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PROCEEDINGSBOOK

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VECCUS
PRE CONGRESS DAY
POINT OF CARE ULTRASOUND

EVECC
EUROPEAN VETERINARY EMERGENCY AND CRITICAL CARE CONGRESS

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VECCUS Symposium, Wednesday 4 June 2025

POINT-OF-CARE ULTRASOUND (POCUS) FOR VASCULAR ACCESS AND PHLEBITIS ASSESSMENT IN SMALL ANIMAL EMERGENCY AND CRITICAL CARE

Kris Gommeren [1]

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Learning objectives:

- Recognize signs of phlebitis

- Know the different techniques described to obtain vascular access

- Know the key facts in human and veterinary medicine regarding POCUS vessel assessment

- Know the key facts in human and veterinary medicine regarding POCUS guided vascular access

Proceeding:

Introduction to POCUS in Veterinary Medicine

Definition and applications of point-of-care ultrasound in small animal practice. Benefits: rapid, non-invasive, and real-time imaging, particularly valuable in emergency and critical care settings.

Vascular Access Using POCUS

Challenges of Traditional Vascular Access: Difficulty in accessing veins in dehydrated, hypovolemic, or critically ill patients. Limitations of blind techniques, particularly in small or collapsed vessels. Advantages of Ultrasound-Guided Vascular Access: Enhanced visualization of vessels. Improved success rates, especially in difficult cases. Reduction in complications such as accidental arterial puncture or hematoma formation. Techniques for Ultrasound-Guided Vascular Access: Equipment overview: high-frequency linear probe for small animals. Preparation: patient positioning, probe orientation, and aseptic technique. Short-axis (out-of-plane) vs. long-axis (in-plane) approaches. Tips for optimizing visualization: applying appropriate pressure and using ultrasound gel. Practical Applications: Placement of peripheral and central venous catheters. Real-time guidance for challenging cases (e.g., small patients, shock, obesity).

Assessment for Phlebitis Using POCUS

Understanding Phlebitis: Definition: inflammation of a vein often due to mechanical or infectious causes, such as prolonged catheter placement or thrombophlebitis. Clinical significance in small animals: pain, swelling, systemic complications. POCUS Evaluation of Phlebitis: Identifying sonographic signs: Vein wall thickening or irregularity. Perivenous hypoechoic or anechoic areas indicating edema or fluid. Echogenic intraluminal material suggestive of thrombosis. Differentiating phlebitis from other conditions (e.g.,

cellulitis, abscess). Monitoring and Follow-Up: Use of POCUS to track progression or resolution of phlebitis. Adjusting catheter management based on ultrasound findings.

Case Examples

Walkthrough of real-life cases illustrating the use of POCUS for vascular access and phlebitis detection. Challenges encountered and lessons learned.

Tips and Best Practices for Implementing POCUS

Training and skill acquisition for small animal practitioners. Equipment considerations and maintenance. Establishing protocols for vascular access and phlebitis assessment.

Conclusion

POCUS is a valuable tool in small animal emergency and critical care for improving vascular access success and diagnosing complications like phlebitis. Incorporating ultrasound into routine practice can enhance patient care and outcomes.

Q&A Session

Opportunity for veterinarians to discuss specific challenges, share experiences, and clarify doubts.

This lecture equips small animal veterinarians with practical knowledge and techniques to enhance their use of POCUS in emergency and critical care settings.

USE OF ULTRASOUND IN ATELECTASIS AND LUNG RECRUITMENT

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Learning objectives:
The aim of this lecture is to discuss the impact of atelectasis in small animals and to demonstrate the usefulness of ultrasonography for detection of atelectasis and for guiding the recruitment maneuvers.

Proceeding:

Atelectasis and Recruiting Maneuvers

Atelectasis frequently occurs during general anesthesia or in recumbent critical care patients. Two mechanisms are described as responsible for atelectasis genesis: compression and oxygen reabsorption. Atelectasis is responsible for decreased lung compliance and can lead to oxygenation impairment.

Healthy patients can generally tolerate mild or moderate atelectasis, but this might not be true for old or sick patients, or patients with respiratory impairment.

Use of positive end expiratory pressure (PEEP) and recruitment maneuvers (RM) during mechanical ventilation can help reducing the incidence and the extent of atelectasis. However, irrespective of how the recruitment is administered, it can lead to complications such as barotrauma, volutrauma or hemodynamic destabilization.

Atelectasis can be detected monitoring lung compliance dynamics or assessment of arterial blood gases. Small amounts of atelectasis or the efficacy of RM can be detected by computed tomography (CT) that is considered the gold standard, but this procedure is not routinely accessible, especially in critical patients.

In recent years, ultrasonography has proven useful to recognize atelectasis early and to guide the clinician during recruitment maneuvers.

A normal healthy lung is characterized by the normal pleural line, mirror-image artifact and A-line artifacts with lung sliding. With the formation of atelectasis, first multiple overlapping B-lines appear, then subpleural consolidations with or without static air bronchogram with frequent B-lines in the margins. During the recruitment maneuver lung modifications are observed in the reverse order: the subpleural consolidations will turn into B-line artifacts and finally will come back to the normal lung image.

Several human studies showed that lung ultrasound (LUS) and the use of specific scoring systems are able to identify and to measure the extent of atelectasis. The comparison between preanesthetic and postanesthetic LUS and atelectasis scores can detect even minimal alterations in lung aeration. Real time ultrasound lung monitoring during RM allows to identify the opening pressure value of PEEP; in this way it is possible to guide a stepwise incremental PEEP recruiting maneuver and cease the increasing pressure at the

value necessary to re-open the alveoli, thus potentially reducing the possibility of lung overinflation. During mechanical ventilation of critical patients, the repeated use of LUS can help the clinician to guide the ventilation mode, the use of PEEP, and of FiO2 and to promptly identify atelectasis areas.

In a critical patient LUS can be used to verify the efficacy of non-invasive ventilatory support methods such as high flow nasal cannula (HFNC) or continuous positive airway pressure (CPAP) in maintaining an aerated lung or in reducing atelectasis.

It is important to remember that atelectasis is seen as consolidation and can be confounded with other cause of consolidation (hemorrhage, pneumonia) thus the importance of a preanesthetic LUS evaluation and the clinical evaluation of the patient. Finally, while LUS can be able to identify atelectasis also at early stages, it is not able to identify overinflation, for this reason it is important to apply an ultrasound-guide recruitment procedure and not to use LUS to just verify the lung at the end of a RM.

In conclusion, routine use of LUS during anesthesia and ventilation can help in customizing the timing and the pressure of recruitment for each patient.

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DIAPHRAGMATIC EXCURSION AND VENTILATION

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Learning objectives:

This lecture discusses the principles, advantages, and practical applications of ultrasonography in assessing diaphragm function in veterinary patients, highlighting its use in diagnosing respiratory dysfunction, guiding treatment, and monitoring recovery.

Proceeding:

Ultrasonography has become an invaluable non-invasive tool in veterinary medicine for assessing various physiological and pathological conditions. One of the more recent applications is the evaluation of diaphragm function. Diaphragm function is crucial in respiratory mechanics, and its proper functioning is vital for the effective exchange of gases. Traditionally, diaphragm function has been assessed using invasive methods like electromyography, but these methods are often impractical in a clinical setting. Ultrasonography offers a non-invasive, real-time, and effective way to assess diaphragm movement and function, providing valuable insights for diagnosing respiratory disorders.

Studies in human medicine showed that ultrasonography helps detecting ventilator-induced diaphragmatic dysfunction (VIDD) in critically ill patients. VIDD is associated with prolonged mechanical ventilation, leading to a decrease in diaphragm strength and effectiveness. Ultrasonography can monitor diaphragm thickness and excursion, helping clinicians identify early signs of dysfunction. M-mode ultrasonography is commonly used to measure diaphragm excursion (DE) during breathing, assessing diaphragm excursion during inspiration and expiration.

B-mode ultrasonography is used to measure diaphragm thickness (DT) and the change in thickness during the respiratory cycle, diaphragmatic thickness fraction (DTF), indicating the diaphragm's contractility and function. Cutoff values for these measurements have been calculated and can help predict which patients are more likely to succeed in weaning from the ventilator. Ultrasonography is used to track the recovery of diaphragmatic function during and after mechanical ventilation. Early transition from controlled ventilation to assistive modes (e.g., pressure support ventilation, positive end-expiratory pressure [PEEP]) can help reverse VIDD and improve diaphragm recovery. Ultrasonography is also used to assess diaphragm function in patients post-surgery (especially thoracic surgery) or those with neuromuscular disorders. It provides an objective measure of diaphragmatic function and helps guide rehabilitation and therapeutic interventions.

In veterinary medicine few studies have been conducted to evaluate the use of ultrasonography for diaphragm function, but the results are encouraging. The mean diaphragmatic excursion (DE) in normal

dogs was reported as 7.29 ± 2.24 mm. A difference of 55% between the left and the right side indicated a unilateral paralysis in a dog.

A study found that the mid-diaphragmatic sublocation in supine position provided the highest DE accessibility. It also noted that while age and sex did not affect DE, body weight was a significant factor among dogs of various sizes. To evaluating the DE, the transducer should be positioned in the craniodorsal direction in the sagittal plane at the costal arch. The stomach or spleen is used as a window to visualize the left hemidiaphragm. The transducer should be adjusted to ensure that the hyperechoic line of the diaphragm is visible, and the mode should be switched from brightness to motion mode for evaluating diaphragmatic movement.

For dogs a formula was described to calculate the diaphragmatic excursion (DE) in dogs with various body weights as following:

DE = 0.0188 x BW + 0.614

where BW represents the body weight of the dog.

A study conducted in cats described also the normal values for this species; the mean diaphragmatic excursions (DEs) for the left and right hemidiaphragms were 0.66 ± 0.16 cm and 0.64 ± 0.18 cm, respectively.The mean diaphragmatic thickness (Tdi) during the end-inspiration phase was 0.13 ± 0.03 cm for the left hemidiaphragm and 0.12 ± 0.03 cm for the right hemidiaphragm, while during the end-expiration phase, it was 0.07 ± 0.03 cm for both hemidiaphragms. The diaphragmatic thickening fraction (DTF) ranged from 23.90–122.1% for the left hemidiaphragm and 38.80–107% for the right hemidiaphragm. Intrinsic factors such as sex, age, body weight, and body condition score did not significantly impact DE, Tdi, and DTF. Cut-off values to distinguish between healthy and diseased cats were established, with the left hemidiaphragm having a cut-off value of 0.458 cm (AUC 0.846), sensitivity of 75.00%, and specificity of 86.36%. The right hemidiaphragm had a cut-off value of 0.423 cm (AUC 0.704), sensitivity of 41.67%, and specificity of 100%.

Even though this technique is promising it presents some limitations that need to be overcome for a spread of its application in the clinical setting. The quality of ultrasonographic images and measurements of diaphragm function (such as thickness and excursion) is highly dependent on the skill and experience of the veterinarian performing the ultrasound. Inexperienced operators may struggle to obtain clear, accurate images, leading to potential misinterpretation.

The effectiveness of ultrasonography can be influenced by the size and species of the animal. In large animals obtaining clear images of the diaphragm can be more challenging due to the depth and size of the body as well as animals with higher body fat, resulting in lower-quality imaging.

Animals, especially those that are ill, anxious, or in pain, may have difficulty staying still during the ultrasound exam. This movement can result in blurry images and inaccurate assessments of diaphragm function. For some species, sedation or anesthesia might be necessary, which could present additional risks and complications. Unlike human medicine, where standardized techniques and protocols are widely used, there are fewer established, evidence-based guidelines for ultrasonographic evaluation of the diaphragm in

veterinary practice. This can lead to inconsistencies in how the procedure is performed and how the results are interpreted.

In conclusion ultrasonography has become a key diagnostic tool for assessing diaphragmatic function in human medicine. It plays a critical role in the diagnosis of VIDD, predicting weaning outcomes, and monitoring recovery, especially in critically ill patients. As a non-invasive, real-time, and cost-effective method, ultrasonography offers significant advantages for improving patient care, particularly in the management of respiratory conditions.

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KEY NOTE: HOW POCUS CHANGED OUR LIVES IN ACUTE HUMAN MEDICINE SETTINGS

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Learning objectives:

- Brief history of POCUS in EM and ICU settings

- Main components of POCUS

- Integration of clinical and ultrasound data into decision-making

- Ultrasound-aided interventions

Proceeding:

Ultrasound has been used in medicine for over half a century but was only recently widely adopted in emergency and intensive care. This is often attributed to the increased availability of suitable devices due to technological advancements. However, the main reason for its relatively late extensive adoption lies in the resistance to changing established habits, which has slowed the integration of this elegant method into standard clinical practice.

POCUS first gained traction in emergency medicine through standardized protocols designed for rapid application in critical scenarios. One of the earliest, the FAST protocol, detects free fluid in trauma patients, indicating the need for urgent surgery. Since then, ultrasound has proven invaluable in many other acute conditions.

The concept of "visual medicine" emphasizes ultrasound use by the clinician at the bedside. This approach extends the physical exam, allowing clinicians not just to look at the patient, but to look into them—literally. Even when performed quickly, without formal measurements, ultrasound enhances the understanding of pathophysiological processes and guides further diagnostics and treatment.

The WINFOCUS organization recognized the need for better defining the scope of ultrasound use by the treating clinician and proposed structured education. This has significantly accelerated the adoption of ultrasound in emergency and intensive care settings.

We advocate for a holistic approach to ultrasound use by clinicians, where answers to clinical questions are immediately integrated into decision-making. A focused ultrasound exam includes a rapid assessment of the lungs, heart, inferior vena cava, serous cavities, abdominal organs, aorta, kidneys, pelvic organs,

proximal leg veins, and other relevant structures as needed. Priority is given to addressing the most clinically relevant questions with greater attention and time.

Lung ultrasound surpasses physical examination and chest X-rays in diagnosing pulmonary edema, pneumonia, pleural effusions, atelectasis (especially in early stages), pneumothorax, and consolidation. French intensivist Daniel Lichtenstein, the founder of lung ultrasound in human medicine, proposed the BLUE protocol, which allows for correctly identifying the etiology of acute dyspnea in 90% of cases based solely on ultrasound. Interestingly, the ability to detect pneumothorax with ultrasound in human medicine was adapted from veterinary medicine.

A basic cardiac ultrasound, performed quickly, based on impression, without measurements, enables assessment of left ventricular systolic function, detection of right ventricular strain, and recognition of significant pericardial effusion. Though estimating volume status based on inferior vena cava diameter has limitations, it provides better clinical orientation than relying solely on physical signs.

In shock patients, identifying the underlying cause is crucial. Ultrasound provides direct insight into the circulatory system, assessing cardiac function, intravascular volume, and lung status. The FALLS protocol guides fluid resuscitation by tracking lung B-lines, indicating early interstitial lung edema. It optimizes preload while preventing fluid overload and is particularly valuable for patients with left heart dysfunction but not suitable for those with right heart failure.

During cardiopulmonary resuscitation, ultrasound simplifies cardiac function assessment compared to pulse palpation. More importantly, it helps diagnose reversible causes of cardiac arrest such as pericardial tamponade, pneumothorax, hypovolemia, and thromboembolic events.

A focused ultrasound of the abdomen and retroperitoneum can reveal significant findings with direct treatment implications.

Identifying the source of infection helps tailor empirical antibiotic therapy and determines the need for drainage of infected fluid collections, often achievable via ultrasound-guided percutaneous intervention.

Ultrasound enhances procedural safety and precision, improving vascular access, pleural drainage, pericardiocentesis, abdominal fluid aspiration, intubation, percutaneous tracheostomy, and the drainage of abscesses or fluid collections.

In our ICU, every patient undergoes a basic ultrasound exam upon admission, followed by daily reassessments during clinical rounds. When needed, comprehensive exams are performed. Integrating ultrasound into daily practice has significantly reduced reliance on radiologic imaging. Additionally, all invasive procedures are performed with ultrasound assistance.

In conclusion, we can say that the adoption of ultrasound as a fundamental tool in the hands of clinicians has enhanced the immediate understanding of a patient's condition, with direct implications for more efficient diagnostic and therapeutic strategies.

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DECODING THE LINES AND SIGNS OF POCUS

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Learning objectives:

- Identify and differentiate the various pleural and lung ultrasound (PLUS) lines and signs described in the human and veterinary literature

- Understanding the significance of different PLUS lines and signs

- Discuss the pathophysiology behind clinically relevant PLUS artifacts.

Proceeding:

Introduction

This lecture will cover the overwhelming number of lines and signs often used in Point-of-Care Ultrasound (POCUS); from A-lines all the way to Z-lines, and everything in between, including B, C, E and W lines and a whole lot more less clinically relevant alphabet signs. The "peek-a-boo", "accordion", "jelly fish", "sail" and many other signs beyond the alphabet will also be covered. Attendees will gain understanding of these artefacts, their clinical relevance (or lack thereof), and how to incorporate or disregard them in their diagnostic decision-making. Clinical examples will be shown, and practical applications will be discussed. Designed for veterinarians and nurses, this 50-minute lecture aims to improve the skills and confidence in using POCUS for clinicians.

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CASE-BASED POCUS

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Learning objectives:

- Describe how POCUS can aid the daily assessment of a critically ill patient

- Describe the aspects of shock pathophysiology that POCUS can aid in understanding

Proceeding:

My approach to daily assessment of a critically ill patient follows a body systems approach. This contrasts with summarising results from the physical examination, monitoring, blood tests, imaging, and other diagnostics separately in sections. Alternatively, I summarise all related data for each body system in turn: neurologic, cardiovascular, respiratory, gastrointestinal and endocrine, renal and fluid/electrolyte, sepsis and haematology, skin and musculoskeletal, and ear/eye/nose/mouth. Rather than just being an alternative approach to organising a medical record, this is an fundamentally different cognitive model that emphasises the interrelated nature of different pieces of evidence regarding the status of each body system. For several systems, data from point-of-care ultrasound (POCUS) forms an essential part of the daily assessment. This session focusses on the cardiovascular, respiratory, and gastrointestinal assessment, though there are applications of POCUS beyond these.

Cardiovascular

Critically ill patients are at risk of cardiovascular instability. Rather than showing the overt, decompensated shock of a newly-presenting emergency patient, critically ill patients often have subtle signs of cardiovascular instability. These patients require haemodynamic optimisation with well-justified therapies, rather than immediate rapid bolus fluid administration. There are several ways in which POCUS of the heart, major vessels, and lungs may aid shock assessment. Many can be accomplished with small amounts of training. Ultrasound assessment of cardiac chamber volume can give a crude assessment of volume status. 2D and M-mode echocardiography can assess myocardial contractility. There is a large body of research into ultrasound of the vena cava through the respiratory cycle. Several locations have been described, but most clinicians assess it as it crosses the diaphragm. Cyclic changes in diameter >25-50% may be suggestive of fluid responsiveness, though further research is needed to validate these findings. Distension of the vena cava suggests that further fluid administration is likely to be poorly tolerated, analogous to a progressively increasing central venous pressure. Lung ultrasound may detect developing fluid overload. Doppler ultrasound techniques may provide even more information, but require advanced

training that is not yet readily available to most veterinary criticalists. Stroke volume (and thus cardiac output) can be estimated from Doppler ultrasound of the left or right ventricular outflow tract, by calculation of the outflow tract velocity time integral in systole. Whilst this may not be the most accurate method of absolute cardiac output measurement, it is well positioned to assess changes in cardiac output following an intervention such as a fluid challenge. This technique is expected to become more widespread in the future, with increased training and improvements in technology.

Respiratory

Respiratory dysfunction is common in critically ill patients. Pulmonary oedema (both cardiogenic and non-cardiogenic), pneumonia, and pleural effusion are common complications of critical illness. Further complications include haemorrhage and pulmonary thromboembolism. In human intensive care medicine, a daily thoracic radiograph is considered standard of care in many settings, as a monitoring tool for early warning signs of these processes before they become clinically relevant. This is rarely practical in veterinary medicine, due to the more challenging logistical considerations. Thus, without POCUS, most critically-ill animals would not have any monitoring for the presence and pattern of pulmonary infiltrates. Whilst POCUS is only able to image the peripheral lung, it can be an early warning sign of development of pathology that may prompt further imaging such as radiography or computed tomography. The distribution of abnormal pulmonary findings such as B lines and shred sign can give clues about the likely underlying process that prompt changes to therapy even while further diagnostics are pending. Furthermore, serial tracking of POCUS can assess response to therapy. Thoracic POCUS can also sensitively detect pleural effusion, which may be an early indicator of fluid overload, decreased oncotic pressure, or increased vascular permeability. Daily ultrasound of the lung and pleural space can therefore be a vital component of respiratory assessment.

Gastrointestinal

Gastrointestinal dysmotility is common in critically ill patients. It predisposes to complications including a negative caloric balance, vomiting or regurgitation, and aspiration pneumonia. Active vomiting or regurgitation are late signs of gastrointestinal dysfunction. POCUS of the gastrointestinal tract allows for early identification of gastroparesis and ileus. Furthermore, the ideal treatment for gastrointestinal dysmotility in critical illness is unknown. Serial POCUS is an ideal method for objectively assessing response to therapy.

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CAT VERSUS DOG POCUS

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Learning objectives:

- Recognise how feline behaviour affects feline POCUS technique

- Understand how feline anatomy affects POCUS

- Interpret POCUS in context of the species and their clinical presentation

Proceeding:

Point-of-care ultrasound (POCUS) is an invaluable tool in the emergency and critical care setting in both the cat and dog. There are, however, species-specific differences in the utility and limitations of POCUS. This lecture will discuss pertinent aspects of feline POCUS highlighting key species differences and gaps in our understanding of feline POCUS.

Probe choice and ultrasound settings

Although pleural space and lung ultrasonographic findings have been found to be similar in dogs and cats when using a micro convex and linear probe, due to the small size of cats, and corresponding narrow intercostal spaces and the relatively large footprint of the linear probe a linear probe is not recommended for pleural space and lung ultrasound in cats. A linear probe may improve assessment of superficial feline abdominal organs such as the kidneys. However, with POCUS rapid stress-free examination is prioritised over perfect image quality.

Therefore a single probe, the microconvex probe is often used for feline lung, pleural space (PLUS), abdominal and cardiac POCUS. Due to the small size of cats the frequency of the chosen probe is often set higher than dogs as the ultrasound beam does not need to penetrate as great a distance. A microconvex probe with frequency of 5-8mHz and depth of 4-6cm is a good basic pre-set for feline POCUS.

Patient scanning

Cats are both predator and prey species and often behave like the latter in new environments. Cats therefore often present as an emergency near-critical and can decompensate rapidly. Careful handling is paramount to minimise stress to the patient. Feline POCUS is often performed with cats in their carriers or in standing position to minimise the stress of handling. Alcohol application should be kept to a minimum as excessive alcohol application has a cooling effect and considering their smaller size and larger surface area

cats are at greater risk of hypothermia than dogs. Proactive placement of a cat on a pet bed or cat-safe heat mat may help minimise further heat loss.

Pleural space and lung ultrasound

PLUS is valuable in both canine and feline emergency and hospitalised patients and various scanning techniques has been described. In both species the choice of technique should be clinically driven, to ensure the clinical question is answered. The operator needs to be aware of important thoracic anatomical differences as well as the aetiopathogenesis of the different causes of respiratory disease in cats and dogs to ensure optimal scanning and interpretation. Cats have a smaller lung volume than dogs, and thus an overall smaller scanning area. However, the shape of the feline thorax is triangular; narrow ventrodorsally and more elongated caudally. This means the caudodorsal lung margins reach more caudally than dogs (T13-L1 versus T12-T13). This is clinically important when assessing the thoracoabdominal borders and identification of the curtain sign, an artefact used to in the diagnosis of a pneumothorax. B lines, vertical hyperechoic artefacts arising from the pleural line are commonly used to identify pulmonary pathology, however, B-lines have been reported in healthy dogs and cats. In both species the incidence of B lines vary depends on lung ultrasound scanning protocol used. Both healthy dogs and cats have increased incidence of B lines in protocol that scan more sites, reported to occur in up to 35% in dogs and 50% cats.

Cardiac POCUS

Cardiac POCUS is a useful tool for detection of cardiac causes of cardiorespiratory disease as well as aid in the assessment of volume status. Both transthoracic and subxiphoid windows can be used to assess the heart in dogs, however, subxiphoid assessment of the heart in cats is more difficult due to the greater extension of the lungs caudally in the thoracic cavity. In both dogs and cats cardiac underfilling is often identified by pseudohypertrophy of the left ventricle. The assessment of volume status is complicated in cats due to the reasonably high prevalence (15%) of hypertrophic cardiomyopathy in the general feline population.

Abdominal POCUS

Distribution and relative echogenicity of abdominal organs varies between dogs and cats, which may affect image interpretation. Important difference includes distinctiveness of the portal veins, the echogenicity of the falciform fat relative to the liver and location of the feline spleen and pancreas. The abdominal vena cava has been assessed at multiple sites (subxiphoid, hepatic, paralumbar view) in both dogs and cats to determine volume status. However, reliability and user agreement between the different site varies between dogs and cats. As for all POCUS, abdominal POCUS needs to be clinically driven, and interpretation of findings should be considered considering the most likely species-specific disease process.

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ULTRASONOGRAPHIC ASSESSMENT IN STATES OF SHOCK

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Learning objectives:

- Suspect hypovolemic shock using cardiac chamber measurement and large vessels measurement and collapsibility.

- Suspect obstructive shock assessing pericardial and pleural spaces.

- Suspect cardiogenic shock with signs of congestion and measurements of cardiac chambers and contractility.

- Suspect distributive shock excluding other types of shock and looking for signs of anaphylaxis

Proceeding:

Shock is a critical condition characterized by inadequate cellular energy production, leading to cellular hypoxia and potential organ failure. The tree of life defines how the oxygen can be correctly distributed to the tissues and any impairment in this tree will lead to shock. Shock can be due to inadequate arterial oxygen content, inadequate arterial to venous pressure gradient or inadequate cardiac output. In both dogs and cats, poor cardiac output shock states can result from various etiologies, including hypovolemia, cardiogenic failure, distributive causes, and obstructive factors.

Perfusion parameters such as pale mucous membrane, prolonged capillary refill time, tachycardia, lethargy, cold extremities and weak pulse will suggest a poor cardiac output but lack of specificity to differentiate the underlying mechanism. Each etiology requires specific management, completely different from one cause to another, which requires initial careful diagnostics of the shock state. Recognizing and diagnosing shock promptly is thus essential for effective intervention. Thus, the clinician should use other additional tests.

Point of Care Ultrasound (POCUS) became an invaluable tool in the assessment and management of shock in dogs and cats. Its non-invasive nature, coupled with the ability to provide real-time imaging, makes it particularly useful in emergency and critical care settings. Let’s review the circulatory shock states and see how POCUS can help us:

Hypovolemic shock: A loss of intravascular fluid volume decreases preload and induces inadequate organ perfusion. This loss of intravascular fluid can be due to hemorrhage or a severe imbalance between fluid intake and loss (through the digestive tract or urine or fluid sequestration). Abdominal and thoracic POCUS can detect a large amount of fluid in the body cavities. After punction, a fluid PCV above 25% of the

patient's PCV will diagnose an hemorrhagic effusion. If no fluid is detected in the body cavities, POCUS can still help the clinician to assess the filling of the cardiovascular system and its potential ability to respond to fluid administration. Thus, the cardiovascular system can be screened with the TANK and the PIPES.

Tank: On a right parasternal short axis view, focusing on the left ventricle, the cardiac cavity will appear "empty" in a hypovolemic patient, and the ventricular walls may appear thickened ("pseudohypertrophy"). In a long axis four chamber view, one might observe a "kissing ventricle" referring to the walls of the ventricle touching due to being underfilled. On the other end, a patient with fluid overload may have an enlarged heart cavity. In this case, examining the left atrium is a reliable indicator. On a right parasternal short axis view at the aortic level, an increased left atrium to aortic ratio (LA:Ao) can indicate fluid overload, although it cannot be differentiated from intrinsic cardiac disease when examined alone.

Pipes: Large veins act as a volume reservoir for the body and can be screened using ultrasound. When assessing the caudal vena cava (CVC), different views are described: sub-xiphoid, transhepatic, and para lumbar. The first two views allow for assessing the CVC diameter and its dynamics regarding the proximity to the thorax and the interactions between the heart and lungs. The last one can provide a view of the aorta for comparing diameters. Patients with low volume status are likely to have a small diameter CVC and highly collapsible vessel. On the other hand, a patient with fluid overload will have an enlarged CVC diameter and it will be poorly collapsible. The CVC diameter can be compared to the aortic (Ao) diameter at the left paralumbar view. A CVC / Ao of less than 0.8 suggests a low volume status and a CVC / Ao above 1.1 suggests overload.

Cardiogenic shock: Systolic and/or diastolic dysfunction will lead to hemodynamic instability in this type of shock. Systolic function can be easily assessed at the right parasternal short axis view using POCUS. Fractional shortening can be calculated using the formula (diastolic left ventricle diameter – systolic left ventricle diameter) / diastolic left ventricle diameter. Breed specific normal ranges are described but a value under 20-25% suggest a poor heart contractility. Systolic and/or diastolic dysfunction can lead to increased hydrostatic pressures in large vessels and congestion. Thus, a left congestive heart failure will cause pulmonary edema, easily detectable with increased number of B lines of the thoracic POCUS. Pleural effusion can also be present in feline left congestive heart failure. On the other hand, right congestive heart failure will cause ascites, easily detectable on abdominal POCUS. After punction, a low cell count and total protein will help to diagnose a transudate.

Distributive shock: Compared to hypovolemic shock, the intravascular volume can be normal but a change in vascular tone will change the distribution of this volume and induce a relative hypovolemia. Sepsis, anaphylaxis or catecholamine excess and the major causes of the redistribution. A correct filling of the tank and pipes in a hypotensive patient will suggest a distributive shock. In dogs with anaphylaxis, a gall bladder "halo sign" is also commonly seen.

Obstructive shock: Venous return can be compromised by compression of the heart or a large vessel. Thus, tension pneumothorax, cardiac tamponade or severe gastric dilation will decrease diastolic filling or preload.

Tension pneumothorax can be detected on the thoracic POCUS if the glide sign is absent of if an abnormal curtain sign is present

Cardiac tamponade can be easily detected on the parasternal views. A pericardial effusion will be associated with a paradoxical movement of a cardiac chamber (most often right atrium) free wall during contraction. The subxiphoid view may help distinguish pericardial effusion from pleural effusion in large dogs.

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WHEN THE PITFALLS ARE UNKNOWN THE ABUSE IS INEVITABLE

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Learning objectives:

- Point-of-care ultrasound (POCUS) in emergency medicine offers rapid diagnostics but has pitfalls. Operator dependency and limited training can lead to misinterpretation.

- Poor image quality in unstandardized positioning and uncooperative patients hamper accuracy.

- Over-reliance on POCUS risks bypassing comprehensive assessments.

- Incomplete protocols and cognitive biases may result in diagnostic errors, impacting patient outcomes.

Proceeding:

Point-of-care ultrasound (POCUS) has revolutionized emergency medicine by providing rapid, real-time imaging that aids in critical decision-making. It is used for diagnosing conditions such as pneumothorax, pericardial effusion, cavitary free fluid, vein thrombosis, and guiding procedures such as central venous catheter placement. However, despite its many advantages, POCUS is not without its limitations and potential pitfalls. These challenges, if not recognized and addressed, can lead to diagnostic errors, patient harm, and a false sense of security. POCUS is highly operator-dependent, meaning its accuracy and usefulness are significantly influenced by the skill, experience, and training of the user. Inexperienced operators may misinterpret images or fail to obtain adequate views, leading to incorrect diagnoses. Even among experienced clinicians, there is variability in technique and interpretation, which can result in inconsistent findings. Unlike radiology-performed ultrasounds, POCUS is often performed in suboptimal conditions such as crowded emergency rooms, poor patient positioning, or when patients are in distress. These factors can compromise image quality and lead to incomplete evaluations. For instance, trembling, bowel gas, and patient movement can degrade the clarity of images, making it difficult to identify pathology accurately. POCUS is meant to be an adjunct to, rather than a replacement for, comprehensive clinical evaluation and other imaging modalities. However, there is a risk of over-reliance on POCUS, leading clinicians to ignore clinical evidence and forgo further necessary imaging studies. This over-reliance can be exacerbated by cognitive biases, such as confirmation bias, where clinicians may interpret images in a way that supports their pre-existing impression rather than objectively analyzing the findings. False-positive findings can lead to unnecessary interventions, while false negatives can result in missed diagnoses. For example:

- Pneumothorax: The absence of lung sliding on ultrasound is often used to diagnose pneumothorax, but conditions such as pleural adhesions and shallow breathing can also produce this finding, leading to a false-positive diagnosis.

- Cardiac Tamponade: A pericardial effusion seen on POCUS does not always indicate tamponade physiology, and clinical correlation is necessary to avoid unnecessary interventions such as emergency tap. POCUS is a focused examination and may not provide a comprehensive assessment. The Focused Assessment with Sonography in Trauma (FAST) can miss retroperitoneal bleeding, small amounts of free fluid, or injuries that require further imaging. Errors in POCUS interpretation or failure to follow up on findings can have legal ramifications. If a clinician makes a critical diagnosis based solely on POCUS and it turns out to be incorrect, they may face liability issues. Additionally, there are concerns about documentation and whether POCUS findings should be formally recorded and archived, as misinterpretations can be scrutinized in legal proceedings. While POCUS is excellent for certain conditions, it has limited utility in others. For instance:

- Pulmonary Embolism (PE): POCUS may suggest PE through right ventricular strain but cannot directly visualize pulmonary emboli.

- Bowel Obstruction: POCUS can detect dilated loops of bowel but is less sensitive than official US for diagnosing the underlying cause of obstruction. POCUS findings can be non-specific and may overlap with multiple conditions.

- B-lines in Lung Ultrasound: While often associated with pulmonary edema, B-lines can also be seen in pneumonia, ARDS, and interstitial lung disease, making it challenging to determine the underlying cause.

- Caudal Vena Cava (CVC) Assessment: CVC collapsibility is frequently used to estimate volume status, but it can be misleading in conditions like pulmonary hypertension, pleural effusion, or increased intra-abdominal pressure. Despite widespread adoption, there is no universal standardization of POCUS training. Variability in curricula, credentialing, and ongoing competency assessment can lead to inconsistencies in how POCUS is performed and interpreted. This lack of standardization increases the risk of errors and limits its effectiveness. To mitigate these risks, providers should undergo rigorous training, use POCUS as an adjunct to comprehensive evaluation, and recognize when further imaging or specialist consultation is required. By understanding its limitations, emergency medicine veterinarians can maximize the benefits of POCUS while minimizing the risks of misdiagnosis and patient harm.

Main Stream, Thursday 5 June 2025

THORACIC TRAUMA: AN ECC APPROACH

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Learning objectives:

- Recognise a unstable respiratory patient after trauma

- Appraise clinical findings & formulate plan for stabilisation

- Perform common respiratory emergency procedures

Proceeding:

Common respiratory pathologies secondary to trauma include pulmonary contusions, pneumothorax, haemothorax, rib fractures and diaphragmatic rupture (Reineke, 2019). Upper airway injuries, such as tracheal rupture, are rare complications but can lead to rapid decompensation and need to be recognised rapidly (Shih, 2021). With these differentials in mind respiratory triage should include respiratory rate, assessment of effort including assessment for altered breathing patterns included focal abnormal thoracic wall movement and the presence of audible respiratory noise. The presence of any wounds and/or subcutaneous emphysema and any thoracic wall defect should be identified and noted. Subcutaneous emphysema may be the only indicator that there has been airway trauma.

Signs of respiratory distress, such as dyspnoea or orthopnoea) and/or the presence of cyanosis indicate an imminent risk of respiratory arrest and necessitate immediate oxygen therapy and/or therapeutic thoracocentesis. Prompt administration of analgesia with a pure opioid such as methadone is recommended to manage pain and facilitate handling. Furthermore, pain is a common non-respiratory cause of tachypnoea and this, unlike respiratory causes, improves with adequate analgesia (Sigrist et al 2011). Non-steroidal anti-inflammatories should be avoided during the emergency stabilisation of a trauma patient due to the likely presence of shock and unknown perfusion status of the gastrointestinal tract.

Oxygen supplementation should be provided to all animals with a history of thoracic trauma. Flow-by oxygen, i.e oxygen provided via a tubing placed close to the patient's mouth, is often the appropriate choice during initial stabilisation in all, aside from comatose patients due to its accessibility.

Pleural space and lung ultrasound (PLUS) is recommended in all animals with a history of thoracic trauma. The technique should only be performed in the position the animal is comfortable in. Care should be taken not to move a polytrauma patient in case there is spinal fracture or luxation. Often polytrauma patients present in lateral recumbency and therefore the scan should be limited to the dependent thorax until

deemed safe to move the patient. PLUS is particularly useful in identifying artefacts compatible with pulmonary contusions, pleural effusion, and ruling out pneumothorax. Trauma patients often have multiple pulmonary injuries and therefore pulmonary contusions and pneumothorax to be present in the same patient. Scanning ventrally will allow detection of pleural effusion and signs compatible with diaphragmatic rupture. The liver is the most commonly herniated organ followed by the spleen, stomach, small intestine and pancreas, which can be readily identified on trans-thoracic scanning. PLUS has been shown to have good agreement with both thoracic radiographs and computed tomography in dogs and cats with thoracic trauma, particularly for the diagnosis of pulmonary contusions (Vidal et al 2024; Dicker et al 2020). Although radiographs may have a role in identification of tracheal, mediastinal, and other thoracic wall injuries due to the stress associated with positioning for radiographs and the difficulties of monitoring patient during the procedure radiographs rarely have a role in the emergency setting.

