.

Dermatologic Manifestations:
Uremic pruritus, a chronic condition affecting 40% of hemodialysis patients, is often resistant to therapies. It involves non-histaminergic mechanisms, including opioid system dysregulation, uremic toxin accumulation, xenobiotica, and microinflammation. Management includes increasing dialysis efficiency, gabapentin or pregabalin, kappa-opioid agonists, mu-opioid antagonists, UVB light, and acupuncture.

Uremic stomatitis causes painful ulcers, necrosis, and pseudomembranes on the oral mucosa and tongue due to high salivary urea concentrations. Oral bacteria with urease enzymes break down urea into ammonia, causing tissue damage. Treatment involves lowering blood urea nitrogen (BUN) through dialysis and local chlorhexidine care.

Calciphylaxis, a life-threatening syndrome of vascular calcification, causes ischemia and skin necrosis. Risk factors include hypercalcemia, hyperphosphatemia, and high PTH levels. Management involves stopping calcium and vitamin D supplements, aggressive phosphate control, and intensifying dialysis.

Conclusion

The "interdialytic" period is dynamic, where renal failure's physiological consequences continue despite intermittent dialysis support. Veterinarians must be vigilant for complications beyond the kidneys, affecting the brain, bones, heart, and skin. Understanding Dialysis Disequilibrium Syndrome, Heparin-Induced Thrombocytopenia, and CKD-MBD allows for proactive prevention instead of reactive management. Successful outcomes depend on dialysis machine efficiency and comprehensive patient management between treatments

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Extracorporeal Therapy for Cats

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Historically, the application of extracorporeal renal replacement therapy (ECT), including hemodialysis (HD), therapeutic plasma exchange (TEP), and hemoperfusion (HP) in cats was limited by technical challenges related to small patient size, vascular access, extracorporeal circuit volume, and hemodynamic instability. Advances in dialysis technology, neonatal circuits, anticoagulation protocols, and critical care monitoring have expanded the feasibility and safety of ECT in cats.

Indications

The primary indication for ECT in cats is severe acute kidney injury (AKI). Candidates for hemodialysis as a type of ECT typically present with persistent azotemia accompanied by clinically significant uremic complications as well as severe oliguria or anuria that fails to respond to appropriate fluid resuscitation and medical therapy. Progressive hyperkalemia, worsening metabolic acidosis, and life-threatening volume overload are the conditions noted in cats with severe oliguria or anuria. Importantly, the absolute magnitude of azotemia alone is not sufficient to determine candidacy. Rather, the decision to pursue ECT is based on the severity of metabolic and systemic complications and the failure of conservative management to stabilize the patient.

ECT also has applications in selected intoxications. Hemodialysis is most effective for toxins that are small, water-soluble, minimally protein-bound, and have a small volume of distribution. TPE is indicated in disorders mediated by circulating pathogenic substances that are large or highly protein-bound and therefore not effectively removed by diffusion-based dialysis. In cats, TPE serves as adjunctive therapy when rapid reduction of circulating antibodies, immune complexes, or paraproteins is clinically necessary. HP therapies may also be considered for the removal of protein-bound toxins or inflammatory mediators. These indications may include conditions such as immune-mediated diseases, selected neurologic immune disorders, and hyperviscosity syndromes secondary to monoclonal gammopathy.

Technical considerations, risk, and complications:

Extracorporeal therapy in cats presents unique technical and physiologic challenges due to small body size and limited circulating blood volume. Vascular access represents a major technical limitation. Compared with dogs, catheter size options in cats are restricted, and placement of large-bore hemodialysis catheters may be technically challenging because of small vessel diameter. Although adequate blood flow is required for effective solute clearance, use of catheters larger than 8 Fr may increase the risk of mechanical and thoracic complications. Large-diameter jugular catheters may predispose to venous obstruction, impaired lymphatic drainage, and development or worsening of pleural effusion, particularly in volume-overloaded patients. Careful catheter size selection, ultrasound- or fluoroscopy-guided placement, and close post-placement monitoring for respiratory compromise are therefore essential. Additional catheter-related complications include malposition, thrombosis, infection, and mechanical irritation, underscoring the importance of meticulous placement technique and ongoing access surveillance.

Even with pediatric dialyzers and blood lines, extracorporeal circuit volume may approach 10–20% of total blood volume in smaller cats. This predisposes patients to hypotension at initiation of therapy and necessitates careful priming strategies. In hemodynamically unstable or anemic patients, blood or colloid priming may be required to mitigate hemodilution and cardiovascular compromise. Continuous monitoring of blood pressure, heart rate, temperature, and perfusion parameters is critical, particularly during the early phase of treatment. Anemia is common during extracorporeal therapy and is multifactorial, resulting from hemodilution, incomplete return of circuit blood, repeated sampling, hemorrhage, and decreased erythropoietin production. Cumulative blood loss over repeated treatments may become clinically significant. In large feline dialysis cohorts,

the majority of cats required transfusion support, and transfusion requirements increased with the number of dialysis sessions performed. In addition to medical risks, transfusion increases the overall cost of ECT and may limit the feasibility of treatment in some cases.

Rapid solute removal during initial treatments may precipitate dialysis disequilibrium syndrome, particularly in severely uremic patients. Although less common with modern protocols, disequilibrium remains a recognized neurologic complication, especially when cats are treated using intermittent hemodialysis platforms. Clinical signs may include agitation, disorientation, seizures, or coma. Gradual reduction of blood urea nitrogen, shorter initial sessions, lower blood flow rates, and sodium modeling are recommended to minimize osmotic shifts and reduce risk.

Anticoagulation requires careful balance between prevention of circuit clotting and avoidance of hemorrhage. Inadequate anticoagulation results in extracorporeal circuit clotting and blood loss, whereas excessive anticoagulation increases bleeding risk, particularly in cats with uremic platelet dysfunction or recent surgical intervention. Catheter-associated thrombosis, including right atrial thrombus formation, has also been reported and may predispose to thromboembolic events. Individualized anticoagulation protocols and appropriate coagulation monitoring are therefore critical.

Therapeutic plasma exchange introduces additional considerations, including selection of appropriate replacement fluids and prevention of citrate-induced hypocalcemia. Plasma removal alters oncotic pressure and coagulation factor concentrations, necessitating careful monitoring. Citrate anticoagulation requires calcium supplementation and monitoring of ionized calcium concentrations. Maintenance of vascular access integrity is particularly important during repeated exchanges. Hemoperfusion and adsorption techniques, although less commonly used in cats, may increase extracorporeal volume and circuit resistance, potentially exacerbating hypotension. These modalities may remove protein-bound toxins or inflammatory mediators but may also unintentionally remove medications or endogenous proteins, requiring careful hemodynamic and laboratory assessment throughout treatment.

Outcomes:

Despite these technical challenges and complication risks, meaningful renal recovery is achievable in a substantial proportion of feline patients. Reported survival to hospital discharge for cats with AKI treated with hemodialysis ranges from approximately 40% to 60%, depending largely on underlying etiology and overall illness severity. Cats with reversible causes such as ureteral obstruction or certain infections generally have more favorable outcomes than those with non-reversible causes, including ischemic injury and certain toxicities (ethylene glycol and lily). Importantly, degree of azotemia alone does not reliably predict survival; reversibility of the inciting insult and systemic stability are more influential prognostic factors.

Long-term survival is attainable in cats that recover sufficient renal function to discontinue dialysis and survive to discharge. Although persistent azotemia and progression to chronic kidney disease are common, some survivors maintain acceptable quality of life for months to years. Mortality most often reflects severity of the primary disease process rather than failure of the ECT procedure itself. Careful case selection, close monitoring, and realistic prognostic assessment remain essential to optimizing patient outcomes.

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Muscle Weakness During Hospitalization: Helping Patients Walk Home

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This lecture focuses on intensive care unit–acquired weakness (ICU-AW), a common complication in critically ill and long-term hospitalized patients that remains poorly defined and insufficiently addressed in veterinary medicine. The aim is to present a new framework for nursing and rehabilitation that emphasizes functional recovery and quality of life (QOL) after survival. Advances in critical care have improved survival rates in severe cases; however, the growing challenge is that many patients struggle to return to their previous level of function after discharge. ICU-AW is therefore emerging as a major issue in veterinary critical care.

In human medicine, ICU-AW is widely recognized as a serious complication associated with sepsis, multiple organ failure, mechanical ventilation, deep sedation, prolonged immobility, corticosteroid administration, and hyperglycemia [1–3]. Its pathophysiology extends beyond simple disuse atrophy and includes rapid skeletal muscle loss, fiber-type changes, peripheral neuropathy, and neuromuscular junction dysfunction [4,5]. These changes are associated with prolonged ventilator dependence, longer ICU stays, increased healthcare costs, long-term functional impairment, and increased mortality [6,7]. ICU-AW is also considered a core component of post-intensive care syndrome (PICS), which encompasses physical, cognitive, and psychological impairments [8]. This concept reflects a shift in critical care goals from survival alone to recovery and reintegration into daily life—an approach that is increasingly relevant in veterinary medicine.

Although long-term functional decline after hospitalization is commonly observed in veterinary patients, it is rarely systematically evaluated, recorded, or treated. The strongest evidence in human medicine for preventing and mitigating ICU-AW supports early mobilization and early exercise therapy, even during mechanical ventilation [9–11]. Avoiding excessive sedation and maintaining appropriate levels of consciousness are also critical. These strategies can be adapted to veterinary practice through nurse-led interventions such as positioning, passive range-of-motion exercises, arousal stimulation, and assisted standing.

ICU-AW is closely linked to pain management, sedation protocols, nutritional support, sleep, and environmental factors. Poor pain control limits mobilization, excessive sedation promotes muscle loss, and appropriate nutrition helps suppress muscle protein breakdown and maximize rehabilitation outcomes [12–14]. Therefore, ICU-AW should be viewed not as a rehabilitation issue alone but as an outcome indicator reflecting the overall quality of inpatient care. Assessment should extend beyond the ability to stand or walk to include posture, weight-bearing capacity, joint range of motion, arousal level, and activity tolerance. Muscle weakness should be recognized as a preventable complication that must be addressed from the early stages of hospitalization.

Veterinary nurses, who have the most consistent patient contact, play a central role in preventing ICU-AW. This lecture aims to redefine ICU-AW as a preventable complication and promote a

continuum of care that extends from life-saving treatment to recovery and return to normal life. The ultimate goal of critical care is not simply survival but a return to life as close as possible to the patient's pre-illness state, with veterinary nurses playing an increasingly vital role in achieving this outcome.

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Pain Management in Hospitalized Patients

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This lecture aims to systematically organize practical and clinically reproducible knowledge and decision-making skills for pain management in critically ill and postoperative patients during hospitalization. Pain is defined as an unpleasant emotional experience; however, beyond sensory perception, it induces excessive sympathetic activation leading to increased heart rate and blood pressure, elevated oxygen consumption and work of breathing, release of inflammatory cytokines, immunosuppression, and metabolic hyperactivity. These responses contribute to delayed wound healing, increased infection risk, higher complication rates, prolonged hospitalization, and poorer prognosis [1,2]. Therefore, inpatient pain management should be regarded not only as a means of ensuring comfort but also as a therapeutic intervention to stabilize cardiovascular, respiratory, and metabolic function.

In critically ill or perioperative patients, pain may be difficult to recognize because sedation, analgesic administration, decreased consciousness, mechanical ventilation, neurologic abnormalities, and environmental stress can mask typical behavioral signs. As a result, observations such as reduced movement, quietness, or lack of vocalization may be mistakenly interpreted as adequate pain control. Inadequately assessed pain can lead to chronic stress and central sensitization, creating a cycle in which even mild stimuli provoke severe pain responses [5]. Pain must therefore be viewed as a dynamic parameter requiring continuous reassessment.

This lecture presents a multidimensional approach to pain assessment, integrating: (1) behavioral indicators such as posture, activity level, responsiveness, avoidance during palpation, facial expression, and muscle tension; (2) physiological parameters including heart rate and blood pressure trends, respiratory rate and pattern, ventilation indices, and sedation depth; and (3) potential pain sources such as surgical wounds, drains, catheters, urinary obstruction, and thoracic or abdominal disease processes [1,3]. Although individually nonspecific, longitudinal assessment of these parameters provides valuable early indicators of worsening pain. Clinical pain scoring systems used in dogs and cats, including the Glasgow Composite Measure Pain Scale–Short Form and the Feline Grimace Scale, will be reviewed with discussion of their scientific validity and limitations in ICU and long-term hospitalization settings [3,4]. Scores may underestimate pain in heavily sedated or critically ill patients; thus, they must be interpreted alongside clinical context and nursing observations.

The pharmacologic properties and monitoring considerations of commonly used analgesics will also be reviewed. Opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), α2-adrenergic agonists, local anesthetics, and NMDA receptor antagonists have distinct mechanisms and adverse-effect profiles, including cardiovascular and respiratory depression, renal and hepatic dysfunction, and gastrointestinal motility impairment [2,6]. In critically ill patients, multimodal analgesia is essential,

and veterinary nurses play a key role in observation, documentation, and communication to ensure safe and effective therapy.

Pain management must be understood as a cyclical process of intervention, observation, reassessment, and adjustment. Pain may recur after nursing care procedures such as repositioning, elimination assistance, rehabilitation, or wound care. Continuous monitoring and communication are therefore critical. The concept of “analgesia-first” is emphasized, distinguishing analgesia from sedation to avoid excessive sedation that may worsen respiratory or hemodynamic stability [1,2]. Effective pain management depends on team communication, including clear reporting of when pain occurs, what triggers it, and how it changes over time.

This lecture aims to reframe pain as a critical clinical indicator rather than merely a symptom and to highlight the essential role of veterinary nurses in evaluating, monitoring, and optimizing pain management in hospitalized patients.

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Basics of shock management

Shiro Watari

Shock is defined as a state in which a marked imbalance between oxygen supply and demand results in inadequate cellular energy production, ultimately leading to cellular death and multiple organ failure [1]. Circulatory function depends on two fundamental components—oxygen delivery and tissue perfusion—and disruption of this balance leads to the development of shock. Although shock is commonly associated with severe emergencies, even seemingly mild clinical signs such as vomiting or diarrhea in veterinary patients may progress to life-threatening shock if left untreated.

This lecture aims to provide veterinary nurses with a practical framework for understanding circulation and recognizing shock early in clinical practice. Circulation refers to the effective delivery of oxygen to every cell in the body, enabling normal organ function. It consists of two major components: (1) the ability to transport oxygen and (2) the ability to deliver oxygen to peripheral tissues.

Oxygen transport involves the diffusion of oxygen from the lungs into the bloodstream and its distribution to the body via cardiac output. This process depends on arterial oxygen content (CaO_2) and cardiac output (CO), where CO is determined by stroke volume (SV) and heart rate (HR). Tissue perfusion refers to the delivery of oxygenated blood to peripheral capillaries and ultimately to individual cells. Although tissue perfusion cannot be measured directly, mean arterial pressure (MAP) serves as an important clinical surrogate. MAP reflects both cardiac output and systemic vascular resistance (SVR), making blood pressure a critical indicator of circulatory status.

Early shock may be difficult to recognize because compensatory mechanisms can mask vital sign changes. Therefore, early recognition, confirmation, and intervention are essential for successful management. Continuous monitoring is ideal, but in many clinical settings, access to advanced monitoring tools may be limited. Practical bedside indicators—such as level of consciousness, blood pressure, urine output, and mucous membrane color/capillary refill time—are valuable for assessing circulatory status and tracking response to treatment.

This presentation will also discuss early intervention strategies and emphasize the importance of recognizing initial signs of shock in everyday clinical practice. The goal is to provide veterinary nurses with practical knowledge that can be immediately applied in clinical settings to improve patient outcomes and support professional growth.

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Nutritional Support for hospitalized patients: Distinct Approaches for AKI and CKD

Chie Ishii

Introduction

Acute kidney injury (AKI) and chronic kidney disease (CKD) are often grouped under the same category of “kidney disease,” yet their metabolic states differ fundamentally. Therefore, a uniform dietary approach can sometimes hinder recovery. The optimal nutritional requirements for AKI remain insufficiently understood, and selecting a renal therapeutic diet based solely on elevated BUN or creatinine is not always the best choice.

This lecture will review critical care strategies for AKI according to disease stage and progression, and contrast them with maintenance strategies for CKD, referencing both human and veterinary guidelines and consensus statements.

CKD Management – Quick Review

Dietary therapy for CKD is widely established in general clinical practice. However, to clarify the contrast with AKI, we will briefly review the basic strategies and common pitfalls.

Key Points in Nutritional Management of AKI

AKI often requires a prolonged recovery period, during which symptoms of uremia such as anorexia and nausea can lead to significant risks of protein-calorie malnutrition during hospitalization. This lecture will also discuss the impact of early nutritional intervention on prognosis and the appropriate timing for such intervention. Furthermore, AKI cases exist in a highly catabolic state under the influence of inflammatory cytokines. Inappropriate protein restriction can accelerate muscle breakdown, impair immune function, and increase mortality rates, as reported in the literature. Therefore, the focus should shift from merely lowering BUN to ensuring adequate supply to support tissue repair. In addition, individualized adjustments are required to correct AKI-specific metabolic abnormalities and changes in fluid, electrolyte, and acid-base balance.

Dietary Adjustment According to Disease Stage

AKI encompasses diverse conditions depending on etiology, case characteristics, and disease stage, and renal failure management must adapt dynamically. During anuria or oliguria, priority is given to addressing fluid overload and hyperkalemia, whereas in polyuric cases, it is necessary to compensate for negative balance caused by urinary losses of electrolytes and water-soluble vitamins. Moreover, nutritional strategies must be modified depending on whether the patient enters the recovery phase or transitions to CKD. In other words, renal failure management should not rely on fixed prescriptions but requires flexible strategies based on careful assessment of disease stage and internal dynamics.