In patients with signs of severe respiratory distress, and a pneumothorax is suspected, diagnostic and therapeutic thoracocentesis should performed in favour of PLUS. In these circumstances the benefit of immediate thoracocentesis outweigh the potential risk (lung injury, haemorrhage, inadvertent injury to abdominal contents. Fluid resuscitation is required in the presence of a tension pneumothorax, a life threatening respiratory and cardiovascular emergency as air continually leaks from the lung the pressure within the pleural space leads to an ongoing reduction in lung volume and decrease in venous return (Brockman 2004).

Thoracocentesis can be performed in the emergency setting with adequate analgesia and light sedation using a needle or butterfly catheter, extension set, three-way tap and collection syringes. The volume of air and/or fluid retrieved should be recorded. Thoracostomy tubes are indicated if air continues to accumulate within the pleural space requiring repeated needle thoracocentesis, or failure to achieve negative pressure. If there is a penetrating thoracic wound then the wound should be cleaned and covered and sometimes a thoracostomy drain is needed (Reinke & Savini 2015). Thoracostomy tube placement using the seldinger technique is achievable in the emergency room, requiring less sedation than large-bore thoracostomy tube placement. In patient with substantial thoracic wall trauma the patient should be positioned with the affected side towards to the table. In cases of diaphragmatic rupture, the patient may be positioned on a slight incline in hope that the abdominal organs move back into the abdomen through gravity. In animals in which the stomach is herniated and showing signs of respiratory distress either passage of a nasogastric tube or needle thoracocentesis into the distended stomach to allow for stomach decompression is indicated (Reinke & Savini 2015)

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THORACIC TRAUMA: DIAGNOSTIC IMAGING

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Learning objectives:

- Improve radiographic technique and develop a systematic approach to assessment of thoracic radiographs.

- Be familiar with radiographic features of common traumatic thoracic lesions, including diaphragmatic rupture, pneumothorax, pleural effusion, thoracic wall trauma and pulmonary lesions.

- Understand the importance of clinical assessment in contextualising radiographic findings.

Proceeding:

Good radiographic technique maximises the benefit of thoracic radiography and aids image interpretation. Ensure a complete study is performed to include three views (dorsoventral (DV), right and left lateral), where possible. In trauma cases, a DV view performed first can enable quick assessment for significant thoracic pathology without further compromising patient stability. Additionally, performing a DV first avoids the confounding effect of atelectasis due to lateral recumbency. Patients should be well positioned, and collimation should include the entirety of the thoracic cavity. Acquisition should be performed in the inspiratory phase to maximise thoracic volume. Be systematic when learning to read thoracic radiographs – developing a repeatable technique will ensure improved lesion detection, which is particularly pertinent in cases with multiple lesions e.g. polytrauma patients.

Traumatic diaphragmatic rupture typically occurs due to an acute increase in abdominal pressure with an open glottis. The resulting increased transdiaphragmatic pressure gradient results in diaphragmatic rupture and may lead to intrathoracic displacement of abdominal viscera. Commonly, the stomach, liver, small intestine and omentum may be displaced, however other organs can be involved. Radiological features include reduced visibility or discontinuity of the diaphragmatic margin, pleural effusion, effacement of thoracic structures by displaced viscera, intrathoracic mass effect, abnormal intrathoracic opacity (e.g. fat or mineralised gastrointestinal content), and cranial displacement or absence of viscera within the abdominal cavity. Complementary imaging techniques can aid with diagnosis if radiographic findings are equivocal, and include contrast radiography, ultrasound and computed tomography. Assessment for incarceration of abdominal viscera within the thorax is essential. Notably, tension gastrothorax can result following gastric displacement - radiographic signs include thoracic displacement of the stomach, marked

gastric distension and contralateral mediastinal shift. If identified, prompt intervention including gastric decompression is indicated.

Common traumatic pleural space diseases include pneumothorax and pleural effusion (typically haemothorax). When identifying pleural space pathology, an attempt should also be made to identify the cause. Additional radiographs performed following thoracocentesis may help with this, particularly in cases where there is underlying primary pulmonary pathology – pulmonary inflation will make parenchymal lesions more conspicuous.

Traumatic pneumothorax may occur secondary to blunt force injury and associated airway / parenchymal rupture ('closed pneumothorax'), or due to extra-thoracic gas entering the pleural space ('open pneumothorax'). Radiological features include retraction of lung margins from the thoracic wall, gas opacity in the periphery of the thoracic cavity and elevation of the cardiac silhouette. Smaller volumes of gas may appear as bubbles in the region of the cardiac apex. When diagnosing pneumothorax, it is important to assess suspected pleural gas for absence of lung markings as several interpretative pitfalls exist. Tension pneumothorax is a noteworthy emergent condition requiring immediate intervention and occurs when a pathological 'one-way' valve effect develops e.g. in the thoracic wall. Features include very large volumes of pleural gas, marked reduction in pulmonary volume, diaphragmatic tenting and contralateral mediastinal shift. If identified, pleural drainage should be initiated without delay.

Traumatic pleural effusion typically reflects haemothorax following thoracic vascular or parenchymal injury, however other types of traumatic pleural fluid have been reported. Features of pleural effusion include widened pleural fissures, retraction of lung lobes from the body wall, lung 'scalloping' and effacement of the cardiac silhouette / diaphragmatic margin.

Assessment for thoracic wall injury should form part of the systematic review in trauma cases and can help corroborate a suspected traumatic cause of intrathoracic pathology. Traumatic lesions of the thoracic wall include muscular disruption, rib fractures or displacement, and soft tissue gas tracking following penetrating injury. Radiographic evidence of two or more adjacent segmental rib fractures facilitates the diagnosis of "flail chest".

Traumatic pulmonary lesions include pulmonary contusions, traumatic pulmonary pseudocysts ("haematocoele") and traumatic bullae ("pneumatocoele"). Pulmonary contusions typically appear as regions of ill-defined alveolar pattern and can be underestimated by radiography. There can be a delay in the development of visible pulmonary contusion of up to 6-12hrs.

Radiography is an excellent screening tool for traumatic pathology but should not precede or replace thorough clinical assessment. Abnormalities identified when performing extensive imaging surveys can be challenging to assign significance to, without complete historical and physical examination data to provide context. Similarly, more subtle lesions of clinical significance may be missed or incompletely included in the imaging examination if clinical evaluation is not performed in advance. Radiographic series should be limited to regions of priority and clinical relevance where possible and be utilised to answer specific clinical questions which may guide treatment planning and aid prognostication in the trauma patient.

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THORACIC TRAUMA: SURGERY

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Learning objectives:

- Be able to approach cases of diaphragmatic rupture and grasp vital concepts for surgical repair.

- Appreciate thoracic wall anatomy and understand potential implications for underlying structures.

- Be familiar with surgical management of thoracic wall injury, for example due to bite wounds.

- Understand how to optimize post-operative care in thoracic trauma cases.

Proceeding:

The diaphragm is a musculotendinous structure that separates the thoracic and abdominal cavities. It is made up of muscular portions; pars costalis, pars lumbalis, pars sternalis and the central tendon. It arises from the thoracic wall at the level of the 8-13th ribs. There are three major openings to be aware of when it comes to diaphragmatic surgery: The caval foramen, the oesophageal hiatus and the aortic hiatus. Motor innervation is provided solely by the paired phrenic nerves.

Most traumatic diaphragmatic hernias (diaphragmatic ruptures) are secondary to road traffic accidents and are thought to be due to a sudden increase in intra-abdominal pressure with an open glottis leading to rupture of diaphragmatic tissue. The most recent publication, which included 49 dogs and 48 cats, found that concurrent orthopaedic or soft tissue injuries were present in 48% of cases, most commonly fractures.

Ruptures are most commonly located on the pars costalis and the most frequently herniated organs are the liver or small intestines. The preferred approach for management of diaphragmatic ruptures is via midline celiotomy for several reasons: it allows inspection of all the abdominal organs, the approach can be extended cranially to a median sternotomy if necessary, postoperative pain is lower compared with sternotomy and it provides access to the entirety of the diaphragm. Treatment involves reduction of herniated organs into the abdominal cavity and repair of the diaphragmatic defect – usually by simple continuous suturing to appose diaphragmatic tissue. In cases of circumcostal tears, sutures may need to be anchored around neighbouring ribs or to the abdominal wall.

Consideration should be given to placement of a thoracostomy tube although absolute elimination of pneumothorax is not necessary and clinicians should be cognizant of re-expansion pulmonary oedema.

Better outcomes have been reported in patients undergoing surgery within 48 hours of diagnosis.

Flail chest occurs with segmental fractures (i.e. each rib fractured in two places to give rise to a 'free' segment of bone") of two or more adjacent ribs. In veterinary practice, pseudo-flail chest is more commonly encountered, which also results in paradoxical movement of the thoracic wall, but can be due to just intercostal muscle disruption. A study of 24 cases of canine and feline flail chest found no difference in outcome between those managed with or without surgical stabilization of the flail segment. It is important to recognize that flail or pseudo-flail chest can often be suggestive of underlying pulmonary injury.

Small-medium breeds are particularly at risk of sustaining severe injuries when bitten by another dog. The external appearance of the wounds can often belie the true extent of injury. For this reason, surgical exploration of bite wounds in these cases is advised, although the clinician should be prepared for the possibility of encountering an open thorax upon skin incision. Higher mortality rates have been associated with presence of pleural effusion, positive bacterial culture, and high Animal Trauma Triage (ATT) scores.

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'FRONT-END' FELINE TRAUMA: HEAD AND HIGH RISE

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Learning objectives:

- Outline initial examination & diagnostics for maxillofacial trauma & HRS

- Describe injuries commonly seen in high-rise syndrome

- Detail types & treatment options for hard palate trauma, mandibular (including wiring mandibular symphysis), pharyngostomy intubation, and caudal fractures-luxation including the temporomandibular joint

- Understand prognosis of these injuries and potential complications

Proceeding:

Maxillofacial trauma is usually associated with RTAs, fights or falls - including 'High Rise Syndrome' (HRS). The innate inclination of cats to turn, so to land on their feet when falling from heights predisposes them to a predictable triad of trauma including the head, thorax and extremities.

On presentation, facial trauma is usually apparent. Assessment includes mentation (note TBI is uncommon), facial asymmetry, ocular function, globe retropulsion and oral/nasal discharge, and otic exam for avulsion/bleeding. Many cats will be open mouthed due to blockage of the nasal cavity, or due to fractures/joint luxation. Some cats will allow palpation of the mandible, with gentle opening and closing (particular after analgesia). Once stable, GA for more extensive examination combined with skull series radiographs, or preferably CT which has twice the diagnostic identification of injuries, should follow. Cats also require thoracic imaging ± any other indicated regions.

At induction, dental occlusion between the maxillary and mandibular arcades should be checked prior to ET tube placement, which prevents full mouth closure. Mandibular symphyseal separations are common, however CT studies show 80% of cats have multiple skull fractures. Dental injuries are also common. Tongue and other intra-oral soft-tissue injuries need debridement and lavage and rarely simple interrupted appositional absorbable sutures. Assessment of mandibular drift/stability, hard-palate damage (particularly common in HRS) follows. Some caudal injuries are difficult to assess but caudal intra-oral bruising can be suggestive. Where there is a symphyseal separation, it is possible to manually reduce it by squeezing the canines together and then assess for other more caudal sources of jaw instability. Fractures can pass

through the alveolus of a tooth, however if the tooth is stable it should be left in place at that stage for stability.

Some mandibular fractures which retain normal dental occlusion may be managed conservatively (soft-food ± tape muzzle). Tape muzzles are reported but not used by the author. Dental occlusion is the key consideration as there is little tolerance for inaccuracy due to the close relationship between the upper and lower dental arcades in cats. Direct fracture repair is preferred to indirect interarcuate stabilisation techniques due to rapid return to normal feline behaviours and reduced risks of asphyxiation. Up to 60-70% of fractures will be open, and due to the excellent blood supply and rapid healing most do not need antibiotics. Mouth closure during intraoperative fracture reduction is essential (unless just a mandibular symphyseal separation), and is achieved via pharyngostomy intubation. Mandibular symphyseal separations are effectively managed with a temporary cerclage wire, removed at 6 weeks. Mandibular body fractures can be managed by interfragmentary wires (suitable for non-comminuted fractures), plating with 1.3-1.5-2.0mm plates, free-form epoxy putty external fixators, or dental composite splinting. Mandibular Ramus fractures are challenging to repair. If the dental occlusion is appropriate and the mandible can be opened and closed, then analgesia and soft food are recommended without fixation. The Ramus Anatomical Plate (RAP) plate has improved options.

Management of split hard palate should be surgical to close any opening into the oronasal cavity and avoid oronasal fistulae. Hard-palate separations may be minimal with intact mucosa, which can be managed conservatively if the dental occlusion is ok. Mucosal split but no overt separation and distortion of the dental arcade/occlusion can be opposed with fine interrupted sutures (4-0 or 3-0 Vicryl). Palate separations and dental arcade distortions are best reduced and stabilised with a pin and tension band wire or dental bridging. Mucosal advancement flaps alone are not recommended as they do not correct dental mal-occlusion. Temporomandibular Joint (TMJ) injuries account for 50% of maxillofacial fractures. Minimally displaced fractures of the condyle or temporal bone can be conservatively managed if there is good dental function, with soft food for 4 weeks. Significantly displaced ones require dental occlusion restoration and interarcuate immobilisation for 2-3 weeks, or condylectomy to avoid ankylosis development. Simple TMJ luxations can usually be reduced closed using ‘the pencil manoever’. Interarcuate stabilisation holds the dental arcades relative to each other whilst early bone healing occurs, and discontinued early at 2-3 weeks for TMJ fractures, but longer for comminuted ramus fractures. An oesophagostomy feeding tube should be placed first. There are several methods including: Interarcade wiring, Maxillary–mandibular external skeletal fixation, Bignathic encircling and retaining devices (BEARD) and Dental splints. Wherever possible interarcuate stabilisation should be avoided due to the high morbidity and mortality associated with acute obstruction.

In general, a non-oral feeding immediately after surgery can be helpful and oesophagostomy feeding tubes are straightforward to place (prior to interarcuate stabilisation), and well tolerated in cats.

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ABDOMINAL TRAUMA: AN ECC APPROACH

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Learning objectives:

- Recognise injuries associated with abdominal trauma

- Appraise clinical findings & perform secondary diagnostic tests

- Formulate a plan for emergency stabilisation

- Recognise indications for emergency surgery

Proceeding:

The most common abdominal injuries in dogs and cats sustaining abdominal injury are hemoperitoneum, abdominal wall rupture and urinary tract rupture (Muir, et al 2006). All of these injuries can lead to both immediate and delayed life-threatening systemic complications including hypovolaemic, distributive shock, systemic inflammatory response syndrome (SIRS) and multiple organ dysfunction (MODS). Often in cases of polytrauma there is often also respiratory and neurological derangements due to thoracic and/or head and spinal trauma occurring the alongside abdominal trauma. Care should be taken to provide oxygen supplementation, provide adequate analgesia and minimise movement during the emergency assessment.

Traumatic hemoperitoneum most commonly occurs secondary to blunt trauma and high-rise syndrome (Hoffberg et al, Lefman et al 2022) and is not always readily identifiable externally. Clinical findings that support the potential for intra-abdominal haemorrhage include signs compatible with shock; obtundation, pale mucous membranes, prolonged capillary refill time, tachycardia and presence of abdominal wall bruising. The secondary survey should include careful inspection of the ventral abdomen, including umbilicus and intrapelvic region for the presence of bruising.

Often patients with hemoperitoneum present hypovolaemic and therefore packed cell volume and total solids are often initially normal. It is important to interpret these values in light of the patients' blood pressure and lactate levels.

Abdominal point-of-care ultrasound has a particularly useful role in the detection of, and serial assessment of, abdominal fluid after a trauma (Boysen, et al 2004). An abdominal fluid scoring system which scores 4 targeted scanning sites as positive or negative for free fluid can help determine the severity of injury as well as monitor progression. Abdominal fluid scores of 3 or 4 have been associated with more severe injury and lower PCV/TP. In one study of dogs who had sustained blunt abdominal trauma 22% of dogs that had serial

scans subsequently developed effusion despite having a negative fluid score at the initial timepoint (Lisciandro, et al 2009).

Other than hemoperitoneum, septic peritonitis and chemical peritonitis (urine or bile) can occur after trauma. Abdominocentesis should be performed, where possible in all animals, unless there is a contraindication to sampling such as severe thrombocytopaenia or coagulopathy. Analysis of fluid should include PCV/TS, cytology, and the exact biochemical analysis (creatinine, potassium bilirubin, bile acids) determined by the clinical presentation and the patient's blood test results (Schmiedt, et al 2001; Pasucal et al 2024). Bile peritonitis and septic peritonitis can occur hours-days after trauma. It is therefore important to monitor these animals closely even if they are stable on initial presentation (Murgia, 2013; Rollings et al 2011).

The presence of severe abdominal pain should alert you to the possibility of septic or chemical peritonitis, intestinal entrapment, or presence of traumatic pancreatitis. Careful palpation for the presence or absence of a bladder and interrogation the inguinal region is required. A high number of animals with uroperitoneum can urinate or have a palpable bladder and therefore the presence of a bladder in isolation cannot be used to rule out a uroperitoneum (Aumann, et al 1998).

Emergency stabilisation of abdominal trauma include management of hypovolaemia, analgesia and provision of antibiotics in the presence of a penetrating abdominal wound or septic peritonitis. Fluid resuscitation should be initiated quickly after presentation in a patient with signs of hypovolaemic shock. Most patients with small volume haemorrhage stabilise with isotonic crystalloid bolus. However, a small percentage of patients may require resuscitation with blood products. A trauma-associated severe haemorrhage (TASH) score, compromised of sex, haemoglobin concentration, systolic blood pressure, abdominal effusion, heart rate, base excess and presence of pelvic/femoral fracture was found to be predictor of transfusion requirement in traumatised dogs. The presence of abdominal effusion was particularly associated with need for a transfusion. (Delgado, et al 2024).Careful application of an abdominal bandage may stop or slow haemorrhage, however, animals should be closely monitored whilst the wrap is in place, and they should be avoided if there is evidence of diaphragmatic rupture or hypoventilation.

Rarely do traumatic hemoperitoneum requires surgical intervention. Indications for surgical intervention include failure to achieve haemodynamic stabilisation, evidence of an increasing abdominal fluid score and dropping PCV/TS. Traumatic uroperitoneum require surgical intervention but this does not need to an emergency procedure so long as urinary diversion can be established with the placement of a urinary catheter and/or abdominal drain. Indication for emergency surgery include a penetrating body wall rupture with incarcerated viscus. When preparing a patient for surgery pre-operative stabilisation is key.

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ABDOMINAL TRAUMA: DIAGNOSTIC IMAGING

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Learning objectives:

- Improve radiographic technique and develop a systematic approach to assessment of abdominal radiographs.

- Be familiar with radiographic features of common traumatic abdominal lesions, including pneumoperitoneum, peritoneal effusion, abdominal wall trauma and urinary tract trauma.

- Understand the importance of clinical assessment in contextualising radiographic findings.

Proceeding:

Good radiographic technique maximises the benefit of abdominal radiography and aids image interpretation. Ensure a complete study is performed to include three views (ventrodorsal (VD), right and left lateral), where possible. A VD projection is particularly important to maximise abdominal volume and aid demarcation of intra-abdominal structures. Patients should be well positioned, and collimation should include the entirety of the abdominal cavity. Acquisition should be performed in the expiratory phase to further maximise abdominal volume. Be systematic when learning to read abdominal radiographs – developing a repeatable technique will ensure improved lesion detection, which is particularly pertinent in cases with multiple lesions e.g. polytrauma patients.

Peritoneal effusion is a frequent non-specific finding in abdominal trauma. Haemoabdomen of variable clinical significance following vascular or parenchymal trauma is commonly identified, but alternative significant causes of effusion should be considered where appropriate e.g. uroabdomen, bile peritonitis, or septic peritonitis. Radiographic features of peritoneal effusion include reduction in serosal detail, wispy opacification of peritoneal fat, and distension of the abdominal contour. Diagnostic abdominocentesis should be pursued where indicated to further characterise peritoneal effusion in the trauma patient.

Pneumoperitoneum is an important radiographic finding in trauma, frequently indicating surgical disease due to hollow viscus rupture or intra-abdominal penetration. Features of pneumoperitoneum include gas bubbles conforming to abdominal visceral margins, or the presence of irregular bubbles that are clearly extraluminal e.g. within peritoneal fluid. Identification of pneumoperitoneum can be challenging, with very small volumes easily missed or larger coalescing gas bubbles being mistaken for intraluminal

gastrointestinal content. Superimposition of cutaneous gas in penetrating trauma can further confound interpretation.

When diagnosis of pneumoperitoneum is equivocal, complementary imaging techniques are useful. Horizontal beam radiography is a sensitive tool which can highlight non-dependent peritoneal gas by removing visceral superimposition. This technique is effective with the patient placed in left lateral or dorsoventral recumbency. Note: right lateral recumbency should be avoided as gas within the gastric fundus can confuse interpretation. Focused abdominal ultrasound can also detect small volumes of gas (0.4ml) within the peritoneal cavity.

Urinary tract trauma is a commonly encountered clinical scenario. Upper urinary tract injury (e.g. to the kidneys or ureters), although less common, may be suspected radiographically if there is a reduction in retroperitoneal serosal detail or retroperitoneal mass effect. Further interrogation of the upper urinary tract with intravenous excretory urography (IVU) is indicated where suspected. Radiographic and computed tomographic techniques are available to assess for leakage of contrast agent from the upper urinary tract, with CT techniques allowing more comprehensive trauma assessment. Sonographic localisation of upper urinary tract trauma is challenging, however ultrasound guided sampling of retroperitoneal fluid can prove informative. Features of lower urinary tract trauma e.g. following urinary bladder or urethral injury include peritoneal effusion and soft tissue thickening of the caudal abdominal and perineal structures. Positive contrast retrograde urethrocystography is an affordable and effective technique for diagnosing lower urinary tract injury and rupture.

Assessment for abdominal wall injury should form part of the systematic review in trauma cases. Traumatic radiographic lesions of the abdominal wall include thickening, heterogeneity or gas accumulation within the abdominal wall; discontinuity or disruption of muscle bellies; avulsion fractures (e.g. of the prepubic tendon / pubis); and abdominal visceral displacement / incarceration. Radiography can underestimate the extent of abdominal wall disruption, and additional imaging techniques (e.g. CT or ultrasound) can facilitate further assessment of regions of concern. Where abdominal visceral displacement is observed, evaluation for signs of strangulation of involved organs is essential. Segmental distension of herniated intestinal loops raises concern for vascular compromise, warranting prompt surgical intervention.

Diagnostic imaging is an excellent screening tool for traumatic pathology but should not precede or replace thorough clinical assessment. Abnormalities identified when performing extensive radiographic surveys can be challenging to assign significance to without complete historical and physical examination data to provide context. Similarly, more subtle lesions of clinical significance may be missed or incompletely included in the imaging examination if clinical evaluation is not performed in advance. Radiographic series should be limited to regions of priority and clinical relevance where possible and be utilised to answer specific clinical questions which may guide treatment planning and aid prognostication in the trauma patient.

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ABDOMINAL TRAUMA: SURGERY

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Learning objectives:

- Be able to approach cases of body wall rupture and grasp vital concepts for surgical repair.

- Appreciate potential challenges in repair of body wall ruptures.

- Be familiar with the surgical technique for managing bladder rupture.

Proceeding:

Traumatic abdominal hernias are classified according to their location, such as pre-pubic, para-costal and dorsal. They are often due to bite wounds or blunt trauma. Herniated content can form adhesions, but lack a true hernia sac. Ideal timing of hernia surgery has not been determined and the clinician must balance patient stability and tissue viability with the risk of organ incarceration/strangulation. Emergent surgery is indicated in patients with incarceration/strangulation or those deteriorating rapidly due to the hernia.

The aim of surgery is to re-establish continuity of the abdominal wall. Typically tissues can be re-apposed in an anatomical manner, and sutured using slowly absorbable suture material. In pre-pubic hernias, the tissue has typically avulsed from the pubis bone. Repair necessitates drilling bone tunnels through to pubis to allow suture anchoring. The pre-pubic tendon takes one year to re-gain 80% of it's original strength. For this reason some authors advocate for the use of non-absorbable suture material in these cases but its use must be considered alongside the individual patient's risk for infection. In very large or chronic defects, direct apposition of body wall tissues may not be possible. The clinician should be mindful of alternative methods of body wall reconstruction, such as the use of local muscle flaps or surgical mesh. Survival is reported to be between 73-100%**.**

In animals with urinary tract injury, it is important to note that they are often still able to pass urine. As such, visualising a urine stream does not preclude urinary tract injury or uroabdomen. Likewise, the presence of a palpable bladder does not rule out bladder rupture. In cases of bladder rupture, urgent cytological evaluation of abdominal fluid is necessary in order to determine the presence/absence of bacteria. Provided the fluid is sterile, uroabdomen patients can typically tolerate a period of stabilisation including placement of an abdominal drain and urethral catheter.

Once the patient is stabilised, a caudal midline coeliotomy approach is made to the bladder. The site of rupture is identified – often omentum will be adherent and may obscure the tissue. Necrotic tissue is

debrided until healthy, viable bladder tissue is encountered. In people it is described that 75% of the bladder can be resected without long-term implications for bladder function. This degree of resection has been reported in dogs with bladder neoplasia, although persistent pollakiuria was noted in some. It is essential to preserve the trigone region of the bladder as this is the insertion site of the ureters as well as the origin of mucosal regeneration in the bladder (from the terminal ureters and urethra). Following debridement the bladder is closed in a single layer, using a simple continuous or simple interrupted appositional suture pattern. Absorbable monofilament sutures such as polydioxanone (PDS) or poliglecaprone 25 (Monocryl) are generally appropriate, although it should be borne in mind that their degradation is variably affected by the presence of bacteria within the bladder. The bladder heals very rapidly, regaining 100% of its tissue strength within 14-21 days.

The most frequent post-operative complication is leakage of urine, although the reported rate is very low at 1.4% of cases. Survival to discharge is reported to be around 74-79%**.**

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'BACK-END' FELINE TRAUMA: PELVIS, BLADDER, NERVES AND TAIL

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Learning objectives:

- Outline regional pelvic anatomy, lower urinary tract and innervation of the bladder.

- Examination, diagnostics (including contrast studies) and prognosticating in these cases.

- List management options for both primary injury (including indications & contraindications) & secondary neurological deficits, including tube-cystostomy.

- Describe common pelvic fracture configurations and outline options for pelvic fracture management and fixation.

Proceeding:

Cats frequently present non-ambulatory and the majority have injuries beyond the pelvic fractures (30% thoracic trauma, 20% blood loss requiring transfusion, 23% neurological dysfunction and Urinary tract damage, (usually bladder or urethra) is not infrequently seen. Following a Major Body Systems Assessment with minimum database & stabilisation, early assessment hindlimb deep pain (note limb withdrawal without a 'cranial' response to a toe pinch is a spinal reflex only), anal and perineal reflexes prior to opioids is prognostically valuable. Nerve damage may be present as hindlimb dysfunction or 'tail-pull' syndrome. Observe whether able to stand unsupported or ambulate, and gently palpate the dorsal aspects of the pelvis for symmetry between the two ilial wings, ischial tuberosities and hip greater trochanters. Palpate the body wall for ruptures and determine if there is a palpable bladder. Other orthopaedic trauma in particular femoral head/neck fractures or hip luxation may also be seen.

A 'Tail Pull Injury", or sacrocaudal luxation, relates to the pattern of neurological signs post traction injury to the tail. Dysfunction is characterised by which nerve roots are damaged, and prognosis depends on severity of damage. A non-functional/flaccid tail may be present, and they may have a large difficult to express 'upper motor neuron bladder', or a soft easy to express 'lower motor neuron bladder'. If anal tone is decreased, it is likely that urinary incontinence will also be present. Assessment should include Pudendal nerve function by anal sphincter tone, bulbocavernosus reflex, and perineal reflexes. Urethral sphincter tone is determined by manual bladder expression. Tail function by tail deep pain response. The presence of an intact anal reflex is a hopeful prognostic sign for return of function. In addition, intact pain sensation (<5cm tail base in first 48hours) is strongly predictive of early return of bladder control. However, 60% of cats without tail base sensation may still go on to recover. Tail function may take several months or longer

to improve – and the question to amputate remains one of opinion. Appropriate bladder management may include daily manual expression in the short term only; indwelling catheterisation for a maximum of 5 days and tube cystostomy - which allows stress-free management as cats tolerate them well for several months, and most owners are happy to manage them. Medical Treatment for urethral spasm would include prazosin or phenoxybenzamine. To encourage bladder emptying, bethanechol can be administered. Diazepam (a striated muscle relaxant) should NOT be used in cats (hepatocellular necrosis).

Diagnostic assessment of orthopaedic injuries follows. Orthogonal lateral and ventrodorsal radiographs (frog-leg and extended) under sedation/GA are often sufficient, as although 60% of fractures are re-classified when CT is performed, in particular for acetabular and sacral, the general indication for management is unchanged. If urogenital trauma is suspected (caudal bruising, free abdominal fluid, biochemical changes) then retrograde urethrocystogram should be performed. Historically, feline pelvic fractures were commonly managed conservatively, however, surgical management is now considered most appropriate where there is pelvic canal narrowing, disruption of the weight-bearing axis (acetabular, ilial or sacroiliac luxations), nerve impingement/intractable pain, inability to ambulate within a few days of injury, or bilateral/concomitant orthopaedic injuries.

Surgery for sacroiliac luxation is recommended for bilateral SI luxation, significantly displaced unilateral, pelvic canal narrowing >45%, or mildly displaced unilateral but in association with significant contralateral 'hemi-pelvis' trauma. Sacroiliac screw placement can be performed open, with or without intra-operative radiography or closed. Most cats require a 2.4 screw which needs to cover 60% sacral width to reduce loosening. Transilial pinning is particularly useful as a low-risk, quick method to stabilise a unilateral SI luxation, or to augment a repair, typically with a 1.6mm pin. Ilial fractures are usually repaired with a 2.0 or 2.4 plate and screws, either laterally (gluteal roll-up), dorsally or orthogonal (gluteal roll-down); and locking screws in the sacral wing are preferred. ESF is also reported. Acetabular fractures are articular and require accurate reduction and rigid stabilisation. Historically, it was suggested that the caudal third of the acetabulum was non-load-bearing and therefore did not need repair. However, biomechanical evaluation of the feline acetabulum demonstrated that weightbearing was focused over the central and caudal thirds. Acetabular fractures are best approached from a dorsolateral approach via a gluteal tenotomy or trochanteric osteotomy. Repair methods include screws-wire-PMMA, straight DCP/LCP, and acetabular plates (most suitable for mid-acetabular fractures only). If severely comminuted and non-repairable, then a femoral head osteotomy or delayed total hip replacement could be considered. Most ischial and pubic fractures do not require repair.

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Advanced Stream, Thursday 5 June 2025

THYROID EMERGENCIES IN SMALL ANIMALS

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Learning objectives:

- Explain the pathophysiology of hypothyroid and hyperthyroid emergencies in cats and dogs.

- Identify the clinical manifestations and risk factors associated with thyroid emergencies in veterinary patients.

- Interpret endocrine test results to accurately diagnose thyroid emergencies.

- Describe pharmacological and supportive care strategies for stabilizing patients with thyroid emergencies.

- Develop a tailored management plan that addresses clinical signs and concurrent conditions contributing to thyroid emergencies.

Proceeding:

Thyroid emergencies, though relatively uncommon, are critical endocrine disorders that require prompt recognition and intervention This lecture will present the pathophysiology, clinical presentation, diagnostic criteria, and therapeutic management of these thyroid emergencies.

Hypothyroid crisis

Myxoedema coma is the classic example of a hypothyroid crisis. This terminology is somewhat misleading as most patients do not present with coma, but may present with an array of clinical signs which include weakness, bradycardia, hypoventilation, hypothermia, hypotension, altered mentation, and thickened subcutaneous tissues or pitting oedema. In dogs, this typically results from untreated primary hypothyroidism, and a concurrent physiological stressor or administration of a thyroid interfering medication. Contributing factors include an infection, anaesthesia, prolonged hypothyroidism, and certain medications such as glucocorticoids and possibly diuretics.[1,2] The clinical presentation and any precipitations factors will increase the suspicion of myxoedema coma in dogs while a 'diagnostic scoring system for myxoedema coma' has been developed in human medicine, which has a reported 100% sensitivity and 85% specificity.[3] Routine diagnostic tests will commonly identify abnormalities which are suggestive of hypothyroidism, such as hyperlipidaemia and increased liver enzyme activities, and a proportion of dogs will present with a non-regenerative anaemia. Definitive diagnosis is achieved with thyroid test results consistent with hypothyroidism (decreased total and or free thyroxine with increased TSH) and response to levothyroxine supplementation. As the presentation of a patient with

hypoadrenocorticism may overlap with many of the clinical signs observed in myxoedema coma, excluding hypoadrenocorticism before starting levothyroxine treatment may be warranted in a subset of patient. Treatment requires administration of thyroid hormone supplementation, cardiovascular, pulmonary and neurologic support and management of any concurrent conditions or withdrawal of any thyroid hormone interfering medications if this is possible. The thyroid hormone supplement of choice is levothyroxine given as 20 mcg/kg q12h, which is administered either intravenously or orally, or if necessary, per rectum. Hypothyroidism induced decreased cardiac contractility can result in congestive heart failure, and this cohort of patients should receive a lower starting dose of levothyroxine which is then gradually titrated to the starting dose of a few days. The mortality rate in humans is around 30% despite condition recognition and appropriate treatment.[4] The mortality rate in dogs is not well described in dogs.

Thyroid Storm

In contrast to hypothyroid emergencies, thyroid storm is a hypermetabolic state triggered by stressors such as infections, surgery, anaesthesia or treatment with iodine containing medications. Excessive circulating thyroid hormones lead to heightened sympathetic nervous system activity, causing tachycardia, arrhythmias, hypertension, hyperthermia, thromboembolic events and multi-organ dysfunction. Whether thyroid storm occurs in cats and dogs is a topic of debate, but there are certainly cases which have been described or the author has been involved in which parallel the condition in humans. A commonly cited article on this topic describes the 'Diagnostic criteria for thyroid storm in cats', which include hyperthermia (temperature >40^{o}C), and varying central nervous system, neuromuscular, cardiovascular, and gastrointestinal clinical signs with blood test results consistent with hepatic dysfunction.[5] Treatment includes decreasing the circulating thyroid hormone concentration which is typically achieved using thyroperoxidase inhibitor medications (methimazole / carbimazole), inhibiting the conversion of T4 to T3 using glucocorticoids, inhibiting the activated sympathetic nervous system using beta blocker medications, and management of concurrent conditions such as hyperthermia and arrythmias and management of any electrolyte and blood glucose derangements which have developed. Removal of any precipitating factors should also be attempted. Humans with thyroid storm may also be prescribed inorganic iodide which can reduce further thyroid hormone, but this treatment is not well described in cats. As with myxoedema coma, the survival rate is not well described in veterinary medicine but the incidence in human medicine has been reported 0.20 per 100,000 per year, with a mortality rate of around 10%.[6]

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MANAGEMENT OF PHEOCHROMOCYTOMA

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Learning objectives:

Following this lecture attendees should be able to:

- Describe the pathophysiology and clinical presentation of pheochromocytoma in dogs

- Identify common clinical signs associated with pheochromocytoma and outline the diagnostic approach for pheochromocytoma

- Explain the principles of the medical management of pheochromocytoma

- Compare surgical and medical management options for dogs with pheochromocytoma.

Proceeding:

Pheochromocytoma is an adrenal medulla tumour arising from chromaffin cells and characterised by excessive catecholamine secretion. This tumour type appears to be uncommonly diagnosed, with 255 cases being recorded in 26 centres over an 11 year period.

The overproduction of epinephrine and norepinephrine results in a variety of cardiovascular and systemic complications, and the clinical presentation of dogs with pheochromocytoma can be highly variable due to the episodic nature of catecholamine release. Common clinical signs include episodic weakness, panting, tachypnoea, collapse, hypertension, tachyarrhythmias, and systemic hypertension. Pheochromocytomas are also diagnosed following investigations for an adrenal mass identified during abdominal imaging.

The diagnostic approach involves a combination of adrenal imaging and endocrine testing. Adrenal masses may be detected via abdominal ultrasound or computed tomography (CT), but distinguishing pheochromocytoma from other adrenal tumors, such as adrenocortical tumors can be challenging. Endocrine testing plays a critical role, with the measurement of plasma or urinary normetanephrines (metabolic byproducts of catecholamines) being the most reliable diagnostic tool. Liquid chromatography-tandem mass spectrometry (LC-MS/MS) is preferred for its sensitivity and specificity.