Practical Approach to Product Selection

Rather than relying on product names, selection criteria should be based on nutrient composition. This lecture will demonstrate how to calculate RER and adjust protein, fat, and electrolytes according to disease stage, using simulations for comparative evaluation.

Conclusion

Nutritional management of renal failure is not a static prescription but a continuous process of dynamic assessment. Rapid decision-making in emergency settings can significantly influence post-recovery quality of life. This lecture aims to reaffirm this principle and provide practical insights that can be applied immediately in clinical practice.
Please note that due to time constraints (one hour including consecutive interpretation), parenteral nutrition will not be covered.

Emergency Nursing Guide: How We Do It in Our Hospital

Satoshi Matsukata, Yuto Mori, Takaomi Nuruki

Following last year's session, this lecture presents a practical approach to the challenges veterinary nurses face during emergencies and the nursing skills required to address them. Emergency nursing plays a critical role in protecting animal lives through close collaboration with veterinarians, particularly during the acute phase when rapid and appropriate responses are essential. This session introduces practical techniques and procedures that veterinary nurses can perform in real clinical settings to strengthen emergency nursing skills.

The lecture focuses on two areas frequently encountered in emergency care and often challenging to manage: **central nervous system abnormalities** and **body temperature disorders**. These clinical signs are common across many emergency conditions, and early recognition and prompt initial intervention by veterinary nurses can significantly influence diagnosis, treatment, and patient outcomes.

First, neurological abnormalities will be addressed. Core elements of neurological monitoring that veterinary nurses can perform—such as assessment of consciousness level, posture, seizure activity, pupil size, and pupillary light reflex—will be reviewed. Practical supportive care for animals during and after seizures, as well as safe handling of patients with altered mentation, will also be discussed. The session will include video-based explanations to clarify the differences between seizures and syncope and to improve recognition of seizure-related clinical signs.

Next, body temperature abnormalities, including hyperthermia and hypothermia, will be examined. Using conditions commonly encountered in emergency practice—such as heatstroke, sepsis, and shock—this lecture will explain the clinical significance of temperature monitoring and how to interpret temperature changes that may indicate rapid deterioration. Stepwise cooling and rewarming techniques will be described, along with precautions to prevent circulatory and neurological complications associated with rapid temperature changes. The importance of integrating temperature management with assessment of peripheral perfusion and mental status will also be emphasized.

Based on real emergency protocols used in our hospital, this lecture will help veterinary nurses review essential knowledge and skills required in urgent situations and acquire practical techniques applicable to daily clinical practice. Early recognition of abnormalities, prompt initial response, and effective teamwork are essential to saving animal lives. We hope this lecture will help strengthen confidence and competence in emergency veterinary nursing.

Entrusted with Blood Pressure Measurement! Essential Techniques and Clinical Application

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Introduction

Blood pressure is the force exerted by circulating blood against the vessel walls and is typically expressed in mmHg. In shock, acute illness, and cardiac patients, blood pressure is a rapid, noninvasive indicator of circulatory status and is indispensable for assessing pathophysiology and treatment response. In many clinical settings, veterinary nurses are entrusted with measuring blood pressure, and the values obtained may directly influence therapeutic decisions. This lecture aims to review the technical fundamentals required for accurate measurement in clinical practice, outline response algorithms when abnormal values are detected, and clarify the essential role of veterinary nurses in circulation assessment.

Technical Standardization and Key Points in Blood Pressure Measurement

Accurate blood pressure measurement begins with a thorough understanding of proper technique. In general practice, oscillometric and Doppler methods are most commonly used. Among potential sources of error, cuff size selection is one of the most critical factors. The cuff width should be approximately 40% of the circumference of the measurement site. Improper cuff selection can result in falsely elevated or falsely low readings and must always be carefully considered.

Whenever possible, the measurement site should be positioned at heart level to avoid hydrostatic error. Minimizing stress and excessive restraint is equally important, as anxiety and struggling can significantly affect results. Clinicians should avoid interpreting a single reading in isolation; instead, multiple measurements should be obtained to evaluate trends over time, thereby increasing reliability and clinical value.

Clinical Response Flow When Abnormal Values Are Detected

When hypotension or hypertension is detected, veterinary nurses are expected to promptly verify the value while simultaneously assessing the patient's clinical status.

If hypotension is identified—defined as systolic arterial pressure (SAP) < 90 mmHg or mean arterial pressure (MAP) < 60 mmHg—the first step is to confirm proper cuff placement and positioning,

followed by remeasurement at an alternative site if needed. Concurrently, perfusion parameters should be evaluated, including level of consciousness, mucous membrane color, capillary refill time (CRT), heart rate, and femoral pulse quality. If signs of hypoperfusion are present, immediate reporting to the veterinarian is required. Importantly, communication should include not only the numerical value but also physical examination findings and patient behavior, enabling rapid decisions regarding fluid therapy or vasopressor administration.

If hypertension is detected—typically SAP exceeding 160–180 mmHg—environmental factors such as stress, pain, or anxiety should first be evaluated. In emergency settings, transient stress-related hypertension is common. However, persistent hypertension carries a risk of target organ damage. Repeated measurements under calm conditions are recommended, along with assessment for retinal hemorrhage, neurologic abnormalities, and historical risk factors. Providing comprehensive contextual information to the attending veterinarian supports timely diagnostic and therapeutic decision-making.

The Evaluator's Perspective Required of Veterinary Nurses

The ultimate goal of blood pressure measurement is not merely recording a number but integrating that value into a comprehensive assessment of circulatory status. Blood pressure reflects the interaction between cardiac output and systemic vascular resistance; therefore, interpretation must consider the underlying pathophysiology.

In shock or acute illness, blood pressure may fall within reference ranges despite severe tachycardia or peripheral hypoperfusion, indicating compensated shock. Conversely, elevated values may warrant therapeutic modification depending on the clinical context. Veterinary nurses play a crucial role in synthesizing numerical data with physical examination findings and communicating objective assessments to the clinical team. This integrative perspective defines the professional function of veterinary nurses in emergency and critical care settings.

Conclusion

Blood pressure measurement is a powerful diagnostic tool that allows veterinary nurses to take an active role in frontline clinical care. By mastering technical accuracy and understanding appropriate response algorithms, veterinary nurses can evolve from simple "measurement operators" into clinical evaluators who contribute meaningfully to team-based decision-making. We hope this lecture enhances the quality of blood pressure assessment in everyday practice and ultimately contributes to saving more lives.

Facing Pet Loss as a Professional: Leadership and Grief Care Required for Emergency Veterinary Nurses

Noriko Niijima
Yamazaki University of Animal Health Technology

Research on pet loss and grief support in veterinary medicine has accumulated across psychological, clinical, and social domains. However, in veterinary emergency and critical care (ECC), unique conditions such as time constraints and unexpected outcomes often concentrate client communication and emotional support responsibilities on veterinary nurses and other healthcare professionals. In many cases, decisions and approaches depend heavily on individual experience and discretion. This situation may intensify client grief while also increasing emotional burden and threatening team sustainability.

This lecture aims to examine grief support not as a psychotherapeutic intervention but as a professional responsibility within veterinary emergency medicine and veterinary nursing education. The goal is to clarify how professionals should assess, engage, and provide support from an academic and neutral perspective. In particular, the traditional role of veterinary nurses as emotional supporters and coordinators will be reframed from an individual “kindness-based” practice into an educable and reproducible professional competency.

The first half of the lecture explores the characteristics of pet loss in ECC settings. Decision-making often progresses without sufficient shared understanding of anticipatory grief, increasing the psychological burden on pet owners. Additionally, healthcare professionals may experience moral distress and emotional labor. The concept of **reality disjuncture** will be used to explain how grief can be intensified by social misunderstanding and institutional gaps, highlighting structural factors that extend beyond individual emotional responses.

The second half of the lecture references the concept of **continuing bonds** to examine how grief may manifest across different stages. It then proposes the development of grief support as a team-based standard operating procedure (SOP) to prevent excessive burden on individuals. Veterinary nurses are redefined not only as providers of emotional support but also as professionals responsible for information organization, communication coordination, and educational bridging. This reframing aims to improve both quality of care and team sustainability.

This lecture does not challenge existing research and practice in pet loss support; rather, it builds upon them to consider how professional involvement in grief care can be positioned and passed on within veterinary emergency medicine and veterinary nursing education. The perspectives presented may serve as a shared language for reflecting on daily emergency practice and fostering team-based discussion.

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Diagnostic approach to cavitary effusion

Seigo Ogasawara, DVM, PhD, DACVP

Introduction: The Mission of Effusion Analysis in Emergency Medicine

In emergency practice, thoracic or abdominal effusions are frequently identified during ultrasound examinations. Once fluid accumulation is detected, clinicians must rapidly and accurately determine the nature of the fluid in order to guide treatment decisions. In this diagnostic process, veterinary nurses (VNs) play a critical role, as they are often responsible for sample collection, handling, and initial evaluation. Although emergency settings demand rapid action, prioritizing speed at the expense of diagnostic accuracy can lead to serious clinical consequences. This lecture highlights practical techniques for effusion analysis that are immediately applicable in emergency settings, while also discussing common pitfalls that may lead to diagnostic errors, illustrated through clinical cases.

The Quality of Sample Handling Determines Diagnostic Accuracy

The diagnostic process effectively begins the moment fluid enters the syringe. The gross appearance of the fluid immediately after collection—including color, turbidity, and viscosity—provides important clues that may predict subsequent laboratory findings. For example, bloody fluid warrants measurement of hematocrit, while milky fluid may indicate the need to evaluate lipid metabolism.

Proper allocation of samples between EDTA tubes and plain tubes is essential. EDTA preserves cellular morphology and allows accurate cell counts, whereas plain tubes are more appropriate for bacterial culture and biochemical analysis of the supernatant, such as glucose or lactate measurement. In emergency situations, the volume of collected fluid is often limited; therefore, prioritizing the most informative tests from a small sample becomes an important skill for VNs.

Special caution is required when using automated hematology analyzers for cell counts in effusion samples. Cells in effusion fluids are generally more fragile than those in blood, and aggregation or cellular debris may result in inaccurate counts. Thus, numerical results should always be interpreted in conjunction with microscopic evaluation of smear preparations.

Preparation of Diagnostic Smear Samples

Cytologic evaluation under the microscope is often the decisive factor in effusion diagnosis. However, improper smear preparation is one of the most common causes of diagnostic error. For samples with low cellularity, sediment preparations obtained after centrifugation are often useful. When preparing smears, minimizing mechanical damage to cells is critical. In particular, when using the squash preparation technique, only gentle pressure—essentially the weight of the slide itself—should be applied.

Another common mistake in emergency settings is inadequate drying of the smear. Rapid and complete air-drying, often aided by a hair dryer, is an essential step in producing diagnostically interpretable specimens.

Contribution to Rapid Diagnosis

This lecture presents representative clinical cases, including inflammatory and cardiovascular conditions associated with effusions, and explains how to interpret laboratory findings in these

contexts. For example, in suspected septic peritonitis, an effective strategy for detecting bacteria is to carefully examine the feathered edge of the smear and areas where cells are concentrated.

The ability to quickly identify key abnormalities—such as nuclear degeneration or intracellular pathogens—and to promptly report these findings to the veterinarian represents one of the most valuable contributions a VN can make to the clinical team.

Additionally, when cytologic interpretation is delayed or inconclusive, chemical analysis of the fluid can serve as a powerful diagnostic tool. Measuring differences in glucose or lactate concentrations between effusion fluid and serum allows rapid assessment of the likelihood of septic peritonitis within minutes. The use of such biomarkers significantly improves the speed and accuracy of diagnosis in emergency settings.

Conclusion

Effusion analysis should not be regarded as a routine technical procedure. When VNs approach each step with an understanding of clinical pathology and the significance of each technique, the results become vital data that directly contribute to patient survival. This lecture aims to organize practical strategies that help maintain a balance between speed and accuracy while improving diagnostic confidence within the veterinary medical team.

Exploring a Career in Emergency Veterinary Nursing

Shun Nakamura
General Animal Hospital – Specialty & Emergency Center

Introduction

How many veterinary nurses choose to pursue a career in emergency and critical care, even temporarily? Emergency medicine is often perceived as intimidating, physically demanding, and highly stressful. Critical care is similarly associated with the fear and pressure of managing severely ill patients. But are these perceptions accurate? Even if they are partly true, might the benefits outweigh the challenges? The reality can only be fully understood by those working in the field. This session brings together three panelists to discuss the real-world experience of emergency veterinary nursing, including both its advantages and challenges, through an interactive discussion with the audience.

Key Topics for Discussion

Common perceptions of emergency veterinary nursing

- Positive impressions (e.g., opportunities to acquire advanced skills)
- Negative impressions (e.g., physical demands and workload)

Is there a gap between perception and reality?

Characteristics of an emergency veterinary nursing career

- Is it too challenging for new graduates?
- Can it be a long-term career path?
- What career opportunities follow experience in emergency care?

How can we encourage more veterinary nurses to pursue this career path?

- Highlighting professional fulfillment and rewards
- Promoting its value as a mid-career pathway
- Emphasizing the transferable skills gained in emergency practice

This session aims to provide realistic insight into emergency veterinary nursing and encourage open discussion about the future of this career path.

JaVECCS 2026 – ICU Ultrasound for Veterinary Nurses
Title: ICU Ultrasound Every Veterinary Nurse Should Know

Go Ohtani

Point-of-care ultrasound (POCUS) has become increasingly recognized over the past 15 years as a rapid bedside tool for evaluating patient status in small animal practice. Thoracic and abdominal FAST examinations performed in emergency settings are examples of POCUS that allow clinicians to quickly identify causes of instability and support triage and stabilization. While POCUS is becoming a standard option for veterinarians, many veterinary nurses may still perceive ultrasound as a diagnostic test performed solely by veterinarians—sometimes even as a passive or routine part of patient restraint.

However, POCUS is not only valuable in emergency settings but also plays an important role in ongoing inpatient care. Its greatest advantage lies in the ability to monitor internal changes repeatedly and noninvasively. Serial POCUS examinations enable clinicians to track improvement or deterioration, detect new complications early, and provide more confident, evidence-based nursing care. As a result, the overall quality of inpatient management can be significantly improved.

In many general veterinary hospitals, ultrasound is not routinely used for hospitalized patients due to limitations such as equipment availability, workflow, staffing, or established practice patterns. Nevertheless, when the indications and benefits of POCUS are well understood, clinicians naturally recognize situations in which bedside ultrasound can provide immediate value—even if patients must be transported to an imaging room.

This lecture focuses on clinical scenarios frequently encountered in the management of critically ill hospitalized patients and explains the physiological background, appropriate indications, and interpretation of POCUS in a clear and practical manner. Alongside the minimum knowledge required for ultrasound image interpretation, case-based examples will be introduced throughout the lecture, including situations such as **1) evaluating decreased urine output, 2) monitoring gastrointestinal function, 3) detecting shock at an early stage, and 4) managing drains in hospitalized patients.**

Although time is limited, this lecture aims to provide practical and accessible guidance on how POCUS can be integrated into inpatient care and nursing practice. Ultimately, the goal is to empower veterinary nurses to use POCUS as a valuable clinical tool in the management of critically ill patients. This session is intended for anyone who has ever felt, “I want to improve inpatient care.” We look forward to meeting you at the conference.

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Nursing and Monitoring of Diabetic Ketoacidosis (DKA)
From “Knowing” to “Doing”: How Veterinary Nurses’ Awareness Can Save Lives

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Diabetic ketoacidosis (DKA) is a systemic metabolic disorder caused by absolute or relative insulin deficiency, resulting in impaired cellular glucose utilization and widespread metabolic dysregulation. Although DKA may be suspected in dogs and cats with known diabetes mellitus, it can also be identified in patients presenting with nonspecific signs such as decreased appetite and reduced activity, highlighting the need for vigilance in clinical practice.

When insulin is deficient, cells are unable to use glucose as an energy source and enter a state of cellular starvation. The body compensates by increasing lipid metabolism and gluconeogenesis to provide alternative energy sources. Enhanced lipid utilization ultimately leads to the production of ketone bodies, which are acidic metabolites. Excess accumulation of ketone bodies shifts systemic pH toward acidosis, resulting in acidemia. This metabolic disturbance can cause a wide range of clinical manifestations, including altered mentation, compensatory respiratory changes, cellular dysfunction, hyperkalemia, and myocardial impairment.

Persistent hyperglycemia further induces osmotic diuresis, leading to dehydration. Consequently, many DKA patients develop hypovolemic shock due to severe fluid deficits. The primary goals of intensive care in DKA are to eliminate ketone bodies through insulin administration, correct acidemia, and maintain hemodynamic stability through appropriate fluid therapy.

Once insulin therapy begins, blood glucose levels decrease; however, rapid reductions in blood glucose can cause neurological complications due to changes in plasma osmolality. Therefore, frequent blood glucose monitoring every one to two hours is essential. In addition, insulin therapy can trigger various electrolyte imbalances, making early detection and careful monitoring of these abnormalities a critical component of DKA nursing care. Successful outcomes depend not only on performing treatments and diagnostic tests but also on proactive nursing that anticipates complications before they occur.

This seminar will review the essential pathophysiology that veterinary nurses should understand, the key elements of initial assessment when encountering DKA patients, and practical nursing approaches and common pitfalls in inpatient care.

NOVEL APPROACH TO PLEURODESIS AND COATING WITH 50% GLUCOSE SOLUTION FOR AIR LEAKAGE AFTER LUNG RESECTION AND SPONTANEOUS PNEUNOTHORAX IN DOGS

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Background: Spontaneous pneumothorax (SP) in dog is a relatively rare and commonly caused by ruptured blebs and bullae (Lipscomb et al, 2003). Due to the limitations of preoperative diagnostics, thoracic exploration by median sternotomy is generally recommended (Puerto et al, 2002). Although surgery has high success rates, recurrence is reported in approximately 14% of cases (Howes et al, 2020). In human medicine, pleurodesis is frequently combined with bullectomy to suppress recurrence (Tokuda et al, 2025). This case report describes the clinical outcomes and potential efficacy of using 50% glucose solution pleurodesis (GP) for postoperative recurrence of SP in a dog.

Case Presentation: A 9-year-old neutered male Labrador Retriever (21.7 kg) was referred with dyspnea. Medical history included atopic dermatitis. Although blood tests showed no significant abnormalities, X-ray examination confirmed pneumothorax. Despite bilateral chest tube placement, air leakage remained uncontrolled. A CT scan suggested bullae in the right pleural cavity, leading to a right thoracotomy. Although the bullae in the posterior and accessory lobes showed no signs of rupture, these lung lobes resection was performed. Due to persistent postoperative air leakage, GP was performed on postoperative day (POD) 2, which successfully resolved the leak. The dog experienced multiple recurrences on POD 167, 217, 351, and 404. Blood patch (BP) was performed at 5 mL/kg (Oppenheimer et al, 2014), but showed limited success. In contrast, GP was performed using 200 mL for the initial dose (referenced from human medicine) and 100 mL for subsequent doses. Repeated GP (6 times in total) effectively managed air leaks. Although a transient increase in blood glucose levels was observed following GP, no other significant complications were noted. At the POD 417 follow-up, no further recurrences were reported.

Unique/New Information: In humans, pleurodesis and pleural coating are used in combination to suppress postoperative recurrence of SP. The method using 50% glucose solution was first clinically applied in Japan, and its efficacy has been demonstrated (Tsuboshima et al, 2018). In veterinary medicine, there have been reports of pleurodesis caused by blood patch (Oppenheimer et al,2014), but to my knowledge, no other agent-based pleurodesis have been reported. In this dog, GP demonstrated superior efficacy compared to conventional BP. While transient hyperglycemia occurred, the procedure was otherwise well-tolerated. Future prospective studies are necessary to substantiate the effectiveness and optimal dosage of GP, and to compare its efficacy and safety with other agents such as talc or OK-432. GP may represent a promising and minimally invasive option for managing refractory canine SP

Tailored Continuous Renal Replacement Therapy for Rhabdomyolysis-Induced Acute Kidney Injury in an Italian Greyhound: A Case Report

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Background: Rhabdomyolysis, characterized by acute skeletal muscle breakdown, can develop from diverse etiologies, including physical causes such as trauma and strenuous exertion, as well as nonphysical causes including drugs and toxins, infections, and autoimmune disorders. Affected patients frequently develop myoglobinuria, and myoglobin-mediated renal injury is a common cause of acute kidney injury (AKI) and other renal complications. This case describes the successful management of rhabdomyolysis-induced AKI using an individualized, tailored continuous renal replacement therapy (CRRT) protocol.

Case Presentation: A 2-year-old neutered male Italian Greyhound was referred to our hospital for renal replacement therapy after a presumptive diagnosis of rhabdomyolysis-associated AKI at a local veterinary clinic, with suspected anuria.
On presentation, the dog was oligouric to anuric, with urine output (UOP) < 1 mL/kg/hr. Serum biochemistry demonstrated severe azotemia (BUN 179 mg/dL; creatinine 6.6 mg/dL) and marked elevations in muscle enzymes consistent with rhabdomyolysis (CK 17,902 U/L; AST 2,222 U/L), with concurrent increases in hepatocellular enzymes (ALT 513 U/L). Serum myoglobin was increased (121.0 ng/mL). Urinalysis identified proteinuria and glucosuria, with yellow urine without myoglobinuria. Urinary Cystatin B was markedly elevated (985 ng/mL). Based on the owner's history, a recent episode of strenuous exercise preceded the onset of clinical signs. There was no history of drug/toxin ingestion, and no clinical or historical findings suggestive of an endocrine disorder or immune-mediated disease. In addition, both blood and urine PCR panels were negative, making an infectious etiology unlikely; therefore, a presumptive diagnosis of exertional rhabdomyolysis was made.
CRRT was initiated immediately and maintained for 13 hours, resulting in improvement of azotemia (BUN to 59 mg/dL; creatinine to 3.0 mg/dL). CRRT was delivered using a patient-tailored prescription specifically designed to prevent dialysis disequilibrium syndrome (DDS) and enhance patient stability. Following CRRT, spontaneous urine production resumed and the patient progressed into a polyuric phase during hospitalization, with peak UOP reaching 29.8 mL/kg/hr. By discharge, clinicopathologic values had markedly improved (BUN 18 mg/dL; creatinine 1.2 mg/dL; ALT 127 U/L; AST 44 U/L; CK 121 U/L), and serum myoglobin had decreased to below the assay's limit of quantification (below the measurable range).

Unique/New Information: This report describes successful recovery from rhabdomyolysis-induced AKI in an Italian Greyhound using a patient-tailored CRRT prescription specifically designed to minimize the risk of DDS. Conventional approaches can result in excessively rapid urea reduction, particularly during the early phase of dialysis; by contrast, the modified strategy applied here targeted gradual solute correction, thereby reducing DDS risk while achieving effective hemodynamic and clinical stabilization in a single patient. Notably, although myoglobinuria is commonly observed in

rhabdomyolysis, this case lacked myoglobinuria despite elevated serum myoglobin at presentation and normalization to below the measurable range by discharge, underscoring that the absence of myoglobinuria does not exclude clinically significant rhabdomyolysis-associated renal injury. The rapid transition from oliguria/anuria to a polyuric phase following CRRT, together with marked improvement in azotemia and muscle enzyme activities, supports CRRT as an effective therapeutic option for rhabdomyolysis-induced AKI when renal replacement therapy is indicated.

MACROSCOPIC AND MICROSCOPIC COMPARISON OF WOUND HEALING IN SURGICAL INCISIONS IN DOGS (*Canis lupus familiaris*) USING AUTOLOGOUS SERUM AND MUPIROCIN OINTMENT

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Objective: Autologous serum is naturally enriched with growth factors, vitamins, and immunoglobulins and has potential in enhancing wound healing during post-operative care. Although serum shares a composition similar to natural tears and contains essential wound healing factors, its potential for treating skin wounds has not been extensively studied. This study aims to see if autologous serum could be a useful alternative to current skin treatment such as mupirocin ointment in skin surgical incisions.

Methods & Materials: Six apparently healthy, adult female, non-descript, dogs for ovariohysterectomy were used for the study. All dogs had two incisions (one from the surgical procedure), one treated with autologous serum, and the other treated with mupirocin ointment. Photo-documentation and punch biopsy of the incision sites were performed on days 7 and 14, to elucidate the macroscopic and microscopic changes in wound healing. Skin samples were stained with hematoxylin and eosin for histologic examination. Grading of macroscopic evaluation included scar, scab and pus formation, hyperemia, wound elevation, and dehiscence, while microscopic evaluation looked into epidermal invagination and thickening, angiogenesis, wound gap, and neutrophil and fibroblastic infiltration. Mann Whitney U test was used to test the difference among treatment groups with significance set at $p<0.05$.

Results: The results showed comparable outcomes of the microscopic and macroscopic evaluation of the wounds. Both proved to show more desired wound healing responses, mitigating observed exacerbations in excessive healing. The cost analysis presented for autologous serum collection was based on hospital procurement prices, and the expense of one collection of autologous serum was proven to be cost-effective and more affordable. Despite being significantly cheaper, autologous serum has proven effective in expediting wound healing.

Discussion: Macroscopically, both treatments similarly produced minimal scabbing, absence of wound dehiscence, and timely progression to the remodeling phase, indicating effective and uncomplicated healing. Microscopic evaluation showed similar histologic responses, including early epidermal proliferation, evident angiogenesis, controlled inflammation, and organized extracellular matrix remodeling, with no statistically significant differences between groups ($p<0.05$). Given its substantially lower cost and ease of preparation, autologous serum may serve as a practical and cost-effective alternative for managing incisional wounds without compromising healing quality.

Conclusion: The results strongly suggest the capability of autologous serum as an effective alternative in accelerating the skin wound healing process. The beneficial properties of autologous serum were also proved in this study. Further studies must be done to determine the definite mechanisms behind the wound healing capacity demonstrated by autologous serum, as well as different reactions to different types of wounds across different species. It is also recommended to determine the effects of the patient's health to the volume as well as the effectivity of the serum produced.

Acute fluid redistribution preceding hemodynamic deterioration in an endotoxemic porcine model: insights from bioimpedance analysis

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Objective: During the acute phase of endotoxemia, rapid and dynamic changes in both hemodynamics and body fluid distribution occur. However, conventional hemodynamic monitoring primarily reflects intravascular circulation and does not directly assess alterations in whole-body fluid distribution. Therefore, this study investigated the temporal relationship between hemodynamic deterioration and acute body fluid redistribution using bioimpedance analysis (BIA) in an endotoxemic porcine model.

Methods&Materials: Seven anesthetized pigs were studied using a porcine endotoxin shock model. Endotoxemia was induced by continuous intravenous infusion of lipopolysaccharide (LPS) derived from *Escherichia coli* (20 μg/kg over 120 min). Hemodynamic monitoring was performed using transpulmonary thermodilution monitoring ($PiCCO_2$, PULSION Medical Systems), including measurements of cardiac output (CO), heart rate (HR), stroke volume (SV), systemic vascular resistance (SVR), global end-diastolic volume (GEDV), pulmonary vascular permeability index (PVPI), and systolic, mean, and diastolic arterial blood pressure (SAP, MAP, and DAP). Blood lactate concentrations (Lac) and hematocrit were also measured (GEM3500, IL Japan; pocH-100iV, Sysmex). Multi-frequency BIA was conducted using a commercial device (InBody M20, InBody), and resistance at zero frequency (R0) was analyzed as an index of extracellular fluid (ECF) distribution. Measurements were obtained at baseline and every 30 minutes after the start of LPS infusion up to 240 min. No fluid resuscitation or vasoactive agents were administered, except for cold saline boluses required for cardiac output measurements and minimal carrier fluids. Repeated-measures data were analyzed using linear mixed-effects models with subject identification as a random effect and time as a categorical variable, with Dunnett's post hoc test (P <0.05). All procedures were approved by the institutional animal care and use committee (approval number: VH24B1).

Results: CO, SV, SAP, MAP, and DAP markedly decreased from 90 min onward, while GEDV decreased after 120 min. HR and PVPI increased from 120 min onward. Hematocrit increased after 30 min, and blood lactate concentrations increased after 120 min, whereas SVR did not show significant changes. In contrast to the delayed hemodynamic deterioration, BIA revealed a decrease in R0 beginning at 60 min.

Discussion: Because R0 is inversely related to ECF volume, the early decrease in R0 suggests functional expansion of the ECF compartment occurring at an early stage of endotoxemia, preceding measurable deterioration in conventional hemodynamic parameters. The early rise in hematocrit, observed prior to changes in GEDV and PVPI, likely reflects hemoconcentration associated with fluid redistribution and altered blood cell distribution rather than true structural vascular leakage. In the later phase, concurrent increases in PVPI and decreases in GEDV indicate progressive vascular hyperpermeability and intravascular volume loss, despite sustained functional ECF expansion. These findings indicate that BIA can capture fluid shifts that precede and accompany hemodynamic collapse during the progression of endotoxin shock.

Conclusion: In this untreated porcine endotoxin shock model, bioimpedance-derived R0 detected early functional ECF redistribution prior to overt hemodynamic deterioration. Combined assessment of conventional hemodynamic monitoring and bioimpedance parameters may provide complementary insights into the temporal evolution of fluid distribution and vascular dysfunction during the acute phase of endotoxin shock, potentially improving pathophysiological assessment and informing therapeutic decision-making.

Precursor-Targeted Immune-Mediated Anemia in a Cat Concurrently Infected with FIV and *Candidatus Mycoplasma haemominutum*: A Case Report

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Precursor-targeted immune-mediated anemia (PIMA) is a serious condition characterized by immune-mediated destruction of erythroid precursors in the bone marrow, leading to profound non-regenerative anemia; however, it is reported to occur less frequently in cats (Lucidi, 2022). However, the nomenclature for this disease in cats is still debated, with various sources referring to it as non-regenerative immune-mediated anaemia (NRIMA), non-regenerative immune-mediated haemolytic anaemia (NRIMHA), and pure red cell aplasia (PRCA) (Maldonado-Moreno *et al.*, 2023). In contrast, immune-mediated hemolytic anemia (IMHA) involves immune targeting of mature red blood cells in the peripheral circulation. In cats, the most frequently identified underlying causes are infectious diseases, particularly hemotropic mycoplasmas, feline leukemia virus (FeLV), *Babesia felis*, and feline infectious peritonitis (FIP) (Garden *et al.*, 2019). Current data offer minimal evidence that feline immunodeficiency virus (FIV) or *Candidatus Mycoplasma haemominutum* have an associative role in PIMA (Garden *et al.*, 2019; Lucidi, 2022).

A 13-year-old male domestic shorthair cat was presented to the Small Animal Teaching Hospital (SATH), Faculty of Veterinary Science, Chulalongkorn University, due to lethargy and hyporexia. On physical examination, the cat had a body condition score of 4/9, a body temperature of 102°F, 5% dehydration, pale pink mucous membranes, a capillary refill time of 2 seconds, normal heart sounds with a heart rate of 200 beats per minute, normal lung sounds with a respiratory rate of 32 breaths per minute, and strong femoral pulses. The systolic blood pressure was 143 mmHg. Hematologic evaluation revealed non-regenerative anemia (0% aggregate reticulocyte, RBC 3.25 x 10^6/µL, hematocrit 16.2%). The anemia was characterized as normocytic and normochromic. Thrombocytopenia (60,000/µL) with the presence of giant platelets, as well as mild leukopenia (4,750/µL), were also noted. A direct antiglobulin test (DAT) for the diagnosis of IMHA, performed using flow cytometry, revealed 0% IgG-positive cells. PCR testing was positive for *Candidatus Mycoplasma haemominutum*, positive for FIV, and negative for FeLV. All blood biochemistry results were within normal reference ranges. The cat received whole blood transfusions to maintain its hematocrit level; however, the hematocrit continued to decline slightly. After the sixth transfusion, the owner consented to the bone marrow biopsy, and the procedure was performed. The section of the bone marrow was normocellular with adequate hematopoietic tissue. Erythroid lineage was decreased, while myeloid lineage revealed normal maturation and proportion. Megakaryocytes were adequate in the examined sections. A few macrophages containing red blood cells (erythrophagocytosis) were present. Prussian blue stain demonstrated iron-positive granules within a

few macrophages. This finding supports ongoing or prior erythroid precursor destruction or hemolysis. PIMA was suspected.

To our knowledge, this is the first reported case of presumptive PIMA in a cat that may have been associated with co-infection by FIV and *Candidatus Mycoplasma haemominutum*. *Candidatus M. haemominutum* is generally considered less pathogenic; however, co-infection with a retrovirus can exacerbate immune-mediated destruction of erythrocytes and lead to a significant decrease in RBC count (Messick, 2003; Greene, 2006; Paes *et al.*, 2012). This finding may enhance veterinary understanding and raise awareness regarding the diagnosis of anemia in cats.

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An Observational Case Series of Unanticipated Perioperative Hyperkalemia in Three Dogs and Three Cats

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Background: Perioperative hyperkalemia is a life-threatening emergency that may result in severe hemodynamic instability and cardiac arrest. In the veterinary literature, reported perioperative risk factors for hyperkalemia include underlying renal disease, prolonged anesthetic duration, extensive tissue injury, and concurrent use of full μ-opioid agonists and dexmedetomidine. However, unanticipated perioperative hyperkalemia remains poorly characterized, particularly in feline patients. This case series describes the clinical features and management of unanticipated perioperative hyperkalemia in dogs and cats.

Case Presentation: Six anesthetized animals that developed perioperative hyperkalemia between November 2022 and July 2025 were retrospectively reviewed. Patients were categorized based on perioperative exposure to dexmedetomidine (DEX group, n = 4) or no exposure to dexmedetomidine (non-DEX group, n = 2).

In the DEX group, one adult dog and three adult cats undergoing oncologic or thoracic procedures developed hyperkalemia despite normal preoperative renal indices and potassium concentrations. Dexmedetomidine was administered as a continuous infusion in all feline cases with concurrent opioid administration, while the dog received a single preoperative intramuscular dose. The mean procedure duration was 383.5 minutes. Hyperkalemia was identified during the late intraoperative period or early recovery. Three patients exhibited peaked T-waves on electrocardiography, and all demonstrated intraoperative hyperglycemia. Treatment with insulin, dextrose, or calcium gluconate normalized serum potassium concentrations within one hour, and all patients recovered uneventfully.

In the non-DEX group, two adult male dogs undergoing orthopedic surgery developed hyperkalemia. Both dogs were clinically healthy with normal preoperative renal indices. Analgesia consisted of a morphine–lidocaine–ketamine intravenous infusion. The mean anesthetic duration was 461 minutes, with hyperkalemia detected at a mean of 411 minutes after induction. Both dogs exhibited bradycardia and peaked T-waves. Management with insulin and dextrose, diuretics, and urinary catheterization resulted in normalization of serum potassium concentration within 12 hours.

Unique/New Information: This case series demonstrates that unanticipated perioperative hyperkalemia can occur even in canine and feline patients without apparent preoperative abnormalities. In the non-DEX group, prolonged orthopedic procedures combined with full μ-opioid analgesic infusions were consistent with previously reported perioperative risk factors of hyperkalemia. In the DEX group, the consistent presence of intraoperative hyperglycemia and the rapid response to dextrose- and insulin-based therapy suggest a possible role of impaired insulin-mediated potassium regulation during anesthesia.

To the authors' knowledge, unanticipated perioperative hyperkalemia in feline patients has rarely been reported. Although causal relationships cannot be established from this small case series, these findings highlight the importance of vigilant perioperative electrolyte monitoring during prolonged anesthesia. The concurrent use of opioids and dexmedetomidine, together with prolonged anesthetic duration, may represent potential risk factors for developing hyperkalemia in cats, similar to those observed in dogs.