Surgical adrenalectomy remains the treatment of choice for pheochromocytoma in dogs, but it carries significant perioperative risks due to the potential for catecholamine surges during anaesthesia and manipulation of the tumour. The benefits of preoperative alpha-adrenergic blockade remain a topic of debate, as the existing veterinary literature presents conflicting evidence on its efficacy. [1–4] The

administration of an alpha blocker, typically phenoxybenzamine for at least 10-14 days before surgery is commonly recommended in human medicine, with a key difference between human and veterinary medicine being the dose of these medications are titrated to effect to control hypertension. [5,6] Beta-blockers, such as atenolol or propranolol, may be added to control tachycardia, but only after adequate alpha-blockade has been achieved to prevent unopposed alpha-mediated vasoconstriction, which could lead to life-threatening hypertension. Intravenous fluids are also recommended to ensure normovolemia before surgery and normovolaemia should be achieved before prescription of beta blockade.

Intraoperatively, careful anaesthetic management is required to mitigate hemodynamic instability. Sudden surges in catecholamine release can result in severe hypertension and tachyarrhythmias. Agents such as phentolamine, nitroprusside, and calcium channel blockers can be used to control hypertension, while lidocaine or amiodarone may be needed for ventricular tachyarrhythmias. Postoperatively, patients must be closely monitored for hypotension, evidence of acute kidney injury, pancreatitis and DIC. Despite the challenges of anaesthesia, surgery and post-operative management, pheochromocytoma excision remains the best option for long-term survival (publication in review).

For dogs that are not surgical candidates or those with inoperable tumors, medical management focuses on controlling hypertension and clinical symptoms. Alpha-blockade remains the cornerstone of therapy, with additional options including calcium channel blockers or angiotensin-converting enzyme (ACE) inhibitors for refractory hypertension. In cases where tumour burden is high or metastases are present, chemotherapy options remain limited, though emerging treatments, including tyrosine kinase inhibitors, may provide future avenues for medical management. Tumour radiotherapy has also been described.

Given the complexity of pheochromocytoma in dogs, a multidisciplinary approach is critical for optimal patient management. Collaboration between soft tissue surgeons, internists, anaesthetists, and critical care specialists enhances the chances of successful treatment.

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STEROIDS IN CRITICAL ILLNESS

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Learning objectives:

- Explain how the HPA axis is affected in critical illness.
- Define critical illness related corticosteroid insufficiency.
- Recognise clinical scenarios in critical illness in which the use of steroids may be beneficial (eg. septic shock, and acute respiratory distress syndrome).

Proceeding:

The use of steroids in acute and critical illness is experiencing a resurgence with evidence of benefit in human patients with septic shock, acute respiratory distress syndrome (ARDS), and community acquired pneumonia. This lecture will review steroid physiology and pathophysiology, including critical illness related corticosteroid insufficiency (CIRCI). Veterinary and recent human literature will be reviewed and suggestions made to guide clinical decision making.

Regulation of cortisol product by the HPA axis in health and disease

Cortisol is produced by the adrenal glands in small amounts in health and in larger amounts in times of physiologic stress.[1] The hypothalamus produces corticotropin releasing hormone (CRH), which stimulates the anterior pituitary gland to release ACTH (also known as corticotropin). Arginine vasopressin (AVP) from the hypothalamus also contributes to the synthesis and release of ACTH from the pituitary into the blood. Circulating ACTH stimulates the zona fasciculata and zona reticularis of the adrenal gland to produce and release cortisol. In health, cortisol has negative feedback on both the hypothalamic release of CRH and the pituitary release of ACTH. Thus when circulating cortisol is low, CRH and ACTH will increase, stimulating the adrenal glands to produce more cortisol. The increased serum cortisol then inhibits the release of more CRH and ACTH.

Binding of pathogen and/or damage associated molecular patterns (PAMPS and DAMPS) to pattern recognition receptors of the innate immune response can also stimulate the HPA axis at multiple levels.[1] DAMPS and PAMPS not only result in the stimulation of the hypothalamus and pituitary directly, but also the autonomic nervous system (ANS). Through this mechanism norepinephrine is produced predominantly in the locus coeruleus which goes on to stimulate CRH synthesis in the hypothalamus. During stress, glucocorticoid synthesis can also be stimulated by ACTH-independent mechanisms via toll-like receptors

(particularly TLR-2 and TLR-4) in the adrenal cortex itself. Additionally, pro-inflammatory cytokines (eg. tumor necrosis factor-a, interleukin-1b) and their receptors are also present in the adrenal glands.

Cortisol metabolism in health and disease

Cortisol is produced from cholesterol stored in lipid droplets as cholesterol esters. Minimal cortisol is stored and thus increased cortisol production during stress relies almost entirely on cortisol synthesis. Circulating cortisol exists in three forms (i) 80-90% bound to cortisol binding protein (CBG, high affinity binding), (ii) 10-15% bound to other proteins such as albumin (low affinity binding), and (iii) the remainder as free cortisol (ie. unbound). The process of CBG binding is saturable, so when there are high circulating concentrations of cortisol, CBG is saturated and there is an increased proportion bound to albumin or free in the plasma. Additionally, since CBG and albumin are negative acute phase proteins, the fraction of free cortisol may increase as these proteins decrease. Cortisol binding globulin can also be cleaved by neutrophil elastases, also reducing its availability during inflammatory states. The net effect of many of these changes is that in the stressed state there is an increase in circulating free cortisol.[2]

Interestingly a recent study in critically ill dogs by Dr Boag and colleagues, published in the journal Domestic Animal Endocrinology, revealed that dogs with sepsis had higher total, bound, and free cortisol concentrations, compared to healthy dogs and dogs with septic shock had higher concentrations of all cortisol fractions than those with sepsis alone.[3] This was with the exception of two dogs that had unusually low total cortisol. Another study, by Sweeney and colleagues, in which dog adrenal function was assessed in an experimental ICU setting, also identified increased total and free cortisol concentrations.[4]

Cortisol signalling

Due to its lipophilic nature cortisol can enter cells passively, and does not require cell surface receptors for entry. In the cytoplasm cortisol binds to one of two types of glucocorticoid receptors; the type 1 receptor, also called the mineralocorticoid receptor (MR), or type 2 receptors, also called glucocorticoid receptors (GRs).[1] Despite their names both receptors can bind both aldosterone and cortisol. The MRs bind to their ligands with high affinity, and are important for signalling during low cortisol concentrations (ie. in health). In contrast, GRs have lower affinity for these ligands but are more important during stress and inflammation. GRa is the most abundant and well-studied GR isoform.

In the resting state, GRs exist in a multiprotein complex along with chaperone proteins, heat shock proteins, and immunoglobulins in the cell cytoplasm. When cortisol binds the GR it undergoes a conformational change, allowing it to dissociate from the other proteins and enter the nucleus or mitochondria. In the nucleus, the glucocorticoid + GR complex binds to glucocorticoid response elements (GREs) in the DNA, the end result of which is determined by the specific GRE (these are called genomic effects). If the GRE is in the promoter region of a target gene the result will be the recruitment of transcription factors and increased transcription of those genes; this is a process called transactivation. In contrast, binding of other GREs (often termed “negative GREs”) to their ligands can down-regulate the action of transcription factors, in a process referred to as transrepression. Glucocorticoids also mediate inflammation through non-genomic effects such as inhibition of phospholipase A2 and subsequently the arachidonic acid cascade.

Pathophysiology of CIRCI

While our understanding of the pathophysiology of CIRCI is evolving, it remains incompletely understood. At the most basic level CIRCI describes impairment of the HPA axis during critical illness, however its aetiology is undoubtedly a complex combination of altered hypothalamic, pituitary, adrenal, hormonal, enzymatic and receptor function that likely varies from patient to patient. Three major mechanisms of CIRCI are recognised (i) decreased cortisol production (ii) alteration of cortisol metabolism, and (iii) target tissue resistance to cortisol.[1, 5] Each is reviewed briefly.

Veterinary studies of CIRCI

Only a few studies have reported or evaluated CIRCI in veterinary medicine. Many were prior to coining of the term CIRCI, at a time when the term relative adrenal insufficiency (RAI) was used. Dr Burkitt and colleagues interrogated the HPA axis in 33 septic dogs by performing a single ACTH-stimulation test.[6] They identified RAI, based on low delta cortisol, in 48% of dogs. Additionally they documented that a delta-cortisol of ≤3ug/dL was associated with hypotension and death. Dr Martin and colleagues studied 31 critically ill dogs with sepsis, severe trauma, and GDV.[7] They measured baseline and ACTH stimulated cortisol, as well as ACTH concentrations. A similar proportion, 17/31 (55%) had HPA insufficiency, and dogs with a delta cortisol ≤3ug/dL were 5.7x more likely to require vasopressors.[7] The early veterinary literature also includes a case report of hydrocortisone responsive septic shock in a 15mo Standard poodle with aspiration pneumonia.[8] More research has investigated the role of hydrocortisone,[9] or dexamethasone,[10] for the treatment of CIRCI and septic shock in dogs.

There is also some evidence for CIRCI in cats. Dr Durkan and colleagues published a case report in JVECC in 2007, describing a diagnosis of RAI in a cat with SIRS secondary to trauma based on fluid-refractory hypotension, a blunted cortisol response to an ACTH stimulation test, and clinical improvement after initiation of glucocorticoid therapy.[11] A study of 20 critically ill cats and 10 control cats was also presented in abstract form in 2003. In this investigation, all sick cats had higher basal cortisol than control cats, but there was no difference in post-ACTH values between control and sick cat groups. As such, the authors concluded that there was no evidence of adrenal insufficiency in this population of critically ill cats during hospitalisation, at least based on ACTH stimulation testing.

Recommendations for Corticosteroid use in critical care conditions (in humans)

Guidelines for the diagnosis and management of CIRCI in critically ill human patients were published in 2017.[1, 5] A few of the recommendations from these guidelines are explained herein.

Corticosteroids in sepsis: The moral of the story here is that steroid use is recommended for septic shock, but not sepsis in the absence of shock. Their specific statements are “We suggest against using corticosteroid administration in adult patients with sepsis but without shock (conditional recommendation, moderate quality of evidence).” “We suggest using corticosteroids in patients with septic shock that is not responsive to fluid and moderate-to-high-dose vasopressor therapy (conditional recommendation, low quality of evidence)…If using corticosteroids for septic shock, we suggest using long course and low dose

(eg. IV hydrocortisone < 400mg/day for ≥3 days at full dose) rather than high-dose and short course in adult patients with septic shock."

Corticosteroids in ARDS: In ARDS, methylprednisolone is recommended as the drug of choice based on the available evidence, and because it has greater lung penetration and longer duration of action. "We suggest use of corticosteroids in patients with early moderate to severe ARDS (P/F < 200 and within 14 days of onset)." The recommended dose of methylprednisolone varied for early vs. late ARDS.

There is also growing evidence of a role for corticosteroids in the treatment of community acquired pneumonia in humans.[12]

Corticosteroids in major trauma: Although trauma has been associated with CIRCI, the guidelines suggest against the use of corticosteroids in major trauma.

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TIPS AND POTENTIAL PITFALLS IN DKA MANAGEMENT

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Learning objectives:

- Discuss the challenges in diagnosing DKA when that are multiple or mixed acid-base disorders or euglycaemia

- Discuss how to formulate a fluid therapy plan for a hypotensive DKA patient

- Discuss how to manage diabetic ketoacidosis patients with co-morbidities such as heart failure and chronic kidney disease

- Discuss the approach to supplementing potassium, phosphorus and magnesium

- Discuss when to consider antimicrobial therapy in the emergency DKA patient

- Discuss methods of administering insulin

Proceeding:

The principles of DKA management include restoration of circulatory volume, management of electrolyte abnormalities and insulin therapy to eliminate ketosis. This is alongside diagnostics and treatment for precipitating causes. Although theoretically straight forward, some patients still present diagnostic and therapeutic challenges.

The diagnosis of DKA can be complicated by mixed-acid base disorders and euglycaemic ketosis. For example, a transient proximal tubular dysfunction with bicarbonate loss can contribute to an acidosis while a co-existing metabolic alkalosis can mask it. Vomiting is likely the origin of a chloride-deplete metabolic alkalosis. Diuretics, hypomagnesaemia, severe hypokalaemia and hyperaldosteronism (adrenal disease or exogenous glucocorticoids) and the impact of hypoalbuminaemia can contribute to a non-chloride-deplete metabolic alkalosis. Euglycemic DKA can occur with a normal glucose, pH and bicarbonate.

Initial resuscitation fluid prescriptions are complicated by shock of multiple aetiologies. Most patients are severely volume depleted and inflammatory comorbidities are frequently seen due to immunosuppression or dysglycaemia. Severe acidosis itself will contribute to tachycardia and peripheral vasoconstriction. Most DKA patients will benefit from rapid intravascular resuscitation followed by high

ongoing rates to account for severe dehydration and ongoing osmotic diuresis. In paediatrics, care pathways suggest severe DKA should be associated with a predicted dehydration status of 10%.

There is no evidence in people for fluid superiority although balanced crystalloids may reduce the incidence of normal-anion gap metabolic acidosis following treatment. Although actual sodium should be used to calculate the anion gap, a high corrected sodium can highlight cases which may benefit from hypotonic fluid administration. High admission corrected sodium has also been associated with increased incidence and severity of acute kidney injury in people with DKA. Furthermore, concurrent cardiac or renal disease may prevent adequate sodium excretion and puts these patients at increased risk of volume overload. Focussed cardiac ultrasound and assessment of fluid tolerance may help guide fluid administration in high-risk patients. Increased concentrations of dextrose (up to 20% centrally) and other medications may eventually be required to limit fluid administration in these individuals.

The main electrolytes requiring management in DKA are potassium, phosphorus and magnesium. All are usually normal or increased at admission but decrease during treatment. Immediate supplementation of potassium is indicated unless hyperkalaemia (in people >5.5mmol/L) or renal compromise exists. Phosphorus is rarely empirically supplemented but monitoring is important to avoid a haemolytic crisis. Even if not measurable in practice, ionised hypomagnesaemia may be suspected in refractory hypokalaemia.

Infections, particularly urinary tract infections, are frequently reported in DKA. However, positive cytology may not be easy to obtain in all infections. High temperature, leukocytosis, bandaemia and other elevated biomarkers such as neutrophil/lymphocyte ratio, and C reactive protein have all been studied as triggers for empiric broad spectrum antimicrobials but evidence guiding usage is still lacking.

There is no evidence that any insulin protocol is superior. Benefits of short-acting insulin are the ability to titrate to affect. This is particularly useful in severe DKA with marked insulin where rates up to 0.2-0.3 IU/kg/h may be required. Insulin infusions are typically started once severe hypokalaemia has resolved.

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ANAPHYLAXIS: PATHOPHYSIOLOGY, DIAGNOSIS, AND TREATMENT

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Learning objectives:

- Describe the unique features of the pathogenesis of anaphylaxis in small animals

- Recognise case presentations suggestive of anaphylaxis

- Formulate a rational treatment approach to anaphylaxis based on the underlying pathophysiology

Proceeding:

Pathophysiology

Anaphylaxis is defined as a life-threatening acute systemic type 1 hypersensitivity reaction that results in multiple organ dysfunction. Anaphylaxis develops rapidly after exposure to a causative allergen, which is not always identified. The typical pathogenesis involves the release of potent inflammatory mediators 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. Cardiovascular dysfunction 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 pruritus. 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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ENVENOMATIONS

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Learning objectives:

- Recall the main venom components and their effects on the envenomed patient (cytotoxins, hemolysins, neurotoxins, myolysins, proteins affecting coagulation)

- Describe the diagnostic principles for snake envenomation

- Describe the role of antivenom in treatment of envenomation

- Recall the findings of recent research investigating small molecule inhibitors for snake venom components.

Proceeding:

Envenomation has the potential to cause a rapid and severe constellation of systemic consequences. This session, focusing on snake envenomation, highlights these consequences from both the perspective of diagnostic and therapeutic considerations. Several case examples will be presented to demonstrate typical clinical presentations and their management. Recent developments in the management of envenomation, including modern antivenoms and small molecule inhibitors of venom components, will be highlighted.

Epidemiology – a brief review

Snake envenomation is a world-wide problem and is classified as a neglected tropical disease in human medicine. The epidemiology of snake envenomation in veterinary medicine is poorly understood, but has recently been enhanced, at least in Australia by the SnakeMap initiative.[1] Literature on snake envenomation in dogs and cats has been published from the Americas,[2, 3] South Africa,[4] Scandinavia,[5] Israel,[6] South Korea,[7] Sri Lanka,[8] Nepal and Cameroon,[9] amongst other regions, describing a wide range of envenomation syndromes.

Venom components and envenomation syndromes

Snake venom is an incredibly complex biologic milieu containing well described protein families, including hemolysins, locally acting cytotoxins, neurotoxins, procoagulants, and myolysins, amongst others. The presence and abundance of different classes of venom proteins varies between snake families (Elapids vs. Vipers), snake species, and amongst individual snakes (based on diet, geography, age, sex etc.). While we often consider particular envenomation syndromes typical of particular

envenoming species, the significant interindividual venom variation means that almost anything is possible in an individual envenomed patient.

Nonetheless, an awareness of the pathology that can be caused by specific venom components is worthwhile. Hemolysins cause red blood cells lysis / hemolysis and may result in anaemia. Hemolysins may also cause erythrocyte cell membrane injury manifesting as echinocytosis on a blood smear. Cytotoxins cause local tissue damage resulting in pain, swelling and tissue damage at the bite site. Neurotoxins act at the neuromuscular junction (pre- and/or post-synaptic) accounting for the paralytic component of envenomation syndromes, that can be so severe in some cases so as to require mechanical ventilation (eg. coral snake Micrurus fulvius, and Australian elapid snakes). A variety of venom components can disrupt normal haemostasis. For example, procoagulant venom components cause venom induced consumptive coagulopathy (VICC), identified upon coagulation testing, which can cause clinical bleeding in some cases. Fulminant pulmonary haemorrhage has been associated with rapid death in Eastern brown snake (Pseudonaja textilis) envenomation in Australia.[10] Myolysins cause direct muscle damage and result in the release of myoglobin, resulting in myoglobinemia, myoglobinuria, and pose a risk for pigment nephropathy.

Diagnosing envenomation

Rapid diagnosis and initiation of treatment is vital to maximise the chances of a good outcome in cases of snake envenomation. Diagnosis generally involves a combination of consistent history, consistent clinical signs, and supportive laboratory test results.

Regarding a consistent history, dogs may be envenomated in their owners yard, or out on a walk. Sometimes envenomation is observed by dog owners, however most cat envenomation is not witnessed. It is thus important to understand what the dog or cat was doing, and where, immediately before the onset of clinical signs. Time of year will also be helpful, with most parts of the world having a snake envenomation “season”. Owners may observe their dog interacting with a snake or notice the onset of clinical signs after the dog has been outside. In parts of the world that are home to numerous venomous snakes, snake identification is important to inform prognostication and treatment but can be challenging.

Consistent clinical signs will depend on the envenoming species. In snake species containing cytotoxins, the bite site with surrounding tissue injury is usually easily identified, however a bite site is not identified in the majority of Elapid species envenomation.

Supportive laboratory tests will also be very dependent on the envenoming species. Envenomation by different snake species can result in abnormalities of complete blood count (eg. echinocytosis), biochemistry (eg. increased muscle enzymes, hypocholesterolemia), and coagulation tests (PT/aPTT, fibrinogen, and/or viscoelastic tests). In some countries, point-of-care tests can specifically detect snake venom in victim body fluids (eg. the snake venom detection kit in Australia), and research is ongoing world-wide to develop similar tests.

Treating snake envenomation

The mainstay of treatment of life-threatening envenomations is the prompt administration of antivenom, and attentive supportive care. In some regions of the world there is still debate over the value of antivenom administration, particularly in cases of less severe envenomation, or when the available antivenom is associated with frequent adverse effects. The development of safer and more effective antivenoms, such as F(ab')2 antivenoms, will undoubtedly improve uptake, and subsequently improve envenomation outcomes. In most parts of the world the ideal dose of antivenom also remains unknown, and further investigation in this field is warranted.

In addition to antivenom, treatment of snake envenomation will likely involve IV fluid therapy, plasma transfusion for clinically significant bleeding, analgesia (for pain associated with myolysis or local cytotoxicity), treatment of gastrointestinal signs, and good supportive care. For neurotoxic envenomation syndromes a proportion of patients also require intubation and ventilation, so recognising these patients early to initiate this life-saving intervention is vital.

Intubation and ventilation are required for dogs and cats that have significant respiratory distress as a result of respiratory muscle paralysis. Usually, the decision for intubation is made shortly after presentation purely based on the observed degree of respiratory distress or in cases that experience a respiratory arrest.[11] Nonetheless, measurement of $PvCO_2$ on a blood gas and documentation of hypercapnia ($PvCO_2$ >55-60mmHg) would be another indication. Additionally, some envenomed dogs and cats will be hypoxemic due to hypoventilation and/or aspiration. An SpO_2 < 93-95% or a PaO_2 < 60-80mmHg despite oxygen therapy would also warrant intubation and ventilation. The duration of respiratory support required seems to be very varied based on the reversibility of the neurotoxic venom components with antivenom. During ventilation good nursing care is vital.

Intravenous fluids are indicated for the treatment of hypovolemia, dehydration, and to provide for maintenance until the patient is able to eat and drink again. The main reason a snake envenomed dog or cat may be hypovolemic or dehydrated is due to fluid losses in vomiting, diarrhoea, and/or salivation. That being said, any fluid deficits are usually relatively mild or moderate. Balanced isotonic crystalloids (eg. LRS or Plasmalyte) would be the fluid of choice. Some clinicians use higher rates of fluid in cases with CK > 5,000 U/L with the goal of reducing the risk of kidney injury; however it is unclear whether or not this is necessary above simply ensuring that the patient is normovolemic and euhydrated. Nausea and vomiting should be treated with antiemetics, while regurgitation or ileus should be treated with prokinetics.

While laboratory defined coagulopathy is common, clinical bleeding occurs in a much smaller percentage of envenomed patients. Generally antivenom (and time) is all that is required, and the clotting times will return to normal (although rechecks are not necessary unless the patient is not responding the way that you would expect to treatment). There are however a small number of dogs (fewer cats) that develop clinically significant bleeding; either bleeding into critical locations (eg. lungs) or large volume bleeding. In these cases, plasma transfusion may be required to aid in the resolution of clinical hemorrhage.[10, 12]

Local tissue injury and myonecrosis that occurs due to the myotoxins are likely painful. As such, pain relief is generally indicated in affected patients. Non-steroidal anti-inflammatory drugs are

contraindicated given the risk of acute kidney injury, and as such opioid analgesics are the drugs of choice. Either buprenorphine 0.01-0.02 mg/kg IV q4-8h, or methadone 0.1-0.3mg/kg IV q4-6h would be appropriate.

Novel therapies?

Snake envenomation remains a significant cause of death and disability in humans worldwide, and thus there remains interest in novel therapies, particularly for use in austere environments. A recently investigated novel therapy that may prove useful in the treatment of neurotoxic snake envenomation in veterinary medicine is the drug varespladib (LY315920). This drug has in vitro potency against snake venom phospholipase A2 (sPLA2), preventing binding at the neuromuscular junction. An experimental study in pigs has demonstrated that delayed oral and IV administration of LY315920 rescues young pigs from the severe neurotoxicity associated with coral snake envenomation.[10]

Marimastat, a broad-spectrum matrix metalloproteinase inhibitor, has also shown promise at inhibiting snake venom induced cytotoxicity.[13] The future of treating snake envenomation will undoubtedly involve using small molecule inhibitors, in combination with antivenom, and supportive care.

Prognosis

Fortunately, with prompt recognition of envenomation, administration of antivenom, and good supportive care, the prognosis for snake envenomation in dogs and cats is excellent.

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SMOKE INHALATION

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Learning objectives:

- Describe the pathophysiology of smoke inhalation, with attention to the time course after exposure

- Propose appropriate diagnostic and therapeutic steps throughout the management of smoke inhalation

Proceeding:

Smoke inhalation causes a broad range of pathophysiologic consequences that culminate in decreased cellular adenosine triphosphate (ATP) production and organ dysfunction. Thus, a broad diagnostic and therapeutic approach is required in these cases.

Carbon monoxide and hydrogen cyanide

Smoke contains variable amounts of the toxic gases carbon monoxide (CO) and hydrogen cyanide (HCN). Fires with limited oxygen supply, primarily those in enclosed areas such as house fires, produce larger amounts of these gases. Carbon monoxide impairs cellular oxygen utilisation through multiple mechanisms. It binds to haemoglobin with high affinity, reducing the binding sites available for oxygen. CO-bound haemoglobin also has a left shift of the oxyhaemoglobin dissociation curve, reducing the ability for oxygen to be offloaded at the level of the tissues. Finally, it also binds to cytochrome-c oxidase in the mitochondria, leading to reduced mitochondrial function. CO inhalation should be assumed to be present in all cases of smoke inhalation, especially those in enclosed spaces, until definitively ruled out. Blood gas analysis with a co-oximeter, that provides a measurement of haemoglobin-bound CO, is necessary for diagnosis. Notably, the presence of CO renders pulse oximetry inaccurate, and the finding of a normal SpO_2 in a smoke inhalation patient should not lead to cessation of oxygen therapy. Provision of oxygen therapy with a high F_iO_2 is the cornerstone of treatment. As well as improving cellular oxygenation, this greatly decreases the half-life of CO. Oxygen support should be provided as early as possible, and can often be provided by first responders at the scene of the fire. CO toxicosis can also cause delayed neurological sequelae. HCN is another toxin that leads to impaired mitochondrial oxygen utilisation. Diagnosis is challenging, and sometimes must be assumed based on evidence of mitochondrial dysfunction, such as a reduced arteriovenous oxygen gradient, in the absence of another cause. Management requires detoxifying compounds such as amyl nitrate, sodium thiosulfate, or hydroxycobalamin.

Upper airway injury

Thermal and caustic injury can cause significant upper airway inflammation. This may cause obstruction, resulting in hypoventilation and increased work of breathing. This may be progressive over the first 24 hours. Classic clinical signs of upper airway obstruction include increased respiratory effort with a prolonged inspiratory phase and upper airway noise (stridor, stertor). Sedation and supplemental oxygen therapy may be sufficient in mild cases. Severe cases may require endotracheal intubation or surgical airway access. In an acute obstruction where endotracheal intubation is not possible, cricothyroidotomy is a faster approach to establishing an emergency surgical airway. Temporary tracheostomy may facilitate early extubation in cases with prolonged upper airway inflammation. Systemic corticosteroids are not recommended as they increase the risk of pulmonary infection. Nebulised corticosteroids and epinephrine may be considered.

Pulmonary injury

Thermal injury from superheated particulates and toxic injury both contribute to pulmonary inflammation. Subsequent increased pulmonary vascular permeability and pulmonary arterial vasodilation contribute to the formation of pulmonary oedema. Irritants may lead to bronchoconstriction. Lower airway obstruction can occur due to particulates, sloughed airway mucosa, and inflammatory exudate. The normal compensatory process of pulmonary hypoxic vasoconstriction is blunted by vasodilatory inflammatory mediators. These mechanisms result in widespread ventilation/perfusion mismatch, hypoxaemia, and may meet the criteria for the acute respiratory distress syndrome (ARDS). Initial therapy involves oxygen supplementation, and escalation to high-flow nasal oxygen or mechanical ventilation if clinically indicated based on refractory hypoxaemia, hypoventilation, or unsustainable work of breathing. Systemic or inhaled bronchodilators are indicated if there is evidence of bronchoconstriction. Saline nebulisation and chest physiotherapy can aid in clearance of lower airway obstruction. Secondary infection is common. Prophylactic antimicrobial therapy may select for a resistant infection. Thus, surveillance for evidence of infection is necessary. If suspected, airway sampling for culture and susceptibility should precede empiric institution of broad-spectrum antimicrobial therapy.

Other supportive care

Careful fluid management is required. Hypovolaemia will compound decreased oxygen delivery, and must be promptly recognised and treated. There may be increased evaporative fluid losses that increase fluid requirements. However, care should be taken to avoid excessive IV fluid therapy, as increased pulmonary capillary hydrostatic pressure leads to pulmonary oedema. The concurrent increased pulmonary vascular permeability caused by pulmonary inflammation magnifies this effect. Concurrent burns may be present, which require local therapy and may complicate fluid management due to increased fluid losses. Anxiolysis and analgesia are often necessary. Corneal injury is common and should be evaluated and treated.

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HEATSTROKE

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Learning objectives:

- List risk factors for heat stroke

- Describe the key pathophysiology and effects of heat stroke on different body systems

- Describe the treatment approach to heat stroke based on a body systems approach

- Understand the prognosis for heat stroke

- Recall the findings of recent research that improves our understanding of heatstroke in dogs

Proceeding:

Heat stroke is a potentially life-threatening condition experienced in dogs, as it is in people. Discussion of heat stroke is timely in this era of climate change, and increasing global temperatures.

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]

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]

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. Of course 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 petechiae or ecchymoses, hematemesis, and/or hematochezia are common. Some dogs have concurrent sunburn.[5]

Diagnostic approach

Blood should be collected for point-of-care venous blood gas and PCV/TS. If hypoglycemia is present this should be immediately treated with an IV bolus of glucose. 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 be used for volume expansion as it also aids in controlling hemorrhage that can occur 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 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 ICP that can occur due to cerebral edema. Seizures can be controlled with benzodiazepines (eg. midazolam), levetiracetam, phenobarbitone, and 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.[6]

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.

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.

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SHOCKINGLY SIMPLE: DIFFERENTIATING TYPES OF SHOCK

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Learning objectives:

- Define the factors that affect oxygen delivery

- Recognize how oxygen delivery is affected in each type of shock

- Differentiate hypovolemic, cardiogenic, and distributive shock

- Describe clinical signs and diagnostic findings associated with each type of shock

Proceeding:

Shock occurs when there is inadequate oxygen delivery to the tissues, resulting in hypoxia. Effective cardiac output and an adequate amount of oxygen in arterial blood are necessary for oxygen delivery. Cardiac output is the amount of blood the heart pumps in a minute, which is affected by heart rate and stroke volume. Stroke volume is impacted when changes occur in preload, afterload, or cardiac contractility. Arterial oxygen content can be altered through changes in hemoglobin concentration, percentage of hemoglobin saturated with oxygen, and the amount of dissolved oxygen. Identifying which parameters of oxygen delivery are affected in each type of shock will help in understanding why specific clinical signs are present and which treatment options are necessary to prevent further decompensation.

Shock can be classified into three major categories: hypovolemic, cardiogenic, and distributive. Hypovolemic shock occurs due to a decrease in circulating blood volume which can result from hemorrhage or severe dehydration. Oxygen delivery is ultimately affected in hypovolemic shock due to decreased preload resulting in decreased cardiac output. Recognition of hypovolemic shock may be made from the patient history if recent trauma occurred. Due to the decreased blood volume, clinical signs can include altered mentation, pale mucous membranes, prolonged capillary refill time, and cool extremities. Tachycardia or bradycardia may be noted depending on the phase of shock the patient is in, with bradycardia indicating the body is no longer compensating.

Point of care ultrasound has often been used during initial assessment to confirm hypovolemic shock due to the presence of fluid in the thorax or abdomen. Caudal Vena Cava Collapsibility Index (CVCCI), Left Atrium to Aorta ratio (La:Ao), and Left Ventricular End Diastolic Diameter (LVEDD) can be used to assess the volume status and the patient's response to treatment. In hypovolemic shock, CVCCI will be

increased and La:Ao and LVEDD will be decreased. The goal of treatment in hypovolemic shock is to improve oxygen delivery by restoring blood volume with intravenous fluid therapy and blood products.

Cardiogenic shock results from failure of the heart to pump adequately despite normal blood volume which can result from underlying heart disease or arrhythmias. Oxygen delivery is also affected due to decreased cardiac output; however, in cardiogenic shock this can result from increased preload and afterload. Clinical signs associated with cardiogenic shock can be similar to hypovolemic shock, including tachycardia, pale or muddy mucous membranes, and cold extremities. Cardiac and pulmonary auscultation may be helpful in identifying signs of a cardiac cause of shock.

Utilizing point of care ultrasound can be helpful as patients with cardiogenic shock will have a decreased or normal CVCCI and a normal or increased La:Ao and LVEDD. Administration of intravenous fluids in cases of cardiogenic shock could further worsen oxygen delivery, so it is imperative to ensure the type is identified prior to initiating treatments. Treatment of cardiogenic shock is focused on improving oxygen delivery by supplementing the cardiac function. This can include diuretics to remove excess volume, inotropic agents to improve contractility, and antiarrhythmics if arrhythmias are present.

Distributive shock occurs due to the body's inability to maintain vasoconstriction leading to systemic vasodilation, which can be seen with sepsis and anaphylaxis. Blood pressure is affected by cardiac output and systemic vascular resistance. Decreased systemic vascular resistance seen in distributive shock will ultimately result in decreased cardiac output which will decrease oxygen delivery. Vasodilation results in different clinical signs as compared to hypovolemic and cardiogenic shock including brick red mucous membranes, rapid capillary refill time, tachycardia, hypotension, and warm extremities.

On point of care ultrasound, CVCCI can be increased and the caudal vena cava can show excessive collapse due to inadequate diastolic pressure. Vasopressors can be used to promote vasoconstriction and restore oxygen delivery, the underlying cause of distributive shock is addressed. Intravenous fluid therapy can be beneficial, but must be monitored carefully to ensure the patient does not become fluid overloaded.

Veterinary nurses should be prepared for patients in shock to arrive in the emergency room and quickly respond to stabilize them. This requires developing a thorough knowledge of the pathophysiology of shock to be able to differentiate which type of shock is occurring and provide appropriate interventions.

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IT'S NOT JUST WATER - FLUID THERAPY AS A DRUG

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Learning objectives:

- Discuss why specific fluids are chosen for a patient

- Review the different reasons for providing fluid therapy

- Recognize how different disease processes require different fluid therapy plans.

- Identify the signs of fluid overload.

Proceeding:

Fluids are the most common treatment administered to hospitalized patients, but it can be easy to forget that they are a drug. Like other drugs, using fluids does carry risks and deciding which fluid to use for a patient needs to take into consideration the desired outcome, disease state and related sequela.

Fluid Balance

Living beings are made up of mostly water. 60% of body weight is from total body water (TBW). TBW is separated into intracellular fluids (ICF) and extracellular fluids (ECF) which are separated by the cell membrane. A simple way to remember the distribution of fluids in the body is the 60:40:20 rule – 60% of a patient's body weight is water, 40% of TBW is ICF and 40% is ECF. Tonicity is the ability of a solution to modify the volume of cells by altering their water content. Administered fluids move between compartments based on their tonicity.

Fluid Therapy

There are three main reasons a patient is given fluid therapy. First to correct hypovolemia (dehydration). Patients become dehydrated from a variety of conditions including trauma, vomiting, excessive urination and/or decreased ingestion. Monitoring perfusion parameters can help us to ensure our patients are appropriately hydrated. Second to improve perfusion by supporting cardiac output. Many critically ill patients are in some form of shock, where tissue and cellular oxygen delivery doesn't meet demand. Tissue oxygen delivery (DO2) depends on cardiac output (CO) and arterial oxygen content (CaO2) and is often shown as: DO2 = CO2 x CaO2. Shock is a life-threatening condition and the goal of fluid therapy is to restore intravascular volume and cardiac output to improve DO2 and relieve the shock state. Finally, fluid therapy is used to maintain hydration. This takes into account previous, ongoing and anticipated fluid losses to keep the body in a state of equilibrium.

Fluid Types

Crystalloids

Crystalloids contain solutes that can enter all body fluid compartments. (Lactated Ringers Solution, Hartmann's Solution, Plasmalyte) Most crystalloids are balanced solutions, meaning that they contain electrolyte concentrations that are similar to that of plasma (isotonic). These fluids are useful for replacing and maintaining hydration and are the most commonly used fluids in the veterinary hospital. Saline solution (0.9% sodium chloride) is sometimes called physiological saline, but it is an unbalanced solution that can cause metabolic acidosis if given in large amounts.

Colloids

Colloids contain larger molecular weight substances that stay in the intravascular space (plasma). They are more effective volume expanders than crystalloids and include blood products, albumin and synthetic colloids. Synthetic colloids have been shown to cause acute kidney injury and coagulopathies in humans and while studies are limited in veterinary medicine it is important0 to be aware of safety concerns when using these products.

Fluid Monitoring

Like other drugs, it is important to be aware of the side effects that come with fluid therapy. Monitoring perfusion parameters, mentation and weight can help prevent fluid overload. Signs of fluid overload include, weight gain of greater that 10% from baseline, tachypnea, clear nasal discharge, abnormal heart or lung sounds (gallop or crackles) and tissue edema. Confirming that the amount of fluid a patient is receiving matches the amount of fluid they are losing (ins and outs) is important to maintain appropriate hydration in our patients.

Conclusion

Fluid therapy should be treated with the same consideration as any other drug treatment we provide our patients. Target fluid therapy addresses the specific needs of each patient and diligent monitoring to ensure patient progress is necessary to avoid adverse effects.

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TURN ON THE LYTES! UNDERSTANDING ELECTROLYTES

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Learning objectives:

- Examine how electrolytes affect the balance of water in the body

- Discuss osmolality and tonicity

- Recognize disease processes that are commonly associated with electrolyte abnormalities

- Identify treatment plans and medications for patients with electrolyte imbalances

Proceeding:

Electrolytes are essential for maintaining homeostasis in the body. They play a vital role in fluid balance and acid base status, as well as regulating cellular, myocardial, musculoskeletal and neurologic function. The kidneys are the main regulator of water and electrolyte balance.