Feasibility and Clinical Utility of Non-anesthetized Short-Duration MRI in Suspected Central Nervous System Emergencies

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Introduction and Objectives: Neurological emergencies such as increased intracranial pressure (ICP) require prompt neurological assessment and timely therapeutic intervention following systemic stabilization. Accurate evaluation of intracranial pathology relies on magnetic resonance imaging (MRI); however, in veterinary medicine, MRI generally requires general anesthesia to ensure patient immobility, and concerns regarding anesthetic risk in patients with suspected elevated ICP may delay or preclude imaging in emergency settings. The objective of this study was to assess the feasibility, safety, and clinical impact of non-anesthetized, short-duration MRI in patients with suspected central nervous system (CNS) emergencies.

Methods and Materials: Medical records of cases in which non-anesthetized MRI was performed for suspected CNS disease at the Neurology Service of the Veterinary Medical Teaching Hospital of our institution between April 2022 and January 2026 were retrospectively reviewed. All MRI examinations were conducted using a 3.0-T MRI system. Clinical data including signalment, neurological status, level of consciousness, modified Glasgow Coma Scale (MGCS) score, MRI acquisition protocols, imaging findings, and outcomes were collected. Conditions under which non-anesthetized MRI was feasible, and the characteristics of eligible cases were analyzed.

Results: Non-anesthetized brain MRI was performed in 16 cases, including 12 dogs and 4 cats. Neurological abnormalities observed in all cases at the time of MRI included loss of postural reactions in the limbs and altered consciousness or responsiveness. The median MGCS score was 15 (range 4–16). Despite relatively high MGCS scores in some cases, all patients exhibited absent responses to auditory stimuli, including loud clapping sounds near their ears. Short-duration two-dimensional T2-weighted imaging was successfully acquired in all cases, with the shortest acquisition time per sequence being 24 seconds. MRI diagnoses included increased ICP (n = 8), severe cerebral atrophy (n = 2), no remarkable abnormalities (n = 4), and other findings (n = 2). No examinations were deemed non-diagnostic due to motion artifacts. No deaths occurred during MRI acquisition. Following MRI, 13 cases received treatment for intracranial disease, 2 cases proceeded to additional diagnostics under general anesthesia, and 1 case was treated for an extracranial condition（hepatic impairment）.

Discussion and Conclusion: Short-duration non-anesthetized MRI was safely and successfully performed in selected neurological emergency patients and provided clinically meaningful information that influenced therapeutic decision-making. This approach was feasible even in patients with relatively high MGCS scores, suggesting potential utility not only for prognostication but also for disease screening and guidance of active treatment strategies. Markedly reduced responsiveness to auditory stimuli appears to be a minimum requirement for successful imaging without anesthesia. Because acquisition time is highly dependent on MRI system performance, broader clinical application of non-anesthetized MRI will require further optimization, including refinement of imaging protocols to reduce scan time, use of sedation or ear protection, and improved restraint techniques to prolong patient immobility.

AMYLOID CAST TUBULOPATHY AND ACUTE KIDNEY INJURY SECONDARY TO GRANULOMATOUS SPLENITIS IN A DOG

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Background: Acute kidney injury(AKI) in dogs can result from diverse etiologies, and histopathological evaluation is required to elucidate the underlying cause. Cast tubulopathy is characterized by intratubular obstruction by pathological casts, leading to renal parenchymal injury. We report a dog with AKI that was managed with repeated continuous renal replacement therapy(CRRT) and intensive medical treatment. Postmortem renal histopathological examination revealed findings consistent with cast tubulopathy, representing a rare renal pathological manifestation in canine AKI.

Case information: A 7-year-old neutered male French Bulldog was referred for hemodialysis because of AKI and oliguria. The dog had a 6-day history of lethargy and anorexia, followed by continuous vomiting and diarrhea. At the initial evaluation, azotemia(Creatinine 12.3mg/dL; ref. 0.5-1.8, Blood urea nitrogen 160mg/dL; ref. 7-27), hypoalbuminemia(1.7 g/dL; ref 2.3–4.0 g/dL), proteinuria, and systemic hypertension(systolic 190mmHg) were identified. No history of underlying conditions or toxin exposure associated with AKI was identified, and blood and urine PCR testing were negative. As the underlying cause of AKI remained unclear, CRRT was performed three times for the management of azotemia. Based on physical examination and blood work, amino acid supplementation and antihypertensive agents (amlodipine, hydralazine, and doxazosin) were prescribed. Because refractory hypertension persisted despite therapy, impaired gastrointestinal absorption was suspected, and intravenous nitroprusside was administered to maintain systolic blood pressure at 155 mmHg. Despite repeated dialysis and intensive care, renal function failed to recover. On 7th day of hospitalization, the dog experienced sudden cardiopulmonary arrest. Cardiopulmonary resuscitation was unsuccessful, and further resuscitation was discontinued at the owner's request.

A necropsy was performed, and histopathological examination revealed eosinophilic fractured casts filling the tubular lumina on hematoxylin and eosin staining, along with nodular lesions in the spleen. To evaluate renal amyloidosis, additional Congo red staining was performed, which demonstrated positive staining within the intratubular casts. These histopathological findings led to a diagnosis of amyloid cast tubulopathy which was considered to be secondary to chronic splenitis.

Unique/New Information: Cast tubulopathy is typically formed through the interaction of light chain amyloid and Tamm–Horsfall protein. In human medicine, this condition is most commonly associated with multiple myeloma and rarely reported with other underlying diseases. In this case, chronic splenitis was considered to have contributed to amyloid deposition within the renal tubules, leading to the development of cast tubulopathy. This report describes a rare occurrence and clinical course of AKI associated with amyloid cast tubulopathy in a dog.

A THREE-DIMENSIONAL ENDOTHELIAL SPHEROID MODEL OF ACUTE PULMONARY INFLAMMATION IN DOGS

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Introduction and Objective: This study aimed to establish a three-dimensional (3D) spheroid culture model of canine lung–derived endothelial cells and to compare inflammatory responses to lipopolysaccharide (LPS) between conventional two-dimensional (2D) and 3D culture systems, with a focus on morphological alterations and microRNA (miRNA) expression profiles.

Methods & Materials: Primary endothelial cells were isolated from canine lung tissue and characterized by flow cytometry. Cells were cultured either as monolayers (2D) or as spheroids using ultra-low attachment plates (3D). Both models were exposed to LPS at concentrations of 0, 50, 100, and 200 ng/mL for up to 72 h. Spheroid morphology was quantitatively analyzed using ImageJ by measuring area, circularity, roundness, and solidity. miRNA expression profiling was performed following LPS stimulation, and differential expression was defined using the following criteria: fold change ≥1.3, average normalized expression ≥1 ($\log_2$ scale), and $p < 0.05$.

Results: LPS induced time- and concentration-dependent morphological changes in endothelial spheroids, characterized by progressive reductions in spheroid area, circularity, roundness, and solidity, with the most pronounced structural collapse observed at 200 ng/mL after 48–72 h. In miRNA analysis, the 3D culture model exhibited substantially broader transcriptional responses to LPS, with 13 upregulated and 13 downregulated miRNAs, whereas the 2D model showed limited changes (2 upregulated and 2 downregulated miRNAs). Only one miRNA was commonly regulated between the two culture conditions, indicating distinct inflammatory response patterns depending on culture dimensionality.

Discussion and conclusion: The results demonstrate that 3D endothelial spheroids exhibit heightened sensitivity to inflammatory stimulation compared with 2D cultures, revealing morphological and miRNA regulatory changes that are not fully captured in monolayer systems. Reduced circularity and solidity in the 3D model reflected structural disruption and surface fragmentation, while the expanded miRNA response suggests a more physiologically relevant activation of endothelial inflammatory signaling. A 3D spheroid culture model of canine lung endothelial cells provides a more sensitive and biologically relevant platform for studying endothelial inflammatory responses to LPS than conventional 2D cultures. This model may serve as a valuable in vitro system for investigating vascular inflammation and for screening anti-inflammatory therapeutics in respiratory disease research.

Management of Severe Hypernatremia Due to Secondary Adipsia in a Dog with a Pituitary Macroadenoma

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Background: Adipsia or hypodipsia associated with hypernatremia has been reported in dogs, most commonly in juvenile patients with congenital forebrain malformations such as corpus callosum abnormalities. However, secondary adipsia caused by acquired intracranial disease in older dogs is rarely described. Pituitary macroadenomas may exert mass effects on hypothalamic structures, impairing thirst regulation and resulting in severe electrolyte imbalance.

Case Presentation: A 9-year-old neutered male mixed-breed dog was referred from a local animal hospital to the Emergency and Critical Care Service at Kangwon National University for lethargy, anorexia, vomiting, and absence of voluntary water intake. The dog had a prior history of polyuria and polydipsia followed by complete adipsia. Physical examination revealed dehydration, poor body condition, and intermittent seizure-like activity. Laboratory evaluation identified severe hypernatremia (190 mEq/L). Abdominal ultrasonography revealed bilateral adrenal enlargement and decreased pancreatic echogenicity. Magnetic resonance imaging demonstrated a pituitary macroadenoma with sellar region mass effect consistent with hypothalamic dysfunction. The patient underwent controlled correction of free water deficit over three days, reducing serum sodium to 163 mEq/L without neurologic complications. The dog was transitioned to outpatient management with trilostane and cabergoline for pituitary-dependent hyperadrenocorticism and pituitary macroadenoma. Appetite, mentation, voluntary water intake, and vital parameters improved, and serum sodium concentrations remained stably around the 150 mEq/L range for approximately six months. Follow-up computed tomography performed three months after initial imaging to monitor tumor size revealed a mild reduction in pituitary mass dimensions.

Unique/New Information: This case describes secondary adipsia caused by pituitary macroadenoma–associated hypothalamic dysfunction as an uncommon cause of severe hypernatremia in an older dog. It emphasizes the importance of gradual, closely monitored correction of hypernatremia in patients with impaired thirst mechanisms, providing clinically relevant guidance for emergency and critical care management.

Management of Refractory Hyperkalemia Induced by Renin–Angiotensin–Aldosterone System Inhibitors Using Desoxycorticosterone Pivalate in Two Dogs with Kidney Disease

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Background

Angiotensin-converting enzyme inhibitors (ACEis) and angiotensin receptor blockers (ARBs) are standard therapeutic agents for reducing glomerular hypertension and proteinuria in dogs with kidney disease. However, by suppressing the renin–angiotensin–aldosterone system, these medications can predispose patients to hyperkalemia, which may escalate to life-threatening levels. Currently, clinical reports detailing long-term management strategies for refractory hyperkalemia that persists even after the discontinuation of ACEi or ARB therapy in dogs remain limited.

Case Presentation

This report describes two dogs with underlying kidney disease that developed severe hyperkalemia following the administration of RAAS inhibitors.

Case 1 was a 10-year-old neutered male Pomeranian (2.95 kg) treated with telmisartan for proteinuria, which resulted in azotemia and marked hyperkalemia that did not resolve despite drug discontinuation and supportive care.

Case 2 was a 9-year-old castrated male Maltese (3.3 kg) receiving enalapril, presenting with generalized weakness and critical hyperkalemia. In both instances, serum potassium concentrations remained elevated long after withdrawal of the offending drugs, suggesting a state of aldosterone deficiency or functional hypoaldosteronism.

To regulate potassium levels, both patients were treated with subcutaneous desoxycorticosterone pivalate (DOCP). Initial and maintenance doses ranged from 0.5 to 1.25 mg/kg, with administration intervals tailored between 2 to 10 weeks based on electrolyte monitoring. Following the initiation of DOCP, serum potassium concentrations stabilized within the reference range, and no further life-threatening episodes were recorded during long-term follow-up, while renal parameters remained clinically manageable.

Unique / New Information

These cases demonstrate that DOCP can serve as an effective long-term treatment option for managing refractory, life-threatening hyperkalemia associated with ACEi or ARB therapy in dogs with chronic kidney disease. Mineralocorticoid supplementation offers a practical and safe strategy for electrolyte stabilization, particularly in emergency patients where hyperkalemia persists despite the cessation of RAAS inhibitors. This approach provides a novel management pathway for cases where functional hypoaldosteronism complicates the treatment of proteinuric kidney disease.

Keywords Chronic Kidney Disease (CKD), Hyperkalemia, Telmisartan, Enalapril, Desoxycorticosterone pivalate (DOCP), RAAS inhibitors

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Successful Management of Refractory Precursor-Targeted Immune-Mediated Anemia (PIMA) using Thrombopoietin and Oclacitinib in Two Dogs

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Background: This report details two canine cases of suspected precursor-targeted immune-mediated anemia (PIMA) that achieved hematologic recovery following treatment with hematopoietic stimulants. The findings highlight the therapeutic potential of these agents and underscore the significant immunologic risks, such as alloimmunization and transfusion-related complications, associated with the long-term management of transfusion-dependent non-regenerative anemia.

Case Presentation: Two dogs presented to the Jeju National University Veterinary Medical Teaching Hospital with persistent lethargy. Diagnostic evaluations, including PCR panels and comprehensive imaging, ruled out infectious, hemorrhagic, and neoplastic causes, leading to a presumptive diagnosis of PIMA. Treatment protocols included Thrombopoietin (Romiplostim, 5–10 μg/kg SC, once weekly), Darbepoetin alfa (1 μg/kg SC, once weekly), and Oclacitinib (0.5 mg/kg BID).

Case 1 was a 8-year-old castrated male Maltese (2.4kg). The patient failed to respond to multiple standard immunosuppressive regimens, but a significant hematopoietic response was observed only after the initiation of thrombopoietin (TPO). The hematocrit (HCT) subsequently rose from 10.1% to 25.3% and has remained stable for nearly two years.

Case 2 was a 6-year-old spayed female Poodle (4.1kg). The patient presented severe transfusion challenges, with 14 out of 17 crossmatches being incompatible, suggesting the formation of multiple alloantibodies. After a transiently compatible transfusion resulted in transfusion-related acute lung injury (TRALI), all further transfusions were permanently discontinued. Following the initiation of combination therapy with TPO, Darbepoetin alfa, and Oclacitinib, the patient's HCT stabilized within six weeks.

Unique/ New Information: These cases illustrate that the combination of TPO and Oclacitinib can lead to successful hematologic recovery in dogs with PIMA that are unresponsive to conventional immunosuppressants and transfusion support. These findings suggest that hematopoietic stimulants and Oclacitinib serve as a potent therapeutic alternative for refractory cases or for patients who have become transfusion-ineligible due to severe alloimmunization or life-threatening adverse reactions like TRALI.

MANAGEMENT OF PRESSURE-INDUCED VESICOURETERAL REFLUX VIA A URETERAL STENT IN A NEUROLOGICALLY IMPAIRED DOG REQUIRING MANUAL BLADDER EXPRESSION

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Background: Ureteral stenting is common for resolving obstructions in small animals but bypasses the vesicoureteral junction's natural anti-reflux mechanism. In patients with spinal injuries requiring manual bladder expression, this lack of a one-way valve can lead to severe, high-pressure vesicoureteral reflux (VUR). While stenting benefits are clear, the mechanical complication of forcing urine into the renal pelvis during manual compression is rarely addressed. This report describes managing a dog with a single kidney where manual expression induced symptomatic reflux through a stent, and the strategy used to mitigate this risk.

Case presentation: A dog with spinal trauma and a prior left nephrectomy presented with acute azotemia and abdominal effusion. Imaging confirmed ureteral calculi and hydronephrosis in the remaining right kidney. Stabilized via CVVHDF (CRRT), the patient underwent surgical ureteral stent placement. A concurrent ureteral rupture and urinoma were identified and successfully repaired during the procedure. Post-operatively, renal parameters and urinary leakage initially stabilized. Two weeks later, the patient was readmitted with fever, elevated creatinine, urinary incontinence, and dyschezia. Imaging revealed retroperitonitis and renal pelvis irritation. It was hypothesized that manual bladder expression, required for the patient’s paralysis, forced urine through the stent (acting as a low-resistance conduit) directly into the renal pelvis. This high-pressure reflux caused mechanical and chemical irritation of the upper urinary tract. To resolve this, a "low-pressure lower urinary tract" strategy was implemented. A continuous indwelling urethral catheter was placed to maintain an empty bladder, ensuring minimal urine was available for reflux during manual manipulation. Following this low-pressure environment, clinical signs and renal parameters improved rapidly. Long-term catheterization risks were managed via systemic antibiotics, antifungals, and regular bladder irrigation with 0.9% NaCl and EDTA to prevent biofilm formation. The patient remains stable with weekly catheter replacement and bladder flushing.

Unique/New Information: This case highlights a critical management conflict: using ureteral stents in patients requiring manual bladder expression. Stents eliminate anti-reflux defenses, turning external compression into a mechanism for renal injury. This report demonstrates that maintaining a "low-pressure" system via continuous drainage is a necessary strategy for such patients. Furthermore, it emphasizes proactive biofilm management using EDTA flushing to ensure the long-term success of permanent urinary implants in chronically catheterized patients

Clinical Interchangeability of a Point-of-Care Blood Gas Analyzer and Laboratory-Based Reference Analyzers for Electrolyte and Glucose Measurement in Canine Venous Blood

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Introduction and Objectives: Rapid and accurate diagnostic results are essential for clinical decision-making in veterinary emergency medicine. Blood gas analyzers are widely used for rapid assessment of electrolytes and glucose; however, data evaluating their analytical agreement with laboratory-based reference analyzers in canine patients remain limited, particularly in large cohorts. Additionally, information regarding clinically relevant differences between serum- and plasma-based reference measurements is insufficient. This study aimed to test the hypothesis that a point-of-care (POC) blood gas analyzer would generate results comparable to those of reference analyzers and to assess their clinical interchangeability.