Fluid Balance

Electrolytes are osmotically active and affect fluid movement across cell membranes. Effective osmoles draw fluid in or out of the cell. Tonicity is the measurement of effective osmolarity. When water is pulled out of a cell into the extracellular fluid the solution is hypertonic, when water is pulled intracellularly the solution is hypotonic.

Sodium (Na+)

Sodium is the major extracellular cation. It maintains water hemostasis and cellular electroneutrality. Disorders are common in critically ill animals.

Hyponatremia

Hyponatremia (<140 mmol/L) occurs due to chronic disease or may be iatrogenic. Patients present with non-specific signs including lethargy and nausea. Neurologic signs are seen with severe hyponatremia. Treatment involves identifying and treating the underlying cause and correcting sodium deficits. Patients should be monitored closely to ensure appropriate hydration and mentation.

Hypernatremia

Hypernatremia (>155 mmol/L) is usually prevented by the normal physiologic mechanisms of the body. It is caused by excessive sodium gain, loss of pure water or loss of hypotonic fluids. Treatment focuses on correcting hypovolemia and treating the underlying cause.

Potassium (K+)

Potassium is the major intracellular cation. Normal levels are necessary for maintaining the proper resting potential of muscle cells, including the heart.

Hypokalemia

Many critically ill patients will develop hypokalemia at some point during their illness. Clinical signs can be seen at levels below 3mEq/L and are related to effects on cardiac and skeletal muscle. At levels less than 2mEQ/L rhabdomyolysis, the breakdown of muscle tissue, occurs. Acute renal failure can follow due to the toxic effects of myoglobin on the kidney. The goals of treatment are to treat the underlying cause and replace potassium deficits. When replacing deficits, it is important to note the maximum amount of potassium that can be supplemented without risking adverse reactions (KMax) is 0.5 mEq/kg/hr.

Hyperkalemia

The kidneys are usually efficient at maintaining potassium balance, but when there is renal dysfunction hyperkalemia can occur. Hyperkalemia (>5.5 mEq/L) causes changes in the cardiac conduction system, including bradycardia and abnormalities in ECG waveforms. Treating the underlying cause often resolves the hyperkalemia, but the patient may need diuresis or medications to achieve resolution.

Chloride (Cl-)

Chloride is the primary anion in extracellular fluid. It is evaluated by its relation to Na+. If there are changes in Na+ or fluid balance it is important to correct the chloride before assuming there is a disorder. The equation for this is: Cl (corrected) = Cl (measured) x Na (normal)/Na (measured).

Abnormalities

Hyperchloremia is often found when there is metabolic acidosis. Conversely, hypochloremia is found with metabolic alkalosis. Both abnormalities require treatment of the underlying disease and correction of blood gas irregularities.

Magnesium (Mg+)

Magnesium plays a pivotal role in the regulation of smooth muscle tone and signal transduction. Studies have shown that patients with magnesium disorders have increased morbidity and mortality.

Abnormalities

Approximately 54% of critical patients have hypomagnesemia (<1.85 mg/dL) from intracellular shifts in disease states, decreased intake and increased loss. Clinical signs will be related to signal transduction and muscle tone including arrhythmias and neuromuscular signs. At levels less than 1.5mg/dL

supplementation may be necessary. Hypermagnesemia is uncommon but can be found in patients with endocrine disorders or may be iatrogenic. In extreme cases respiratory depression will occur.

Phosphorus (P-)

Phosphorus is the most abundant intracellular anion and is a major segment of adenosine triphosphate (ATP), the energy source for all cells.

Abnormalities

Causes of hypophosphatemia (<2.5mg/dL) include decreased intestinal absorption, increased renal loss and transcellular shifts. Clinical signs are varied and affect high energy cells such as erythrocytes and skeletal muscle. Treatment most often includes supplementation. The most common cause of hyperphosphatemia is Tumor Lysis Syndrome. Symptomatic treatment is warranted and if severe a phosphate binder is needed.

Calcium (Ca+)

99% of calcium is found in bone but is poorly bioavailable. The 1% that is found extracellularly is only useable in its ionized form. Calcium is responsible for intracellular transport of ions, neuron excitability and is a coagulation factor (IV).

Abnormalities

Hypercalcemia is uncommon but life threatening. Excess calcium is toxic to cells and causes mineralization of tissues. Hypocalcemia is often seen in lactating mothers who have calcium intake that is insufficient to keep up with the demand of newborns. Clinical signs are acute and include seizures or tremors, lethargy and dyspnea. It is imperative to supplement with intravenous or oral calcium and no longer allow the babies to nurse.

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BALANCING FLUID THERAPY IN THE CARDIAC PATIENT

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Learning objectives:

- Understanding of the cardiac patient's circulation and cardiogenic shock
- Comprehension of pathophysiology of the renin-angiotensin-aldosterone system
- Overview of fluid overload and the risks this carries
- Provision of skills to monitor patients for fluid overload

Proceeding:

In a healthy patient, the need to maintain plasma volume, mean arterial blood pressure and tissue perfusion, drives normal circulation. Mean arterial blood pressure and plasma volume contribute to the cardiac output of the patient as well as the stroke volume and systemic vascular resistance, which are crucial to the central circulation. There are several physiological factors that will affect the systemic vascular resistance and cardiac output, many of which are visible within multiple heart diseases and congestive heart failure (CHF). Plasma volume plays a pivotal role in venous pressure and cardiac filling, with serum sodium concentration playing a pivotal role in determining the plasma volume. Various homeostatic mechanisms within the body, such as the renin-angiotensin-aldosterone system (RAAS) and hormones such as mineralocorticoids are factors in regulating sodium balance. The development of CHF will often concurrently come with sodium excretion abnormalities. Heart disease is characterised by cardiac dysfunction, cardiac structural remodelling and changes to preload and afterload in most patients. In patients with heart disease or CHF that have concurrent issues, such as acute or chronic renal injury/failure; trauma or toxin induced blood loss, and sepsis, the patient's homeostatic mechanisms are activated to preserve the perfusion to the brain and heart. This results in increased systemic vascular resistance, sodium retention to increase plasma volume to true or perceived loss of volume, and in patients with CHF there may even be cardiac dilation or hypertrophy. These responses to the perceived fluid loss may initially serve the patient in maintaining the normal circulation, however, patients with heart changes will experience the inability to effectively maintain cardiac output due to poor contractility, increased heart rates and increased preload. In addition, sodium retention will then contribute to continuing increased plasma volume, leading to oedema and effusions, as well as electrolyte imbalances.

Renin-angiotensin-aldosterone System

Renin is released in response to reduced renal blood flow, which is most commonly due to blood flow due to heart failure or to volume depletion which may be seen in diuretic therapy or in hypotension which could be due to blood loss or obstruction to blood flow. The release of renin triggers a pathway in which the renin cleaves angiotensinogen, from the liver, to angiotensin I. Angiotensin I circulates to the lungs, where angiotensin-converting-enzyme (ACE) converts this into angiotensin II. Angiotensin II further contributes to the release of aldosterone from the adrenal gland, and together they increase sodium retention in the kidneys and increase the systemic vascular resistance to maintain blood pressure and volume. This system can become maladaptive over time if the glomerular filtration rate decrease is not due to a decrease in blood volume, i.e. heart disease/ CHF and will further exacerbate these conditions.

Fluid Therapy in The Cardiac Patient

In most cardiac patients, the use of fluid therapy is actively discouraged due to the hearts inability to cope with additional preload and afterload. However, in patients with concurrent disease processes, fluid therapy may be a life saving treatment. Often in cardiac patients the administration of fluid therapy may push them into congestive heart failure and it is important to monitor closely for fluid overload. Often, the first indication of volume overload is increased resting respiratory rate, which should be checked every hour during fluid administration. Heart rate may also increase if fluids are poorly tolerated, as well as the caudal vena cava collapsibility index changing on point of care ultrasound. Monitoring for increased body weight is also recommended to monitor for fluid overload. Judicious use of intravenous fluids is crucial in these patients with administration of smaller aliquots than usual and rapid reassessment of cardiovascular parameters such as:

- Heart rate & pulse quality
- Blood pressure
- Mucous membrane colour
- Capillary refill time
- Mentation
- Urine output
- Blood lactate level

Fluid therapy choice is equally as important, considering sodium levels and the sodium content in the maintenance fluids being administered. Normasol-M with 5% dextrose or 0.45% saline may be a better choice for maintenance due to their lower sodium content, in those patients with increased sodium levels. Consideration for the type of fluid lost is equally as important- the use of plasma may help with volume expansion with smaller volumes of fluid therapy administered. If the patient does not need shock replacement, consideration of use of oral fluids will reduce the need for intravascular fluids and therefore the risk of overload. Finally, the therapeutic goals for fluid therapy in the cardiac patient include the fine balance of increasing myocardial contractility, decreasing preload and afterload, counteracting RAAS, and improving vasodilation.

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UNDER PRESSORS

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Learning objectives:

Delegates will be:

- Able to assess which patients may need vasoactive agents

- Able to compare different therapies

- Able to describe the physiological effects on the patient

- Able to carry out appropriate nursing management for these patients

Proceeding:

Which patients might need vasoactive agents?

Patients who are in shock or have severe hypotension (e.g. sepsis, systemic inflammation), and who have not responded to repeated intravenous fluid therapy (IVFT) boluses, are likely to need vasoactive agents to improve their overall cardiovascular status.

Patients who are hypovolaemic will respond appropriately to IVFT boluses or blood component therapy depending on why they are hypovolaemic, we should see changes in their vital signs as their IVFT bolus has finished e.g. decrease heart rate and increase in blood pressure. When the patient no longer responds to IVFT boluses and remains hypotensive we have to look at what drug therapies we can give to help the body maintain perfusion to our tissues and organs.

Vasoactive agents

Vasoactive agents are drugs which when administered will have an effect upon the blood vessels, the effect will depend on the type of drug therapy used e.g. vasoconstriction or decreasing systemic vascular resistance(SVR).

The therapies I will be discussing are inotropes and vasopressors. Inotropes increase the force of myocardial contractions, which improves contractility and therefore cardiac output (CO). Vasopressors cause vasoconstriction, which increases SVR therefore increasing the patients mean arterial blood pressure (MAP) leading to tissue and organ perfusion. Many drug therapies have both inotropic and vasopressor effects, this is down to which receptors the drugs will affect, Alpha (α) receptors or Beta (β) receptors either separately or both.

α_1 Receptors are found in the smooth muscle cell of the blood vessel wall, when activated cause vasoconstriction increasing systemic vascular resistance.

β Receptors are found on the surface of cells in tissues and organs, β_1 receptors are in the heart and when activated have positive chronotropic effects (increased heart rate) and positive inotropic effects (increased myocardial contractility). β_2 receptors are found in smooth muscle cells of the bronchi and blood vessels, when activated they cause bronchodilation.

Norepinephrine

The "go to" vasopressor in sepsis, as recommended in the surviving sepsis campaign. This drug is a neurotransmitter and a hormone, acting on both α and β receptors but mainly on α receptors. By activating the α receptors, the smooth muscle in the vessels it causes vasoconstriction and increases SVR. This increases MAP and improve our patients CO. β_1 receptors are also active, causing an increase in heart rate and contractility which helps to maintain our CO.

Dobutamine

Dobutamine is primarily an inotrope activating β_1 receptors, which improves our CO by increasing contractility, CO = heart rate x stroke volume, stroke volume is combination of three factors preload, afterload and contractility. Dobutamine does have effects on α_1 receptors at higher doses, causing an increase in SVR but may come with side effects of tachycardia. Vasoactive agents should be tailored to the patient specific needs, dobutamine may assist maintaining CO in patients with sepsis and have cardiac dysfunction when used along side norepinephrine.

Dopamine

Dopamine is the precursor to norepinephrine, dopamine acts on dopaminergic receptors type 1 and 2 (D1 & D2) which are important neurotransmitters in the brain as well as having vasopressor effects (α1 and β1 receptors). The dose of dopamine administered influences which receptors it acts on, lower doses of dopamine can have vasodilatory effects causing decrease in SVR, therefore a low MAP and an overall negative effect on our patients CO. Intermediate rates of dopamine act on the β1 receptors increasing heart rate and contractility and higher rates will have α1 effects causing vasoconstriction and increase MAP.

Adverse effects

Vasopressors and inotropes will have different adverse effects depending on the type of drug used and the dose rate it is administered at. Dysrhythmias, myocardial hypoxia and hypoperfusion to the gastrointestinal tract can be some of the effects of these therapies.

Monitoring

Patients need continual monitoring of their blood pressure either via non-invasive or invasive methods, heart rate, ECG and frequent point of care ultrasound (POCUS) of the heart to assess their cardiac function. If our patient is on vasoactive agents then they are usually critical, in which we are already monitoring respiratory rate, pulse rate, pulse quality, urine output, temperature, capillary refill time and

mucus membrane colour. These drugs can cause tissue necrosis if extravasation occurs, keeping an eye on these patients intravenous catheters (IVC) is important, we could be titrating our infusion rate higher if their blood pressure is suddenly dropping, when actually the vein integrity could have been compromised and they need a new IVC placement.

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NURSING THE INTENSIVE CARE PATIENT – FOLLOWING KIRBY'S RULE OF 20

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Learning objectives:

- Be familiar with Kirby's rule of 20

- Why it is an important tool in ECC nursing

- How it can be applied to a multitude of patients

Proceeding:

Introduction

Critical patients that are nursed within an ICU ward require time intensive nursing. It is important that all factors are evaluated frequently and by following a checklist such as Kirby's rule of 20 ensures that nothing is overlooked. Intensive care patients can involve all disciplines and can include (but are not limited to) cases such as;

Surgical cases –GDV, septic peritonitis

Medicine cases –DKA, CRGV

Neurology cases –Tetanus, Status epilepticus

Cardiology cases –Heart failure, arrythmias

ECC cases –Pneumothorax, mechanical ventilator patients

Kirby's rule of 20 can be adapted to individual patients and treatment guided accordingly.

Kirby's Rule of 20 – A brief outline

Fluid Balance –Replace losses to maintain adequate tissue perfusion

Albumin and Oncotic Pull– Maintain COP to prevent oedema and 3rd spacing of fluids

Electrolyte and Acid Base –Correct derangements for normal cellular function

Mentation –If inappropriate then can indicate a decreased tissue perfusion and therefore cerebral perfusion

HR, Rhythm and Contractility –Abnormalities can affect cardiac output which consequently will affect tissue perfusion

Perfusion and BP –Blood pressure is a vital indicator of perfusion. Fluid therapy +/- vasopressor therapy should be initiated if hypotensive. Hypertension should also be treated

Temperature –Hypothermia, hyperthermia, pyrexia

Oxygenation and Ventilation –Is the patient moving air effectively and is the body able to deliver the oxygen to tissues for adequate perfusion. Oxygen supplementation or mechanical ventilation may be required

RBC + Hb Concentration –Body will attempt to compensate for lack of RBC's and Hb in order to maintain delivery of oxygen to the tissues

Coagulation –Coagulopathies can lead to haemorrhage or thrombosis and can occur due to multiple disease processes. Signs of petechiation, ecchymoses and oozing at surgical wound/venepuncture sites should be monitored

Renal Function –Essential for fluid regulation, BP regulation and excretion of waste products

GI Motility and Mucosal Integrity -Malnutrition can increase morbidity and mortality. Bacterial translocation can occur if mucosal integrity is compromised

Nutrition –Vitally important to initiate early to aid patient healing

Glucose –Important to check if mentation decreases. Especially important in diabetic, anorexic, juvenile, and septic patients as well as toy breeds.

Immune Status and Antibiotics –Consider following the surviving sepsis guidelines. Broad spectrum antibiotics should be initiated until culture and sensitivity results are available

Wound Care and Bandages –Gloves should be worn for wound care. Bandages and dressing should be kept clean and dry. Check bandages regularly

Drug Dose and Metabolism –Monitor how the patient responds to medication. Caution in hypoproteinaemic patients with protein bound drugs

Pain Control –A validated pain scoring system should be utilised to monitor the patient's pain levels prior to and after administering analgesia to check if adequate. A muti-model analgesia protocol should be used

Nursing Care –Regular tube and line care to check if clean and patent. Recumbency care

TLC –Groom if unable to themselves. Interaction and enrichment

Conclusion

It is beyond the scope of these proceedings to go into further detail on each rule but it is evident that there are many factors that should be considered when monitoring a critical patient. Ultimately it is the veterinary surgeon who will direct treatments and make clinical decisions but as nurses we are responsible for obtaining the information required to support this. Adopting a whole team approach can be beneficial to the patient as the nurses are the ones that spend the most time with the patient and can detect subtle signs of improvement or deterioration. By identifying any abnormalities or changes in the patient's condition treatment can then be initiated/adapted swiftly. Intensive care patients can be challenging and time consuming but can also be incredibly rewarding.

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WHAT DO WE DO WHEN IT GOES WRONG - USING PROCESSES AND SYSTEMS TO SUPPORT TEAM AND IMPROVE PERFORMANCE

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Learning objectives:

- For all learners to be aware of the common traps we often fall into following an adverse event and consider how to avoid them.

- All learners will be able to describe what a systems approach is reviewing an adverse event.

- Attendees will be able to explain what contributory factors are and begin to apply them to their own reflections.

- The foundations of a significant event will be introduced with signposting to resources in order to develop this in their own practice

Proceeding:

When I was a trainee nurse we worked night shifts. Alone, with no one else in the building to assist or help us. One of my fellow student nurses had an unfortunate incident involving a subcutaneous injection of a once daily medication and a wriggly puppy. Whilst she was trying to administer the medication the puppy moved, and unluckily for everyone the needle caught the puppy's eye.

She felt awful and the reaction after this made those feelings even more intense. The team were horrified that she could have made such a simple mistake, what on earth was she doing? However, on reflection this was incredibly unfair and what we should have been considering was why was she being asked to that alone? Did it really need to be done at 10pm when no one else was around?

The way we respond to an adverse event can hugely affect how the individuals involved feel. When we know the negative impact that these events can have on our mental health. The individuals involved often become the second victim and at a time when burn out is so high how can we support our teams. How do we avoid falling into the blame trap and how do we do better?

It's incredibly easy to blame people. Often, it's our default setting. From a personal experience I feel remorse at how I handled some events with the nursing team I was managing. Quick to blame those involved rather than take a step back and drill down into why the event happened. Surprising when 90% of medical errors within veterinary are attributed to the systems in which our teams work (Low and Wu 2022)

We can all recall the events that have shaped us, modified our behaviours and perhaps influenced the knowledge we pass on. Why should we have to make an error to learn from it though, is it not enough to learn from others. As Churchill and the philosopher Santayana are known for saying "Those who do not remember the past are condemned to repeat it" if we take that forward sharing of learnings should become integral to our practice.

Initially after every event we should be conducting a debrief. Within the UK Ambulance service both 'hot' and 'cold debriefs' have been introduced. To support this process the University of Edinburgh have created the 'STOP5' model to provide a framework for this process. They were trialled in their emergency resuscitation cases and were well received, with 90% of the team rating the process from good to excellent. It is worth noting that over 70% stated the optimum time for a debrief was just 5 minutes (Walker et al 2020) Surely, we have 5 minutes after an adverse event to check in with the teams?

By performing the debrief immediately after the event the debrief becomes 'hot' and if were to repeat the process a little bit after the event they become 'cold'. Perhaps we could consider displaying the 'STOP5' poster around the hospital, or could this be worked into our crash recording documents? One hospital found that when they introduced post cardiac arrest 100% of the participants felt it improved their practice (Gilmartin et al 2020)

Once the 'hot debrief' has performed consideration should be given to what happens next. A systematic review will help us analyse why the event occurred. Identifying opportunities for improvement and potentially stopping them from happening in the future. There are a couple of names you may hear this process referred to as, the most common being a Significant Event Audit (SEA) or a Morbidity and Mortality round (M and M) Within human healthcare the process of event review began with an M&M, but they have progressed to contributing factors identification and root cause analysis (Raja et al 2021)

The key part with either of these is the use of a structured process, that focusses on the systems and processes, not the individuals. Why did the event happen? The RCVS Knowledge has numerous resources on their website, as does the Veterinary Defence Society. When we are analysing an event, using a systemic process we are establishing the contributing factors. The factors that caused the event to happen. The resources mentioned will go into more detail but let's consider the fishbone diagram as a start point. Within this diagram categories of contributing factors are listed.

These include, but not limited to, task factors, team factors, organisational factors. In the scenario I described at the start we could initially identify a patient factor; the fact it was young wriggly puppy. Some organisational factors, doing a night shift solo and the timing of the medication. By doing this we have already highlighted areas that if we were to plan that medication again, we would do it differently.

The process of identifying factors and then modifying what we do is critical to using adverse events to shape our practice for the positive. We look at why something was done, and we suggest changes that will shape our actions in the future. An SEA or M&M should be done after every adverse event. Led by an individual, but with the whole team involved. Caution should be taken to ensure this is not a blame process and by using the process mentioned above keeping focussed on the systems and what was happening at the time of the event.

We can then use these learnings to make changes within the practice, or our own way of working. Considering a range of actions that could be introduced, perhaps a check list or even a physical adaptation. Implementing changes to minimise errors and create a safer environment for our patients and colleges. Helping us to protect ourselves from the impact these events can have on our mental health.

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IMPOSTER SYNDROME

Marlaina Hrosch [1, 2]

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Learning objectives:

- Define imposter syndrome and how it affects wellbeing

- Identify the 5 subtypes associated with imposter syndrome

- Recognize strategies to overcome the imposter phenomenon

Proceeding:

Imposter syndrome is a feeling of doubt in one's skills or knowledge, despite evidence to the contrary. Individuals experiencing imposter syndrome may fear their achievements were based solely on luck or timing and they will eventually be uncovered as a true imposter. This can be exhibited in behaviors such as perfectionism, anxiety, and fear of failure.

This phenomenon was first introduced in a study published in 1978 by psychologists Dr. Pauline Clance and Dr. Suzanne Imes. This study found many successful and accomplished women to feel that they aren't actually intelligent or successful. Imposter syndrome in these women often originated due to social expectations in early childhood, either from being labeled less intelligent than another sibling or from unrealistic expectations of success. Therapy and self reflection practices to improve self-awareness were utilized to help the women in overcoming their imposter syndrome.

In 1985, Dr. Clance additionally developed the Clance Imposter Phenomenon Scale (CIPS) which was further validated in 1995. CIPS is a free self-assessment tool to assess factors relating to imposter syndrome including depression, self-esteem, social anxiety, and self-monitoring. This assessment asks respondents to assess how accurate each statement is to provide an assessment of how frequently an individual is experiencing imposter syndrome and how it may be interfering with their well-being. In studies conducted using CIPS to evaluate the presence of imposter syndrome among veterinary professionals, imposter syndrome was found to be more common in young females with less years of veterinary experience.

Dr. Valerie Young identified five subtypes that individuals with imposter syndrome may be classified into. The perfectionist will strive for flawlessness in their work and often focus on what went wrong rather than what went well. The superhero will take on many different roles and feel that they failed if they fall short in any one of them. The expert values expertise and does not want to be caught with a lack of understanding or a knowledge gap. The natural genius believes that success should come easily and does

not feel successful when they struggle. The soloist feels that success is measured based on their individual accomplishments and does not want to ask for help.

Feelings of imposter syndrome can lead to decreased performance, depression, burnout, and feelings of isolation. It is important to start managing and overcoming imposter syndrome before it escalates. Engaging in self-reflection practices to track accomplishments can help provide a reminder that success was earned and not a matter of luck. Setting reasonable goals can also help in measuring success. It is important to remember to celebrate when each goal is achieved along the way. Given the prevalence in veterinary medicine, having the vulnerability to speak with colleagues about imposter syndrome can be helpful in growing a support system.

Promoting psychological safety within a veterinary hospital plays a vital role in helping individuals overcome imposter syndrome. By creating an environment where mistakes are seen as valuable learning opportunities and open conversations among team members are encouraged, the hospital becomes a space for growth amongst the entire team. When team members feel safe communicating their mistakes and receive encouragement instead of punishment, they are less likely to doubt their abilities moving forward. A positive and supportive team that celebrates successes and recognizes individual wins can make a meaningful impact in reducing the prevalence of imposter syndrome.

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Literature Review, Thursday 5 June 2025

ECC YEAR-IN-REVIEW

Simon Cook [1], Tommaso Rosati [2]

[1] Royal Veterinary College, Royal Veterinary College, London, United Kingdom
[2] University of Zurich, Zurich, Switzerland

Proceeding:

These sessions will explore the most exciting and impactful literature in the field of small animal emergency and critical care from the preceding 12 months. Sessions are designed to review articles that shed new light on the understanding of the pathophysiology of relevant clinical conditions and advancements in clinical treatments. The discussion will be led with a critical appraisal approach to the selected articles and ensure evaluation of their impact and implications. The primary focus will be veterinary literature, but human landmark publications will also be covered.

NEUROLOGY YEAR-IN-REVIEW

Abbe Crawford [1]

[1] Royal Veterinary College, Clinical Science and Services, North Mymms, United Kingdom

Learning objectives:

- Refresh on recent literature relating to Veterinary Neurology.

- Identify key take home messages from pertinent papers.

- Develop skills in critical analysis of the veterinary literature.

Proceeding:

Keeping on top of the current literature can be daunting. In this “Neurology year-in-review” session we will recap and review key journal articles published over the last year from various Journals including JVECC, JVIM, JSAP and Frontiers. The emphasis will be on papers that apply to emergency and critical care, but a range of topics will be covered including seizure management, trauma, intervertebral disc disease, inflammatory and infectious diseases of the nervous system. The main findings of each paper will be discussed, alongside any pertinent features of the methods and any implications for current practice. The goal is to provide an overview of new understanding and current research focuses in veterinary neurology.

INTERNAL MEDICINE YEAR-IN-REVIEW

Christopher Scudder [1]

[1] Royal Veterinary College, Clinical Science and Services, Potters Bar, United Kingdom

Learning objectives:

- Review and summarise key internal medicine studies published between 2024 and 2025, focusing on those with implications for clinical practice.

- Interpret their study design, outcomes, and identify the limitations of these key studies and systematic reviews.

- Assess how recent evidence may be integrated into clinical practice.

- Identify knowledge gaps and discuss how these may be further investigated.

Proceeding:

The presentation will cover the cornerstone publications in internal medicine from 2024 to 2025. It will highlight the most significant diagnostic advancements, and outcomes from clinical trials and descriptive studies relevant to canine and feline medicine. These studies will be critically appraised, and how to integrate the insights from the literature into evidence-based decision-making will be discussed.

Main Stream, Friday 6 June 2025

THE GENERAL APPROACH TO THE POISONED PATIENT

Sigal Klainbart [1]

[1] Koret School of Veterinary Medicine, The Hebrew University of Jerusalem, Small Animals Emergency and Critical Care, Rehovot, Israel

Learning objectives:

- Recognize the key steps in assessing and stabilizing poisoned veterinary patients.
- Understand the various decontamination techniques available and their indications.
- Identify contraindications for common decontamination methods.
- Gain insight into advanced extracorporeal therapies for toxin removal.
- Appreciate the role of standardized treatment protocols in improving patient outcomes.

Proceeding:

The General Approach to the Poisoned Patient in Veterinary Medicine

Effective management of poisoned patients in veterinary medicine requires a structured and evidence-based approach. Rapid assessment and timely intervention are crucial to minimizing the harmful effects of toxic substances and improving patient outcomes. Management involves thorough history gathering, a comprehensive physical examination, and appropriate decontamination and stabilization techniques.

Initial Assessment and Stabilization

Telephone Triage

Rapid preliminary assessment through direct communication with the pet owner.

Obtain initial information about the incident to determine urgency.

History Gathering

Identify the potential toxin (active ingredient, commercial product name).

Determine dosage, timing, and route of exposure.

Assess dosage form details (e.g., sustained-release or extended-release formulations).

Review any home interventions or treatments attempted.

Verify product details with the manufacturer or pharmacy when necessary.

Evaluate the pet's medical history and any underlying conditions that may affect treatment.

Evaluate the clinical status of the pet (e.g. asymptomatic, neurological signs, GI signs)

Physical Examination

Assess vital signs (temperature, heart rate, respiratory rate, blood pressure).

Evaluate neurological status and signs of shock or organ dysfunction.

Initial Stabilization

Administer oxygen, intravenous fluids, and supportive therapies as indicated.

Continuously monitor vital parameters to guide further management.

Decontamination Techniques

Decontamination aims to prevent further absorption and enhance toxin elimination. The method depends on toxin characteristics and route of exposure.

Ocular Decontamination

For corrosive or caustic substances: Lavage with 0.9% saline or tap water for 15–25 minutes at home, or 20–30 minutes in-clinic under sedation if needed.

Avoid decongestant eye drops; apply ointments only if the corneal epithelium is intact.

Dermal Decontamination

For corrosives: Gentle lavage with room-temperature water for 20–30 minutes.

For oil-based contaminants: Use dish detergent (not dishwasher detergent) followed by thorough rinsing.

For sticky substances (e.g., glues): Apply vegetable oil or peanut butter, then bathe with detergent.

Inhalation Decontamination

Remove the patient from exposure immediately.

Ensure adequate ventilation and administer supplemental oxygen as needed.

Recognize that inhaled toxins may cause pulmonary damage requiring veterinary intervention.

Gastrointestinal Decontamination

Emesis

Indicated for recent ingestion in asymptomatic patients or when the toxin remains in the stomach for prolonged periods.

Contraindicated in cases of corrosive or hydrocarbon ingestion, symptomatic patients, or those at high risk for aspiration pneumonia.

Agents:

Dogs: Apomorphine, tranexamic acid, or ropinirole.

Cats: Alpha-2 adrenergic agonists (e.g., dexmedetomidine, xylazine).

Gastric Lavage

Indicated when emesis is unproductive, contraindicated, or in cases of massive ingestion.

Contraindicated for corrosives, hydrocarbons, or sharp object ingestion.

Adsorbents

Activated Charcoal: Administer orally, with or without a cathartic, to bind toxins (ineffective for alcohols, xylitol, ethylene glycol, heavy metals, corrosives, and hydrocarbons).

Cholestyramine: Effective for toxins undergoing enterohepatic recirculation (ensure formulation does not contain xylitol).

Urine Decontamination

Ion Trapping

Alters urine pH to prevent toxin reabsorption.

Techniques:

Alkalinization with sodium bicarbonate for weak acids (e.g., ethylene glycol, salicylates, barbiturates).

Acidification with ammonium chloride for weak bases (e.g., amphetamines).

Requires urine pH, serum electrolytes, blood pH, and blood pressure monitoring.

Intravascular Decontamination

Intravenous Lipid Emulsion (ILE)

Used for lipophilic toxins via the “lipid sink” mechanism.

Effective for ivermectins, lidocaine, naproxen, pyrethrins, calcium channel blockers, and tremorgenic mycotoxins.

Extracorporeal Therapies

Hemodialysis

Removes small-molecule, water-soluble, low-protein-binding toxins with a low volume of distribution.

Effective for alcohols, analgesics, antibiotics, anticonvulsants.

Hemoperfusion

Used for toxins with large molecular weights, high protein binding, and high lipid solubility.

Effective for NSAIDs, baclofen, salicylates, barbiturates.

Therapeutic Plasma Exchange

Removes highly protein-bound toxins with a low volume of distribution.

Involves plasma removal and replacement with donor plasma or suitable solutions.

Conclusion

A systematic approach to poisoned patients in veterinary medicine enhances outcomes through prompt assessment, comprehensive decontamination, and targeted interventions. By integrating these strategies—from initial stabilization to advanced extracorporeal therapies—clinicians can improve patient care. Standardized treatment protocols and ongoing education remain essential in managing toxic exposures effectively.

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BLOOD-PATCH PLEURODESIS FOR PERSISTENT PNEUMOTHORAX - WHAT WE LEARNED IN 15 YEARS

Efrat Kelmer [1]

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Learning objectives:

- Review the veterinary literature regarding autologous blood-patch pleurodesis since 2010
- Discuss indications for the procedure
- Provide tips on when and how to perform it
- Review clinical cases

Proceeding:

Persistent air leak following traumatic or spontaneous pneumothorax in dogs can be managed conservatively with a thoracostomy tube or surgically by removing the affected lung lobes. (1) An additional option reported in the human literature is the injection of irritating substances into the pleural space in order to induce pleurodesis. (2) Substances that have been used for this purpose include tetracycline, talc powder and autologous blood. (2) When comparing these substances, autologous blood was shown to have the shortest air leak cessation time, and the least side effects. (2) Autologous blood-patch pleurodesis has been used successfully in humans for treatment of air leakage following surgery, and in cases of primary and secondary spontaneous pneumothorax, with reported success rates ranging from 75-85%. (2-5)

Autologous blood-patch pleurodesis (ABP) was first described by Robinson et al in 1987 for treatment of patients with spontaneous pneumothorax. (4) Since then, a number of studies have investigated the efficacy and complications of this procedure. Rivas et al performed ABP on 6 patients with non-small cell lung cancer who suffered of persistent air leak postoperatively for more than 10 days (6). After 24 hours, no air leak was detected in any of the patients and radiographs confirmed full lung expansion. Lang-Lazdunski et al reported 11 patients who underwent pulmonary resection for a variety of reasons. (7) Patients with a persistent air leak of >7 days were treated with ABP. Air leak resolved within 12 hrs in 8 patients, and by 48 hrs it resolved in all of them. Complications were encountered in 3 patients. Pneumonia developed in one patient 24 hours after ABP and two patients developed low grade fever with isolation of Staphylococcus from their pleural fluid. Shackcloth et al. tested the technique in a prospective randomized controlled study (8). Twenty patients suffering from prolonged air leak after a lobectomy were randomized to receive ABP or continued to be treated by tube thoracostomy alone. The

duration of the air leak and time to discharge was significantly shorter in the patients who were treated with ABP.[8]

We previously described a single case report in which ABP was successfully used for the treatment of persistent leakage of air following surgery to correct a chronic diaphragmatic hernia in a pregnant dog (9). During surgery, many adhesions were present between the uterus, liver and lung lobes. Pneumothorax persisted for 4 days post-operatively. Once ABP was performed, the pneumothorax resolved and the dog made a full recovery. Following this case report, we described a series of 8 cases undergoing ABP. Autologous blood-patch pleurodesis was performed with the dogs either anesthetized or sedated. All dogs had the chest evacuated from air prior to the procedure either by thoracocentesis or via the thoracostomy tube. The skin over the jugular vein and ipsilateral intercostal spaces 4 to 7 were clipped and prepared aseptically. Blood was collected from the jugular vein either with a newly or previously placed jugular catheter, or with a syringe and butterfly-needle, with no additives, and injected immediately into the pleural cavity. The amount of blood collected ranged between 5% and 10% of body weight and was collected in 20-50 ml increments until the calculated total dose was achieved. In 4 dogs, blood was injected into a previously placed thoracostomy tube, and in 4 other dogs blood was injected into the pleural space via a 16g Teflon catheter or a 19g butterfly needle. Following the procedure, the tube was flushed with 10-20 ml of saline and was not used to evacuate the chest for at least 4 hours, unless the dog was dyspneic. In dogs with bilateral pathology, half of the total calculated volume of blood was collected and injected into each hemithorax. Resolution of pneumothorax was objectively assessed by a stable respiratory rate and a negative thoracocentesis and subjectively assessed by respiratory effort. Post-procedure radiographs were performed in 6 of 8 dogs and all dogs received a short course of antibiotic treatment following the procedure.

The procedure was successful in 7 of 8 dogs. The median duration of pneumothorax until the ABP was performed was 4 days (range 2-6 days). Pneumothorax resolved immediately after one treatment in 4 cases. ABP was repeated once in 3 cases, and twice in 1 case after which it resolved in 3 of the 4 cases. Out of a total of 13 ABP procedures in 8 dogs, 5 (62.5%) were successful after one procedure and success rate increased to 87.5% after additional procedures. One dog failed ABP and was euthanized 3 days later due to continued deterioration and a hospital acquired pneumonia. Mild to moderate complications occurred in 2 other dogs and resolved in both.

In addition to our publications, successful ABP was described post-operatively in 3 cats with persistent pneumothorax after correction of a diaphragmatic hernia,[15] in a King-Charles cavalier with bullae and blebs[16], in a dog with pulmonary MCT[17] and in 5 dogs (2 with congenital bulla, 1 trauma, 1 lungworm and 1 neoplasia)[18]. In the latter, the procedure was successful in 4/5 dogs (80%) and unsuccessful in one dog suffering from pulmonary hemangiosarcoma. In addition, canine xeno-blood patching was reported in a cat with iatrogenic pneumothorax. [19]

To conclude, ABP is a viable, minimally invasive options for treatment of pneumothorax. Case selection is important, as, in the author's experience, in severe cases (e.g large bullae), and in those with underlying neoplasia, the likelihood for resolution without surgical intervention is low. In addition, recurrence rate is most likely higher with ABP compared to exploratory thoracotomy and surgery remains the gold

standard. In the author's opinion, ABP should be offered when surgical intervention is not possible or when air leak persists post-operatively.

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ACID-BASE EVALUATION IN THE EMERGENCY ROOM

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Learning objectives:

- List the step-by-step approach to traditional acid-base analysis.

- Describe the expected compensation for each of the four primary acid-base disturbances.

- List differentials for each of the four primary acid-base disturbances.