Methods and Materials: Canine patients presenting to the Seoul National University Veterinary Medical Teaching Hospital between March 2024 and November 2025 were retrospectively identified when blood gas and reference analyses were concurrently performed on the same sample. After exclusion of unsuitable specimens, 187 serum-based and 143 plasma-based reference electrolyte results, along with 265 serum-based reference glucose results, were analyzed. Whole blood was immediately analyzed using a lithium-heparin syringe for blood gas testing, while remaining samples were submitted for reference analysis within 1 hour. Blood gas measurements were obtained using the GEM 5000 POC blood gas analyzer, whereas reference measurements were performed using the EX-D/Ds analyzer for electrolytes and the AU 480 analyzer for glucose. Agreement between instruments was evaluated using Passing–Bablok regression and Bland–Altman analysis. Observed differences were further assessed relative to American Society for Veterinary Clinical Pathology (ASVCP) - recommended total allowable error (TEa), and Mann–Whitney U tests were used to compare absolute bias between serum- and plasma-based reference measurements. Statistical analyses were conducted using R software and GraphPad Prism.

Results: Passing–Bablok regression showed no significant proportional or constant bias for sodium, chloride, or glucose, as slope confidence intervals included 1 and intercept confidence intervals included 0. In contrast, potassium demonstrated both proportional and constant bias in both serum and plasma samples. Bland–Altman analysis revealed that the blood gas analyzer tended to report lower values than reference analyzers for sodium (−4.10 mmol/L in serum; −4.28 mmol/L in plasma), potassium (−0.22 mmol/L in serum; −0.23 mmol/L in plasma), chloride (−0.22 mmol/L in serum; −1.05 mmol/L in plasma), and glucose (−2.89 mg/dL). Most sodium, chloride, and glucose results fell within TEa limits, whereas potassium exceeded TEa thresholds more frequently. No significant difference in absolute bias was observed between serum- and plasma-based reference measurements.

Discussion and Conclusion: Blood gas results demonstrated generally acceptable agreement with the reference analyzer for sodium and chloride, and although potassium showed minimal statistical bias, its higher rate of TEa exceedance suggests that clinical interpretation should be made with caution. Glucose measurements exhibited a significant constant bias and wide limits of agreement, limiting interchangeability between the two analyzers. Given the retrospective nature and limited sample size, further studies incorporating larger datasets and evaluating additional preanalytical factors are warranted.

CASE REPORT: MANAGEMENT THERAPY OF FIBROSARCOMA VAGINAL MASS IN CRAB-EATING MACAQUES (*Macaca fascicularis*)

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Background

Exotic and wildlife animals medication in clinical settings have limited data as guidelines in managing and diagnosing their diseases. There is an increasing need for documentation of rare disease, including neoplasia cases in exotic pets to help veterinarians working with these species and improve its welfare and care.

Case Presentation

A female, 8 years old crab-eating monkey *(Macaca fascicularis)* presented in our clinic for protruding mass on her anal area. The owner informed me that the condition has been recurring for the past year but usually the protruding mass will recover by itself again. The owner thought it was a hemorrhoid condition. The monkey was very aggressive so it was not possible to do a physical examination without sedation. Sedation was performed using a combination of ketamine-medetomidine (ketamine 5mg/ml [IM], medetomidine 0.5mg/ml [IM]). Upon sedation, we did a physical examination and found that the protruding mass was not coming from anus but from the vulva area. It was thought that a prolapsed uterus occurred and we prepared to do reposition surgery and ovariohysterectomy.

However during the surgery it was discovered that the protruding mass was not uterus and has encapsulated form adhered to the bladder outer wall. The condition of the monkey was slightly jaundiced and we were unable to differentiate the line of the mass and bladder, making it risky to perform mass removal. We performed ovariohysterectomy and mass removal through vagina route. The mass was shaped 5 cm and had multiple mass attached on vagina wall. The sample was sent to the laboratorium for biopsy. Post surgery therapy was done by giving long acting antibiotic injection (Ceftiofur 20mg/kg q5d SC, Excede), pain killer injection (Meloxicam 0.2mg/kg q24h SC, Metacam) and antibiotic ointment.

Biopsy results showed a medium mitotic cell activity of spindle fibroblast cell interlacing bundles in collagen stroma. This result indicated a fibrosarcoma neoplasma condition and was highly possible to recurring again.

Fibrosarcomas in monkeys are malignant tumours that are sometimes linked to viruses like Simian Sarcoma Virus (SSC) and Rous Sarcoma Virus (RSV). It needs to be studied to understand cancer progression. Treatment for fibrosarcoma should be surgical excision with wide margins and followed by chemotherapy. However, due to the site of the fibrosarcoma that is difficult to approach, wide margin excision was not possible.

The animal was discharged after 7 days of inpatient care in our clinic with complete healing of the surgery site. However, within 3 months the monkey came again to the clinic with recurrence of the tumor.

In this case, it was highly suggested to continue treatment with chemotherapy after surgery to prevent recurrence and metastasis of the tumor cell. This procedure was not performed due to unavailability of the medication. Beside chemotherapy, the hygiene of the animal's cage in home needs to be ensured to prevent secondary infection. This patient died 6 months after the first surgery due to secondary infection of tetanus because of repeated wound in her vaginal area and contaminated cage in her house environment.

Successful Management of Severe Immune-Mediated Thrombocytopenia in a Dog with Pyometra

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Background

Pyometra is a potentially life-threatening inflammatory disease in intact female dogs and is typically managed by ovariohysterectomy following medical stabilization. In some cases, the systemic inflammatory response associated with pyometra may contribute to immune dysregulation, leading to complications such as immune-mediated thrombocytopenia and increased surgical risk. This case report describes the successful management of a dog with pyometra complicated by severe thrombocytopenia.

Case Presentation

An 8-year-old intact female Poodle was referred for evaluation of lethargy, anorexia, fever, and hemorrhagic vulvar discharge. Hematologic evaluation demonstrated severe thrombocytopenia(<1K/Ul), leukocytosis, and mild anemia, and the patient fulfilled the criteria for systemic inflammatory response syndrome (SIRS). Abdominal ultrasonography revealed echogenic material within both uterine horns.

Broad-spectrum antimicrobial therapy was initiated, and plasma transfusion was performed to address potential consumptive thrombocytopenia associated with hemorrhagic pyometra. PCR testing was concurrently submitted to exclude infectious causes of thrombocytopenia. After infectious causes were ruled out, immunosuppressive therapy (PDS 1.5mg/kg q12h, MMF 10mg/kg q12h) was initiated for suspected immune-mediated thrombocytopenia. Also, Human intravenous immunoglobulin was administered, while vincristine was withheld because of concerns regarding infection exacerbation related to bone marrow suppression. Romiplostim (7.5ug/kg q 12h) was used to promote platelet production. Following a marked increase in platelet counts to a surgically acceptable level, ovariohysterectomy and planned splenectomy were performed without major hemorrhagic complications. Postoperatively, the patient recovered uneventfully with stable platelet counts and histopathologic examination of the removed spleen revealed a benign lesion

Unique/New Information

In dogs with pyometra, severe immune-mediated thrombocytopenia may preclude immediate ovariohysterectomy because of an unacceptably high risk of hemorrhage. Early diagnosis and treatment of immune-mediated thrombocytopenia prior to definitive surgery allowed platelet recovery and successful surgical management in this case.

Atypical Hepatic Cysts Mimicking Ductal Plate Malformation in a cat with a history of peritonneopericardial diaphragmatic hernia

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Background

Feline polycystic liver disease is characterized by the presence of multiple cysts within the hepatic parenchyma and may arise from embryologic developmental abnormalities. It is frequently observed in association with polycystic kidney disease. This condition is closely related to ductal plate malformation (DPM), an embryologic disorder resulting from dysfunction of the primary cilium. Abnormal primary ciliary function disrupts normal tubulogenesis, ultimately impairing bile duct development.

Case Presentation

An 18-month-old neutered male Norwegian forest cat was referred for evaluation and drainage of hepatic cysts. The patient had previously been diagnosed with a peritoneopericardial diaphragmatic hernia (PPDH) at six months of age during a preoperative assessment for neutering, which was surgically corrected. Upon presentation to our hospital, physical examination revealed persistent tachypnea and abdominal distension. Abdominal radiography revealed a soft tissue opacity measuring approximately 13 cm in diameter. Abdominal ultrasonography identified two anechoic cystic structures in the right hepatic lobe and one in the left lobe, without evidence of biliary ductal dilatation. Abdominal CT confirmed four well-defined, cystic structures originating from the liver. The hepatic cysts were surgically excised.

Histopathologic evaluation of hepatic tissue biopsies demonstrated multifocal portal arteriolar hyperplasia, periportal and perivascular fibrosis. Histological examination of the cystic structures revealed cuboidal epithelial lining and extensive hepatocellular atrophy, raising suspicion for a congenital biliary cyst, possibly ductal plate malformation (DPM). Immunohistochemical staining for smooth muscle actin, factor VIII, and CD31 was positive, supporting a final diagnosis of a primary hepatic vascular malformation. Additionally, genetic testing confirmed a PKD1 gene heterozygous mutation.

Unique/New Information

In feline patients of multiple hepatic cysts, concurrent biliary dilatation and elevated liver enzyme levels are frequently observed, often leading to a poor prognosis. In this case, however, the condition was successfully managed surgically, with no recurrence observed over a three-year follow-up period. Congenital multiple hepatic cysts have been reported in association with other congenital anomalies such as peritoneopericardial diaphragmatic hernia (PPDH) and polycystic kidney disease (PKD), and therefore should be considered in the differential diagnosis.

Temporary Use of Tilapia Fish Skin Grafts for Management of Extensive Burn Injuries in a Dog

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Background: Management of large-surface-area burns in veterinary patients remains a significant clinical challenge due to prolonged healing times, pain control, and high demands for intensive wound care. Tilapia fish skin grafts have emerged as a novel biological dressing with promising regenerative properties in veterinary medicine. This case report describes the application and clinical outcome of temporary tilapia skin grafts in a dog with extensive burn injuries.

Case Presentation: A 3-year-old, intact male, mixed-breed dog was referred for treatment of severe burn injuries following exposure to a large-scale wildfire. Burn injuries involved the head, neck, thoracic and pelvic limbs, and trunk, with an estimated total body surface area (TBSA) involvement of 60%. The depth of injury varied by region, ranging from superficial (first-degree) to full-thickness (third-degree) burns. After a 3-day period of stabilization and comprehensive burn assessment, surgical debridement was performed. Post-debridement wound management included daily hydrotherapy, removal of necrotic tissue, topical therapy, and honey-based moist dressings. Despite intensive care for 1 month, wound healing of the large burn lesions remained delayed, and the patient required prolonged daily dressing time and exhibited persistent pain reactions even under analgesia. Considering the delayed healing associated with the extensive burn area, the severity of the patient's pain, and the prolonged dressing time, the application of second-intention wound healing with tilapia fish skin graft was considered, given its structural properties, collagen richness, antibacterial activity, and overall cost-effectiveness and accessibility. Tilapia fish were procured from an aquaculture facility and underwent a three-day pretreatment process before clinical application in the patient. Tilapia fish skin grafting was applied twice, each application maintained for a duration of one week, after confirming the absence of infection through PCR and culture testing prior to grafting. Post-grafting, wounds were kept consistently moist, and weekly assessments were carried out to monitor wound healing progression, dressing time, and pain levels. Favorable wound healing was achieved following the application of tilapia fish skin grafts. No major complications occurred, except for transient focal areas of suspected hyper-epithelialization. Notably, the grafts contributed to a reduction in dressing time and subjective pain scores, facilitating improved patient comfort and cooperation.

Unique/New Information: In a canine patient with extensive burns, short-term application of tilapia fish skin grafts also effectively promoted wound healing and proved to be a safe, low-cost treatment. Moreover, this approach reduced pain and dressing time, enhancing patient comfort and facilitating clinical management.

Severe Thrombocytopenia, Petechiae, and Mucosal Bleeding Following Sentinel Lymph Node Mapping in an Anesthetized Dog with Low-grade Cutaneous Mast Cell Tumor

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Background: Mast cell tumor (MCT) is the most common cutaneous neoplasm in dogs. Mast cells contain granules rich in histamine, heparin, proteases, and cytokines, and degranulation during manipulation is a well-known surgical risk. Clinical manifestations of mast cell degranulation include swelling of the peritumoral tissue, hypotension, local or systemic hemorrhage, and coagulopathy, but the onset and severity are unpredictable. Sentinel lymph node mapping has become a widely used staging technique of cutaneous MCT; however, published evidence directly linking peritumoral injection during mapping to subsequent mast cell degranulation remains scarce. This case report describes an acute and severe adverse event consistent with degranulation and secondary coagulopathy occurring after the mapping procedure in an asymptomatic anesthetized dog.

Case Presentation: A four-year-old spayed female mixed-breed dog was anesthetized for excision of a reddish, soft-to-firm, movable cutaneous mass on the ventral abdomen. Cytology revealed numerous mast cells scattered individually and with moderate granularity and anisocytosis, supporting a tentative diagnosis of MCT. The dog was otherwise healthy without other medical issues. On the surgery day, the dog was premedicated with intravenous injection of diphenhydramine, dexmedetomidine and ketamine, induced with diazepam and propofol, and then maintained with isoflurane. Before surgery, abdominal ultrasonography and fine-needle aspiration of the spleen and liver were performed, followed by sentinel lymph node mapping through injection of iopamidol into the subcutis one to two cm around the mass. Vital signs monitored during anesthesia were within normal ranges.

Twenty minutes after the mapping procedure, the dog regurgitated dark-brown gastric fluid with blood odor. During cleaning of the oropharynx, erythema and mild bleeding of bilateral tonsils were noted. The skin surrounding the mass became bruised, and multiple ecchymoses and petechiae appeared on the ventral thorax and abdomen. Buccal mucosal bleeding time was markedly prolonged and repeat complete cell count revealed severe thrombocytopenia compared to pre-operative values. Therefore, the surgery was canceled, and medical therapy including diphenhydramine, omeprazole, sucralfate, prednisolone, and tranexamic acid was initiated. After three weeks, platelet count normalized and skin lesions resolved. Surgical excision was later performed without major complication. Histopathology confirmed low grade MCT without lymph node involvement. Mild postoperative anemia occurred without clinical signs of coagulopathy, and the dog recovered uneventfully.

Unique/New information: This case illustrates the unpredictable and potentially severe nature of mast cell degranulation, which may occur independently of tumor grading or clinical presentation preoperatively. Peritumoral injection during sentinel lymph node mapping may precipitate acute degranulation, resulting in severe thrombocytopenia and systemic hemorrhage resembling a paraneoplastic syndrome. Clinicians should remain vigilant for unexpected systemic reactions during staging or diagnostic procedures in dogs with MCT. Awareness of this risk and prompt medical management can prevent progression to life-threatening coagulopathy.

Fatal Systemic Inflammatory Response Syndrome and Multi-Organ Failure Following Rescue Rabacfosadine Therapy in a Dog with B-Cell Lymphoma

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Background Rabacfosadine (Tanovea®) is a novel double prodrug utilized as a rescue agent for canine lymphoma. While it is generally well-tolerated, its safety profile in heavily pre-treated patients remains largely undescribed in clinical literature. This report details a fatal case of suspected rabacfosadine-associated toxicity that manifested as severe systemic inflammatory response syndrome (SIRS) and irreversible multi-organ failure.

Case Presentation An 11-year-old spayed female mixed-breed dog with CHOP-refractory B-cell lymphoma was treated with a THOP protocol to induce partial remission. Subsequently, rabacfosadine was administered at 1 mg/kg IV as a last-line rescue therapy following a 4-week washout period. Adverse events, including dermatopathy and diarrhea, appeared one week after the first dose and worsened significantly following the second dose. On Day 39, despite a marked reduction in tumor size, the patient presented in critical condition with profound hypotension (systolic BP 40 mmHg) and tachycardia, indicating hypovolemic shock. Physical examination revealed generalized dermatopathy characterized by extensive redness, exudate, desquamation, and crusting. Diagnostic evaluation confirmed catastrophic metabolic derangement, including IRIS Grade IV acute kidney injury (creatinine 8.06 mg/dL), severe anemia (Hct 20.8%), and severe pancreatitis (cPL >2000 µg/L). The patient eventually died due to progressive cardiopulmonary collapse despite aggressive resuscitation efforts.

Unique / New Information The clinical course in this patient suggests a "toxicity cascade" where the synergistic effect of extensive cutaneous barrier loss and mucosal compromise precipitated severe fluid loss and hypovolemia. This systemic stress likely imposed an additional ischemic insult that triggered secondary injury to the pancreas and kidneys, leading to SIRS. This case highlights that rabacfosadine can trigger fatal systemic decompensation in debilitated patients. Clinicians should recognize early dermatologic or gastrointestinal signs not merely as localized toxicities but as potential indicators of life-threatening shock. Immediate intervention is necessary to prevent progression to irreversible multi-organ failure.

[Keywords] Canine B-cell lymphoma, Rabacfosadine, Systemic inflammatory response syndrome (SIRS), Multi-organ dysfunction syndrome (MODS), Dermatopathy, Rescue therapy.

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Prolonged Survival in Phenobarbital-Associated Hepatocutaneous Syndrome Managed with Periodic IV Amino Acid Infusions

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Background: Hepatocutaneous syndrome (HCS), also known as Superficial Necrolytic Dermatitis (SND), is a rare and often fatal metabolic skin disorder characterized by the "Red, White, and Blue" histopathological pattern and a "Swiss cheese" liver appearance on ultrasound. Long-term phenobarbital administration is a known risk factor. The underlying pathophysiology involves Aminoaciduric Canine Hypoaminoacidemic Hepatopathy Syndrome (ACHES), causing widespread metabolic perturbations. Historically, the prognosis is poor, with a median survival time (MST) of only 3–6 months. This report describes a case of phenobarbital-associated HCS managed with intravenous amino acid (IV-AA) infusions and dietary modification.