Proceeding:

Acid-base (AB) analysis is a valuable tool for use in patients that present to the ER. The first goal of AB analysis is to define the primary AB disturbance, of which there are four: respiratory acidosis, respiratory alkalosis, metabolic acidosis, and metabolic alkalosis. After defining the primary AB disturbance, a list of differentials should be considered to identify the cause.

Steps to Performing Traditional AB Analysis

1. Confirm appropriate sample collection and handling.

2. Evaluate the pH and describe as normal or as contributing to an acidemia or alkalemia.

3. Evaluate the pCO_2 (respiratory component) and describe as normal or as contributing to an acidosis or alkalosis.

4. Evaluate the HCO_3- or BE (metabolic component) and describe as normal or as contributing to an acidosis or alkalosis.

5. Define the primary process as normal, respiratory acidosis, respiratory alkalosis, metabolic acidosis, or metabolic alkalosis.

6. Evaluate for compensation.

7. Describe overall AB analysis as the primary process with or without expected compensation.

Mechanisms and Differential Diagnoses for Primary AB Disturbances

Respiratory acidosis: Respiratory acidosis is most commonly due to diseases that decrease CO_2 excretion through reductions in respiratory rate, tidal volume, or both. Differentials for respiratory acidosis include:

Compensation for metabolic alkalosis

Decreased CO_2 excretion through alterations in respiratory rate, tidal volume, or both

Central respiratory depression – Drugs, brain injury

Cervical myelopathy

Peripheral nervous system disease - Myasthenia gravis, tetanus, botulism, polyradiculoneuritis, tick paralysis, diaphragmatic paralysis, neuromuscular blocking agents, generalized myopathy, hypokalemia

Upper airway obstruction - Foreign body, mass, tracheal collapse, brachycephalic obstructive airway syndrome, laryngeal paralysis

Small airway disease - Asthma

Pulmonary disease, usually end-stage or severe disease - Pneumonia, pulmonary edema, ARDS, PTE, pulmonary fibrosis

Pleural space disease - Pneumothorax, pleural effusion, diaphragmatic hernia, pleural space mass

Chest wall trauma / flail chest / rib fractures

Anterior displacement of the diaphragm due to abdominal distension / GDV

Ineffective mechanical ventilation

Cardiopulmonary arrest

Increased CO_2 production - Hyperthermia, heat stroke, fever, seizures, malignant hyperthermia

Respiratory alkalosis: Respiratory alkalosis is most commonly due to increased CO_2 excretion associated with increases in respiratory rate, tidal volume, or both. Differentials for respiratory alkalosis include:

Compensation for metabolic acidosis

Increased CO_2 excretion through alterations in respiratory rate, tidal volume, or both

Central respiratory stimulation - Brain injury, liver disease, Cushing's disease, sepsis, drugs, heat stroke, exercise, pain, fear / anxiety

Decreased oxygen delivery - Hypoxemia, anemia, hypotension, decreased cardiac output, hypovolemia

Pulmonary parenchymal disease due to hypoxemia or due to stimulation of pulmonary stretch receptors and nociceptors

Airway inflammation

Metabolic Acidosis: Metabolic acidosis may be due to the accumulation of acids, the loss of bicarbonate through the GI tract or kidney, or through the administration of chloride. When a metabolic acidosis is

diagnosed, the anion gap (AG) should be calculated, and the disturbance should be further defined as an increased AG metabolic acidosis or a normal AG acidosis.

The four most common causes of an increased AG metabolic acidosis include (LUKE):

Lactic acidosis, uremic acids, ketones, ethylene glycol

Less common causes of an increased AG metabolic acidosis include:

D-lactic acidosis, salicylate toxicity (aspirin), methanol toxicity

Differentials for a normal AG / hyperchloremic metabolic acidosis include:

Compensation for respiratory alkalosis

GI loss of bicarbonate through diarrhea

Renal loss of bicarbonate - renal tubular acidosis, proximal or distal

Hypoadrenocorticism

Administration of IV fluids, especially those with high chloride concentrations

Administration of carbonic anhydrase inhibitors or cationic amino acids

Metabolic alkalosis: Metabolic alkalosis is due to the loss of acid through the GI tract or kidney or through the accumulation of bicarbonate. Differentials for a hypochloremic metabolic alkalosis include:

Compensation for respiratory acidosis

Loss of chloride through the GI tract - Vomiting, GI tract obstruction

Loss of chloride through the kidney - Administration of a loop diuretic, Cushing's disease

Accumulation of bicarbonate - Decreased effective circulating volume, administration of sodium bicarbonate

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DIAGNOSIS AND MANAGEMENT OF URGENT AND EMERGENCY CASES: A SHARED CLINICAL PATHOLOGY AND ECC APPROACH

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Learning objectives:

- Recognize the importance of microscopic evaluation of blood and cytology smears in the diagnostic work-up and treatment of emergency/urgent care cases.

- Evaluate blood and cytology samples using a standardized approach.

- Recognize key pathologic cell changes and prioritize findings to aid in interpretation and formulation of hematological and cytological diagnoses that can assist treatment/management decisions.

- Review important aspects of sample preparation to provide the best diagnostic yield.

Proceeding:

Introduction:

Emergencies can occur at any time—late at night or over the weekend—when access to a clinical pathologist with a short turnaround time is often limited or non-existent. However, many diseases can be diagnosed bedside with the help of cytology and clinical pathology, allowing for timely decision-making and improved patient outcomes.

This session provides a review of how to effectively approach evaluation of blood smears and cytology smears from dogs and cats that may present in an emergency/urgent care setting. This session will be primarily case based, focussing on clinical pathology imagery and data used in the diagnosis and management of case examples that present to an ER, including various causes of anemia, platelet and leukocyte abnormalities, body cavity effusions and other pertinent cytology specimens.

Hematology cases

Evaluation of a blood smear alongside numerical CBC analyzer results is an essential component in the overall assessment of a patient's blood sample, often aiding in efficient diagnosis of common hematological disorders such as hemolysis, infection, inflammation, and leukemia.

Good blood smear preparation technique is key in producing a smear that contains the important components for effective evaluation and interpretation, including a monolayer (for evaluation of individual cell morphology) and feathered edge (for evaluation of platelet clumps and/or larger atypical cells).

Approaching microscopic review of a blood smear:

Start with a brief scan of the whole slide at low power (10X) to look for any evidence of larger parasites (microfilaria for example), cell aggregates including agglutination, and/or platelet clumps, both of which can be present throughout the smear.

A follow-up quick scan of the monolayer area at low power (10X) can provide an idea of overall leukocyte numbers (increased or decreased) and red cell density (e.g. presence of increased white space or an extended monolayer (>1 field of view at 10X) can signal the presence anemia); a rough leukocyte estimate, if needed, can be performed at low power (total # leukocytes in 4-5 fields(10X)/# fields viewed = average of leukocytes per 10X field/4 = estimated #leukocytes X 10^9/L).

Lastly, at low power (10X), review of the feathered edge is important in assessment for platelet clumps and/or atypical nucleated cells.

Return to the monolayer area and change focus to higher power (40-100X depending on the objectives available) to evaluate individual cell morphology and background of the smear. It is here that identification of key erythrocyte, leukocyte and platelet pathologies are made. A rough platelet estimate (total # platelets in 8-10 fields (100X)/# fields viewed = average of platelets per 100X field x 15 = estimated #platelets X 10^9/L) and a leukocyte differential (count and identify 100 leukocytes to calculate percentages of each cell type) can be performed if needed in this monolayer space at this higher power. An absolute count for each leukocyte type can be calculated if the total leukocyte count (X10^9/L) is known.

Diagnostically important erythrocyte pathologies to identify can include spherocytes, eccentrocytes, Heinz bodies, and ghost cells in cases of hemolytic anemias; colour changes in erythrocytes like hypochromasia and polychromasia can be imperative to identification of iron deficiency anemia and regeneration respectively; identification of other shape changes such as schizocytes (or schistocytes), keratocytes, acanthocytes or a combination of these termed poikilocytosis, may be helpful when there is suspicion of underlying systemic disease such as liver disease, microthrombotic disease and/or vascular neoplasia. See vetclinpathimages.com for images of these and other erythrocyte pathologies.

Leukocyte pathologies important to recognize on a blood smear include immature or band neutrophils, toxic change in neutrophils, reactive versus neoplastic lymphocytes or other leukemic cells, and other atypical cells (e.g. increased mast cells). Identification of the various leukocyte types and the ability to recognize pathology in these cells on a blood smear can be very useful when interpreting unexpected or odd numerical CBC results produced by bench top analyzers – blood smear review alongside a CBC printout has the potential to provide clinically useful data for the patient that we may otherwise miss.

Platelet numbers can be easily estimated from a blood smear (as described above), and the identification of platelet clumping can discern artifactual platelet number decreases from true thrombocytopenia. Observation of enlarged platelets or macroplatelets, can potentially signal active thrombopoiesis in thrombocytopenic patients, or be an indication of normal platelet function in the presence of low platelet numbers but adequate platelet mass in some dog breeds affected by inherited macrothrombocytopenia (e.g. Cavalier King Charles Spaniels).

A brief scan of the background of a blood smear may provide important diagnostic information, especially in cases of suspected infectious agents such as Mycoplasma sp. (hemotropic organisms that attach to the surface of erythrocytes and can “fall off” during sample storage, they can be observed in the background of smears).

Common artifacts to be aware of when reviewing blood smears:

Stain Precipitate: can occur when using outdated staining solutions or with inadequate rinsing of slides after staining. Stain precipitate can overlay erythrocytes, sometimes closely resembling epicellular parasites or bacteria, or potentially obscuring cellular detail and leading to diagnostic errors. Regularly refreshing stain solutions and thoroughly rinsing slides can help minimize this issue.

Water Artifact: can create a “moth-eaten appearance” in erythrocytes, often appearing as refractile bodies. This artifact usually results from water contamination in the fixative solution in poorly maintained rapid stains. Again, ensuring that staining reagents are fresh and properly maintained can prevent the formation of these distracting artifacts.

Platelet Confusion: platelets may occasionally sit on top of erythrocytes on a blood smear, landing there during the smearing process. They can sometimes then be mistaken as infectious agents. In this instance, comparison of the suspected cells to free-floating platelets elsewhere on the slide can be helpful.

Cytology cases

Emergency/urgent care cases where in house cytological evaluation can be most helpful include those animals that present with cavitary effusion, in cases where there is concern for septic inflammation, or potentially those in decompensation from underlying neoplasia.

In emergency medicine, recognizing effusion type is critical to guiding the diagnosis and determining the next steps in treatment. Pleural and peritoneal effusions are particularly important and often qualify as “do not miss” cases due to their potential severity. Accurate identification of the effusion type, based on total protein and cell type, helps narrow down differential diagnoses quickly.

There has been much discussion recently as to the best way to classify effusions from a clinical pathology perspective. Traditionally the classifications of transudate, modified transudate, and exudate have been applied based on fluid protein content and total number of nucleated cells; more recently there has been recommendation to classify fluids that are not notably coloured, i.e. are not overtly red (hemorrhagic), greenish brown (bilious) or milky-white (chylous) as protein-poor or protein-rich transudates versus exudates, while others recommend classification based on etiology. A modified approach combining these latter two schemes was published in 2011 as a practical approach to effusions in a clinical setting. In essence, this includes a first assessment based on the gross colour and viscosity of the fluid, followed by protein evaluation via refractometry and/or bench top analyzer (depending on the analyzer) and microscopic evaluation of well-prepared cytology smears to look for evidence of inflammation, infection and/or neoplastic cells. Combined assessment of these three components will aid in determining the origin of the fluid and subsequent diagnostic and treatment options for the patient.

For cytological evaluation of fluid samples, both direct smears (a sample taken straight from the EDTA collection tube) and/or concentrated smears (prepared from a centrifuged sample) can be made. If the fluid is clear-colorless and relatively thin, preparation of a concentrated smear will be most helpful in identification of any nucleated cells present. If the fluid is intensely coloured, opaque, and/or more viscous, then a direct smear may be all that is needed to identify disease-associated cells or organisms.

Approaching a cytology smear:

Start with a brief scan of the whole slide at low power (10X) to look for nucleated cells, and how/where they are dispersed. Cells may be present individually or in aggregates depending on cell type, thickness of fluid and spreading technique.

Evaluate cells more closely at higher power (40X-100X) and assess morphology, cell type and presence of microorganisms.

Conclusion

In veterinary emergency medicine, accurate and timely identification of hematologic and cytological changes, especially in cases that include effusion, is critical for guiding diagnosis and treatment. Whenever possible, a confirmatory cytology should be sent to a local clinical pathology laboratory for definitive analysis. The growing capabilities of AI and remote clinical pathology services provide valuable tools for supporting hematologic and cytologic diagnoses; however, these technologies should be seen as complementary rather than definitive, especially in cases where financial constraints or technical issues might arise. It is essential for emergency veterinarians to feel confident in performing basic cytology and blood smear analysis, as these can provide quick, cost-effective results that may be crucial in urgent situations. Having a solid understanding of these basic tools allows veterinarians to make informed decisions in time-sensitive cases while awaiting more comprehensive diagnostic results.

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FOCUS ON THE SPECIFICS OF THE RECOVER 2024 UPDATES

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Learning objectives:

- Demonstrate the proper compression techniques for wide-chested animals, small dogs, and cats to maximize blood flow during CPR.

- Execute effective ventilation techniques for intubated and non-intubated patients, utilizing tight-fitting masks and alternative airway management approaches.

- Implement evidence-based drug protocols by selecting and delivering the appropriate doses of epinephrine, atropine, vasopressin, and antiarrhythmics during resuscitation efforts.

- Interpret $ETCO_2$ values to assess the effectiveness of chest compressions and adjust resuscitation techniques to enhance patient outcomes.

Proceeding:

Key Updates in the 2024 RECOVER CPR Guidelines

Chest Compressions in Wide-Chested Animals

The 2024 RECOVER guidelines introduce significant updates regarding chest compressions for wide-chested animals, a group that includes breeds whose thoracic cavities are broader than they are deep. The primary goal of these changes is to optimize blood flow generation during cardiopulmonary resuscitation (CPR), ensuring that chest compressions are as effective as possible.

Basic life support (BLS) remains the foundation of successful resuscitation, as it directly drives blood flow to vital organs. Without properly executed compressions, the efficacy of advanced interventions diminishes significantly. While core principles like maintaining a compression rate of 100–120 compressions per minute and allowing full chest recoil remain unchanged, specific recommendations for wide-chested animals have been adjusted.

For these animals, when placed in dorsal recumbency, the recommended compression depth is now set at approximately 25% of the anterior-to-posterior chest diameter. This is a departure from the previously advised one-third to one-half compression depth used for other body types. The adjustment reflects the anatomical reality that a significant portion of the posterior chest in these animals is occupied by noncompressible structures, such as the spine and epaxial muscles. Compressing to a depth of 25% of

the chest's anterior-to-posterior dimension ensures that adequate circulation is maintained while minimizing the risk of excessive compression and related injuries.

Recumbency During Intubation for Wide-Chested Animals

Intubation is a crucial aspect of CPR, ensuring effective ventilation and oxygenation. However, wide-chested animals, particularly brachycephalic breeds, pose unique challenges during intubation due to excessive soft tissue that can obstruct the airway. The updated guidelines now recommend initiating chest compressions in lateral recumbency until intubation is secured. Once intubation is complete, repositioning the animal into dorsal recumbency for compressions is suggested. To minimize interruptions in CPR, this transition should take place during the brief pause between compression cycles.

However, if dorsal positioning is unstable or fails to generate sufficient end-tidal CO_2 ($ETCO_2$), lateral compressions may be continued as an alternative. Additionally, dorsal recumbency intubation is reasonable for teams that have experience with this technique and can perform it efficiently without prolonging interruptions in compressions. These recommendations aim to balance the need for effective ventilation with maintaining high-quality chest compressions.

Compression Techniques for Small Dogs and Cats

The 2024 guidelines now include three distinct chest compression techniques tailored for small dogs and cats, replacing the traditional two-handed method used for larger animals. The primary focus of these techniques is to optimize blood flow while minimizing the risk of overcompression and thoracic injury. The updated techniques target the ventricles of the heart directly, rather than compressing over the heart base, which can obstruct circulation and reduce CPR effectiveness.

Circumferential (Two-Thumb) Technique: This method involves encircling the chest with both hands, with the thumbs placed on one side of the chest and the fingers on the opposite side. Compressions are applied using the thumbs, directly compressing the heart's ventricles between the thumbs and fingers. This technique is particularly useful for neonatal and very small patients, offering stability and direct control over compression depth.

One-Handed Technique: The dominant hand wraps around the chest, with the thumb on one side and fingers on the other, applying compressions directed from the bottom of the ventricles toward the atria. The nondominant hand stabilizes the patient's back. This technique offers precise control and minimizes the risk of overcompression, making it ideal for small dogs and adult cats.

Heel of the Hand Technique: This technique uses the heel of one hand to apply compressions over the ventricle region, avoiding the heart base. Positioning closer to the sternum can enhance blood flow and improve $ETCO_2$ levels. It is particularly useful for small dogs with chests that are too stiff for the one-handed technique.

These updated techniques emphasize individualized approaches based on patient size and anatomy, ensuring optimal circulation during CPR.

Non-intubated Ventilation

The 2024 RECOVER guidelines emphasize that ventilation remains critical in CPR, even in non-intubated patients. Intubation remains the gold standard for delivering ventilation, as it allows continuous airflow while compressions are ongoing. However, in cases where intubation is not immediately possible, alternative methods have been recommended.

For non-intubated patients, the guidelines now prioritize ventilation over compression-only CPR. The preferred method is using a tight-fitting mask with a resuscitator bag, allowing for oxygen supplementation and reducing risks to the rescuer. If a resuscitator bag is unavailable, mouth-to-nose ventilation may be used if there is no risk of zoonotic disease transmission or exposure to hazardous substances. If such risks exist, compression-only CPR is acceptable until intubation can be achieved.

The guidelines also recognize the challenges posed by brachycephalic breeds and neonates in achieving effective ventilation. Efforts are underway to develop smaller, species-specific masks and resuscitator bags to accommodate different facial structures. Techniques such as occluding the gastrointestinal pathway during ventilation are being explored to reduce air entry into the stomach, preventing gastric distension.

Increased $ETCO_2$ Targets in CPR

$ETCO_2$ levels serve as an indicator of CPR quality, with higher values correlating with improved survival outcomes. The 2024 guidelines raise the target $ETCO_2$ level during CPR from 15 mmHg to 18 mmHg, reflecting evidence that suggests better circulation and oxygen delivery with higher $ETCO_2$ values. This adjustment is based on studies demonstrating that patients achieving higher $ETCO_2$ levels during CPR have improved chances of return of spontaneous circulation (ROSC) and better post-resuscitation outcomes.

High-Dose Epinephrine Removed

High-dose epinephrine (HDE) has been removed from the guidelines due to concerns about its effects on coronary and cerebral blood flow. While historically believed to increase ROSC rates, recent evidence suggests that HDE may cause excessive vasoconstriction, compromising perfusion to vital organs. Studies have shown no survival benefit associated with HDE use in veterinary patients. Standard-dose epinephrine remains the recommended protocol for non-shockable rhythms such as asystole and pulseless electrical activity (PEA).

Atropine Use

The revised guidelines recommend administering a single dose of atropine (0.04 mg/kg IV or IO) early in the resuscitation process for non-shockable rhythms when high vagal tone is suspected as a contributing factor to cardiac arrest. This change is based on concerns about atropine accumulation, which could have adverse effects on myocardial oxygen consumption. Simplifying atropine administration ensures efficient resuscitation management while minimizing risks.

Changes to Treating Refractory Shockable Rhythms

Updates to treating refractory shockable rhythms, such as ventricular fibrillation (VF) and pulseless ventricular tachycardia (PVT), include refined defibrillation protocols and optimized medication use.

The first defibrillation should be at 2 J/kg using a biphasic defibrillator.

If unsuccessful, a second shock should be at 4 J/kg.

All subsequent shocks remain at 4 J/kg, as further dose escalation lacks supporting evidence.

Vasopressin (0.8 U/kg IV or IO) is now the preferred first-line vasopressor, with epinephrine recommended only if vasopressin is unavailable. Lidocaine (2 mg/kg IV or IO) is advised for dogs with refractory VF or PVT, while amiodarone (5 mg/kg IV) is recommended for cats due to their sensitivity to lidocaine. Esmolol (0.5 mg/kg IV or IO) has also been introduced for persistent shockable rhythms, as it mitigates excessive catecholamine effects and improves ROSC rates.

Conclusion

The 2024 RECOVER guidelines represent a refined approach to CPR in veterinary medicine, integrating new research to improve resuscitation outcomes. Updates focus on optimizing compression techniques, improving ventilation strategies, increasing $ETCO_2$ targets, and refining medication use. These changes simplify CPR protocols, enhance efficiency, and prioritize patient survival with meaningful neurologic recovery.

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GARY STAMP MEMORIAL LECTURE: NEWBORN RESUSCITATION

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Learning objectives:

- Develop an approach to resuscitation in the newborn puppy or kitten based on heart rate.

- Explain why ventilation is prioritized over circulation in newborn resuscitation efforts.

- Describe the indications for the use of epinephrine during CPR in newborn puppies and kittens.

- Describe the correct use of oxygen supplementation during newborn resuscitation.

Proceeding:

The newborn puppy or kitten must rapidly transition its physiology to survive in the environment. The first few minutes may be the most life-threatening in dogs and cats. The RECOVER 2.0 Newborn guidelines are targeted at management of dogs and cats during the transition phase, which occurs from birth to the first few hours of life.

Identification of Newborns in Need of Resuscitation

Given the physiologic alterations of the newborn, resuscitative efforts may be required before apnea and cardiac arrest have occurred. Patient selection in this population is based upon identifying those **not** in need of CPR. The recommended criteria are: (1) normal parturition; (2) mother able to provide care; (3) vigorous: breathing (RR> 15 bpm), clear vocalization and a vigorous response when testing for reflex irritability. Any newborn dogs and cats not exhibiting all 3 of these criteria may require resuscitative measures, even if they are still breathing and still have an obvious heart beat and pulses. In addition, all animals born by C-section require resuscitative efforts.

The First 1-2 Minutes

Management of hypoxia is critically important in the newborn and includes the following therapeutic steps: (1) establishing a patent airway, (2) supplementation of oxygen, (3) ventilation. Fetal membranes should be removed immediately, and the airway cleared by gentle aspiration using a suction bulb or DeLee suction catheter if an obstruction to spontaneous breathing is evident. Oxygen should be supplemented if the patient is cyanotic or bradycardic, but routine administration of 100% oxygen is currently not recommended in newborns due to the associated harm, including reduced survival rates compared to breathing room air. In veterinary medicine, it is reasonable to administer flow-by oxygen as needed if respiratory issues persist after airway clearance. In addition, tactile stimulation accomplished

by rubbing of the animal with a warm towel may stimulate and improve ventilation and circulation and should be initiated as early as possible. Newborn puppies and kittens are at high risk of hypothermia, and maintenance of normothermia using heat support is important.

If the patient is apneic or gasping, ventilation should be actively supported by administering breaths with a tight-fitting face mask at a rate of 20-30 breaths per minute. A small gauge needle placement into GV26 may stimulate ventilation. Doxapram is likely not effective. The heart rate is used to guide resuscitation measures, with bradycardia being commonly associated with hypoxemia. Further intervention is recommended in newborn puppies and kittens with progressive, severe bradycardia (e.g., < 120 bpm). These interventions include positive pressure ventilation (PPV) with a tight-fitting mask, more aggressive oxygen supplementation, and use of a continuous heart rate monitor to assess for response to therapy should be considered. Atropine is likely not effective as bradycardia is the consequence of hypoxia rather than high vagal tone. Naloxone should be administered if the dam/queen received opioids prior to delivery of the newborn (0.1 mg/kg SQ, IM, preferentially IV/IO, consider intranasal/mucosal).

Resuscitation After the First 1-2 minutes

Resuscitation in newborns is fundamentally different than in older patients in that effective ventilation, as opposed to chest compressions (as recommended in adults), has primacy. Endotracheal intubation can be challenging but can be accomplished with small uncuffed endotracheal tubes or venous catheters. It is reasonable to deliver 20-40 short breaths per minute (e.g., 1 breath every 2 seconds) with chest excursion commensurate to the size of the animal to address clinical hypoxia.

With more severe bradycardia, (<50 bpm) despite optimal ventilation, chest compressions should be initiated by positioning the thumb and indicator fingers of one hand on opposite sides of the chest just over the heart by approximately 30-50% of the chest width. There are two fundamentally different aspects from adult CPR: (1) chest compressions are initiated during bradycardia, (2) effective ventilation in newborns precludes concurrent chest compression. It is recommended that compressions and ventilations be delivered at a ratio of 3:1, administered at a rate such that 120 chest compressions and 30 breaths can be delivered in a minute (i.e., 150 events per minute).

Epinephrine is less important, as the core issue is asphyxiation. However, if CPR has continued for > 2 minutes without an increase in heart rate, intravenous or intraosseous epinephrine should be considered (0.01-0.03 mg/kg IV/IO). Hypoglycemia can occur during prolonged resuscitation and should be addressed.

Conclusions

In the newborn, focus on managing asphyxia is key, even if the clinical signs are primarily bradycardia. Early intervention to support respiration is essential to newborn resuscitation.

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GARY STAMP MEMORIAL LECTURE: MONITORING DURING CPR: ECG DIAGNOSIS ALGORITHM, $ETCO_2$

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Learning objectives:

- Explain the role of ECG and ETCO2 monitoring during CPR and how they influence decision-making

- Apply an algorithmic approach to diagnosing cardiac rhythms and adjusting CPR interventions.

- Explain how direct arterial blood pressure monitoring can be used to guide vasopressor therapy during CPR

Proceeding:

ECG Rhythm Diagnosis

Although the ECG is highly susceptible to motion artifact and is of limited use during ongoing chest compressions, an accurate rhythm diagnosis is essential to guide drug and defibrillation therapy. The goal of ECG monitoring during CPR is to diagnose which of the four most common arrest rhythms are present: (1) asystole, (2) pulseless electrical activity (PEA), (3) ventricular fibrillation (VF), or (4) pulseless ventricular tachycardia (pulseless VT). Rhythms 1 and 2 are the “non-shockable” arrest rhythms and rhythms 3 and 4 are the “shockable” arrest rhythms. During the brief (< 10 seconds) pause in chest compressions between 2-minute cycles of BLS, the pulse should be palpated, and the ECG should be quickly evaluated while compressors are being rotated. If a pulse is present despite the absence of chest compressions, the patient has achieved return of spontaneous circulation (ROSC), CPR should be terminated, and the team should move on to post-cardiac arrest care.

If the patient does not have a palpable pulse, all members of the team should contribute to the ECG rhythm diagnosis using a simple algorithm.

Are there consistent, repeating complexes in the ECG?

-- If yes, is the rate > 200 per minute?

-- -- If yes, this is pulseless ventricular tachycardia (pVT), a shockable arrest rhythm.

-- -- If no, this is pulseless electrical activity (PEA), a non-shockable arrest rhythm.

-- If no, is the ECG a flat line?

-- -- If yes, this is asystole, a non-shockable arrest rhythm.

-- -- If no, this is ventricular fibrillation (VF), a shockable arrest rhythm.

The shape of any ECG complexes does not affect CPR ECG rhythm diagnosis, and over-interpretation of the ECG should be avoided. The rhythm diagnosis should be called out to the group by the team leader, and differing opinions on the diagnosis should be solicited. Discussion about the rhythm diagnosis should not prevent rapid resumption of chest compressions.

End-tidal CO_2 Monitoring

$ETCO_2$ data can be used in multiple ways during CPR, and regardless of the technology used is highly resistant to motion artifact. The presence of measurable CO_2 by $ETCO_2$ monitoring is supportive of (but not definitive for) correct placement of the endotracheal (ET) tube. If $ETCO_2$ readings are low (< 5 mmHg), rescuers should reassess the placement of the endotracheal tube using techniques such as direct visualization of the tube passing through the arytenoid cartilages or lung auscultation during pauses in compressions.

Because $ETCO_2$ is proportional to pulmonary blood flow, it can also be used as a measure of chest compression efficacy under conditions of constant ventilation at the correct rate (10 breaths per minute). The RECOVER 2.0 guidelines recommend a minimum target of 18 mmHg for $ETCO_2$ during CPR. Upon return of spontaneous circulation (ROSC), $ETCO_2$ dramatically increases due to the rapid increase in circulation and therefore is a valuable early indicator of ROSC during CPR.

Direct Arterial Blood Pressure Monitoring

If an arterial catheter is in place during CPR, direct arterial blood pressure monitoring can allow for more specific titration of vasopressor therapy, targeting a diastolic arterial blood pressure of > 30 mmHg may result in better coronary perfusion and improve myocardial oxygen delivery. Adjusting chest compressions and administering additional doses of vasopressors to patients with diastolic blood pressures below this target have the potential to improve myocardial perfusion and outcome.

GARY STAMP MEMORIAL LECTURE: POST-CARDIAC ARREST CARE AND NEUROPROTECTION

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Learning objectives:

- Devise a therapeutic and monitoring plan to meet ventilation and oxygenation goals in a patient in the post-cardiac arrest period.

- Use global hemodynamic targets to develop a plan to optimize perfusion to tissues in a patient in the post-cardiac arrest period.

- Implement neuroprotective measures to improve neurologic function and outcome in the post-cardiac arrest period.

Proceeding:

After cardiopulmonary arrest (CPA), patient outcome is largely determined by the events that led to and the duration of CPA, but the processes that occur during and after reperfusion can also play a major role. Abnormalities in dogs and cats in the PCA phase result from a combination of anoxic brain injury, postischemic myocardial dysfunction, the systemic response to ischemia and reperfusion, and the persistent precipitating pathology. Consequently, the clinical abnormalities in these PCA cases are highly variable, and therapy should be aimed at alleviating the clinical signs resulting from these abnormalities. The overall approach to PCA care consists of respiratory optimization, cardiovascular optimization, and neuroprotection.

Respiratory Optimization

Short-term mechanical ventilation to ensure optimal arterial oxygen tension (80 to 100 mmHg) and CO_2 concentration (35 to 40 mmHg), and to prevent respiratory arrest in the comatose PCA patient is optimal if available, but is not required for patients that are ventilating sufficiently. In all cases, adequate ventilation should be monitored using end-tidal CO_2 monitoring or blood gas analysis.

It important to avoid both hypoxemia and hyperoxemia in the PCA period. Hypoxemia can be treated with supplemental oxygen administered by mask, nasal cannula, nasal catheter, or using an oxygen chamber. When this is not sufficient to meet oxygenation targets (SpO_2 of 94-98%), mechanical ventilation may be required. Titration of oxygen supplementation during the PCA period to a maximum SpO_2 of 98% will reduce oxidative injury while maintaining adequate tissue oxygenation.

Hemodynamic Optimization

Central venous oxygen saturation of at least 70% or normalization of lactate should be used as global perfusion metrics and end points for resuscitation. Cerebral autoregulation of blood flow may be impaired and adequate cerebral blood flow may depend on a sufficiently high cerebral perfusion pressure. Therefore, hemodynamic optimization should be focused on maintaining a mean arterial blood pressure of 80-120 mmHg. Mild hypertension in the PCA period has been shown to be beneficial in human PCA patients, although more severe arterial hypertension should be avoided.

For hypotensive patients (MAP < 80 mmHg, SAP<100 mmHg), fluid resuscitation should be used first. Patients with evidence of peripheral vasodilation or who do not respond to fluid boluses may benefit from vasopressor therapy. PCA myocardial dysfunction is common in human PCA patients, and patients not responding to fluids and pressors may require inotropic support as well. Normotension does not necessarily equate to adequate perfusion. Once normotension is achieved, measures of oxygen delivery (central venous oxygen saturation and lactate) as previously described should be evaluated. In patients not meeting the targets despite normotension, blood transfusions may be required.

Neuroprotection

Because the cerebral cortex is more sensitive to ischemic injury than the brainstem, cortical function tends to deteriorate first, leading to patients with intact brainstem reflexes but deficits in cognitive, motor and sensory function. Markers of cellular injury like heat shock proteins 72 and 32 are severely increased in patients with ischemic injury but not in patients with hypoxia, supporting the more damaging nature of ischemia compared to hypoxia alone. The patient with CPA has both hypoxia and loss of circulatory compensation, leading to more severe neurologic injury.

There are several pathophysiologic mechanisms underlying the brain injury induced by ischemia. First, glutamate, an excitatory neurotransmitter, is released in significantly higher amounts in ischemic conditions than in purely hypoxic conditions. Excitotoxicity from excessive glutamate release leads to neuronal calcium and sodium influx as well as potassium efflux, ultimately causing cellular edema and injury. Lactic acid and hydrogen ions accumulate due to anaerobic metabolism, leading to a severe cerebral acidosis. This ischemic environment also leads to release and activation of neurotoxic enzymes, including proteases, nucleases and lipases. All of these processes contribute to brain swelling, increases in intracranial pressure (ICP), and decreased cerebral perfusion during CPR.

When large amounts of oxygen are delivered to any previously ischemic tissue, reperfusion injury can result, leading to continued tissue damage despite improved oxygen delivery. The brain is particularly sensitive to reperfusion injury when ROSC is achieved in patients with CPA. Free radical formation is an important underlying mechanism for this injury, leading to cell membrane damage, loss of ion gradients, and cell death. Iron from micro-hemorrhage, polyunsaturated fatty acids from cell membranes, and nitric oxide produced by endothelial cells can all act as sources of free radicals. Glutamate is also released from injured neurons during reperfusion, leading to further excitotoxicity and intracellular calcium accumulation.

There have been many neuroprotective drugs targeted at specific pathophysiologic processes in experimental models of HI-BI, but none have shown efficacy in actual clinical studies. It should be noted that hyperoxemia should be avoided to reduce the production of reactive oxygen species; this is achieved by titrating oxygen supplementation to maintain an arterial oxygen saturation of 94-98%. Mild hypertension has been shown in some studies to be beneficial in the immediate PCA period, and strategies to reduce blood pressure should be employed only for cases of extreme arterial hypertension. Specific neuroprotective strategies shown to be effective include mild therapeutic hypothermia, osmotic therapies such as mannitol or hypertonic saline, and seizure prophylaxis / aggressive seizure control. Although not well investigated in veterinary clinical studies, these therapies are reasonable to consider in the PCA patient with persistent neurologic deficits.

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Advanced Stream, Friday 6 June 2025

STRUCTURE AND FUNCTION OF THE ENDOTHELIUM

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Learning objectives:

- Describe the structure of the endothelial cell and endothelial glycocalyx.

- List the functions of the endothelial cell.

- List the functions of the endothelial glycocalyx.

- Describe the role of the endothelial cell and endothelial glycocalyx in regulating vascular permeability.

Proceeding:

Endothelial Cell Structure

All blood vessels in the body are lined by a single layer of endothelial cells (ECs). ECs are anchored to the basal lamina, which is the main scaffold of the vessel. ECs may be linked to each other by three types of cell-to-cell junctions, depending on the type of vessel and permeability requirements. Tight junctions create a continuous endothelium, adherens junctions create a fenestrated endothelium, and gap junctions create a discontinuous endothelium.

Endothelial Glycocalyx Structure

The endothelial glycocalyx (EG) is a gel matrix that lines the luminal aspect of blood vessels, between the blood and the EC. The components and thickness of the EG varies throughout the vasculature. The EG is composed of proteoglycans, glycoproteins, and glycosaminoglycans. It has a net negative charge, which is largely due to the glycosaminoglycan side chains. The EG and other soluble components, including albumin and anticoagulants, that are suspended in a plasma layer are called the endothelial surface layer (ESL).

Functions of the Endothelial Cell

Hemostasis / Thrombosis / Fibrinolysis

ECs play an important role in hemostasis, thrombosis, and fibrinolysis. In normal conditions, ECs have an anti-thrombotic, anti-coagulant, and pro-fibrinolytic phenotype (Feletou 2011).

Regulation of vascular tone

NO is a vasodilator formed in the EC. It has an important role in the maintenance of basal vasodilator tone of the blood vessels and is also important in preventing platelet and leukocyte activation and adhesion. Endothelium-derived hyperpolarizing factor is another important vasodilator.

The balance in production of PGI_2 and TXA_2 is very important to homeostasis. PGI_2 allows for relaxation of the smooth muscle and TXA_2 causes platelet aggregation and vasoconstriction. Endothelin is a vasoconstrictor which is stimulated by the production of inflammatory molecules.

Leukocyte trafficking

The steps of leukocyte trafficking generally include capture, rolling, adhesion/arrest, and diapedesis. Under normal conditions, leukocytes do not adhere to endothelial cells. After activation, the leukocyte binds to the EC and the rolling process begins. This process relies on the expression of adhesion molecules on both the EC and the leukocytes.

Regulation of Vascular Permeability

In health, continuous and fenestrated endothelium are impermeable to macromolecules. However, cytokines and other inflammatory mediators can disrupt the tight and adherens junctions between ECs.

Functions of the Endothelial Glycocalyx

Regulation of microvascular tone

The EG acts as a mechanotransducer as it transmits the forces of shear stress or increased flow to the ECs. This force leads to release of nitric oxide from the EC, causing vasodilation. In this way, the EG acts as an effector of metabolic coupling between organ function and local hemodynamics.