Case Presentation: A client owned 13-year-old intact male Jindo dog, treated with phenobarbital for seizures since 2020, presented with severe hyperkeratosis of the footpads, crusting, and erythema. Diagnosis was confirmed via skin biopsy showing superficial necrolytic dermatitis and abdominal ultrasound revealing a diffuse hypoechoic nodular ("Swiss cheese") liver pattern. Notably, serum liver enzymes and bile acid stimulation test results were within normal limits, initially obscuring the hepatic origin. Treatment consisted of high-protein dietary management and periodic IV-AA and lipid infusions to correct hypoaminoacidemia. The infusion protocol began weekly and was tapered to every 4 weeks. Although oral nutritional supplementation was suboptimal, the patient showed significant improvement in systemic signs. The dog survived over 12 months post-diagnosis, far exceeding historical expectations.

Unique/New Information: This case highlights a critical diagnostic disconnect: normal liver enzymes and bile acids do not rule out HCS/ACHES. Diagnosis must rely on clinical pillars—characteristic skin lesions, ultrasonographic appearance, and histopathology—especially in patients with a history of phenobarbital use. Furthermore, this report suggests that aggressive core therapy with regular IV-AA infusions and a high-protein diet can significantly extend survival (>12 months) and improve quality of life, even when "optimal" multi-modal oral supplementation is not fully sustained. Early presumptive treatment based on imaging and clinical signs is crucial for prognosis.

CASE REPORT: PYOGRANULOMATOUS PNEUMONIA IN A CAT WITH NON-EFFUSIVE FELINE INFECTIOUS PERITONITIS

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Background: Feline infectious peritonitis (FIP) remains one of the most significant and fatal infectious diseases in cats. Clinical manifestations of FIP are highly variable and reflect the distribution of vasculitis and pyogranulomatous lesions. Pleural effusion is the most common respiratory manifestation; however, in rare cases, pulmonary involvement may occur in the form of pyogranulomatous pneumonia, leading to severe dyspnea and a guarded prognosis. This report describes a case of pyogranulomatous pneumonia in a cat with a presumptive diagnosis of non-effusive FIP that responded successfully to antiviral therapy.

Case Presentation: A three-year-old male domestic shorthair cat from a multi-cat household was presented with decreased appetite and reduced activity without respiratory abnormalities. The cat had received up-to-date core vaccinations, routine deworming, and ectoparasite preventive treatments. Physical examination revealed a normal body temperature, normal heart and lung sounds, normal heart and respiratory rates, and no nasal discharge. Three days later, the cat was re-presented with acute onset of dyspnea. Clinical examination revealed tachypnea, mixed-type dyspnea, abnormal respiratory sounds, mild fever, weight loss, and mild dehydration. Emergency management was initiated, including oxygen supplementation via an oxygen cage and fentanyl continuous rate infusion (CRI), prior to diagnostic evaluation. Thoracic Focused Assessment with Sonography for Trauma (TFAST) revealed B-lines, nodular signs, and shred signs throughout the thorax. Thoracic radiographs demonstrated diffuse interstitial pulmonary infiltrates with multiple nodular opacities. No evidence of pleural effusion was detected on either modality. Complete blood count revealed mild leukocytosis with neutrophilia and a decreased reticulocyte count. Serum biochemistry showed hyperglobulinemia with a decreased albumin-to-globulin ratio (A/G ratio = 0.4). Screening tests indicated positive Feline Leukemia Virus (FeLV) antigen, negative Feline Immunodeficiency Virus (FIV) antibodies, negative Toxoplasma gondii IgG and IgM antibodies, and positive Feline Coronavirus (FCoV) antibodies. Based on clinical history, imaging, and laboratory results, the cat was diagnosed with pyogranulomatous pneumonia, with presumptive diagnoses of non-effusive FIP and concurrent FeLV infection. The cat was hospitalized in an oxygen cage and treated with oral GS-441524 at 15 mg/kg once daily (q24h). Dyspnea improved by day 4, and complete resolution of thoracic imaging abnormalities was observed on day 12. Treatment was continued for 12 weeks.

Unique/New Information: There are only a limited number of published reports indicating that pyogranulomatous pneumonia is a rare clinical manifestation of feline infectious peritonitis (FIP), particularly in cases presenting with diffuse pulmonary consolidation and non-effusive disease. When present, this manifestation tends to progress rapidly and severely, leading to acute respiratory compromise. Such an aggressive disease course highlights that early recognition, rapid diagnosis, and prompt initiation of appropriate therapy are critical**,** as clinical deterioration may occur within a short time frame. In this context, timely therapeutic decision-making is essential, as clinicians are effectively racing against disease progression to improve patient outcomes.

COMPLETE RESOLUTION OF A NASOPHARYNGEAL ABSCESS IN A CAT TREATED WITH SYSTEMIC ANTIBIOTICS

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Background: Nasopharyngeal disease can result in upper airway obstruction and severe respiratory distress in cats. Nasopharyngeal neoplasia and polyps are the most common causes of nasopharyngeal disease in cats, whereas nasopharyngeal abscesses are exceedingly rare, with only a single case reported to date.

Case Presentation: A 6-year-old spayed female Persian cat was presented with an 11-day history of hypersalivation, retching, and increased inspiratory effort. The cat had previously been treated with doxycycline and bromhexine, with no clinical improvement observed. Physical examination revealed 8% dehydration, open-mouth respiration, and stertor. Thoracic radiography revealed no remarkable abnormalities. After rehydration and supportive care, computed tomography (CT) was performed to evaluate the upper airway structures. CT revealed a homogeneously isoattenuating soft-tissue mass (2.3 x 1.3 cm) in the posterior nasopharynx. The mass was aspirated and cytological examination revealed numerous neutrophils with intracellular bacteria. A nasopharyngeal abscess was suspected and systemic antibiotics, including amoxicillin-clavulanate and marbofloxacin, were initiated. Three days later, surgical exploration was attempted to remove the mass; however, no grossly visible lesion was identified. Repeat CT confirmed complete resolution of the mass. Eight days after presentation, the cat showed no clinical signs and was successfully discharged. Oral antibiotics were maintained for 21 days and discontinued 22 days after presentation. No recurrence of clinical signs has been reported to date.

Unique/New Information: This case demonstrates that medical management with systemic antibiotics can result in complete resolution of the lesion and clinical improvement in a cat with a nasopharyngeal abscess. Furthermore, our findings provide representative CT images of a nasopharyngeal abscess and suggest that cytology may be helpful in differentiating nasopharyngeal abscesses from nasopharyngeal neoplasia or polyps.

Successful Management of Severe Post-dialysis Polyuria and Sepsis in a Dog with Leptospirosis-Induced Acute Kidney Injury

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Background: Leptospirosis is a common infectious cause of acute kidney injury (AKI) in dogs and may progress to severe azotemia with oliguria requiring renal replacement therapy. Although recovery of renal function can occur, the post-dialysis polyuric phase may be profound and difficult to manage, particularly when accompanied by systemic complications such as pancreatitis and sepsis.

Case Presentation: A 5-year-old neutered male Bichon Frise was presented for acute onset azotemia and decreased urine output. The chief complaint was lethargy and progressive deterioration in renal parameters identified at a referring hospital. The dog had no significant prior medical history. On presentation, severe azotemia, hyperphosphatemia, metabolic derangements, and oliguria unresponsive to diuretic therapy were identified, and leptospirosis was diagnosed based on urine PCR test. Concurrent severe pancreatitis with hemorrhagic diarrhea was observed during hospitalization. Due to refractory oliguria and worsening azotemia, a single session of intermittent hemodialysis was initiated, resulting in rapid improvement of uremic parameters. Following dialysis, a marked polyuric phase developed. While polyuria in canine AKI is typically defined as urine output >2 mL/kg/h, with reported mean urine output of approximately 4.7 mL/kg/h in surviving dogs, urine output in this case increased dramatically, peaking at 36 mL/kg/h. This extreme diuresis required prolonged hospitalization with carefully titrated fluid therapy, electrolyte monitoring, strict in and out recording, daily body weight assessment, and focused cardiac ultrasonography (FoCUS) to guide volume management. During the polyuric phase, a systemic inflammatory response progressed to a state suspected to represent sepsis, potentially secondary to gastrointestinal barrier dysfunction and bacterial translocation associated with severe pancreatitis. Prompt antimicrobial escalation and intensive supportive care led to clinical stabilization. Over time, urine output gradually normalized, renal parameters improved, and gastrointestinal signs resolved, allowing discharge in stable condition.

Unique/New Information: This case highlights successful recovery from severe leptospirosis-induced AKI complicated by refractory oliguria, extreme post-dialysis polyuria (up to 36 mL/kg/h), and sepsis. The use of an integrated fluid management strategy combining strict in and out monitoring, daily body weight, and FoCUS-based intravascular volume assessment was particularly valuable. Because AKI is often accompanied by catabolic weight loss, reliance on body weight alone may underestimate the risk of volume overload during aggressive fluid therapy. FoCUS helped prevent iatrogenic hypervolemia while maintaining euvolemia during profound diuresis. Additionally, this case highlights the need for careful monitoring for potential sepsis or SIRS during AKI recovery, given the possibility of pancreatitis-associated gastrointestinal barrier disruption and bacterial translocation.

Pericardial effusion identified following cardiopulmonary resuscitation in a dog with advanced myxomatous mitral valve disease

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Background: Atrial rupture is a recognized complication of advanced myxomatous mitral valve disease (MMVD) and may result in acute hemopericardium and cardiovascular collapse. Management decisions in post–cardiopulmonary resuscitation (CPR) patients can be challenging, particularly when pericardial effusion is identified and the etiology is uncertain.

Case Summary: An adult dog with known advanced MMVD presented in cardiopulmonary arrest with agonal respirations and absent pulses. Cardiopulmonary resuscitation was initiated and return of spontaneous circulation (ROSC) was achieved. Following stabilization, point-of-care ultrasound (POCUS) was performed. Findings included a large pericardial effusion with hyperechoic fluid consistent with hemorrhage, marked left atrial enlargement, and moderate bilateral B-lines (pulmonary contusion versus pulmonary edema). The findings were most consistent with suspected atrial rupture associated with advanced MMVD. However, the temporal relationship between CPR and development of the effusion could not be definitively determined, and CPR-associated trauma could not be excluded.

Management and Outcome: Pericardiocentesis was not performed due to concern for ongoing hemorrhage, potential intrapericardial clot formation, and risk of recurrent bleeding. Management was guided by the underlying cardiac disease, physiologic stability, and risk–benefit assessment. Supportive care and close monitoring were instituted. The patient maintained hemodynamic stability, survived to hospital discharge, and remained clinically stable at three-month follow-up.

Conclusions: Pericardial effusion identified following CPR may have uncertain etiology. In dogs with advanced MMVD, management decisions should prioritize physiologic status rather than certainty of cause. The presence of suspected intrapericardial clot may contraindicate pericardiocentesis. Conservative management may be appropriate in selected cases.

LOMUSTINE AS CONTINUING SINGLE-AGENT ORAL CHEMOTHERAPEUTIC IN A 7-YEAR OLD INTACT MALE LABRADOR RETRIEVER WITH MULTICENTRIC LYMPHOMA

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Background: Canine multicentric lymphoma is a hematopoietic neoplasm characterized by marked uncontrolled clonal expansion of lymphoid cells. Clinical presenting signs involve generalized lymphadenopathy with or without systemic signs.Paraneoplastic signs and possible metastasis may be concurrently observed. In the Philippines, lomustine and other oral chemotherapeutic drugs are vastly inaccessible, while the Cyclophosphamide-Hydroxydaunorubicin (Doxorubicin)-Oncovin (Vincristine)-Prednisone (CHOP) protocol remains to be the first-line of treatment for malignant lymphoma and other neoplasias. Palliative care or euthanasia are offered as other options often due to the client's limitations.

Case Presentation: A 7-year-old intact male Labrador Retriever was referred to the veterinary hospital to rule out lymphoma. The owner recalled that they had been to other veterinary clinics that only offered euthanasia for their pet. The patient was presented with a low BCS, generalized lymphadenopathy and pyrexia. Physical examination, hematology, serum biochemistry, and fine needle aspiration biopsy (FNAB) of the lymph nodes were performed. Hematology revealed anemia, lymphocytosis, and thrombocytopenia while serum biochemistry revealed low amylase levels. FNAB showed atypical lymphocyte morphology and mitotic figures which are consistent with canine lymphoma. Apart from these, degenerative mitral valve disease and a cardiac mass in the left ventricular free wall were also inferred upon cardiac consultation, deeming the patient unfit to undergo the CHOP chemotherapy protocol. Four initial monthly sessions of vincristine-prednisone chemotherapy were performed, while awaiting the arrival of lomustine, which was given as sole agent starting on the 5th session. The initial vincristine-prednisone chemotherapy protocol was able to initiate partial remission after 4 sessions, while after 2 sessions of monthly lomustine, complete remission was observed. The client reported minimal side effects after oral administration, including fever, and mild gastrointestinal disturbance. This case report highlights the importance of effective integration of detailed patient information and diagnostic procedures to arrive at suitable treatment protocols for the patient. Further research and clinical trials involving the use of lomustine as a single agent, compared to lomustine-prednisolone and the CHOP protocol is recommended.

Unique/New info: Lomustine is deemed expensive and generally with limited availability in pharmacies and even human hospitals in the Philippines, which makes it even more difficult to access for veterinary patients. If further proven to be effective as a single agent chemotherapeutic drug, coupled with better availability locally, then it can be a potential chemotherapeutic option for tumor-bearing veterinary patients.

PENILE FIBROSARCOMA IN A 6-YEAR OLD INTACT MALE MIXED BREED DOG

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Background: Fibrosarcoma (FSA) is a malignant and locally invasive mesenchymal tumor arising from fibroblasts capable of producing a collagen matrix. This tumor originates from connective tissues, and is more commonly observed on the skin and subcutaneous tissues. This is considered to be a rare type of tumor, accounting for less than 5% of all primary canine tumors.

Case Presentation: A 6-year-old, intact, male, mixed-breed dog was brought to the veterinary hospital as a referral case from another veterinary facility for Transmissible Venereal Tumor (TVT) treatment. Upon presentation, the patient was observed to have an erythematous cauliflower-like mass on the tip of the glans penis, and the patient was licking and biting the said lesion. Impression smear (IS) and Fine Needle Aspiration Biopsy (FNAB) were performed, but both did not reveal cells consistent with TVT. Partial or complete penile amputation were the offered surgical options and due to its conservative and non-radical nature, partial penile amputation was chosen by the owner. The excised mass was measured at 1.7 cm x 1.6 cm. Histopathology revealed well-differentiated, moderately pleomorphic, spindle-shaped neoplastic cells arranged in irregular interwoven and herringbone patterns, consistent with a diagnosis of grade 2 penile FSA. No documented cases of canine penile FSA are encountered as common locations are the skin, limbs and oral cavity, making this a novel presentation. The surgical intervention resulted in good post-operative healing with no observed local recurrence, inflammation and discharge after 14 days. In another post-operative follow up performed after 84 days, the owner noted that there was no more licking and biting of the area observed. Conclusion: Histopathologic findings of the excised mass confirmed grade 2 penile FSA documenting its first reported incidence. Conservative surgical excision without adjunct therapy resulted in favorable short-term outcomes with regular monitoring for local recurrence and metastasis.

Unique/New info: Canine penile growths/masses are usually diagnosed as TVT after cytology. Fibrosarcoma itself is a rare type of tumor in canine patients, occurring at less than 5%, and with the tumor occurring at the tip of the glans penis, this case makes it more novel in this regard.

Antimicrobial Resistance Influencing Pre-, peri- and post-operative Management of Urinary Bladder Leiomyoma in a 2-year-old Intact Female Shihtzu

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Background:
Hematuria and dysuria are nonspecific clinical signs that may arise from multiple overlapping urinary tract conditions, making differentiation a step-by-step diagnostic process. This overlap often results in empirical or prolonged antimicrobial use, which, when inappropriately used, may contribute to the development of antimicrobial resistance (AMR). In critical care settings, AMR can significantly complicate diagnostic evaluation as well as pre-, peri-, and post-operative decision-making. Although urinary bladder leiomyoma is an uncommon and typically low-priority differential, it should be considered in cases of persistent or recurrent lower urinary tract signs, particularly when prolonged clinical disease occurs concurrently with suspected or confirmed bacterial cystitis. This case highlights the influence of AMR on clinical decision-making throughout the pre-, peri-, and post-operative phases in a young dog with urinary bladder leiomyoma.

Case Presentation:
A 2-year-old intact female Shih Tzu was presented with persistent hematuria following previous surgical management for urocystolithiasis and at least one month of empirical antimicrobial therapy with one type of antibiotic. Clinicopathologic evaluation revealed hypochromic anemia, leukocytosis with neutrophilia, proteinuria, and marked bacteriuria. Urine culture obtained via cystocentesis identified a coagulase-negative Staphylococcus exhibiting multiple antimicrobial resistance, prompting discontinuation of ineffective antimicrobial therapy during the pre-operative period and limiting medical treatment options.

Diagnostic imaging, which included pneumocystography and ultrasonography, demonstrated focal thickening (5–6 mm) at the urinary bladder neck, raising concern for structural pathology rather than persistent infection alone. Due to continued hematuria, failure of medical management, and the presence of AMR, surgical intervention was recommended. During the pre, and post-operative period, antimicrobial management was tailored, including a trial of adjunctive non-conventional antimicrobial support (herbal therapy) and the selective use of an antimicrobial with known urinary bladder affinity that was not included in the susceptibility testing of the isolated organism. A partial cystectomy involving approximately 20% of the urinary bladder was performed.

Histopathologic examination of the excised tissue confirmed a diagnosis of urinary bladder leiomyoma. Post-operatively, the patient recovered uneventfully, with complete resolution of urinary clinical signs. Follow-up care emphasized wound monitoring, surveillance for secondary infection, and avoidance of unnecessary antimicrobial use.