Regulation of fluid movement and vascular permeability

The revised Starling principle demonstrates the importance of the EG in the regulation of transvascular fluid movement. The EG has an important role in the regulation of vascular permeability through its net negative charge and structure. GAGs are important contributors to the net negative charge of the EG. The fiber-matrix theory describes the EG as a fibrous mesh that acts as a molecular sieve for the vessel and explains why the EG excludes large molecules but is permeable to small molecules.

Regulation of leukocyte trafficking

In health, the EG is anti-inflammatory and the net negative charge of the EG repels WBCs. In addition, the EG completely covers WBC adhesion molecules which are expressed at the base of the EG near the EC. This prevents circulating WBCs from binding adhesions molecules in normal conditions.

Inhibition of coagulation

In health, the EG exhibits anti-coagulant properties. The net negative charge of the EG repels RBCs and helps to maintain laminar flow within the vessel. In addition, the thickness of the EG prevents platelets from interacting with von Willebrand factor and platelet adhesion molecules located on the EC. There are many anticoagulant molecules located within the EG, including antithrombin. Thrombomodulin is

expressed on the EC, where it plays an important role in activating the protein C anticoagulant pathway. Tissue factor pathway inhibitor plays an important role in inhibiting thrombosis formation through inhibition of the TF-FVII complex (Gaudette 2020).

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ENDOTHELIAL DYSFUNCTION IN SEPSIS

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Learning objectives:

- Describe the endothelial changes that occur in sepsis.

- Describe the differences in endothelial dysfunction in different organs.

- Summarize the techniques and limitations of research techniques for studying the endothelium.

- Summarise the recent animal literature on endothelial dysfunction in sepsis.

Proceeding:

Controlled endothelial cell (EC) activation during infection is required to limit bacterial dissemination. However, an unregulated host response can result in inappropriate systemic EC activation. Consequences of EC dysfunction include altered neutrophil trafficking, decreased barrier function, prothrombotic phenotype and dysregulated NO metabolism. These can all contribute to microcirculatory dysfunction, which contributes to multiorgan involvement in patients with sepsis.

Despite an identical genome, ECs have heterogeneous phenotypes that are influenced by environmental signals from the tissue microenvironment. This is particularly apparent in organs with highly specialized vasculature, e.g., kidneys, liver, and lungs. Although some common modes of septic endothelial dysfunction exist, organ-specific EC dysfunction also occurs. For example, ECs in fenestrated glomerular capillaries are especially vulnerable to ultrastructural alterations leading to reductions in glomerular filtration rate. In the liver, loss of the ‘sieve-plate architecture’ of sinusoidal ECs contributes to neutrophil transmigration. The pulmonary circulation is unique in that transmigration occurs preferentially from the capillary bed. Different septic endotypes are likely to have divergent effects on ECs.

There is a myriad of techniques for assessing the endothelium but all have their limitations.

Biomarkers include shed adhesion molecules or glycocalyx products, and these have been associated with sepsis severity. However, many studies are limited by a lack of standardized assays and do not report threshold values or receiver operating characteristics. Alternative sources of these EC biomarkers (e.g., epithelial and immune cells) and mechanisms of shedding and clearance (e.g., as an anti-inflammatory response or following fluid resuscitation) also currently limit their prognostic use. Animal models of EC dysfunction in sepsis cannot mimic the heterogenous phenotypes seen in clinical septic populations, nor the presence of chronic EC dysfunction. However, genetic knock-out mice are useful for investigating specific mechanisms and trialing therapeutics. Experiments requiring culture of ECs are

complicated by phenotypic drift that occurs when ECs are uncoupled from extracellular cues, e.g., shear stress and signaling from the microenvironment. Some of this can be mitigated by culturing in physiologically relevant flow conditions and using organ-specific primary endothelial cells. In spite of their limitations, they facilitate repeatable exploration of specific pathways identified in animal models or clinical settings. Techniques such as sidestream darkfield imaging to visualize microvascular abnormalities have been used to try and stratify septic populations for more personalized treatment, but are still limited due to equipment availability, challenges in image acquisition and lack of robust data for clinical application. High-throughput -omics has the potential to both characterize molecular mechanisms and identify septic endotypes. A better mechanistic understanding of EC dysfunction in sepsis, especially across organs damaged early on, such as the lungs, liver, and kidneys, may elucidate new therapeutics, whilst establishing endotypes may help stratify patients for treatments. Unfortunately, -omics in relation to EC dysfunction is still in its infancy.

Studies on EC dysfunction in small animals are limited. Currently validated assays exist only for hyaluronic acid, VEGF, and angiopoietin. Those that exist have shown increased endothelial biomarkers in sepsis, and angiopoietin has been shown to discriminate survivors from non-survivors.

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HOSPITAL-ACQUIRED AKI

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Learning objectives:

- Identify risk factors that can lead to hospital-acquired AKI

- Understand AKI diagnosis, including the utility of scoring systems

- Detail a rational treatment approach to hospital-acquired AKI

Proceeding:

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 aetiology. 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 (normovolaemia/euvolaemia) 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 haemodynamic 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 (see “Urine AKI Biomarkers”).

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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LEPTOSPIROSIS: FROM INJURY TO RECOVERY

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Learning objectives:

- Review the pathophysiology of leptospirosis.

- Detect organ injury by Leptospira, from the classic ones to the less common.

- Choose the best diagnostic test.

- Adapt prognosis to each clinical presentation.

Proceeding:

Leptospira is a spirochete bacteria responsible for leptospirosis. This zoonotic disease is widespread worldwide and primarily infects dogs among companion animals, although cats can also be infected or act as reservoir hosts. Although there are hundreds of serovars and serogroups, only some strains are pathogenic. In dogs, *Leptospira interrogans*, *Leptospira borgpetersenii*, and *Leptospira kirschneri* are the main pathogenic strains.

Exposure occurs through contact with the bacteria shed from the renal tubules of reservoir hosts into the environment. The bacteria enter the organism via mucous membranes, leading to bacteremia. The bacteria can disrupt the endothelial membrane, allowing it to infiltrate tissues and damage the vascular endothelium. Its primary tropism is for the kidneys and liver, but other organs and systems can also be affected, resulting in severe systemic disease. By invading the renal tubules, the bacteria typically cause acute tubulointerstitial nephritis, resulting in varying degrees of acute kidney injury. In the liver, hepatocyte function can be impaired, leading to liver dysfunction. However, more commonly, the bacteria disrupt the intercellular junctions of hepatocytes, resulting in bile leakage and leading to cholestatic hepatopathy. Biliary mucocele is also reported in up to 30% of dogs affected. Leptospiral pulmonary hemorrhage syndrome (LPHS) is a complex and severe form of leptospirosis that may result from a combination of the bacteria's ability to disrupt the endothelial cell barrier and the host's immune response and state of systemic inflammation. Coagulation disorders are frequently reported, with common findings including thrombocytopenia, disseminated intravascular coagulation, and dysfibrinogenemia. These result from a combination of direct leptospiral effects, liver dysfunction, and systemic inflammation. Virtually all organs can be invaded by the bacteria, and some clinical

manifestations of leptospirosis that are reported, though less commonly, include intussusception, myocarditis, myositis, pancreatitis, uveitis, and meningitis.

Diagnosis can be challenging, depending on the stage of the disease and clinical factors such as prior antimicrobial use. The gold standard is a coupled MAT serological test, but a combination of clinical suspicion, biological markers, and judicious use of available serological and pathogen detection tests can help confirm the diagnosis.

Etiological treatment is based on antimicrobial therapy. Intravenous ampicillin, amoxicillin, or penicillin G should be initiated, followed by oral doxycycline once the patient has shown good tolerance, with doxycycline continued for two weeks. Supportive treatment should also be provided, tailored to the specific organ dysfunction of each patient. Pain management, antiemetics, and nutritional support can benefit all patients. Some may require oxygen therapy. Particular attention should be given to fluid balance, electrolytes, and acid-base disturbances. Extracorporeal kidney support therapy can be indicated in a subset of patients with AKI.

Prognosis is variable. Mortality up to 70% is described in a case of leptospiral pulmonary hemorrhage syndrome. Identified negative prognostic factors include anuria, respiratory complications, hyperbilirubinemia, hypocoagulability, and pancreatitis. Some biomarkers may help to sharpen the prognosis.

Vaccination is considered the most effective way to prevent leptospirosis. Vaccines are available for some of the most common serovars of Leptospira. Hygiene and control of rodent populations are also essential preventive measures.

Once the patient's physical condition improves, the azotemia stabilizes, and it is non-dependent on fluid therapy, discharge can be considered. Some patients will still be azotemic at this time and need a follow-up for several months to years, as full renal regeneration or chronic kidney disease may occur. Today, no biomarker allows the clinician to anticipate renal regeneration.

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URINE AKI BIOMARKERS

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Learning objectives:

- Understand the physiology of urine AKI biomarkers.

- Describe the potential utility of urine AKI biomarkers in clinical practice and research.

- Summarize the current evidence for major urine AKI biomarkers.

Proceeding:

Early identification of acute kidney injury (AKI) is challenging. Traditional serum/plasma biomarkers of glomerular filtration rate (GFR) detect AKI relatively late. The need for an earlier biomarker of active AKI has led to research in a breadth of potential urine AKI biomarkers. Most of these biomarkers are posited to detect tubular injury, either active tubular damage and/or tubular dysfunction from previous damage. The ability to detect early AKI with these biomarkers may serve two distinct purposes. The first is as a sensitive research tool for detecting the potential of a disease, intervention, or other physiological alteration to cause AKI. The other is as a clinical diagnostic test for the identification of AKI in at-risk veterinary patients. Whilst some urine AKI biomarkers have been successfully used for research purposes, clinical utility remains in its infancy.

Indexation of Urine Biomarkers

A major challenge in measuring urine biomarkers is the large variation in urine concentration. Thus, most studies index urine biomarkers to a marker of urine concentration, most frequently urine creatinine concentration. An alternative is indexation to urine osmolality, which may be more accurate but is harder to measure. The final approach is to index to total urine output, which is often not practical in veterinary medicine.

Neutrophil Gelatinase-Associated Lipocalin

Neutrophil gelatinase-associated lipocalin (NGAL) is both a marker of active tubular injury and tubular dysfunction. It is produced by many cell types in the body, freely filtered at the glomerulus, and reabsorbed by tubular cells. In AKI, there is decreased reabsorption from dysfunctional tubular cells. Furthermore, injured tubular cells secrete further NGAL into the urine. Increased urine NGAL may also result from systemic or urinary tract inflammation. Measurement of NGAL currently requires immunoassay techniques, limiting its utility to a research setting. Immunoassays for NGAL may vary in which forms of NGAL are detected. Measurement of serum NGAL concentration and calculation of the

urine-to-serum NGAL ratio can aid in ruling out increases due to excessive circulating NGAL from systemic inflammation. Evaluation of urine cytology for the presence of pyuria can aid in determining if lower urinary tract infection should be considered. Research use of NGAL has included detection of AKI in models of haemorrhagic shock and fluid resuscitation. Several studies have evaluated urine NGAL in naturally occurring AKI in small animals. There is evidence that urine NGAL concentration differs between dogs with AKI and healthy dogs, dogs with chronic kidney disease, and dogs with lower urinary tract diseases. There is also evidence that it may predict postoperative AKI in dogs.

Gamma-Glutamyl Transferase and Alkaline Phosphatase

Gamma-glutamyl transferase (GGT) and alkaline phosphatase (ALP) are present as brush border enzymes in the proximal tubular cells. Increases in the urine activity of these enzymes reflect active tubular injury. They are not filtered by an intact glomerulus. However, prostatic fluid contains ALP. An advantage of these biomarkers is that they can generally be measured by the widely available and inexpensive traditional biochemical techniques used to measure their activity in serum. A major disadvantage of these enzymes is their instability under storage. Thus, it is recommended to measure these biomarkers immediately on fresh urine. Furthermore, acidic or alkaline urine can decrease enzyme activity. Urine GGT has been used to detect AKI in experimental models, including our haemorrhagic shock model. Other studies have investigated the utility of these enzymes, mostly GGT, in naturally occurring kidney disease.

Cystatin C

Cystatin C is a 14 kDa cysteine protease inhibitor that is released at a relatively constant rate by most nucleated cells, freely filtered by the glomerulus, and completely reabsorbed and catabolized by functional proximal tubular epithelial cells. An increased concentration in the urine reflects proximal tubular cell dysfunction. Measurement of cystatin C requires immunoassay techniques. ELISA, multiplex assays, and a particle-enhanced turbidimetric immunoassay (PETIA) are available. Cystatin C has been measured in several experimental models. Clinical research is limited.

Cystatin B

The 11 kDa cysteine protease inhibitor cystatin B, in contrast to cystatin C, does not circulate in measurable concentrations in health. It is released from renal tubular cells during active injury. Species-specific ELISA assays are necessary, as the protein structure in cats and dogs differs from that in humans. A commercial laboratory now offers a proprietary agglutination immunoassay, though validation data do not seem to be publicly available. Cystatin B has not yet been widely used to detect AKI in experimental models. A small number of clinical studies published in the last 5 years have reported preliminary data about its utility in naturally occurring clinical disease.

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IV FLUIDS FOR NEPHROTOXINS – WHAT'S THE EVIDENCE?

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Learning objectives:

- Describe the evidence for fluid therapy for nephrotoxin exposure.

- Create individualised fluid therapy plans for patients exposed to common nephrotoxins.

Proceeding:

Exposure to potentially nephrotoxic substances is a common reason for dogs and cats to be presented to emergency practices. Common examples include non-steroidal anti-inflammatory drugs (NSAIDs), lilies (for cats), and grapes (for dogs). In addition to gastrointestinal decontamination, most references recommend admission to hospital for administration of intravenous fluid therapy (IVFT) at greater than calculated maintenance requirements (usually two to three times) for several days (usually 48-72 hours). The stated rationale is that this will induce a diuresis, which results in more rapid renal excretion of the toxin and/or prevention of processes causing acute kidney injury (AKI) in the response to the toxin. This assertion is not supported by evidence and does not have a strong physiologic rationale.

But doesn't inducing a diuresis enhance renal excretion of the toxin and prevent tubular damage?

Clearly, the administration of above maintenance IVFT to these animals results in the production of more urine. The commonly stated rationale for IVFT diuresis explains this phenomenon with an increase in glomerular filtration rate (GFR), which both aids toxin elimination and increases tubular flow rate. These factors are suggested to prevent AKI. However, the physiology is not that simple. The effect of IVFT on urine output is mostly not a direct renal effect but mediated by increased production of atrial natriuretic peptide (ANP), with a myriad of effects on the kidney. Many of them, such as decreased sodium reabsorption in the distal nephron, are unlikely to have any beneficial effect in this context. Furthermore, ANP can have detrimental effects, such as inducing shedding of the endothelial glycocalyx. Given the complexity of the physiology, knowing whether increased ANP enhances elimination of toxins would require pharmacokinetic studies for the specific toxins, which have not been performed. Whether the effects of ANP would limit AKI would depend on the mechanism of nephrotoxicosis, and thus require research for each toxin of interest. Notably, the mechanism of many common toxins has not been well described.

But surely there is some evidence that such a common approach is helpful?

Unfortunately, just because a medical practice is common does not mean it is evidence-based. No veterinary studies evaluating the efficacy of this technique could be located. In human medicine, 'forced diuresis' by administration of IVFT alone, or more commonly in combination with a loop diuretic, has fallen out of favour due to a lack of evidence of benefit and the potential for harm.

But shouldn't we give IVFT just in case? After all, it won't do any harm, will it?

Most otherwise healthy dogs tolerate above maintenance IVFT, because they can increase urine output as discussed above. However, if AKI does begin to develop due to the nephrotoxin, the ability to excrete excess fluid becomes impaired and hypervolaemia can rapidly result. Early signs of hypervolaemia are subtle and easy to miss, and fluid overload with resultant interstitial oedema can easily occur. Interstitial oedema is harmful to many organs, through mechanisms including impairment of oxygen and nutrient diffusion through the expanded interstitial space, distortion of normal tissue architecture, impaired paracrine signaling, impairment of perfusion, and obstruction of venous and lymphatic outflow. The kidney is exceptionally sensitive to the adverse effects of interstitial oedema due to its rigid capsule, meaning that oedema results in increased intraparenchymal pressure that opposes perfusion and glomerular filtration. Thus, overzealous IVFT may actually contribute to AKI, rather than preventing it.

So, are you saying we should never give nephrotoxin-exposed animals IVFT?

Until there is clear evidence that the theoretical benefits exist and outweigh the risks, I do not recommend a mandatory period of above maintenance IVFT. This does not mean that IVFT should never be administered to nephrotoxin-exposed animals. Individualised therapy is recommended. The core guiding principle for IVFT is that it should be used to achieve and maintain normal blood volume (normovolaemia/euvolaemia) and normal interstitial hydration (normohydration/euhydration). For many animals, this is achieved in less than the traditional 48-72 hour recommended period of IVFT.

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EXTRACORPOREAL THERAPIES FOR ACUTE CONGESTIVE HEART FAILURE AND DIURETIC RESISTANCE

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Learning objectives:

Ultrafiltration is a therapeutic option for acute congestive heart failure (CHF) with diuretic resistance or fluid overload. By removing excess fluid, it alleviates pulmonary congestion and improves hemodynamics. Ultrafiltration offers precise volume control, reducing hospitalizations and symptoms. However, it requires specialized equipment and expertise, carries risks like hypotension, and is costly. Careful patient selection and monitoring are essential to maximize benefits while minimizing complications in acute CHF management.

Proceeding:

Acute congestive heart failure (CHF) is a critical condition that often requires aggressive medical management to alleviate fluid overload and restore hemodynamic stability. While diuretics remain the cornerstone of treatment, a subset of patients exhibit diuretic resistance, necessitating alternative therapeutic strategies. Extracorporeal therapies, including ultrafiltration (UF), hemodialysis, and hemofiltration, have emerged as viable options for managing fluid overload in these patients. Diuretic resistance occurs when patients fail to respond adequately to loop diuretics, despite escalating doses. Contributing factors include sequential renal tubular hypertrophy, chronic kidney disease (CKD), neurohormonal activation, sodium retention, and vasopressin-induced free water accumulation. Prolonged use of loop diuretics can also lead to maladaptive renal changes, reducing their efficacy over time. In such cases, extracorporeal therapies offer an alternative means of fluid removal. Ultrafiltration is a mechanical process that removes isotonic fluid directly from the plasma via a semipermeable membrane. This therapy provides controlled fluid removal, independent of renal function. UF has been shown to be particularly beneficial in patients with volume overload refractory to diuretics. The UNLOAD trial in humans demonstrated that UF leads to greater fluid removal and reduced re-hospitalization rates compared to intravenous diuretics. For CHF patients with concurrent renal impairment, hemodialysis may be required. Hemodialysis removes both fluid and solutes, making it beneficial for patients with electrolyte disturbances or severe azotemia. Extracorporeal therapies offer several advantages over conventional diuretic therapy.

These include:

- Predictable Fluid Removal: Unlike diuretics, which rely on renal function, these therapies provide direct and controlled fluid removal.

- Reduction in Neurohormonal Activation: Diuretics can stimulate the renin-angiotensin-aldosterone system (RAAS), worsening CHF progression. UF mitigates this response.

- Enhanced Sodium and Fluid Removal: Unlike diuretics, which promote sodium excretion through the renal tubules, UF removes isotonic fluid, reducing the risk of worsening sodium retention.

- Improved Symptoms and Outcomes: Studies in humans suggest that patients receiving UF experience reduced symptoms of congestion and improved quality of life compared to those on diuretics alone. Despite their benefits, extracorporeal therapies come with several challenges. These include the need for specialized equipment and trained personnel, risks of vascular access-related complications, potential hypotension, and electrolyte imbalances. Additionally, the cost of extracorporeal therapies may limit their widespread use.

EXTRACORPOREAL THERAPIES FOR SEPSIS

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Learning objectives:

Extracorporeal removal of cytokines and lipopolysaccharides (LPS) offers a novel approach in managing septic shock by targeting the dysregulated immune response. Techniques such as hemoadsorption and high-cutoff hemodialysis aim to reduce excessive pro-inflammatory cytokines, mitigating the cytokine storm. LPS removal via polymyxin B hemoperfusion directly neutralizes endotoxins, improving hemodynamics and potentially reducing mortality. While these therapies show promise, evidence remains limited, and clinical benefits vary. They require specialized equipment, carry risks like hypotension or clotting, and increase costs. Optimizing timing, patient selection, and integration with standard care is essential to harness their potential in improving septic shock outcomes.

Proceeding:

Sepsis and septic shock are life-threatening conditions resulting from a dysregulated host response to infection, leading to widespread inflammation, secondary organ dysfunction, and high mortality rates. Despite advances in antimicrobial therapy, fluid resuscitation, and vasopressor support, mortality remains significant. Extracorporeal therapy, including extracorporeal blood purification (EBP) techniques such as hemoperfusion, high-volume hemofiltration, hemodialysis (HD), and extracorporeal membrane oxygenation (ECMO), has emerged as a potential adjunct in managing sepsis and septic shock. Sepsis involves a complex interaction between pro-inflammatory and anti-inflammatory responses, often leading to endothelial dysfunction, microcirculatory failure, and multiple organ dysfunctions. In septic shock, persistent hypotension and cellular dysfunction contribute to mortality. Traditional treatment focuses on infection control, hemodynamic support, and organ support, but these measures may not adequately remove circulating inflammatory mediators. Extracorporeal therapies aim to eliminate these mediators, modulate the immune response, and provide organ support in critically ill patients. Hemoperfusion involves passing blood through an adsorbent material to remove toxins and inflammatory mediators. Hemoadsorption devices such as CytoSorb and Toraymyxin target cytokines and endotoxins, respectively. Studies suggest that these therapies may help stabilize hemodynamics, reduce vasopressor requirements, and improve organ function, though their impact on mortality remains uncertain. High-Volume Hemofiltration (HVHF) and Mid-Cut-Off (MCO) Membrane hemodialysis are used in septic patients with acute kidney injury (AKI) to provide renal support and remove inflammatory mediators. They involve the removal of large plasma volumes with fluid replacement to reduce cytokine burden. Despite promising theoretical benefits, clinical trials have yet to demonstrate a definitive mortality benefit. Plasma Exchange (TPE) involves removing and replacing plasma to eliminate

immune complexes. It is mainly considered in conditions such as septic shock complicating autoimmune or immune-mediated processes or thrombotic microangiopathies. Limited evidence supports its routine use. Extracorporeal Membrane Oxygenation (ECMO) is used in refractory septic shock or sepsis-induced cardiopulmonary failure. It provides circulatory and respiratory support, allowing sepsis treatment and organ recovery time. Despite its ability to sustain patients in critical conditions, ECMO is associated with high complication rates, including thrombosis, bleeding, and secondary infections. Extracorporeal therapies offer several potential benefits, including:

- Reduction of Inflammatory Mediators: Removing cytokines and endotoxins may prevent excessive immune activation and reduce organ dysfunction.

- Hemodynamic Stabilization: Some studies report decreased vasopressor dependence and improved perfusion following extracorporeal interventions.

- Organ Support: HD and ECMO support renal and cardiopulmonary function in critically ill patients. However, challenges remain:

- Lack of Definitive Evidence: Despite theoretical advantages, randomized controlled trials have not consistently demonstrated a survival benefit.

- High Cost and Resource Utilization: These therapies require specialized equipment, trained

- Potential Complications: Vascular access-related infections, hemodynamic instability, and coagulopathy are notable risks.

Extracorporeal therapies hold promise in sepsis and septic shock management by targeting inflammatory mediators and providing organ support. While current evidence suggests potential benefits in select patient populations, widespread implementation is hindered by high costs, technical complexity, and inconsistent clinical outcomes. Further research is necessary to define the optimal use of extracorporeal therapy in sepsis management. Until then, these interventions should be considered on a case-by-case basis in critically ill patients with refractory sepsis.

Nurse & Tech Stream, Friday 6 June 2025

ADVANCED NURSING TECHNIQUES: CENTRAL VENOUS CATHETER AND DIALYSIS CATHETER MANAGEMENT

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Learning objectives:

This lecture covers the essential role of advanced nursing skills in handling central venous catheters (CVCs) and dialysis catheters in veterinary critical care. Attendees will:

- Understand the indications and contraindications for CVC and dialysis catheter placement.

- Learn the aseptic techniques for insertion and maintenance of these catheters.

- Explore best practices to prevent complications such as infection, thrombosis, and catheter occlusion.

- Gain insight into the role of dialysis in veterinary medicine, why central venous catheters are necessary, and how dialysis functions

Proceeding:

Introduction

Central venous and dialysis catheters are crucial in veterinary emergency and critical care. They allow for essential treatments, frequent blood sampling, and, in some cases, life-saving dialysis. Proper placement, maintenance, and infection prevention are key to successful treatment. Veterinary nurses play a vital role in ensuring these catheters are handled safely and effectively.

Central venous catheter

CVCs are used when patients need multiple IV medications, continuous fluids, frequent blood tests, or central venous pressure monitoring. They provide more stable and long-term access than standard IV catheters. However, they should not be used in animals with blood clotting problems, infections at the insertion site, or abnormal blood vessels.

Placing a CVC requires strict sterility. The most common method is the Seldinger technique, which involves inserting a needle into a large vein (mostly jugular), advancing a guidewire, and then threading the catheter over it. The guidewire is then removed, and the catheter is flushed with sterile saline or heparinized solution. Proper fixation and a chest X-ray help confirm placement and rule out complications. The site must be checked daily for infection, the catheter flushed regularly, and dressings changed when dirty or loose.

Dialysis in Veterinary Medicine

Dialysis is a life-saving treatment for animals with severe kidney problems, dangerous electrolyte imbalances, or poisoning. Hemodialysis, the most common method, filters toxins from the blood using a machine before returning the blood to the patient. It removes waste products like urea and creatinine while maintaining electrolyte balance.

To perform hemodialysis, a large central vein is needed for high blood flow. Peripheral veins are not suitable, so a special dialysis catheter is placed, usually in the jugular vein. These catheters are larger than standard CVCs and have two tubes—one for withdrawing blood and the other for returning it after filtration.

Complications and Nursing Considerations

Veterinary nurses play a crucial role in preventing and managing complications related to CVCs and dialysis catheters. The main risks include:

Infection: Signs like fever, redness, swelling, or pus at the insertion site should be quickly addressed. Infection can lead to longer hospital stays and serious illness. To reduce infection risk, strict hygiene, proper handwashing, and minimal handling of the catheter are essential.

Blood clots and blockages: Regular flushing with saline or heparinized solution helps prevent clot formation. If a blockage occurs, clot-dissolving medications may be needed.

Torsion and kinking: The catheter may twist or fold at the insertion point, reducing or blocking blood flow. Nurses should monitor for resistance during flushing and aspiration and ensure the catheter remains in a straight position.

Patient monitoring: Vital signs, hydration, and electrolyte levels should be closely watched, particularly in dialysis patients. Rapid fluid shifts can cause low blood pressure.

Pain management: Some patients may feel discomfort after catheter placement. Proper pain relief should be provided, and any signs of pain should be addressed quickly.

Conclusion

CVCs and dialysis catheters are essential in veterinary emergency and critical care, providing crucial access for treatments and life-saving procedures. Proper placement, maintenance, and prevention of complications are key to successful outcomes. Veterinary nurses play a vital role in managing these catheters, making advanced nursing skills essential for critical care practice.

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THE STORY OF MURRAY: POST ARREST NURSING CARE - A CASE DISCUSSION

Melissa Evans [1]

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Learning objectives:

- Review the RECOVER Guidelines on Post-Arrest Care

- Identify the complications that can come from resuscitation

- Understand the mechanisms that lead to post-arrest syndrome

- Discuss the special patient care considerations for post-arrest patients

Proceeding:

The main goal of performing cardiopulmonary resuscitation (CPR) is a return of spontaneous circulation (ROSC). However, that is not the final goal, as only 2-10% of patients who achieve ROSC after cardiopulmonary arrest (CPA) survive to discharge. It is believed that this is due to post-arrest syndrome, a combination of the effects of CPA and ROSC. The interventions that are initiated post ROSC can improve a patient's outcome significantly.

Post-arrest syndrome

Post-arrest syndrome is defined as the consequences that occur as a result of ROSC. These include tissue ischemia and hypoxemia, myocardial dysfunction, reperfusion injury and any underlying condition which may have led to the arrest. Reperfusion injury is damage that is caused by the return of blood flow to the tissues. The reintroduction of oxygen from blood flow to tissues that have been deprived of oxygen produces inflammatory cytokines. This inflammatory response leads to increased permeability of the capillaries which contributes to coagulopathies and intracranial pressure. Gastrointestinal damage from reperfusion injury leads to bacterial translocation and sepsis. These sequelae can lead to multiple organ dysfunction syndrome (MODS), systemic inflammatory response syndrome (SIRS) and disseminated intravascular coagulation (DIC). Treatment for post-arrest syndrome is focused on providing cardiovascular and respiratory support to maintain tissue perfusion.

Recover initiative algorithm

The RECOVER Initiative guidelines have provided an algorithm for post-cardiac arrest care which highlights respiratory optimization, hemodynamic optimization and neuroprotection to improve the chances a patient will survive to discharge and beyond.

Respiratory optimization

It is recommended to avoid both hyper and hypoxemia to prevent oxidation injury that could exacerbate reperfusion injury. Maintaining normocapnia is also important to prevent further neurologic damage. Pulse oximetry (SPO2) should be maintained at 94-98% while arterial partial pressure of carbon dioxide (PaCO2) should be kept at 32-43 mmHg for dogs and 26-36 mmHg for cats. Mechanical ventilation may be warranted if patients are not spontaneously breathing.

Hemodynamic optimization

Arterial blood pressure is the force that brings oxygenated blood to the tissues. Keeping a mean arterial pressure of 80-120 mmHg ensures that all tissues are perfusing appropriately. Patients are often hypotensive post-arrest. Fluid boluses, vasopressors or a positive inotrope can help achieve normotension. Studies have shown that mild hypertension is beneficial after ROSC. Moderate to severe hypertension is contraindicated however and analgesia or an anti-hypertensive may be necessary.

Lactate is a product of anerobic metabolism, making it a reliable indicator of tissue hypoperfusion. It should be less than 2.5 mmol/L, levels higher than this are associated with increased mortality.

Neuroprotection

The brain is particularly sensitive to ischemic injury. Common neurological sequela include coma, blindness and seizures. Neurological exams are recommended every 2-4 hours for the first 48 hours. Hyperthermia relates to increased mortality. Patients should be warmed up slowly, no more than 0.5°C per hour. Therapeutic hypothermia has been shown to be beneficial to ROSC patients. This involves cooling a patient down to 32 – 34° Celsius for the first 24 to 48 hours post-arrest. These patients need sedation, mechanical ventilation and intense nursing care making it difficult in most clinics. Increased intracranial pressure (ICP)should be suspected in any patient where respiratory and hemodynamic optimization has been achieved but has neurologic deficits and can be treated with osmotic agents. Seizures may occur and can be treated with benzodiazepines.

Nursing care

Patients will be attached to continuous ECG, oxygen supplementation, blood pressure, pulse oximetry and end-tidal carbon dioxide monitoring. Multiple catheters may be needed to allow for numerous infusions. A central jugular line is contraindicated due to the risk of increased ICP. Instead, a multilumen peripheral central line can be used. An arterial catheter for blood gases is ideal for these patients. Urinary ins and outs need to be monitored for these patients. As they are often unable to walk or move themselves, it is recommended to place a urinary catheter. In addition to making measuring urine production simple, this will help keep the animal clean and dry. After ROSC many animals are recumbent or comatose, necessitating recumbent patient care. Passive range of motion exercise, flipping sides, oral and eye care will need to be performed at least every 4 hours. Elevating the head 30 degrees can encourage venous drainage and help ICP.

Prognosis

Prognosis is varied. Poor prognostic signs include extended unconsciousness, absence of reflexes and absence of spontaneous breathing. Positive prognostic indicators include awareness of surroundings, appropriate reflexes and spontaneous breathing.

Conclusion

Using the RECOVER Algorithm can help veterinary staff ensure patients in the post-arrest phase are being appropriately treated and give them the best chance to survive to discharge.

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STABILISATION AND NURSING CONSIDERATIONS FOLLOWING A SUSPECTED TOXIN INGESTION

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Learning objectives

- Understand the steps involved in the initial stabilisation of the toxicological emergency patient.

- Recognise the nursing considerations for the initial and ongoing management of a patient following toxin ingestion.

Proceeding:

Presentation and initial management

A 6-year-old female neutered Miniature Schnauzer dog presented in status epilepticus, with horizontal nystagmus, cyanotic mucous membranes and severe hyperthermia following suspected ingestion of an unknown toxin. The patient presented as a first opinion out of hours emergency and was transferred to the emergency and critical care referral department for initial and ongoing management. Emergency stabilisation comprised of rectal administration of diazepam, gaining venous access, flow-by oxygen supplementation and evaporative cooling. Intravenous fluid therapy was administered, and bolus administration of sedation and anti-epileptic medication included diazepam, midazolam and levetiracetam, which seizures were refractory to. Continuous rate infusions of midazolam and dexmedetomidine were started, intravenous lipid emulsion therapy was initiated, and methocarbamol was administered per rectum. Patient mentation improved slightly, however, regurgitation, ongoing seizures and worsening anxiety resulted in anaesthetising with propofol and intubating to secure the airways. Tremors ceased upon use of sevoflurane as an inhalant anaesthetic, and manual ventilation was required under general anaesthetic due to apnoeic episodes. Stabilisation occurred in the Emergency Room, and the patient was transferred to the High Dependency Unit of the Intensive Care Unit for monitoring and intensive nursing care.

Diagnostics

Blood sampling was performed via the newly placed intravenous catheter on admission. Blood gas analysis indicated a marked hypocapnia, hyperkalaemia, hyperlactataemia and hyperglycaemia. Haemoconcentration was evident from an elevated packed cell volume and total solids. In-house coagulation testing was performed due to bruising at venepuncture sites; this revealed a coagulopathy, with parameters above the reportable range. No abnormalities were detected on point of care ultrasound of the thorax and abdomen on admission. B-lines were noted bilaterally the following day, consistent with findings suggestive of aspiration pneumonia.

Treatment

Gastric lavage was performed under general anaesthetic via orogastric tube, with retrieval of green-brown gastric fluid and no obvious toxin or foreign body. Activated charcoal was used for gastrointestinal decontamination via the orogastric tube. Several hours after admission, a fresh frozen plasma transfusion was administered in response to the coagulopathy. Hyperosmolar therapy with hypertonic saline was initiated 12 hours after presentation due to episodes of hypertension, bradycardia and miotic pupils. Antimicrobial therapy with amoxicillin-clavulanic acid was initiated upon suspicion of aspiration pneumonia. Intensive nursing care was required for ongoing monitoring and supportive therapies.

Nursing considerations

Veterinary nursing care for this patient was varied, involving acute management of status epilepticus and hyperthermia, ongoing general anaesthetic monitoring, and the necessary critical care nursing necessary during hospitalisation. Key nursing considerations throughout included careful monitoring of:

- The cardiovascular system, considering cardiovascular stability and perfusion parameters.

- The respiratory system, considering management of airways and the potential and actual risk of aspiration pneumonia.

- The neurological system, considering the identification of seizure activity.

- Temperature, considering active cooling and warming methods.

- General anaesthetic, considering initial manual ventilation and the depth of anaesthesia.

- Fresh frozen plasma transfusion, considering the signs of a transfusion reaction.

Recumbency care was required whilst anaesthetised, with nursing considerations to include:

- Turning and changing recumbency.

- Physiotherapy

- Oral and ocular care.

- Urinary and faecal management.

- Nutritional support.

Outcome

The patient was under general anaesthetic with propofol and sevoflurane for 34 hours before successful extubation and a slow recovery. She was discharged home on day three of hospitalisation and was bright, ambulatory and appetent, with no further tremors or seizures. The presentation, diagnostics and response to treatment were consistent with ingestion of a neurological toxin, most likely a tremorgenic mycotoxin.

Conclusion

This case underscores the challenges and complications associated with managing a severe toxicological emergency. The patient's emergent presentation required rapid interventions including venous access, evaporative cooling, seizure management and decontamination. The successful outcome highlights the importance of a coordinated approach by the veterinary team. Vigilant nursing care and effective interprofessional communication were essential for recognising and addressing patient deterioration and contributed to patient recovery.

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OPTIMIZING NUTRITION FOR HOSPITALIZED DOGS AND CATS

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Learning objectives:

This lecture focuses on the importance of tailored nutritional strategies for hospitalized dogs and cats to promote faster recovery and improve clinical outcomes. Attendees will:

- Gain insights into evidence-based methods for evaluating nutritional requirements, developing feeding strategies, and managing patients with critical conditions or special dietary needs.

- Understand the role of feeding tube options, such as nasogastric and esophagostomy tubes, in delivering essential nutrition to patients unable to eat independently.

- Learn how these nutritional techniques can positively impact healing, immune function, and overall well-being.

Proceeding:

Introduction

Nutrition is a crucial component of the management and recovery of hospitalized dogs and cats. Malnutrition can negatively impact immune function, wound healing, and overall prognosis, leading to increased morbidity and mortality. Providing appropriate nutritional support early in hospitalization is crucial for optimizing patient outcomes. Veterinary nurses play a fundamental role in recognizing nutritional needs, implementing feeding strategies, and ensuring patient compliance with feeding protocols.