Unique/New Information:
This case demonstrates how antimicrobial resistance can significantly influence surgical timing, diagnostic prioritization, and antimicrobial decision-making in veterinary critical care. Identification of a multidrug-resistant organism prior to definitive diagnosis highlighted the risks of prolonged

empirical antimicrobial therapy in young patients with atypical urinary disease. Early culture and susceptibility testing, combined with timely surgical intervention, were key in resolving clinical signs while minimizing further antimicrobial exposure. This report reinforces the integration of antimicrobial stewardship principles across all phases of care when managing complex urinary tract cases.

Rapid Diagnostic and Supportive Strategies in Veterinary Emergency and Critical Care: Improving Survival Outcomes in Acute Animal Trauma.

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Abstract

Veterinary emergency and critical care (VECC) has emerged as a vital specialty aimed at reducing mortality and morbidity in animals experiencing life-threatening conditions. Acute trauma, shock, sepsis, and respiratory distress remain among the most common emergencies presented to veterinary hospitals, requiring rapid diagnosis and immediate intervention. This poster highlights the role of timely diagnostic approaches and evidence-based supportive care strategies in improving clinical outcomes in veterinary emergency settings.

The study focuses on the integration of point-of-care diagnostics, including blood gas analysis, lactate measurement, and rapid hematological testing, to facilitate early clinical decision-making. These tools allow clinicians to assess tissue perfusion, oxygenation status, and metabolic disturbances within minutes, enabling prompt stabilization. Additionally, the poster emphasizes the importance of standardized triage protocols, fluid resuscitation strategies, pain management, and oxygen therapy in the initial management of critically ill animals.

Supportive care remains the cornerstone of veterinary critical care, particularly in resource-limited settings. Proper monitoring of vital parameters, early recognition of deterioration, and timely referral to intensive care units significantly enhance survival rates. The poster also discusses the role of laboratory sciences in emergency diagnostics, highlighting how collaboration between clinicians and diagnostic laboratories improves accuracy and speed of treatment.

In conclusion, rapid diagnostics combined with structured supportive care protocols play a crucial role in veterinary emergency and critical care. Strengthening laboratory-based diagnostic capacity and promoting standardized emergency response systems can substantially improve patient outcomes. This poster aims to contribute to ongoing discussions on advancing VECC practices, particularly in developing veterinary healthcare systems.

Keywords

Veterinary emergency; Critical care; Acute trauma; Rapid diagnostics;

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A Sequential Therapeutic Approach to Right-Sided Heart Failure and Pulmonary Hypertension in a dog

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Background : Tricuspid valve dysplasia (TVD) combined with pulmonary hypertension (PH) is rare in small-breed dogs and complicates therapeutic decision-making. While sildenafil is often prioritized for PH, managing the immediate consequences of right-sided heart failure, such as venous congestion and ascites, may require a more tailored, stepwise approach.

Case Presentation : A 10-year-old spayed female Pomeranian presented with a chief complaint of abdominal distension due to transudate ascites and concurrent coagulopathy. The medical history revealed progressive lethargy. Physical examination and echocardiography confirmed severe right atrial and ventricular dilation with abnormal tricuspid valve morphology, consistent with TVD and Group 1 PH. The patient was treated with a protocol focusing on preload reduction, including furosemide, spironolactone, and pimobendan. Sildenafil was intentionally withheld to prioritize the management of venous congestion.

Unique/New Information : This case demonstrates that ascites in concurrent TVD and PH may be primarily attributable to TVD-related venous congestion rather than PH itself. The rapid resolution of effusion and 7-month stability without sildenafil suggest that a stepwise therapeutic trial—prioritizing diuretics for preload reduction over initial pulmonary vasodilation—can be an effective and focused strategy in complex right-sided heart failure cases.

Post-Anesthetic Gastroesophageal Reflux–Associated Esophageal Stricture in a Cat: Diagnosis and Successful Endoscopic Balloon Dilation

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Esophageal stricture is an abnormal narrowing of the esophageal lumen and is most commonly caused by gastroesophageal reflux during general anesthesia, medication-induced injury, foreign bodies, or mass lesions. The incidence of gastroesophageal reflux during anesthesia ranges from 16% to 55% and may precede esophageal stricture formation in up to 65% of cases. This case report describes a post-anesthetic esophageal stricture in a cat and emphasizes the importance of early recognition of anesthesia-associated esophageal injury and its successful management.

A 2-year-old male British Shorthair cat presented to Vetville Animal Hospital Hospital for persistent vomiting. The cat had undergone castration under general anesthesia 21 days prior, and subsequently developed vomiting up to 10 times per day, occurring 1 minute to 4 hours after feeding. On physical examination, the cat's body condition score was 3/9, body temperature 100.8°F, conscious, 5% dehydration, pink mucous membrane, and normal heart and lung sounds. Cervical esophageal palpation was nonpainful, and a cough reflex could not be elicited. Oral examination revealed no masses or ulceration. Laboratory testing showed mild leukocytosis with hepatic, renal, and pancreatic enzyme parameters within reference ranges. Plain thoracic and abdominal radiographs revealed no abnormalities. A contrast study using liquid iohexol did not identify megaesophagus, esophageal stricture, or gastrointestinal obstruction. Due to persistent clinical suspicion, the cat was referred to the Small Animal Teaching Hospital, Faculty of Veterinary Science, Chulalongkorn University, for contrast fluoroscopy, which demonstrated obstruction of positive contrast at the distal cervical esophagus, confirming an esophageal stricture. Treatment consisted of a total of four esophagoscope balloon dilation sessions performed at two-week intervals. Transendoscopic triamcinolone injection was administered during the third procedure. No vomiting was observed following any of the procedures. Medical management consisted of an acid suppressant, a prokinetic, and an antiemetic drug. During the treatment period, the cat was maintained on semisolid food. After the final dilation procedure, the cat showed complete resolution of dysphagia and vomiting, was able to eat normal dry food, and remained clinically normal without recurrence during a 3-month follow-up.

This case report describes a rare occurrence of post-anesthetic esophageal stricture in Thailand. Gastroesophageal reflux during anesthesia may result from anesthetic-induced reductions in lower esophageal sphincter tone, leading to injury of the esophageal muscularis layer. Subsequent fibroblastic proliferation and tissue contraction can progress to esophageal stricture formation. Contrast fluoroscopy and upper endoscopy are considered the diagnostic gold standards. Balloon dilation combined with intralesional corticosteroid therapy represents an effective treatment strategy. This case highlights the importance of recognizing and diagnosing esophageal strictures in feline patients.

TEMPORARY CRICOTHYROTOMY FOR DIFFICULT EXTUBATION AFTER VENTRAL SLOT SURGERY IN A DOG WITH SUBCLINICAL TRACHEAL COLLAPSE

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Background: Ventral slot surgery is a common procedure for cervical intervertebral disc decompression, with respiratory distress being a rare but potentially life-threatening complication. This report describes a dog without overt clinical signs of tracheal collapse that developed acute postoperative respiratory compromise manifested as difficult extubation following ventral slot surgery, requiring emergent airway intervention.

Case Presentation: A 10-year-old, intact male, overweight Yorkshire Terrier was presented for evaluation of progressive non-ambulatory tetraparesis and was diagnosed with C3–C4 intervertebral disc extrusion. Comorbidities included myxomatous mitral valve disease and radiographic evidence of subclinical tracheal collapse, with only occasional exercise-induced coughing reported. Pre-anesthetic respiratory examination revealed no clinically apparent airway abnormalities. Intraoperatively, mild hypercapnia was noted. Following extubation, the patient immediately developed respiratory compromise characterized by dyspnea and hypoxemia. Initial supportive management involving optimized analgesia and sedation, corticosteroids, and oxygen therapy failed to improve respiratory status, resulting in repeated re-intubation. Due to persistent airway compromise, laryngoscopy and tracheoscopy were conducted to evaluate airway structure and function, revealing grade IV dynamic collapse at multiple regions of the cervical trachea. To bypass the obstructed airway, a temporary cricothyrotomy tube was placed. Despite 12 hours of supportive care, increased respiratory effort and persistent stridor recurred after attempted tube removal. Definitive airway surgical correction was achieved through placement of an extraluminal tracheal stent. After surgery, the patient was successfully extubated with improved respiratory function, supplemental oxygen was discontinued within 24 hours, and the patient was discharged three days later.

Unique/New Information: This case demonstrates that subclinical tracheal collapse may manifest as severe perioperative airway compromise following cranial ventral slot surgery. Unexpected difficult extubation should prompt early consideration of endoscopic airway evaluation to facilitate accurate diagnosis and avoid repeated intubation attempts. This case supports consideration of temporary cricothyrotomy as an effective temporary airway management technique for postoperative cervical tracheal collapse, allowing relief of airway obstruction while avoiding more invasive procedures such as tracheotomy.

Upper Airway Obstruction and Delayed Tracheal Injury in a Dog with Inhalation Injury

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Background

In human medicine, inhalation injury generally refers to respiratory tract damage caused by inhalation of smoke, heated gases or steam, and toxic combustion products. In humans, upper airway obstruction due to laryngeal edema may occur early after injury, whereas delayed airway obstruction at the tracheal and bronchial levels, associated with mucosal necrosis, sloughing, and pseudomembrane or airway cast formation, may also develop over time. Although similar pathophysiological mechanisms may be considered in dogs, reports of inhalation injury in dogs remain limited. While sporadic canine cases describing respiratory compromise or hypoxemia following smoke inhalation have been reported, detailed clinical descriptions of delayed airway obstruction involving both laryngeal pathology and suspected tracheal mucosal injury remain scarce. Here, we report a dog with inhalation injury that developed delayed airway obstruction and was successfully managed with temporary tracheostomy, providing insight into the pathophysiology of inhalation injury in dogs.

Case Presentation

A 1-year-8-month-old spayed female mixed-breed dog weighing 2.9 kg, with no notable past medical history, was presented on day 1 after injury with dorsal burn wounds caused by kerosene ignition, facial edema, and coughing. At presentation, intermittent coughing was observed without marked abnormalities in respiratory pattern, and thoracic radiographs were unremarkable. Blood examination revealed marked elevations in muscle leakage enzymes and C-reactive protein, as well as metabolic acidosis. During hospitalization, inspiratory effort gradually worsened, and on day 4, the dog developed respiratory distress associated with upper airway obstruction, prompting emergency endotracheal intubation.

Computed tomography performed immediately after intubation revealed extensive non-contrast-enhancing, low-attenuation material along the left side of the tracheal lumen extending from the thoracic inlet to the tracheal bifurcation, as well as circumferential low-attenuation areas with poor contrast enhancement in the tracheal wall caudal to the larynx. In contrast, laryngoscopic examination demonstrated laryngeal edema and degeneration of the arytenoid cartilages, resulting in laryngeal dysfunction, which was considered the primary cause of respiratory distress. Therefore, a temporary tracheostomy was performed to secure the airway.Postoperatively, arterial blood gas analysis confirmed adequate ventilation and oxygenation, and the dog was recovered from general anesthesia. Persistent coughing was observed after recovery, and within approximately one hour, necrotic tracheal mucosal fragments were expelled through the tracheostomy site. Following this event, the respiratory pattern improved markedly. The respiratory condition remained stable thereafter, and the tracheostomy tube was removed on day 16 after confirmation of resolution of upper airway obstruction. No further respiratory deterioration was observed until discharge on day 93.

Unique / New Information

This case highlights that, in dogs with inhalation injury, acute upper airway obstruction due to laryngeal edema and delayed tracheal mucosal injury can coexist and contribute to airway compromise within the same clinical course. While delayed tracheal complications following smoke

inhalation have been reported in dogs, the present case provides a detailed clinical description of how laryngeal dysfunction and suspected delayed tracheal mucosal injury may sequentially and independently influence respiratory status.In this case, airway compromise was initially dominated by laryngeal pathology, prompting temporary tracheostomy for airway protection. Subsequently, imaging findings and clinical progression suggested the involvement of delayed tracheal mucosal injury, which became clinically apparent after airway stabilization. Accidental expulsion of necrotic tracheal mucosal fragments through the tracheostomy site resulted in clinical improvement; however, this should be regarded as an incidental finding rather than a therapeutic effect.In human medicine, therapeutic bronchoscopy is considered the standard approach for managing delayed airway obstruction following inhalation injury. The present case underscores the importance of recognizing that, in dogs with suspected severe inhalation injury, airway pathology may evolve over time and involve multiple anatomical levels. Accordingly, in addition to addressing laryngeal dysfunction, clinicians should consider the potential contribution of tracheal and bronchial lesions and evaluate the indication for bronchoscopic assessment and intervention when appropriate. This report contributes to a more nuanced understanding of the temporal and anatomical complexity of inhalation injury in dogs.

A Case of Secondary Hydrocephalus and Cerebellar Herniation Caused by a Cerebellar Abscess in a Cat

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Background: Infectious intracranial diseases in cats represent neurological emergencies. Acute and severe increases in intracranial pressure or involvement of the brainstem are generally associated with a poor prognosis.

Case Description

A 5-year-old neutered male mixed-breed cat with indoor–outdoor access presented with acute, progressive neurological deterioration. The cat had a two-month history of recurrent ulceration and purulent discharge from a bite wound on the right cheek. Three days prior to admission, acute ataxia and right-sided weakness developed, progressing to bilateral forelimb paresis the following day. On presentation, the cat was non-ambulatory. Neurological examination revealed depressed mental status, lateral recumbency, upper motor neuron tetraparesis, vertical nystagmus, hypersensitivity, and no cranial nerve deficits.

Screening tests suggested an infectious intracranial disease, and magnetic resonance imaging (MRI) was performed. MRI revealed a well-demarcated ring-enhancing mass lesion with internal low signal intensity (11 × 7.5 × 8.8 mm) in the right cerebellar hemisphere, consistent with a cerebellar abscess. Diffuse cerebral sulcal effacement, dilation of the rostral ventricular system, narrowing of the caudal ventricular system at the level of the mesencephalic aqueduct, and cerebrospinal fluid flow obstruction were observed. Caudal cerebellar herniation through the foramen magnum with compression of the caudal medulla was also identified. Blood culture yielded anaerobic bacteria.

Based on these findings, a diagnosis of cerebellar abscess secondary to bite wound infection, complicated by severe cerebral edema, secondary hydrocephalus, and cerebellar herniation, was made. Intensive medical management was initiated on day 1 and included mechanical ventilation, antimicrobial therapy, corticosteroids, and osmotic diuretics. Follow-up MRI on day 3 demonstrated reduction of the abscess and improvement of cerebral edema, although findings suggestive of caudal medullary injury secondary to herniation persisted.

Due to concerns regarding prolonged mechanical ventilation and neurological sequelae, a nasogastric tube and tracheostomy were placed on day 6. After gradual ventilator weaning, successful extubation was achieved on day 21. Although tetraparesis and lateral recumbency persisted, the cat's general condition and appetite were stable, and it was discharged on day 30. By day 58, the cat regained postural control, was able to crawl forward, and demonstrated avoidance responses to noxious stimuli. Persistent urinary dysfunction remained as a chronic sequela.

Discussion and Conclusion:

This case demonstrates that favorable outcomes may be achieved in cats with cerebellar abscesses complicated by severe secondary hydrocephalus, cerebellar herniation, and brainstem dysfunction through aggressive medical management combined with mechanical ventilation. Mechanical ventilation may contribute to stabilization by reducing ICP fluctuations, optimizing oxygenation, and enabling close monitoring of respiratory and hemodynamic status. Tracheostomy may facilitate successful weaning from prolonged mechanical ventilation; however, its indications and associated risks in feline patients warrant further investigation.

INTRODUCTION AND CHALLENGES OF HOME-BASED THORACOCENTESIS AS A COMPONENT OF EMERGENCY VETERINARY CARE: A CASE OF MALIGNANT PLEURAL EFFUSION IN A CAT REQUIRING PHYSICAL RESTRAINT BY AN ELDERLY OWNER DUE TO TEMPERAMENT

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Background: Requests for veterinary home-based palliative care have increased, particularly among elderly caregivers who face medical, psychological, or practical difficulties with regular clinic visits. In routine practice, invasive procedures such as thoracocentesis are performed in hospital settings, where staffing and equipment allow safer restraint and monitoring. When animals experience marked stress in clinical environments, or when caregivers are unable to pursue standard care, veterinarians are often required to make complex decisions that extend beyond conventional clinical protocols. In the present case, an elderly individual living alone requested home-based care because of previous bereavement-related distress and the cat's severe intolerance of clinic visits. Although restraint by trained staff is normally required, owner-assisted restraint was permitted after careful discussion of potential risks and limitations.

Case Presentation: A 7-year-old spayed female rescue cat, positive for feline immunodeficiency virus (FIV), was examined for progressive dyspnea associated with pleural effusion caused by metastatic mammary adenocarcinoma. After discussion of procedural risks, minimal sedation was selected to reduce stress and improve safety. Thoracocentesis was performed at home with supplemental oxygen and owner assisted restraint, resulting in temporary improvement of respiratory signs. Over the following weeks, the cat's respiratory condition fluctuated, while the caregiver's own physical condition gradually worsened, reducing her ability to participate safely in care. During a later episode of marked respiratory distress, immediate hospital transfer was not feasible. In this situation, the veterinarian proceeded with urgent intervention from an animal welfare perspective and accepted full clinical responsibility for the procedure.

Unique/New Information: For some terminally ill animals, clinic-based treatment may not represent a realistic or humane option. In such situations, thoracocentesis performed at home may provide meaningful symptomatic relief when carefully planned and limited in scope. At present, however, veterinary home care lacks the structured support systems commonly available in human home healthcare, and clinical decisions are frequently left to individual practitioners. When animals tolerate handling only by their owners, caregivers inevitably become part of the care process. At the same time, the physical condition of the caregiver may limit what can be safely performed in an emergency. Strong emotional commitment may delay recognition of declining health, and continued treatment focused solely on the animal can gradually become difficult to sustain. These cases suggest the need for earlier discussion of contingency plans and clearer assessment of caregiver capacity, particularly when elderly individuals living alone are supporting animals with advanced disease. Closer coordination between veterinary services and human healthcare or social support systems may be necessary to protect the well-being of both patients and their caregivers.

Ante-mortem diagnosis of myocardial infarction in a dog with a hypercoagulable state using two-dimensional speckle-tracking echocardiography

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Background: Myocardial infarction (MI) is a life-threatening emergency in humans; however, it is rarely diagnosed antemortem in dogs. In veterinary practice, the definitive diagnosis of MI is particularly challenging because confirmatory coronary imaging is highly invasive, requires general anesthesia, and is often infeasible in hemodynamically unstable patients. Consequently, a rapid, cage-side, non-invasive diagnostic approach is needed in veterinary ECC settings. Two-dimensional speckle-tracking echocardiography (2D-STE) enables the quantitative assessment of myocardial deformation and has demonstrated a high diagnostic performance for MI in human medicine.

Case presentation: A 6-year-old spayed female Boston Terrier was referred for refractory diarrhea and poor general condition, with a two-year history of chronic enteropathy treated with prednisolone and cyclosporine. At the initial presentation, the dog was diagnosed with lymphangiectasia and sepsis secondary to uterine stump pyometra. Laboratory testing revealed leukocytosis, elevated C-reactive protein, hypoalbuminemia, hyperbilirubinemia, and a hypercoagulable state close to disseminated intravascular coagulation. Surgical resection of the uterine stump was performed on day 10 to control the sepsis. On day 16, the patient acutely deteriorated with respiratory distress and a non-ambulatory status. Abdominal ultrasonography revealed a thrombus at the aortic trifurcation. Echocardiography revealed spontaneous echo contrast in the left ventricular cavity, regional wall thinning, and pericardial effusion. 2D-STE demonstrated a markedly reduced global longitudinal strain with regional abnormalities localized to the vascular territory of the left paraconal interventricular branch of the left coronary artery, thus leading to a diagnosis of MI secondary to thromboembolism. Coronary computed tomography angiography performed after hemodynamic stabilization confirmed the complete occlusion of the corresponding coronary artery.

Unique/New Information: This report describes the first ante-mortem diagnosis of canine MI secondary to systemic thromboembolism using 2D-STE in an ECC setting. This case has highlighted that MI may accompany aortic thromboembolism in hypercoagulable dogs, and that cage-side 2D-STE may serve as a practical, noninvasive screening tool when invasive imaging is not immediately available.

A Dog with a Systemic Capillary Leak Syndrome–Like Condition Following Vaccination

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Background: Systemic capillary leak syndrome (SCLS) is a rare disease characterized by a triad of hypotension, hypoalbuminemia, and hemoconcentration. Although its pathogenesis remains incompletely understood, SCLS is thought to result from increased vascular permeability caused by endothelial dysfunction, leading to extravasation of plasma and plasma proteins into the interstitial space. In recent years, SCLS has been increasingly reported as a delayed and severe adverse reaction following COVID-19 vaccination in human medicine. In contrast, no cases of SCLS as a delayed severe adverse reaction to vaccination have been reported in veterinary medicine to date. We report a canine case that developed a clinical presentation resembling SCLS following vaccination.

Case Presentation: An 18-month-old intact male Jack Russell Terrier with no notable medical history presented with acute lethargy and respiratory distress two days after multivalent vaccination. Blood pressure could not be measured at presentation. Hematologic analysis revealed a hematocrit (Hct) of 54.4% and hypoalbuminemia (albumin 1.8 g/dL). Diagnostic imaging identified pleural effusion. Pleural fluid analysis showed a specific gravity of 1.024, a total protein concentration of 2.5 g/dL, a nucleated cell count of 100/μL, and a pale yellow appearance, consistent with a low-cellularity, protein-containing effusion. The dog was hospitalized and treated with intravenous fluid therapy and corticosteroids. On the second day, despite maintenance fluid therapy, the hematocrit increased to 57.9% while the albumin level decreased further to 1.3 g/dL. The dog developed worsening respiratory distress, severe nausea, and died later that day.

Unique/New Information: In this case, circulatory failure with hypotension, hypoalbuminemia, and hemoconcentration was observed after vaccination. These findings are consistent with the triad reported in human SCLS. In addition, the presence of a protein-containing, low-cellularity pleural effusion supports increased vascular permeability as an underlying pathophysiological mechanism. Taken together, these findings indicate that this dog exhibited a clinical presentation resembling SCLS and suggest that similar pathophysiological responses may occur in canine patients after vaccination.

EMERGENCY HEMORRHAGE CONTROL USING THE PRINGLE MANEUVER AND AUTOLOGOUS BLOOD TRANSFUSION IN A DOG WITH A RUPTURED LARGE HEPATIC MASS

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Background: Hemoperitoneum caused by rupture of hepatic tumors is relatively common in veterinary emergency medicine. However, depending on the volume and rate of hemorrhage, some patients present without obvious signs of shock and remain in a compensated circulatory state, making assessment of severity and timing of intervention challenging. In cases involving large hepatic masses, strategic hemorrhage control during surgery is particularly important.

Case presentation: A 13 year 7 month old castrated male mixed-breed dog weighing 12.75 kg was referred to our hospital after presenting to the primary care clinic with acute lethargy, where a hepatic mass and abdominal effusion were identified. On physical examination at presentation, the heart rate was 174 beats/min, the visible mucous membranes were pale, and blood pressure was 168/108 mmHg (mean arterial pressure 120 mmHg). The dog was mentally alert and able to stand unassisted. Hematologic examination revealed anemia with a hematocrit of 22.5%. Abdominal ultrasonography identified a large hepatic mass, and hemoperitoneum was diagnosed. Computed tomography further confirmed that the mass originated from the left hepatic division. Based on these findings, an emergency exploratory laparotomy was performed. During surgery, persistent hemorrhage from the mass obscured visualization of the hepatic hilum, and manual hemostasis was ineffective. Therefore, the Pringle maneuver was applied to temporarily occlude hepatic inflow from the hepatic artery and portal vein. This rapidly reduced hemorrhage and improved surgical visualization, allowing left lateral hepatic lobectomy to be performed. The total duration of hepatic inflow occlusion using the Pringle maneuver was 2 min 53 s. Concurrently, 420 mL of salvaged autologous blood was transfused to stabilize hemodynamics. Histopathological examination of the resected hepatic mass revealed a diagnosis of undifferentiated sarcoma and hepatocellular adenoma. Postoperative recovery was uneventful, and the short-term outcome was favorable. At the final follow-up,14 days postoperatively, the patient was in good general condition.

Unique/New Information: In this case, application of the Pringle maneuver enabled effective hemorrhage control and facilitated intraoperative surgical decision-making. In dogs with ruptured large hepatic masses, continuous bleeding may limit surgical manipulation and compromise treatment decisions. The combination of the Pringle maneuver and autologous blood transfusion represents a practical and effective emergency hemorrhage control strategy in veterinary emergency and critical care practice.

PHENYLEPHRINE-INDUCED VASOSPASTIC ANGINA IN AN ANESTHETIZED DOG

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Background: Hypotension is a common complication of anesthesia in small animals. This report describes a canine case in which phenylephrine administered to treat general anesthesia-induced hypotension was associated with ST-segment elevation, followed by ventricular fibrillation. The dog was diagnosed with vasospastic angina.

Case Presentation: A 10-year-old male Yorkshire Terrier was brought for follow-up evaluation after completing six sessions of radiation therapy for aortic body tumor. Pre-anesthetic vital signs were as follows: heart rate: 160 bpm, respiratory rate: 36 breaths/min, body temperature: 38.4°C, and blood pressure: 175/120 mmHg. Preanesthetic echocardiography revealed no abnormalities. Given these clinical findings, the anesthetic risk in this case was low. Atropine and butorphanol were administered, followed by propofol for induction and isoflurane for maintenance. Immediately after induction, the dog presented with hypotension (mean blood pressure: 60 mmHg). Phenylephrine (2.5 μg/kg IV) was administered, but the femoral pulse and blood pressure remained undetectable. Because ECG showed ST-segment elevation, isoflurane was discontinued. Thereafter, frequent premature ventricular contractions occurred, which progressed to ventricular fibrillation. Cardiopulmonary resuscitation was initiated immediately, and spontaneous circulation was achieved within 10 s. ST-segment elevation was resolved after circulation restoration, which was followed by transient ST-segment depression. The dog recovered from the anesthesia without other complications. After recovery from anesthesia, mild increase in serum cardiac troponin I levels and an absence of regional wall motion abnormalities on speckle-tracking echocardiography indicated vasospastic angina rather than infarction.

Unique/New Information: To the best of our knowledge, this is the first report describing phenylephrine-associated vasospastic angina during anesthesia in a dog. This case demonstrates that in a patient deemed to be at low anesthetic risk, the administration of phenylephrine for intraoperative hypotension might induce transient myocardial ischemia (initiated from vasospastic angina). Vasospastic angina resembles acute ST-segment elevation myocardial infarction, characterized by localized ST-segment elevation and reciprocal ST-segment depression. However, unlike acute ST-elevation myocardial infarction, these ECG changes are transient and reversible and not usually associated with myocardial necrosis. In this case, the diagnosis was corroborated by mild elevation of serum cardiac troponin I and absence of significant changes on pre- and post-anesthetic speckle-tracking echocardiography, distinguishing it from acute ST-segment elevation myocardial infarction. This case highlights that to prevent drug-induced myocardial ischemic events, cautious vasopressor selection and vigilant cardiovascular monitoring is required even in patients with low-risk anesthesia.

Case Report: Maintenance Hemodialysis Using Infusion Pumps for Canine Renal Impairment

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Background
In veterinary medicine, long-term maintenance hemodialysis is rarely performed. In this case, two-months of maintenance hemodialysis using commercially available infusion pumps was attempted to treat renal impairment and refractory hyperkalemia. We report the complications that arose and the countermeasures taken during two months of maintenance dialysis in a dog.

Case Presentation
The patient was a neutered male Miniature Schnauzer, 12 years and 9 months old at the time of the initial examination, weighing 7.26 kg. His medical history included chronic kidney disease (IRIS stage 2), mitral valve insufficiency (ACVIM stage B1), bladder stones, and immature cataracts.
The patient was referred to our hospital for frequent vomiting and hyperkalemia as the main complaints (Day 1). In the blood tests conducted at our hospital, BUN was 115.8 mg/dl, Cre was 6.36 mg/dl, and K was 7.6 mEq/L. Ultrasound examination revealed dilation of the left and right renal veins and mild accumulation of pleural and abdominal fluid. Based on various findings, renal AKI (IRIS AKI classification: grade 4) was diagnosed.
Intravenous infusion therapy was initiated on the first day of admission. However, as bradyarrhythmia due to hyperkalemia persisted, hemodialysis was initiated on the second day of admission. On the fourth day, fludrocortisone was administered to treat hyperkalemia. By the seventh day, hemodialysis had been performed four times. As serum creatinine and urea levels decreased and potassium levels were maintained at 6.0 mEq/L or lower, an attempt was made to discontinue hemodialysis.
On the 33rd day, worsening azotemia and hyperkalemia, decreased appetite, vomiting, and necrosis at the tip of the tongue were observed, and hemodialysis was resumed. Whole blood transfusions were administered on the 37th and 50th day. Hemodialysis was performed 14 times between the 33rd and 69th days. On the 65th and 69th days, dialysis was discontinued because bradycardia progressed to cardiac arrest, and cardiopulmonary resuscitation was performed. On the 69th day, the patient died at home.

Unique/New Information
A total of 18 hemodialysis sessions were performed by the 69th day. During the treatment period, the dialysis catheter was replaced six times due to poor blood drainage.
Excluding the two sessions in which dialysis was interrupted midway, the average URR for the 16 maintenance dialysis sessions was 41.8%, and the average dialysis time was 6.9 h. Before starting dialysis, the average BUN was 119.5 mg/dL, and the average potassium concentration was 7.1 mEq/L. After dialysis, the average BUN was 69.0 mg/dL and the average potassium concentration was 5.5 mEq/L.
The dialysis system used in this case employed commercially available infusion pumps, which is considered to have enabled cost reduction compared with a CRRT device. Furthermore, because this system does not require water supply and drainage equipment necessary for IRRT devices, there are no restrictions on the treatment location, making long-term dialysis easier to perform.

CLINICAL IMPACT AND ADVERSE EVENTS OF SILDENAFIL IN DOGS WITH PULMONARY HYPERTENSION SECONDARY TO LEFT HEART DISEASE

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Introduction and Objectives: Canine pulmonary hypertension (PH) is an emergent condition characterized by dyspnea, syncope, and collapse. In Group 2 PH secondary to left heart disease (LHD), pulmonary vasodilators are generally not recommended because of the risk of pulmonary edema. However, refractory clinical signs may persist in some dogs with severe PH despite appropriate management of the underlying LHD. This study aimed to evaluate the therapeutic effects and adverse events of sildenafil in dogs with Group 2 PH.

Methods and Materials: A retrospective multicenter study was conducted between September 1, 2020, and September 30, 2024. Dogs diagnosed with Group 2 PH with a high probability of PH based on the American College of Veterinary Internal Medicine consensus statement were included. Data collected included sildenafil administration status and clinical reasons for use, underlying causes of PH, changes in clinical signs and echocardiographic variables before and after treatment, and adverse events (mild: requiring escalation of left-heart treatment at follow-up reassessment without clinical deterioration; severe: marked clinical deterioration).

Results: Among 262 dogs diagnosed with Group 2 PH, 17 received sildenafil therapy. The underlying disease in all cases was myxomatous mitral valve disease (stage B2: 5, C: 8, D: 4). Reasons for sildenafil administration included syncope (14 dogs), right-sided heart failure (3 dogs), and dyspnea (3 dogs). Clinical improvement was observed in 16 dogs (94.1%). Echocardiographic pulmonary vascular resistance significantly decreased after treatment (pre: 3.10; post: 1.90; $p = 0.01$). Left-sided indices showed worsening trends but did not reach statistical significance. Adverse events occurred in 7 dogs (41.2%), including mild events in 5 dogs (29.4%) and severe events in 2 dogs (11.8%). Pulmonary edema developed on the day following sildenafil initiation in 1 dog and 10 days after initiation in 1 dog. Escalation of LHD therapy around the time of sildenafil initiation was more frequent in dogs with adverse events than in those without (6/7 vs. 2/10 dogs, $p = 0.02$). A main pulmonary artery–to–aorta ratio cutoff value of 1.12 was identified for adverse event detection. At study end, 14 dogs had died, with a median survival time of 134 days (range: 7–640 days). Causes of death included respiratory failure (10 dogs), right-sided heart failure (2 dogs), and sudden death (2 dogs).

Discussion and Conclusion: In this study, a reduction in echocardiographic pulmonary vascular resistance was accompanied by clinical improvement, indicating that sildenafil may reduce right ventricular afterload and improve clinical signs in dogs with Group 2 PH. However, adverse events, including pulmonary edema, tended to occur in dogs with more severe pulmonary hypertension and greater left-sided cardiac loading, highlighting the potential risks associated with its use.

Furthermore, the association between main pulmonary artery–to–aorta ratio and adverse events suggests that this parameter may help identify dogs at higher risk of adverse events, regardless of treatment for LHD. Therefore, sildenafil should be used cautiously in dogs with symptomatic PH secondary to LHD, with close monitoring for left-heart decompensation.

Initial Management of Acute Canine Glaucoma and the Involvement of Veterinary Nurses at a Specialized Ophthalmology Facility

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Background

Acute glaucoma in dogs is the one of ophthalmic emergencies that can rapidly compromise visual prognosis due to a sudden increase in intraocular pressure, resulting in retinal ganglion cells damage. Although urgent intervention is required even at specialized ophthalmology facilities, reports examining the role of veterinary nurses in such emergency cases are limited. This report evaluated the importance of the involvement of a veterinary nurse in the initial management of a dog with acute glaucoma.

Case Description

A 13-year-old spayed female Toy Poodle presented at Veterinary Ophthalmology Specialized Clinic with a chief complaint of elevated intraocular pressure in the left eye, which had been identified by rDVM the previous day. The patient's ophthalmic history included chronic glaucoma in the right eye, for which an intrascleral silicone ball implant had previously been placed.

At presentation, no obvious pain-related behavior was observed; however, mild photophobia and conjunctival hyperemia were noted. A veterinary nurse performed an initial interview and measured intraocular pressure, which was found to be elevated, and promptly reported the findings to the veterinarian. Under veterinary instruction, prostaglandin eye drops were administered while awaiting examination. In anticipation of surgical intervention planned for the following day, the veterinary nurse also collected information regarding the patient's systemic condition and confirmed examination results from the primary care veterinarian, facilitating a smooth clinical workflow prior to examination.

Unique/New Information

Acute glaucoma requires rapid intervention, and early intraocular pressure measurement and initiation of pressure-lowering treatment after presentation are essential. Medical assistant work such as IOP evaluation, ocular ultrasound and other ophthalmic diagnostic tests by veterinary nurses could be also utilized in the hospital management of animals that have undergone intraocular surgery.

In specialized ophthalmology facilities, emergency care must often be provided with limited staffing. However, by providing appropriate training in ophthalmic examinations to nationally certified veterinary nurses, they can proactively participate in the initial response and work in cooperation with veterinarians, which is thought to be an important factor in protecting the vision of dogs with acute ocular disorders.

In this case of acute canine glaucoma as we reported, the veterinary nurse contributed to initial management by performing immediate history-taking, intraocular pressure measurement, and assessment of clinical signs of ocular diseases, and by promptly sharing findings with the veterinarian. This collaboration was considered to facilitate rapid therapeutic intervention. Establishing an emergency initial response system that utilizes the expertise of veterinary nurses was

considered to contribute to improved quality of veterinary care not only in ophthalmology specialty facilities but also in general veterinary practices.