Nutritional Assessment and Planning

A systematic nutritional assessment helps identify patients at risk and guides proper interventions. Body condition score (BCS) and muscle condition score (MCS) are essential tools to evaluate nutritional status and detect muscle loss, which is critical in hospitalized patients. The resting energy requirement (RER) is calculated using the formula:

RER = 70 × (Body weight in kg)^0.75

The calculated energy requirement is adjusted based on the patient's condition, such as sepsis, trauma, or post-surgical recovery. The choice of diet depends on the patient's condition and the mode of feeding. High-protein, highly digestible diets are often recommended to support recovery.

Proper nutrition is vital for hospitalized pets, strengthening their immune system and aiding recovery. Malnourished animals heal slower and are more prone to infections. Protein supports antibodies and tissue repair, while omega-3s reduce inflammation. Vitamins A, C, and E protect cells, and zinc and selenium boost immunity. Providing balanced nutrition tailored to each pet's needs enhances healing and infection resistance. Furthermore, in anorexic animals, albumin levels may drop, and since albumin is not available in every country, the only way to restore it is through nutrition.

Enteral Nutrition Strategies

Some very sick pets have trouble eating and may need extra help. If a pet refuses to eat, appetite stimulants like mirtazapine may be useful, and making the feeding area more comfortable can encourage eating. If the pet has vomiting or diarrhea, the diet may need to be adjusted to a more digestible or specialized food. It is also important to check and maintain proper levels of essential nutrients like potassium, phosphorus, and B vitamins. However, if voluntary food intake is insufficient, enteral nutrition should be initiated. The preferred method depends on the severity of the condition and the anticipated duration of support.

Nasogastric tubes (NGT) are commonly used for short-term feeding (up to seven days) in anorexic patients with a functional gastrointestinal tract. These tubes are minimally invasive and do not require general anesthesia but necessitate liquid diets. For longer-term feeding, esophagostomy tubes are preferred as they allow for thicker diets and can be managed at home. Placement requires anesthesia, and there is a risk of infection at the entrance site. In cases where extended feeding support is required, gastrostomy and jejunostomy tubes provide direct access to the stomach or intestine. These options involve more invasive placement techniques and necessitate careful monitoring to prevent complications such as peritonitis.

Feeding Protocols and Monitoring

Proper implementation and monitoring of enteral nutrition are essential to avoid complications and ensure optimal nutritional support. Feeding should begin with one-third of the calculated RER on day one, gradually increasing over three days to reach full energy requirements. Refeeding syndrome should be monitored, particularly in severely malnourished patients.

Patients should be positioned in sternal recumbency during feeding to reduce the risk of aspiration. Tubes must be flushed with water before and after feeding to prevent clogging, and food should be administered slowly over ten to fifteen minutes at room temperature. Complications such as tube blockage, vomiting, or regurgitation must be rapidly addressed. Regular flushing with warm water or carbonated fluids can prevent and resolve blockages. Reducing feeding volume or administering prokinetic medications like metoclopramide may be necessary in cases of vomiting. Infection prevention involves maintaining proper hygiene at tube insertion sites and changing bandages as needed.

Conclusion

Optimizing nutrition for hospitalized dogs and cats significantly enhances recovery, immune function, and overall patient outcomes. Veterinary nurses are integral in assessing nutritional needs, implementing feeding strategies, and managing enteral nutrition. By incorporating evidence-based

feeding protocols and closely monitoring patients, veterinary professionals can ensure optimal care and improve survival rates in critical cases.

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NURSING THE TRAUMA PATIENT

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Learning objectives:

- Using triage and trauma scales to prioritise patients

- Applying primary and secondary surveys to aid in identifying life threatening issues

- Being familiar with different shock types and monitoring for improvement/deterioration

- What we as nurses can do in these cases

Proceeding:

Introduction

Patient's that have experienced trauma require urgent veterinary care and will generally be seen in general practice first. Veterinary staff need to be familiar and confident in dealing with these cases as often they can present without warning and therefore without time for staff to prepare. Non-clinical staff are often the first to see these patients come through the door and it is important that they are trained in identifying cases that need immediate veterinary attention.

Triage

The implementation of trauma/triage scales within your practice can be vital to help all staff members identify what patients need immediate attention and how to prioritise them. This can be especially important for non-clinical staff (as well as less experienced staff such as student nurses etc) that may be answering the phone or greeting clients as they arrive.

The primary and secondary surveys are vital to assess the condition of the patient. Nurses should be confident in conducting these surveys as a veterinary surgeon may not be present immediately.

Primary Survey

The primary survey should take 30-60 seconds and needs to be a look/listen/feel assessment of the 3 major body systems;

Respiratory

Are they breathing and moving air?

Do they have an increased respiratory effort (RE)?

Body position. Are they orthopneic?

Cardiovascular

Perfusion parameters; Capillary refill time (CRT), mucous membranes (MM), pulses, heart rate (HR), extremity temperature, mentation

Neurological

AVPU; Alert, voice responsive, pain responsive, unresponsive

Ambulation

STABILISE IMMEDIATE LIFE-THREATENING ISSUE FIRST.

Altered mentation, seizuring or active haemorrhage requires immediate intervention.

Don't get distracted by blood and visual injuries. Apply pressure and treat most concerning issue.

Secondary Survey

Once the primary survey has been completed the secondary survey can be started. This is a more in-depth evaluation of the patient and should include a full examination of the patient and diagnostics. Vitals and observations that should be included are;

HR, respiratory rate (RR), blood pressure (BP), rectal temperature, SPO2, ECG

Auscultation

Modified Glasgow Coma scale (MGCS), brainstem reflexes

Pain score and appropriate analgesia administered

An emergency database should be taken and a thoracic and abdominal Point Of Care UltraSound (POCUS) performed (+/- radiographs). A full clinical history should be taken from the owner and AMPLE is an anacronym that can be utilised for this;

A –Does the patient have any?

M –Are they on any regular medications?

P –Past history. Previous health issues.

L –When did the patient last eat, drink, pass urine/feces or have any meds?

E –What event immediately preceded them presenting to the practice?

It is vital that a RESUS status is obtained from the owner if you have not already done so.

Throughout the primary and secondary survey's, we as nurses can be providing oxygen supplementation, attaching monitoring equipment, obtaining vascular access, drawing blood and initiating treatment (IVFT, analgesia etc.) as directed by the veterinary surgeon.

Shock

It is essential that we are aware of the different types of shock that a patient can present with in order that the correct treatment is given.

Hypovolaemic- Decrease in intravascular volume leading to insufficient oxygen delivery to tissues

Haemorrhagic- Leads to direct hypoxic injury

Distributive -Inappropriate vasodilation or ineffective vasoconstriction leading to a relative hypovolaemia

Obstructive - Decreased delivery of oxygen to tissues due to decreased venous return and therefore decreased cardiac output

Cardiogenic - Inadequate cardiac output due to ineffective forward blood flow

Monitoring trends for signs of improvement/deterioration in a shock state when stabilising patients can guide the need for further treatment/intervention. Perfusion parameters to monitor include are;

Mentation, BP, Pulse quality, HR, CRT, urine output (UOP), Blood Lactate +/- shock index (SI)

Analgesia

Analgesia is a critical part of stabilising trauma patients. This is not only for patient welfare but physiologically will aid in healing and reduce/normalise vital parameters. If the patient is comfortable but vital parameters are still not within normal limits, then you can rule out a pain response and investigate other avenues in order to guide treatment appropriately. A validated pain scoring system should be used regularly and by adopting a multimodal analgesia treatment plan the patient's nociception levels can be addressed and controlled swiftly.

Other considerations

Nutrition: Nutrition is often underestimated for hospitalised patients. Adequate nutrition is necessary to promote healing and reduce hospitalisation times. Dependant on the patient's condition and injuries sustained a feeding tube (naso-oesophageal/naso-gastric or oesophagostomy) should be considered so there is no delay in initiating feeding.

Recumbency care: A urinary catheter can be beneficial in recumbent patients to avoid urine scalding and aid in patient comfort. Regular turning in non-ambulatory patients and introducing physiotherapy techniques such as PROM can aid in joint mobility and prevent muscle contracture and atrophy.

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THE TETANUS PATIENT, MANAGEMENT AND NURSING CONSIDERATIONS

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Learning objectives:

- Etiology and pathophysiology
- Presentation and diagnosis
- Treatment options
- Management and nursing care
- Prognosis

Proceeding:

Eitiology

Tetanus is caused by the bacterium Clostridium tetani (C. tetani). C. tetani is an obligate spore forming, gram positive rod which is found in infected soil. This is the most common source of C. tetani infection, where it passes to the patient through an open wound. C. tetani thrives in anaerobic conditions and causes the release of a potent neurotoxin, tetanospasmin. The binding of tetanospasmin to the presynaptic sites of inhibitory neurones is irreversible and recovery therefore requires new growth of the nerve terminals, which is why the recovery process is so long in these patients. Initial clinical signs can take up to 4 weeks to develop, however are more commonly noted after 4 to 12 days.

Presentation

A human based grading system is used to gauge severity of disease process and prognosis for dogs.

Grade 1: ambulatory but have facial signs including risus sardonicus, erect ears, lock jaw and potentially have hypersensitivity to light, noise and touch.

Grade 2: ambulatory but have a more stiff gait, erect tail or ‘sawhorse stance’ in combination with some or all of grade 1 signs. Dysphagia may also be evident.

Grade 3: have some or all of the signs evident with grade 1 and 2 plus the patient will be recumbent and be exhibiting muscle twitching, spasm and/or seizure activity.

Grade 4: autonomic signs as well as some or all of grade 1, 2 and 3 signs. High risk of respiratory arrest and hypoventilation due to respiratory fatigue and apnoea. Respiratory paralysis may occur in severe disease and mechanical ventilation may be indicated.

Diagnosis

The best way to diagnose the condition is by taking a thorough clinical history to find out whether there have been any recent injuries. Occasionally, the patient will present with no visible wound as this may have already started to heal. Owners' are likely to initially notice reluctance to eat due to difficulty in opening the mouth. It is important to rule out other possible causes such as temporomandibular dislocation or osteoarthritis.

A severely elevated creatine kinase (CK) is consistent with muscle damage from constant muscle spasticity. Dehydration may be noted on PCV and TS and a mild leukocytosis with neutrophilia as well as a left shift due to infectious process. A gram stain from a smear of the open wound may help identify gram positive rods.

Treatment

The treatment of tetanus aims to neutralise the toxins outside of the central nervous system and destroy the organisms in the body to prevent further toxin release. Tetanus equine or human antitoxin can be administered to neutralise any toxin, however only if these toxins have not already bound to the CNS. Therefore early intervention is key for this to have any success. Antitoxin doses should not be repeated, as this increases the chance of anaphylaxis. Human antitoxin immunoglobulin (IG) is reported to have a higher chance of anaphylaxis in our patients. Adrenaline, glucocorticoids and an antihistamine should be available in case a reaction occurs.

All obvious wounds need to be debrided radically, potentially with digit or limb amputation to ensure as much of the contaminated tissue has been removed. Hydrogen peroxide can be used as a flush as this inhibits anaerobic organisms although this may impair wound healing. Mildly affected animals generally recover well from neurological dysfunction with appropriate wound debridement and antimicrobial therapy.

To minimise the effects on the CNS, it is important to control the muscle rigidity and spasticity as this can cause severe discomfort, pain and anxiety.

Nursing

Any stimulation can trigger anxiety levels and potentially worsen muscle spasms, so these patients should be kept in a darkened, quiet room to minimise overstimulation.

Analgesia, sedation and antimicrobials are indicated. Respiratory depression is linked with higher doses of opioids so fentanyl may be preferred as it can be titrated up and down with quick effect.

Almost all tetanus patients admitted to hospital require intense nursing care and management. As nurses it is vital we advocate for our patients. The use of a multi-parameter monitor and the placement of urinary catheters, central lines, feeding tubes and arterial lines can make a huge difference to allow

for a more hands off approach. Recumbency management is essential along with passive range of movement physiotherapy where feasible. Nutrition should be provided as early as possible, including gastro protectants and motility agents due to a reduced gastrointestinal function and increased metabolic rate causing rapid weight loss.

In some cases tracheostomy tubes can be placed, and relevant additional nursing care for these will need to be provided. Risk of aspiration pneumonia is high due to excessive secretions, regurgitation and inability to swallow, so daily thoracic point of care ultrasound scans are recommended, as well as careful positioning of the patient.

During the recovery period it is still encouraged to monitor and manage anxiety and pain. As these patients have had such prolonged muscle rigidity and spasticity, it's likely they will have continued discomfort which needs to be addressed and the pain plan should be titrated as required. It is still encouraged to monitor and manage continued anxiety and pain. As these patients have had such prolonged muscle rigidity and spasticity, it is likely they will have continued discomfort which will need to be addressed. The key to success is good nursing care, anxiety management and managing the clinical signs, including autonomic signs if present.

Prognosis

Prognosis depends on classification. Cats generally have localised tetanus, and their prognosis is good. Localised tetanus is where muscle rigidity is seen only in or around the area where the C. Tetani enters the body. It can cause muscle rigidity and spasm next to the affected area, or of the whole limb. Localised tetanus can progress into generalised tetanus, but not always.

In dogs with class 1 or 2 signs, a study reports that all of these dogs survived. In patients where class 3-4 signs were observed, only 58% of these animals survived. It is also reported that a full recovery may not be possible in around 15% of surviving dogs, but continued improvement is possible over the coming 3-5 months. The recovery process depends on new axonal terminals forming, and the majority of patients will start to improve within 5-12 days providing there are no autonomic abnormalities. There is no evidence to suggest that a natural infection provides any immunity against tetanus. So there is a risk that these animals can become infected again. Ocular and facial changes are the most common initial signs, with 50% of dogs with generalised tetanus progressing to recumbency within 14 days. Most patients that die have concurrent autonomic signs, including pyrexia, vasoconstriction and respiratory compromise.

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THE ROOM IS SPINNING! VESTIBULAR SYNDROME IN COMPANION ANIMALS

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Learning objectives:

- Examine the anatomy of the vestibular system

- Describe the signs associate with vestibular disease

- Discuss peripheral and central localization of vestibular disease

- Review nursing care needed for patients with vestibular disease

Proceeding:

The vestibular system is responsible for balance, posture and orientation of the body within space. There are two segments of the vestibular system, the peripheral, made up of the inner ear and cranial nerve VIII (CNVIII), and the central, made up of the brainstem and cerebellum. Together these components detect rotational and linear movement and head tilting relative to gravity and turn this information into nerve signals that are sent to the brain. The brain interprets these signals and helps the body adjust to avoid falling and dizziness. The brain receives input from a vestibular organ (the ear) on either side. When an animal tilts or falls to one side the ipsilateral (same side) vestibular structures are stimulated.

SYMPTOMS

Symptoms of vestibular dysfunction are often acute and can be very dramatic. When these animals present in clinic, they are often nervous and their owners are extremely stressed due to the sudden onset of symptoms. The patient most commonly presents with a head tilt and is ataxic. Strabismus and nystagmus are also common symptoms. A simple test of the vestibular system involves moving the head from one side to another. The eyes will stay focused (slow phase) while the head rotates until the vestibular system kicks in and the eyes correct back to the center of the head (fast phase).

CAUSES

There are many things that can cause vestibular symptoms. The most common is idiopathic vestibular syndrome. This is sometimes called “old dog disease” because these animals are usually senior pets who show no other signs of illness. When treated supportively they usually recover quickly on their own. Other causes of vestibular symptoms include, severe otitis interna, neoplasia, metabolic disease, meningoencephalitis and certain medications. Treatment for these causes may be more involved.

Localization

A full neurologic exam should be done on any patient that presents with vestibular symptoms. The exam will help determine where the lesion is located and what part of the vestibular system is being affected. Determining if the patient has a central or peripheral lesion is important for helping diagnose the cause of the disorder.

Peripheral

Acute onset of clinical symptoms is a defining characteristic of peripheral syndrome. These patients show no or little change in mentation and are usually bright and alert. Hypersalivation and/or vomiting are not uncommon. The balance center of the brain is very close to the chemoreceptor trigger zone (CTZ), the part of the brain which induces vomiting.

Central

Central vestibular syndrome is due to a lesion that affects the vestibular nuclei in the brainstem. In general, central vestibular syndrome is more serious and difficult to diagnose than peripheral. Clinical signs are similar to those of peripheral disease. The animal will be weak ipsilateral to the lesion and tilt, roll or fall ipsilaterally as well. Unlike peripheral disease the patient will often have a dull or obtunded mentation. Mentation change is a key sign that points towards central syndrome. The patient may have conscious proprioceptive (CP) deficits indicating brainstem involvement.

Paradoxical and bilateral

Paradoxical vestibular syndrome is a form of central disease. In this uncommon presentation the clinical signs of dysfunction are contralateral, or on the opposite side of the lesion. Bilateral vestibular syndrome usually occurs with peripheral dysfunction and is due to injury to both sides of the vestibular system.

Diagnostics

Full blood work should be run, and a complete history is needed. Magnetic resonance imaging (MRI) is considered gold-standard for diagnostic imaging in vestibular syndrome.

Nursing Care

The treatment goals for these patients are twofold. Treat any underlying cause and provide supportive care for symptoms. Vestibular patients will need extra padding in their cages to help them avoid injury if they fall or are rolling around in their cage. Antiemetics are used to help with nausea. Recumbent care is essential if the patient is unable to turn or move themselves. The animal may need assistance to stand to urinate and defecate due to weakness or ataxia. Monitoring for any mentation changes is particularly important in patients where there is concern for central disease but should be done for all patients.

Prognosis

The prognosis for vestibular syndrome depends on the type of disease. Idiopathic disease tends to resolve with supportive care. Symptoms begin to clear up within 2-3 days. Patients with central vestibular disease have a guarded to poor prognosis. The involvement of the brain causes these patients to be considered critical.

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WHEN THE BRAIN GOES BOOM: MONITORING THE HEAD TRAUMA PATIENT

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Learning objectives:

- Understanding brain dynamics

- Know how to triage and stabilize these (poly)trauma patients

- Understand the importance of continuous monitoring and intervention strategies to prevent complications.

Proceeding:

Traumatic Brain Injury (TBI) requires an understanding key pathophysiology aspect.

The volume of the cranial content consists of 80% brain tissue, 10% blood, and 10% cerebrospinal fluid. These components are balanced with each other, with the intracranial pressure (ICP) remaining between 5-12 mmHg. The cranium is a rigid structure, offering little room for additional fluid or tissue. The Monro-Kellie hypothesis or intracranial compliance explains how the body compensates for changes in volume, but this mechanism is exhausted in TBI, with can lead to dangerous increases in ICP.

Cerebral Perfusion Pressure (CPP) is the pressure that determines blood supply to the brain and is expressed as mean arterial pressure (MAP) minus ICP. Under normal circumstances, autoregulation (where the cerebral vasculature adjusts in diameter in response to changes in CBF) can maintain cerebral blood flow (CBF) within a range of MAP 50-150 mmHg. TBI can disrupt this autoregulation, increasing the risk of hypoxia, hypotension, and ischemic damage. The goal is to maintain MAP between 80-100mmHg to support adequate CBF.

Chemical autoregulation is a direct response of the brain's vasculature to arterial carbon dioxide pressure (P_aCO_2). Increased PaCO2 causes vasodilation and can increase ICP, while decreased PaCO2 leads to vasoconstriction, which can lower both ICP and CBF

Primary brain trauma refers to brain injury caused directly by the traumatic impact.

Secondary brain trauma occurs due to biochemical changes that occur minutes to days after the initial trauma. Such as hypoxia, hypo/hypertension, increased ICP etc. These processes can worsen the damage and ultimately lead to cell death.

Triage and stabilization

Our goal is to quickly identify and stabilize life-threatening conditions using the **ABC(DE).** Hypotension, hypovolemia, and hypoxia must be rapidly identified and treated because these can worsen secondary brain injury. Our aim is to maintain a mean arterial pressure (MAP) of 90mmHg, Systolic AP (SA) of 100mmHg and a SpO2 of >95%.

After initial stabilization, a neurological assessment **(D)** is conducted using the Modified Glasgow Coma Scale (MGCS), which helps evaluate the patient's consciousness, pupil responses, and posture. Keep in mind that decreased mentation may result from shock or hypoglycemia, not just TBI.

With External assessment **(E)** you check for external injuries such as bleeding and fractures especially in polytrauma patients. Fractures of the cranium can be a serious complicating factor in the treatment of TBI. Temperature regulation may fluctuate due to shock or brain injury.

Fluid therapy

When TBI is suspected resuscitation with hypertonic saline should be used as first fluid. Followed with crystalloids when needed. Mannitol is not preferred for hypovolemic TBI patients due to its diuretic effect. Vasopressors may be required if the patient doesn't respond to volume resuscitation.

Further investigations, such as CT or MRI scans, may be needed to assess trauma severity. Surgical decompression may be necessary for compression fractures, intracranial hemorrhage, or high ICP.

Pain management is crucial as pain can cause increased MAP and ICP. Effective pain control can improve patient comfort, reduce anxiety, and potentially aid in the healing process.

Monitoring

Hypotension and hypoxia are the primary culprits as they can exacerbate secondary brain trauma.

Until hypoxia is ruled out as a danger, every patient should receive oxygen therapy, and their ventilation and oxygenation ability evaluated with an arterial blood gas. Once the animal is stabilized, a period of intensive monitoring follows. During this period, the animal is carefully observed for signs of deterioration in:

- Neurological symptoms using the MGCS

- Cardiovascular changes such as hypotension and early detection of the Cushing reflex (bradycardia, hypertension and abnormal breathing pattern)

- Respiratory changes: d/t neurogenic or respiratory origin.

- Oxygenation and ventilation assessment

- Glucose control: increased glucose is linked to higher mortality.

- Hypo/hyperthermia: prevent hyperthermia as it increases cellular metabolism.

Avoid actions that could trigger coughing or sneezing, as these can increase ICP, like jugular venipuncture or nasal tubes. Keep the head and shoulders elevated by 30 degrees to promote venous drainage. Ensuring jugular vein is not obstructed.

Promote general patient wellbeing through sufficient pain relief, comfortable bedding, nutrition and, depending on the patient's status: repositioning, physiotherapy, eye ointments, and oral care (with intubation).

In human medicine, direct ICP monitoring is standard practice. Although studies on ICP monitoring methods in veterinary medicine are promising, it is not yet widely used.

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Leadership Stream, Saturday 7 June 2025

DESIGNING AND MANAGING MODERN SMALL ANIMAL ICUS

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Learning objectives:

- Understand key principles of ICU layout and patient flow optimization
- Recognize the importance of ergonomic design in veterinary ICUs
- Implement effective hygiene and infection control strategies
- Integrate digital tools to enhance ICU efficiency
- Foster staff well-being through strategic ICU design

Proceeding:

The modern small animal ICU is a dynamic and complex environment, requiring thoughtful design and management to ensure optimal care for critically ill patients. As veterinary medicine advances, so too must our approach to ICU layouts, patient workflow, infection control, and staff well-being. A well-designed ICU not only improves patient outcomes but also enhances efficiency, minimizes stress, and fosters a better working environment for veterinary professionals. This lecture explores the fundamental elements of modern ICU design and offers practical insights into creating effective, high-performing critical care spaces.

Patient and Workflow

Efficient patient workflow is at the core of a well-functioning ICU, allowing for seamless transitions between triage, diagnostics, treatment, and recovery. Implementing the 5S methodology—Sort, Shine, Set in Order, Standardize, and Sustain—can significantly improve workflow efficiency and organization. This approach ensures that essential supplies and equipment are easily accessible, reducing time wasted searching for items and minimizing staff frustration.

Sort: Identify and categorize essential, frequently used, and rarely used items. This reduces clutter and ensures only necessary equipment and supplies are readily available.

Shine: Clean and maintain workspaces regularly, ensuring that all equipment is in good condition and potential hazards are removed. A clean environment supports hygiene and minimizes risks.

Set in Order: Arrange items logically based on workflow requirements. High-use items should be within easy reach, and lesser-used items should be stored systematically to prevent inefficiencies.

Standardize: Implement uniform procedures across different workstations, ensuring consistency in organization and accessibility. This improves workflow predictability and reduces staff confusion.

Sustain: Maintain these standards through regular audits, staff training, and management support. A sustained 5S system enhances long-term efficiency and workplace organization.

Flexibility is another key component, as certain equipment, such as ventilator stations and incubators, must be modulable and movable to adapt to changing patient needs. Adequate storage areas must be available to accommodate these adjustments, preventing clutter and ensuring quick access to necessary tools. By integrating the principles of 5S and prioritizing adaptable design elements, ICUs can maximize efficiency while maintaining a high standard of patient care.

Hygiene

Hygiene and infection control are paramount in any ICU, where the risk of disease transmission is high. To maintain the strictest hygiene standards, continuous hand hygiene protocols must be enforced, with strategically placed hand sanitizing stations at entry points and in close proximity to all patient areas. Frequent and thorough hand hygiene practices among staff significantly reduce the spread of infections. According to the World Health Organization (WHO), proper hand hygiene can reduce hospital-acquired infections by up to 50%.

Hand hygiene should be performed:

- Before and after patient contact

- Before performing any aseptic procedures

- After exposure to bodily fluids

- After touching patient surroundings

Additionally, ICU layouts should include adequately isolated stations to ensure optimal patient monitoring while adhering to hygiene requirements. These spaces should be designed to allow direct observation of critical patients while minimizing unnecessary movement, reducing contamination risks. By incorporating high-efficiency air filtration, seamless surfaces, and dedicated isolation areas, ICUs can maintain the highest levels of cleanliness and infection control.

Staff and Patient Well-being

A well-designed ICU should foster the well-being of both patients and veterinary professionals. Adequate lighting sources are essential, with a combination of natural light and adjustable artificial lighting to support optimal visibility and reduce fatigue. Proper ventilation is equally critical, ensuring clean air circulation and preventing the buildup of harmful airborne pathogens.

Sound control measures, including acoustic paneling and designated quiet zones, help minimize noise-related stress for both staff and patients. Music therapy has also been shown to create a calming atmosphere, benefiting patient recovery and reducing staff stress levels.

Ergonomic and quiet workstations should be incorporated to support staff comfort and efficiency. Height-adjustable workstations, anti-fatigue flooring, and strategically placed equipment ensure that veterinary professionals can perform their tasks with minimal physical strain. These measures collectively contribute to a healthier and more sustainable work environment.

Digital Integration

Technology plays an essential role in modern ICU management, improving efficiency and patient care through digital integration. Telemetry devices allow for continuous patient monitoring, providing real-time data on vital signs such as ECG, oxygen saturation, and blood pressure. This technology enables rapid response to changes in patient conditions, improving outcomes.

Triage boards and electronic medical records (EMRs) streamline communication and facilitate efficient information sharing among staff. Implementing CPR alarms that connect multiple rooms ensures immediate team response in emergency situations. Additionally, transitioning to a paperless system enhances workflow efficiency, reducing documentation errors and improving data accessibility.

Beyond streamlining processes, digital integration can significantly limit the occurrence of medical errors by automating various procedures, reducing reliance on manual data entry, and ensuring consistency in record-keeping. Automation also assists in fundamental tasks such as patient information transfer, allowing seamless updates between departments and reducing miscommunication. By fully embracing digital integration, ICUs can optimize patient care, enhance safety, and reduce administrative burdens on staff.

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PATIENT SAFETY IN ECC – WHAT DO WE KNOW AND WHAT CAN WE DO ABOUT IT?

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Learning objectives:

This lecture explores common patient safety risks in Emergency and Critical Care (ECC) using global incident data. It focuses on understanding why errors occur and identifying high-risk areas. Attendees will learn practical strategies—such as communication tools, medication protocols, and system improvements—to minimise errors and enhance safety for patients, owners, and teams.

Proceeding:

Content

Emergency and Critical Care (ECC) environments present unique challenges in patient safety due to the fast-paced, high-stakes context. Incidents in ECC can lead to serious harm to patients, distress for pet owners, and significant emotional and financial costs for veterinary teams and clinics. This lecture explores the most common patient safety risks in ECC using global incident reporting data from small animal veterinary care. By understanding why errors occur and where the biggest risks lie, veterinary professionals can implement targeted strategies to enhance safety and reduce preventable harm.

Why Do Errors Occur?

Patient safety incidents often arise from a combination of systemic issues and human factors. High workload, equipment features, time pressure, communication breakdowns, and cognitive overload increase the likelihood of errors. Additionally, unpredictable case severity and the need for rapid decision-making further heighten risk. By analysing real-world incident data, we can identify patterns and underlying causes, allowing teams to proactively address vulnerabilities rather than reacting after harm has occurred.

High-Risk Areas in ECC

Data from global incident reporting systems highlight several critical risk areas in ECC:

- Medication: Incorrect dosing, drug mix-ups, and administration errors are common in high-stress situations.

- Communication breakdowns: Miscommunication between team members, unclear instructions, and handover mistakes contribute to preventable incidents.

- Equipment and Procedure incidents: Issues such equipment set up, misplaced IV catheters, or oxygen supply failures can lead to serious complications.

- Decision-Making Under Pressure: Errors in triage, diagnostics, or emergency treatment plans often result from cognitive overload or fatigue.

Strategies to Minimise Errors and Enhance Safety

- Communication Tools: Implementing structured handovers (SBAR or IPASS), closed-loop communication, and checklists can improve clarity and prevent misunderstandings.

- Medication Protocols: Standardized dosing charts, labelling of syringes, and double-check systems reduce medication-related incidents.

- Training and Simulation: Regular team training, emergency drills, and case debriefs help teams develop strong decision-making skills under pressure.

- A Culture of Learning: Encouraging and normalising open discussions about incidents, incident reporting, and focusing on system-wide improvements rather than individual blame foster a proactive safety culture.

By integrating these strategies, ECC teams can mitigate risk, improve patient outcomes, and create a safer working environment. This lecture provides actionable insights to help veterinary professionals recognize high-risk areas and implement effective safety interventions in their practice.

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THREAT AND ERROR MANAGEMENT

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Learning objectives:

This session introduces Threat and Error Management (TEM) as a proactive approach to patient safety. It emphasises identifying and mitigating risks in dynamic clinical settings rather than eliminating all threats. Delegates will learn from real-world veterinary examples how teams can apply TEM to prevent harm and create safer systems in real time.

Proceeding:

Content

Patient safety is frequently reactive, looking at why a patient was harmed and how we can make the system safer for the future. While this is important and should form a part of quality improvement activities at all practices, clinical teams must and do create safety as they work. Threat and error management (TEM) is a framework for looking at safety in a different way. It does not rely on eliminating all risks or expect people to be perfect and never make mistakes. Instead the focus in on how to identify threats and spot errors early so they can be mitigated.

What is TEM?

TEM is a framework developed in the aviation industry which can be used to help us understand the interaction between human performance and safety in a dynamic and challenging context. It has three components:

Threats - events or conditions that are outside the control of the clinical team, increase risk and complexity, and must be managed to maintain safety. An example could be having two patients with the same name in the hospital.

Errors - action or inaction by a member of the clinical team that results in a deviation from individual or team intentions or expectations. An example might be a team member mis-identifying the patient.

Undesired states – where an unintended situation has resulted in reduced safety. Often considered the last stage before an adverse event or accident. This could be the wrong patient being brought from the ward for a procedure.

Mismanaged threats greatly increase the chance of errors and failure to effectively manage both threats and errors can lead to an undesired state.

Managing threats, errors and unintended states

Some threats can be anticipated as they are already known or expected while others are unexpected and result from a changing situation. We will never be able to eliminate threats as they are inherent in everyday work. Because of this we can tend to normalise them, but it must be remembered they are still threats and need to be managed.

The effect of an error on safety depends on whether it is detected and responded to before an undesired state is reached. Errors, like threats, are a normal part of work. Human error is neither unusual nor unexpected so we need to focus not on who made a mistake but how we can quickly detect and respond to errors.

Once an undesired state is reached, it represents the last opportunity to manage the situation before it results in an adverse event.

Systemic: Automated alerts on equipment, standard operating procedures, briefings and professional training.

Individual and team:

Good leadership and communication (information flow and teamwork)

Planning – preparation, briefings, contingency plans (managing anticipated and unexpected threats)

Execution – monitoring and cross-checking (error detection and error response)

Review/modify – evaluation of plans, inquiry (managing changing conditions)

We can also look at patient safety event reporting data to identify the most common threats and errors present in our work and use this to inform team training.

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Nurse Advanced Stream, Saturday 7 June 2025

LET'S STICK TOGETHER- DIVING INTO THE NITTY GRITTY OF COAGULATION, COVERING COAGULOPATHIES, PLATELET'S ROLE, CASCADE AND VISCOELASTIC TESTING

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Learning objectives:

- Identify between the classic coagulation cascade and the cell-based model

- Understand the role of viscoelastic testing in critical patients

- Describe the platelets' role in sepsis and inflammation

- Summarise how sepsis and inflammation effect coagulation

- Identify which patients may be at risk of hyperfibrinolysis

Proceeding:

Cascade vs cell-based model

The coagulation cascade is a simplified view of coagulation and is separated into the extrinsic pathway, intrinsic pathway, and the common pathway. In this example the pathways act dependently of each other but in reality, the pathways are all working together at the same time. This is not a current view of how we know coagulation happens within the body but is it useful for understanding of coagulation and when using coagulation diagnostics in-house to give us an idea of which factors are affected.

The Cell-Based Model of coagulation shows us the coagulation process is happening all at the same time in different phases overlapping each other. And is how we currently view coagulation. This is divided into 3 phases.

Cell-Based Model:

Initiation Phase

- TF within a cell is exposed to haemorrhage.

- FVIIa binds to TF, to activate more FVII and FIIa.

- This then activates FIX and FX.

- FXa binds to FVa

- Small amount of thrombin (FIIa) is created.

Amplification Phase

- Small amount of thrombin breaks away to activate platelets around the site of haemorrhage.

- Thrombin binds to the surface of the platelets, where receptors are causing the platelet to change shape.

- Granules are released from the platelets containing proteins ADP and thromboxane A2.

- These proteins aggregate and activate more platelets to come to the site of haemorrhage.

- Ca^{2+} helps platelets adhesion and activation, as well as activating several coagulation factors.

- Thrombin separates vWF from FVIII (which circulate together), vWF then aids with platelet adhesion and aggregation to help stabilise the platelet plug.

- FVIII is activated to FVIIIa (fibrin).

Propagation Phase

- Intrinsic tenase complex is form on the surface of the activated platelet.

- This made from binding FVIIa and FIXa.

- FXa is generated on the platelet surface and binds to FVa.

- This aids in converting prothrombin to thrombin (FII to FIIa).

- A prothrombinase complex is formed.

- A large amount of thrombin is generated, which results in fibrinogen.

- This turns into a mass of fibrin (FXIII).

- Thrombin activates FXIII to FXIIIa.

- Cross line of fibrin strands form and stabilise the clot further.

Sepsis and inflammation

Platelets have an important role in inflammation and the immune response. Pro-inflammatory cytokines are released during times of inflammation within the body. These cytokines regulate inflammation, different types of cells can release cytokines including platelets. Platelets secret cytokines and release immune modulators to attract neutrophils, monocytes and lymphocytes causing further inflammation. Platelets as they pass by in circulation, they will adhere to white bloods cells that have accumulated at a site of damage, platelets interact with neutrophils creating “cell traps” neutrophil extracellular traps (NETs), which tap and kill bacteria. If the body produces too many NETs this increases thrombus formation along with a reduction is how fast clots are broken down (hypofibrinolysis), leading to accumulation of clots within organs causing multiple organ dysfunction (MODS).

Hyperfibrinolysis

Fibrinolysis is a natural process of breaking down fibrin to enable normal circulation to continue to the tissues post healing process. Hyperfibrinolysis is where this process has been sped up in body, this can be seen in trauma induced coagulopathies (TIC) and hepatopathies and is probably much more commonly seen in sighthound breeds such as greyhounds as they seem to be predisposed to hyperfibrinolysis. This is due to a either deficit in an inhibitor of fibrinolysis e.g. Plasminogen activator inhibitor type-1 (PAI-1) or an excess of an activator of fibrinolysis e.g. Tissue plasminogen activator (tPA), these are enzymes that work within the coagulation process. PAI-1 is released from the liver into circulation where it meets tPA. tPA is found within the endothelial walls of the vessels, converts PAI-1 to plasmin which lysis clots into fibrin degradation products (FDP), FDP can be measured but increased levels of FDP can be due to any disease or inflammatory process.

Antifibrinolytic drug therapies like tranexamic acid (TXA) can help reduce the rate at which the clots are broken down therefore reduce haemorrhage, these can be given prophylactically before surgery to sighthound breeds or if a patient who has been hit by a car and has suffered trauma, TXA can reduce the effects of TIC hyperfibrinolysis. Caution TXA will induce emesis if given quickly intravenously, best to give as a slow continuous rate of infusion over 10-20minutes.

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FINDING THE BALANCE TO ACID-BASE: SOLUTIONS AND BUFFERS

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Learning objectives:

- Gain deeper understanding of the Henderson-Hasselbalch equation and strong ion theory

- Provide the ability to assess electrolytes in line with acid-base balance

- Define the mechanisms in which acid-base is changed within disease processes

Proceeding:

The Basics

pH is the conventional way of describing the concentration of hydrogen ions. It is a logarithmic scale that allows representation of a wide range of hydrogen ion concentrations in a simplified way. Because it's a logarithmic scale, a small change in pH represents a significant change in hydrogen ion concentration. The pH considered compatible with life from 6.8 to 8, with normal for most mammalian species being around 7.4 and kept within a very tight range. Acidaemia is a blood pH lower than normal (an increase in blood H+ concentration), this differs from acidosis-a pathophysiologic process causing accumulation of an acid (containing protons) or loss of a base (both will increase H+), which lowers the pH. This may or may not result in an acidemia. An acidosis can be respiratory or metabolic in origin, i.e. primary respiratory or metabolic acidosis. Alkalaemia is a blood pH higher than normal. This is important to distinguish from the term's acidosis and alkalosis, pH (or a decrease in blood H+ concentration). Again, this differs from alkalosis- a pathophysiologic process causing accumulation of a base or loss of an acid (both will decrease H+), which increases the pH. This may or may not result in an alkalemia. An alkalosis can be respiratory or metabolic in origin, i.e. primary respiratory or metabolic alkalosis. When analysing acid-base, sample collection is particularly important because the pCO2 of the sample will rapidly decrease as it equilibrates with air. The main factors that will result in erroneous results are time delays, exposure to air (including bubbles within the sample) and over-dilution of the sample with anticoagulant such as heparin. When referring to a primary acid-base disturbance, there is a major abnormality that will drive the direction of pH changes. For example, a primary metabolic acidosis will decrease the pH. Secondary or compensatory acid-base disturbance is a compensatory response by the body in an attempt to correct or normalise alterations in pH (specifically, H+) caused by the primary disturbance. The pH can sometimes return to normal within this compensation, but it often overshoots and goes the other way. Simple (uncomplicated) disturbance indicates that there is a single primary acid-base disturbance. This may or may not be accompanied by the expected compensatory response. A mixed disturbance indicates that there is more than one primary disturbance (two or three), e.g. a primary

respiratory acidosis and primary metabolic acidosis. You will never find a mixed respiratory disturbance (i.e. alkalosis and acidosis).

Henderson-Hasselbalch Equation

pH= 6.1 + log ([HCO3-]/[0.03 x PCO2])

An acid is a hydrogen ion (or proton) donor, a base is a hydrogen ion acceptor. The traditional approach to acid-base analysis is based on the Henderson-Hasselbalch equation. From this equation, it is clear that pH is directly related to bicarbonate (that is, as bicarbonate increases, pH increases) and inversely related to pCO2 (so as pCO2 goes up, pH goes down). From this equation it is evident that pH is a consequence of the ratio of pCO2 and bicarbonate. When an abnormality in one side of the system occurs, the other side of the system attempts to return the ratio of bicarbonate and pCO2 back to normal, in order to restore a normal pH, this is called compensation. The body has 3 major processes to maintain acid-base balance: regulation of pCO2 by ventilation, buffering of acids with buffer systems (both bicarbonate and non-bicarbonate), and altering renal excretion of hydrogen ions or bicarbonate. The respiratory component of the system is represented by pCO2, whereas bicarbonate and base excess represent the metabolic component, which is influenced by both buffering systems and renal excretion. Carbon dioxide acts as an acid because of its ability to react with water and produce carbonic acid, as the equation shows. As the pCO2 increases, the equation is driven to the right and the hydrogen ion concentration will increase. As we've said, pCO2 represents the respiratory side of the system. Bicarbonate and base excess are both ways of quantifying the effect of the metabolic side of the system. Bicarbonate concentration is an easier concept to understand: bicarbonate is basic (as opposed to acidic) so more bicarbonate means more base, which is an alkalotic process. Less bicarbonate means less base, which is an acidotic process.

Electrolytes and Base Excess

Base excess (BE) is a more complicated concept: it is the amount of acid that would need to be added to a sample of oxygenated whole blood to restore the pH to 7.4 assuming a pCO2 of 40mmHg and a temperature of 37°C. Theoretically, a normal individual should not have an excess or deficit of acid or base, so the base excess should be zero. The major advantage of base excess over bicarbonate is that it is independent of the respiratory side of the system because it is not influenced by pCO2. Within the body there are cations (sodium and potassium) and anions (made up of chloride, TCO2 and the anion gap which is made up of predominately albumin, but also things such as phosphates and are the 'unmeasured' anions that help to maintain electroneutrality against the measured cations). The cations and anions will always counterbalance the other- and this is how to assess when simple metabolic acid-base disorders are occurring. The secondary profile consists of the potassium and the urine pH. These will often change in retaliation to the changes in the right column. For example, the potassium will rise in acidosis; there is a decrease in metabolic alkalosis. A frequently cited mechanism for this is that acidosis causes potassium to move from cells to extracellular fluid (plasma) in exchange for hydrogen ions, and alkalosis causes the reverse movement of potassium and hydrogen ions.

Strong Ion Theory

This non-traditional theory created by Peter Stewart in 1981, works on the basis that strong ions and proteins will have affect on the acid-base status. This theory treats body fluids as a system, and states that not just the H+ and HCO_3^- determine the pH. To simplify this theory, there are dependent variables (H+, OH–, HCO3–, HA [weak acid], A– [weak anions]) that are reliant on the independent parameters and try to create equilibrium. The independent variables (pCO_2, ATOT [total weak non-volatile acids], SID [net Strong Ion Difference]) control the acidity in the arterial plasma. These independent variables can be predicted through equations. The body works to maintain electroneutrality through the dissociation of equilibria with weak electrolytes. Strong ions are those ions that dissociate completely at a pH of 7.4. SID= [strong cations; Na^+, K^+, Ca^{2+}, Mg^{2+}] – [strong anions; Cl^- and SO_4^{2-}]. Since Na^+ and Cl^- are the most abundant strong ions, it is relatively simple to calculate the SID from Na^+ and Cl^-. Anions also include L-lactate and urate, which can have great influence over the pH. A high SID equates to alkalosis due to a loss of Cl^- or a gain of Na^+. A low SID, or acidosis, is due to a gain of Cl^- or loss of Na^+. ATOT are acids that are not fully dissociated at a pH of 7.4 and will not cause changes to the respiratory or renal mechanisms, and these include proteins such as albumin and phosphate. Unmeasured strong ions include lactate, ketones (β-hydroxybutyrate, acetoacetate), and sulfates. These ions are attributed to BE; since unmeasured strong ions are anions, they will cause an acidosis and decrease the BE.

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EXPLORING RENAL REPLACEMENT THERAPIES

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Learning objectives:

- Identify four extracorporeal therapy modalities that are available

- Recognize the different mechanisms of each modality

- Distinguish the ideal patient populations for intermittent hemodialysis vs. continuous renal replacement therapy

- Explain the indications for therapeutic plasma exchange

Proceeding:

Extracorporeal therapies have been used in the treatment of kidney disease, toxin ingestion, and autoimmune diseases in dogs and cats. While there are limitations in availability and cost, early referral for renal replacement therapy when indicated can help increase chances of success. These modalities are commonly used to allow the kidneys to recover from injury while maintaining critical functions like fluid and electrolyte balance and removal of waste products. Extracorporeal therapies that are currently used include peritoneal dialysis, intermittent hemodialysis, continuous renal replacement therapy, and therapeutic plasma exchange.

Peritoneal dialysis uses the patient's peritoneum as a semipermeable membrane to promote removal of waste products through diffusion and osmosis. This modality can be performed more readily in many veterinary hospitals as compared to other extracorporeal modalities that require a dialysis machine. A peritoneal dialysis catheter is placed into the abdomen to allow infusion of dialysate. Dialysate solutions are hyperosmolar and contain electrolytes to draw in excess fluid and solutes over time prior to being removed. This process is repeated continuously while monitoring the patient's response. Complications associated with peritoneal dialysis include clogging of the catheter, electrolyte disturbances, peritonitis, pleural effusion, and hypoalbuminemia.

Intermittent hemodialysis (IHD) and continuous renal replacement therapy (CRRT) utilize a hemodialysis machine, dialyzer, and circuit. A dialysis catheter is placed in the jugular vein to allow adequate blood flow through the circuit. Within the dialyzer, blood flows through thin tubes encased in a semipermeable membrane. Dialysate flows around these thin tubes allowing for diffusion of solutes from areas of high concentration to areas of low concentrations to help correct kidney values, electrolyte, and acid-base disturbances. Ultrafiltration promotes the removal of excess water through a pressure gradient created

between the semipermeable membrane. This movement of water will also create a solvent drag, further contributing to the removal of solvents through convection.

IHD consists of approximately three weekly treatments that are three to four hours. IHD can be beneficial in toxin ingestions and acute or chronic kidney disease. CRRT treatments occur over 12 or more hours to more closely imitate normal kidney function and require a longer period of hospitalization. CRRT can be beneficial in cases of fluid overload and acute or chronic kidney injury. Complications associated with IHD and CRRT can include hypotension, coagulation abnormalities, anemia, hypothermia, and dialysis disequilibrium syndrome.

Therapeutic plasma exchange (TPE) is used to remove unwanted toxins and antibodies from the blood by removing the patient's plasma. A jugular catheter is placed to allow adequate blood flow through the circuit. Centrifugal TPE utilizes a centrifuge to separate plasma from the blood, whereas membrane-based TPE utilizes a filter to separate plasma from the blood. The plasma that is removed through either process is then discarded. The separated red blood cells are returned to the patient along with fresh frozen plasma and crystalloid fluids to restore intravascular volume. TPE has been found to be beneficial for highly protein bound toxins like NSAIDs, immune mediated hemolytic anemia, immune mediated thrombocytopenia, and myasthenia gravis. Complications associated with TPE include filter clotting, hypocalcemia, vomiting, and hypersensitivity reactions.

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DYSNATREMIA

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Learning objectives:

- Understand the body's mechanisms for maintaining sodium balance

- Identify clinical symptoms of hypo and hypernatremia

- Review treatments of dysnatremia

- Discuss specific nursing care needed for patients with sodium abnormalities

Proceeding:

Sodium abnormalities are common in critical patients. Studies in human medicine have shown that even small changes are associated with an increased mortality risk.

Sodium

Sodium is the major extracellular cation. It is closely related to water balance within the body and is the primary determinant of serum osmolality. The link between sodium and water balance means that changes in circulating volume affect or may be secondary to the sodium levels in the body. The kidneys are the main regulator of sodium balance and normally sustain a narrow normal range to achieve equilibrium. This process is supported by physiologic mechanisms within the kidney.

Hyponatremia

Hyponatremia is identified when sodium levels drop below 140 mEq/L. Calculating the osmolality of serum can help identify the cause of the hyponatremia. Normal serum osmolality is 290-310 mOsm/kg in dogs and 308-355 mOsm/kg in cats. Serum osmolality can be calculated using the formula: 2(Na+K) + glucose/18 + BUN/2.8. Hyponatremia with hyperosmolality (>340mmol/L) is typically seen with hyperglycemia or mannitol administration. Hyponatremia with hypoosmolality is usually due to sodium loss or increased water conservation from a disease state. Less commonly patients may present with normal osmolality due to hyperlipidemia or hyperproteinemia.

Clinical Signs

Patients who present with hyponatremia may be lethargic, weak, inappetant and have an altered mentation. Neurologic signs are common including seizures and coma that may lead to death.

Treatment

Treatment is directed at addressing the underlying cause. In patients with mild clinical signs correction of sodium levels may not be necessary. Fluid intake should be closely monitored and be placed on a balanced crystalloid solution with a sodium content as close to current plasma sodium levels as possible. When correction is indicated hyponatremia should be corrected slowly at no more than 0.5 mEq/L/hr to prevent further neurologic harm. Volume status should be closely monitored on these patients, including serial weights, fluid intake and urine output.

Hypernatremia

Hypernatremia (>160mEq/L) is less common because normally the physiologic mechanisms of the body prevent it. When it does occur, it is due to one of three causes. The least common cause is sodium gain. This occurs when a patient ingests an excess of salt. This causes an increase in osmolarity in the extracellular fluid compartment (ECF) and therefore a shift of fluid from intracellular fluid (ICF) to ECF leading to hypervolemia. Animals who have no access to water or who have disease processes involving vasopressin abnormalities may present with hypernatremia from pure water loss. This is also known as a free water deficit, and it causes the ECF to become hypertonic pulling water from the ICF. The most common cause of hypernatremia is from loss of hypotonic fluid, often due to non-renal losses, such as vomiting and diarrhea. Patients will present as hypovolemic.

Clinical Signs

Severity of clinical signs depends on how quickly the hypernatremia developed. Generally, signs are not seen until sodium levels rise above 170mEq/L. Symptoms can be vague, including lethargy, weakness and seizures. As serum osmolarity increases water moves from brain cells into ECF causing decreased cerebral cellular volume which can lead to cerebral hemorrhage and permanent brain damage. If the hypernatremia is chronic, symptoms may be less severe because the body has had time to acclimate to the change with the creation of idiogenic osmoles to protect the brain by keeping water in the intracellular space.

Treatment

Treatment can be difficult, and many factors must be considered. In addition to identifying the underlying cause of the abnormality, hydration status must be closely monitored and the rate of sodium correction carefully considered. If the patient has an acute development of hypernatremia, it can be corrected rapidly with minimal risk for further cerebral sequela. Idiogenic osmoles take 24 hours to develop, with chronic hypernatremia, or if the amount of time is unknown, the body may have already put into place mechanisms to protect itself. Sodium concentration therefore needs to be corrected more slowly (over 48-72 hours). Electrolytes should be measured every 2-4 hours to ensure sodium levels do not change to rapidly. A packed cell volume and total solids will help monitor patient response to fluid therapy. In light of neurologic complications, patient mentation must be closely monitored. Patient comfort may indicate placement of a urinary catheter for recumbent patients and central venous catheter for serial blood draws.

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Main Stream, Saturday 7 June 2025

HOW TO ASSES VOLUME STATUS USING POCUS?

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Learning objectives:

- Recall the different abnormal volume status: hypo vs hypervolemia / fluid overload

- Understand the concept of static versus dynamic assessment of volume status

- Understand that correct volume assessment of a patient will improve their medical management and outcome

- Learn how to use cardiac POCUS, large vessels size and dynamics and lung POCUS to better assess volume status

Proceeding:

Volume status can be defined as low (hypovolemia), normal (euvolemia), or high (hypervolemia or fluid overload). It usually refers to the blood volume of a patient at a set time. Fluid responsiveness is another concept that help predict a favourable or unfavourable outcome from fluid administration to a patient by increasing its cardiac output. Volume status assessment cannot be static; it needs to be reevaluated during hospitalization as patients may be receiving fluids, medications, or, on the other hand, experiencing significant losses.

Volume status assessment should always begin with an appropriate and thorough history taking and clinical examination. Perfusion parameters (heart rate, mucous membrane colour, capillary refill time, pulse quality, mentation, and extremities temperature) are excellent first-line tools to assess a patient for hypoperfusion. Decreased perfusion parameters are unfortunately not specific to the type of shock (hypovolemic, cardiogenic, obstructive, distributive). Parameters of the tree of life are affected differently in these scenarios, and some patients may require fluids while others may need vasopressors or inotropes. In a patient with fluid overload, physical indicators may include increased weight, peripheral oedema, retrograde jugular pulse, and chemosis.

The first use of POCUS to identify patients in need of fluids involves the evaluation cardiac cavities. A hypovolemic patient may present specific abnormalities. On a right parasternal short axis view, focusing on the left ventricle, the underfilled cardiac cavity will appear "empty" compared to usual, and the ventricular walls may appear thickened ("pseudohypertrophy"). In a long axis four chamber view, one

might observe a "kissing ventricle" referring to the walls of the ventricle touching due to being underfilled. On the other end, a patient with fluid overload may have an enlarged heart cavity. In this case, examining the left atrium is a reliable indicator. On a right parasternal short axis view at the aortic level, an increased left atrium to aortic ratio (LA:Ao) can indicate fluid overload, although it cannot be differentiated from intrinsic cardiac disease when examined alone.

Large vessels, particularly large veins, serve as high-capacitance vessels that provide valuable information about both volume status and fluid responsiveness. When assessing the caudal vena cava (CVC), different views are described: sub-xiphoid, transhepatic, and para lumbar. The first two sonographic views allow for assessing the CVC diameter and its dynamics regarding the proximity to the thorax and the interactions between the heart and lungs. The last one can provide a view of the aorta for comparing diameters. Patients with low volume status are likely to have a small diameter CVC and the vessel will look collapsible. On the other hand, a patient with fluid overload will have a large diameter CVC, and the vessel will appear distended and less collapsible.

Lung POCUS is also an excellent tool for evaluating fluid overload. Pulmonary oedema can be visualized by detecting an abnormal increase in the number of B-lines. Pleural effusion is also a common finding in (feline) patients with fluid overload. Abdominal POCUS can provide additional clues regarding volume overload. Finding effusion or gallbladder oedema—though not specific and not always present—can be associated with fluid overload.

Understanding the multitude of POCUS assessment points for volume status and fluid responsiveness will enhance clinicians' accuracy and effectiveness and help to adjust therapeutic plan. Volume status can be defined as low (hypovolemia), normal (euvolemia), or high (hypervolemia or fluid overload). It usually refers to the blood volume of a patient at a set time. Fluid responsiveness is another concept that help predict a favourable or unfavourable outcome from fluid administration to a patient by increasing its cardiac output. Volume status assessment cannot be static; it needs to be reevaluated during hospitalization as patients may be receiving fluids, medications, or, on the other hand, experiencing significant losses.

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KEY TAKEAWAY FROM THE VECCUS POCUS DAY

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Proceeding:

This lecture will summarize the key insights gathered during the VECCUS Congress Day, focusing on the evolving role of Point-of-Care Ultrasound (POCUS) in emergency and critical care settings. Throughout the day, esteemed speakers have shared their expertise on the benefits of POCUS techniques across a range of applications, from vascular access to lung ultrasound and shock assessment. This session will distil essential take-home messages, emphasizing both the advantages and the potential pitfalls when implementing these novel techniques in clinical practice.

The lecture will begin by reviewing POCUS applications in vascular access and phlebitis assessment, highlighting its role in improving success rates while minimizing complications.

The discussion will then move to ultrasound in atelectasis and lung recruitment, led by Angela Briganti, followed by an exploration of diaphragmatic excursion and ventilation, presented by Angela Briganti and Chiara Di Franco. Additionally, the session on decoding POCUS lines and signs by Hugo Swanstein and Søren Boysen offers crucial insights into image interpretation, an essential skill for avoiding diagnostic errors. These topics will emphasize the integration of lung ultrasound in respiratory management and its implications for patient outcomes.

A pivotal keynote by Radovan Radonic provided a broader perspective on how POCUS has revolutionized acute care in human medicine. This will be used as a foundation to explore parallels and lessons applicable to veterinary medicine.

The afternoon discussions have also tackled the nuances of POCUS in different species, comparing its application in cats vs. dogs, and assessing its value in states of shock, as presented by Alexandra Nectoux. The day concluded with an important discussion led by Alessio Vigani on the dangers of misusing POCUS when its limitations are not well understood, underscoring the importance of proper training and clinical context in interpretation.

This lecture will serve as a structured review, ensuring attendees leave with a comprehensive understanding of the day's discussions. By reinforcing the benefits while clearly identifying potential pitfalls, we aim to equip practitioners with the knowledge to implement POCUS effectively and responsibly in their clinical settings.

KEY NOTE: BETTER IMPLEMENTATION OF CRRT: FROM ANIMAL MODELS TO HUMAN STUDIES AND VICE-VERSA

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Learning objectives:

- To show studies and advancements in CRRT on animal studies and how a critical care physician can implement this to his routine practice

- To show new approaches and indications verified in human studies and how can that be extrapolated to veterinarian medicine

Proceeding:

To show the latest publications and achievements in the field of CRRT in both human studies and animal studies and how these results can be implemented in a standard, every-day clinical practice.

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THE GASTROINTESTINAL TRACT AND CRITICAL ILLNESS

Claire Sharp [1]

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Learning objectives:

At the end of this lecture, you will be able to:

Describe the various contributors to gastrointestinal tract dysfunction in critical illness. Recall the varied clinical indicators of gastrointestinal tract dysfunction, and how some of these are incorporated into a scoring system in human medicine. Describe a systemic approach to the prevention and treatment of gastrointestinal tract dysfunction in critically ill dogs and cats.

Proceeding:

Gastrointestinal (GI) tract dysfunction can be considered an organ dysfunction in dogs and cats with systemic inflammation and sepsis. This may result in dysmotility, feeding intolerance, barrier dysfunction, and malnutrition. This lecture will discuss clinical recognition of GI tract dysfunction and discuss a wholistic treatment approach including treatment of the underlying disease, identifying and reducing risk factors for dysmotility, early mobilisation, appropriate IV fluid therapy, and early enteral nutrition. Other components of treatment include the appropriate use of antiemetics, prokinetics, antacids, gastroprotectants, antibiotics, and probiotics.

Normal GI tract function and Dysfunction in critical illness

Normal GI function includes nutrient absorption, immune and barrier function, and motility including mechanical digestion. Gastrointestinal dysfunction should be considered in organ dysfunction in critically ill patients,[1] with consequences including malabsorption/malnutrition, barrier dysfunction, dysmotility, and feeding intolerance.

Regarding the barrier function the GI has both immunologic and non-immunological defence functions. Non-immunologic aspects of barrier dysfunction are commonly affected by critical illness and our treatments. For example, gastric acid secretion is interrupted with antacid therapy, peristalsis can be impaired by drugs, epithelial cell permeability barrier may be increased secondary to decreased oxygen delivery (DO2), and enterocyte malnutrition, while sphincter function can be interrupted by tube placement, and some surgical procedures (eg. Bilroth).

Normal GI motility is also vital for normal GI function.[2] Compromised GI motility in our dog and cat patients is undoubtedly multifactorial, with contributors including the effects of stress, medication, surgical intervention / disruption, and compromised DO2, in addition to specific gastrointestinal diseases

(eg. GDV, foreign body obstruction).[3] Indeed even just hospitalising healthy dogs significantly increases gastric emptying time.[4] Medications that can delay gastric emptying include opioids, anticholinergics, and calcium channel blockers.[5]

Diagnosis of GI dysfunction

Clinical indicators of GI dysfunction may include nausea, vomiting, reflux / regurgitation, abdominal distention, abdominal pain, intra-abdominal hypertension, enteral feeding intolerance, GI haemorrhage (eg. haematemesis, haematochezia, and/or melena), reduced gut sounds, diarrhoea, or constipation. In human medicine a Gastrointestinal Dysfunction Score (GIDS), that could be incorporated into a SOFA score, has been developed for critically ill patients.[6] This, grades patients from 0 (ie. No risk of GI dysfunction), through to 5 (ie. GI failure with severe impact on distant organ function) which is life-threatening GI dysfunction.

Treatment approach

Treatment considerations for GI dysfunction should include treating the underlying disease, early mobilisation, appropriate IV fluid therapy, early enteral nutrition (EN), adjusting other therapies to minimise adverse effects on the GI tract, appropriate use of GI medications (including antiemetics, prokinetics, antacids, gastroprotectants), as well as appropriate use of antibiotics and probiotics.

IV fluid therapy is of course indicated for resuscitation from hypovolemic shock, to resolve dehydration, provide for maintenance and replace ongoing losses when the patient is unable to do so by drinking. Appropriate IV fluid therapy can be lifesaving, but fluid overload or overhydration can be deleterious, with the GI tract particularly susceptible to injury. The ROSE model of fluid therapy is a useful consideration here.[7]

Early EN advocates feeding as soon as possible after haemodynamic stability is achieved, rather than delaying nutritional intervention by several days. Numerous publications report the findings of studies investigating early EN in veterinary medicine including in diseases such as acute pancreatitis,[8, 9] septic peritonitis,[10, 11] and canine parvovirus gastroenteritis[12]. Pros and Cons of each type of feeding tube, and the various diet options, should be considered in the context of the individual patient.

Adjusting other therapies to minimise adverse effects on the GI tract may involve using multimodal analgesia to reduce the use of opioids, optimising serum electrolyte concentrations, and even considering laparoscopic surgery instead of open abdominal surgery.

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HOW HAVE NUTRITIONAL RECOMMENDATIONS CHANGED FOR CRITICALLY ILL PATIENTS

Daniel Chan [1]

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Learning objectives:

- Review how the role of nutritional support has evolved in critical care

- Understand how the opinion on timing to initiation of nutritional support has changed

- Review how the role of parenteral nutrition in critical care has changed

Proceeding:

For many years, the approach with critically ill patients was to delay provision of nutrition until they were well into their recovery. Early in critical illness the focus was on improving cardiovascular stability and oxygenation. When the effects of malnutrition on patient morbidity and mortality were realised, a reactive movement ensued in critical care and many patients were fed quite aggressively, leading to the term "hyperalimentation," which we have now recognised as causing its own set of problems. Much like other aspects of critical care, our paradigm of critical care nutrition constantly changes; previously held assumptions become less relevant and new research uncovers novel strategies.

Timing Of Nutritional Support:

In dogs, a period as short as 3 days of anorexia has been documented to produce metabolic changes consistent with those seen associated with starvation in people. In cats' detectable impairment of immune function can be demonstrated in healthy cats subjected to acute starvation by day 4, and so recommendations to institute some form of nutritional support in any ill cat with inadequate food intake for more than 3 days have been made. In both dogs and cats, there is some consensus that there is an urgent need to implement a nutritional intervention (e.g., place feeding tube) when a dog or cat has not eaten for more than 3 days. Currently, the major recommendation for support of the critically ill animal is to initiate early enteral feeding as soon as it is feasible. Some veterinary studies have implemented enteral nutrition as early as within 10 hours of admission. Although it is difficult to assess whether these measures actually influence outcome, one thing that has been established is that many patients previously assumed to require "bowel rest," actually tolerated early enteral feeding. The most notable studies demonstrating this tolerance to feeding evaluated puppies with parvoenteritis, cats with acute pancreatitis and most recently, dogs with severe and acute pancreatitis. It is therefore reasonable to pursue enteral feeding even in cases believed to have significant gastrointestinal dysfunction.

Parenteral Nutrition: Bad Intervention or Just Bad Timing?

The optimal timing of implementing parenteral nutrition (PN) is very controversial at the moment, even in people. Publications specifically evaluating timing of initiating PN support (eg, early enteral followed by early PN vs early enteral followed by delayed PN) have suggested that early initiation (ie, within 48 h of admission to ICU) was associated with higher morbidity and mortality. Other studies have corroborated these findings and there is a growing consensus against initiating PN until day 8 of ICU admission in people. However, there should be considerable caution in extrapolating these results to companion animals. First, the differences were rather modest (6% increase in likelihood of being discharged alive) and the clinical relevance of such modest differences needs to be considered. A perhaps more important distinction in the provision of nutritional support, is the optimal calorie target. There is ample evidence in both the human and veterinary literature demonstrating the deleterious effects of over-feeding in both morbidity and perhaps mortality. For these reasons, recent veterinary recommendations have centred on simply targeting RER. Although there are similar recommendations in human ICUs, an analysis of the caloric target in the recent studies, which demonstrated longer hospitalisation times, longer ICU stays, shows that patients who did worse were essentially “aggressively fed” and likely overfed compared to. In animals, where a combination of enteral and parenteral nutrition was associated with better outcome, the total target was less than RER. It is therefore possible that positive outcomes can be achieved with supplemental PN, as long as the total caloric target is set very conservatively at RER.

Changes In Veterinary Nutritional Support:

The standard approach to feeding critically ill animals is to initiate enteral nutrition as soon as possible and so feeding tubes are commonly employed in veterinary ICUs. Another recent assumption, which has been challenged is the perception that critically ill animals need to be fed via continuous rate infusions rather than receive boluses of food into their feeding tubes. A recent trial found no difference in tolerance or ability to reach nutritional targets by feeding animals using boluses rather than continuous rate infusions. Overall effect on outcome is unfortunately beyond these small trials but emphasises the need to challenge notions, especially when not based on credible evidence.

Summary

Nutritional support continues to be an evolving topic in critical care. From the problem of under-feeding to grossly overfeeding, these may be important areas requiring further refinement. The timing of intervention, the optimal caloric goal and even composition of diet should be further investigated. Previous assumptions about feeding the critically ill animals have been successfully challenged and the way we approach some critically ill animals has dramatically changed. Newer techniques, newly developed diet formulations will also enhance the way we support patients. These innovations along with new research findings illustrate how dynamic the field of critical care nutrition is, and highlight the need to continually reassess and question our knowledgebase.

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REVISITING THE POTENTIAL ROLE OF ANTIOXIDANT THERAPY IN CRITICAL CARE

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Learning objectives:

- Review the role of oxidative stress in health and disease

- Understand how oxidative stress may be targeted therapeutically

- Understand why decades of antioxidant therapy in critical illness may not have improved outcomes

- Understanding how new approaches to oxidative stress may be required in managing critical illness

Proceeding:

Classically, critical illness has been described as a state with marked redox/oxidative imbalance where there is increased production of oxidant and free radical species and consumption of endogenous antioxidant defences. There is also ample evidence showing an association between multiple organ damage/failure, poorer patient outcomes and this oxidative imbalance. As the relationship between oxidative stress and diseases has been well established, there has been a considerable interest in targeting this imbalance and exploring the role of nutrition in potentially improving outcome. Despite decades of clinical research, the use of antioxidants in critical care has had inconsistent beneficial results.

What has been tried in terms of antioxidant use in critical care?

One strategy has been to use exogenous untargeted antioxidants such as N-acetylcysteine (NAC) in patients with severe liver injury, sepsis and acute respiratory distress syndrome (ARDS).[5] Administration of NAC aims to replenish glutathione and scavenge ROS. Whilst one can demonstrate an improvement in glutathione concentrations, studies have not been able to show improvements in mortality. A different approach has been to attempt to replenish endogenous antioxidants by supplementing antioxidant such as vitamins A, C, E and selenium. Again, inconsistent results have been shown with such an approach and in some populations, a surprising increase in mortality.

Why antioxidants may not have worked?

Similar to disappointments with blocking cytokines such as TNF-alpha, IL-1, antioxidant therapy in sepsis, ARDS and critical illness has not yielded the results hoped. One thought to reconcile the fact that although critical illness is indeed associated with oxidative stress, and yet antioxidant therapy has not improved outcomes could be that disrupting ROS may have unexpected detrimental effects. It is important to remember that ROS serve as important signalling molecules for normal cellular function.

For example, in combating infections, immune cells rely on respiratory burst to kill pathogens. Furthermore, ROS are also involved in optimal activation of lymphocytes and monocytes which critical to fight infections. Therefore, it is possible that exogenous antioxidants may be curtailing cellular processes required for optional response to stresses such as infections. There is some thought that there may be aspects of the inflammatory response that actually require oxidative stress, and so blunting oxidative stress may explain some of the negative results seen in some trials.

Conversely, when modulation of oxidative stress is believed to be beneficial, it may be that the use of small molecules therapeutically (eg, vitamin C, vitamin E, beta-carotene, polyphenols) may have been disappointing largely due to, as described by Forman et al, as an "overly optimistic and incorrect assumptions about how antioxidants work." For example, attempts to use small antioxidant molecules to quench or scavenge hydroxyl radicals is too impractical since antioxidant enzymes react thousands to millions of times more rapidly with those oxidants than small molecules do. It would be far more effective to prevent the formation of the hydroxyl radical by reducing hydrogen peroxide production in the first place. It has also come to light that most antioxidant defence within cells is not provided by either exogenous or endogenous small molecules acting as scavengers, but by antioxidant enzymes using their specific substrates to reduce oxidants. Therefore, the major therapeutic opportunities lie in the prevention of oxidant production that cause injury, inhibiting downstream signalling by oxidants that results in inflammation or cell death, and increasing both antioxidant enzymes and their substrates.

Part of lack of success of using antioxidants therapeutically is the fact that once oxidant damage begins (ie, oxidant stress being the primary cause of pathology), antioxidant therapy cannot inhibit the progression of tissue injury as other factors become dominant in this pathological process.

Refocused antioxidant therapeutic strategies

Given mixed results with small antioxidant molecules, there are several antioxidant enzyme mimics being evaluated in clinical trials. Superoxide dismutase (SOD) is the only enzyme that can eliminate oxygen free radicals in mammalian cells and is a key component in defence against oxidative stress. Using its mimic is a current strategy being evaluated in several trials.

The induction of antioxidant enzymes, particularly through transcription factors (namely, nuclear factor erythroid 2-related factor or NRF2 activators), is a major way in which antioxidant therapy is being developed. Dysregulation of NRFF2 signalling does appear to be implicated in many oxidative stress-related disease processes, therefore are regarded as potential agents to enhance antioxidant defences.

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TOOLS TO EVALUATE NEUROLOGICAL FUNCTION IN THE ICU PATIENT: EEG AND BEYOND

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Learning objectives:

- To build confidence in performing serial neurological assessments of the ICU patient.

- To understand the potential utility of electroencephalography in assessing neurological function.

- To recognise additional monitoring tools that could be considered to aid prognostication in the ICU, such as advanced imaging and serum biomarkers.

Proceeding:

After resuscitation from out-of-hospital cardiac arrest, 80% of people admitted to an intensive care unit are comatose. Decision making over whether life support should be continued is guided by multiple modalities including serial neurological assessments, advanced imaging of the brain, serum biomarker evaluation and electrodiagnostic testing. The European Resuscitation Council and European Society of Intensive Care Medicine guidelines recommend delaying neurological prognostication for at least 72 hours after return of spontaneous circulation (ROSC) in people.[1] To date, no equivalent recommendations exist in veterinary medicine and our efforts at prognostication are often limited to serial clinical assessments. Application of a more multimodal approach could support clinical decision making to avoid intensive and prolonged efforts at rehabilitation if the ultimate recovery and associated quality of life will not reach a sufficient level.

Neurological examination

The presence of initially severe neurological abnormalities does not preclude acceptable functional outcome in patients with brain injury. Regular reassessments of key neurological parameters such as mentation, cranial nerve evaluation and posture, supported by objective grading systems such as the modified Glasgow Coma Scale (GCS), can identify changing neurological status and guide prognostication attempts. Detectable neurological improvements within the first 72 hours, and continued improvement every 48 hours thereafter, might suggest a better prognosis and justify continued ICU care.[2]

Advanced imaging

Magnetic resonance imaging (MRI) is routinely used in comatose people to characterize lesion extent and severity. In particular, identification of diffuse brain injury on MRI, specifically attenuation of the gray and white matter interface, is a negative prognostic indicator.[3-5] In veterinary medicine, the high cost of MRI can preclude its use, and we currently lack an evidence base to guide prognostication.

However, application of principles used in human medicine could help in guiding clinical decision making and form the basis for prospective veterinary studies.

Electrodiagnostic testing

There is a growing interest in electroencephalography (EEG) in veterinary medicine. EEG records electrical activity arising from cortical neurons. Its primary utility is in the detection of epileptiform discharges, but assessment of background activity provides an indication of alertness and brain function. EEG is routinely used in the evaluation of people after cardiopulmonary arrest, with continuous EEG monitoring enabling assessment of the evolution of brain activity over time.[6] Patients typically show suppressed background activity patterns (<10 µV) immediately after cardiac arrest, with gradual increases in amplitude and continuity thereafter. Progression toward continuous normal voltage background activity within 24 hours of cardiac arrest has been shown to be associated with good outcome. A poor prognosis was associated with suppressed background activity, a non or poorly responsive EEG pattern (i.e. no response to auditory, visual, tactile, or noxious stimuli) and the presence of periodic generalized phenomena.[7,8]

Brainstem auditory evoked potentials (BAEPs), another patient side electrodiagnostic test, offer a means to assess the integrity of the central hearing pathways as an indicator of brainstem function. The absence of BAEPs has been documented in comatose people and dogs. A study in dogs reported that BAEPs were sensitive for the detection of brainstem lesions, while additionally providing information on location and extent of lesions.[9] BAEPs are rapid and relatively straight forward to perform, and their serial assessment in ICU patients could support prognostication.

Serum biomarkers

Neuron specific enolase (NSE) is released from injured neurons and its plasma concentration correlates with the severity of global hypoxic-ischaemic brain injury in people following cardiac arrest. [10] Other biomarkers of interest include S100-beta (released from damaged glia) and protein tau (released from damaged axons). In veterinary medicine, various proteins have been evaluated as potential biomarkers in different diseases, such as neurofilament light chain in meningoencephalitis of unknown aetiology[11] and cognitive decline.[12] Serum glial fibrillary acidic protein plasma concentration has been shown to predict outcome in dogs with complete spinal cord injury.[13] Veterinary studies are needed to evaluate the clinical utility of serum biomarkers in the ICU patient, and to develop rapid patient side tests.

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ASSESSING FLUID RESPONSIVENESS

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Learning objectives:

- Describe the difference between assessing volemic status and fluid responsiveness

- Describe the benefit of using dynamic parameters (versus static parameters) for monitoring fluid responsiveness

- Describe techniques for assessing dynamic parameters of fluid responsiveness

- Describe techniques for measuring cardiac output

Proceeding:

Volemic Status and Fluid Responsiveness

There is an important difference between assessing volemic status and assessing fluid responsiveness. Assessment of volemic status involves determining if the patient has a normal, decreased, or increased intravascular volume at one point in time. Fluid responsiveness is the dynamic assessment of volume status to determine if stroke volume (SV) or cardiac output (CO) increase after a fluid bolus.

In a patient that has been assessed to be hypovolemic, intravenous fluids are administered to restore the intravascular volume which will increase CO. However, the human literature has shown that 40-50% of critically ill patients do not have an increase in CO after the administration of a fluid bolus. The decision to continue or stop fluid resuscitation is based on determining if the patient is fluid responsive or not. Fluid responsiveness has been defined as a 10-15% increase in SV after the administration of 500 mL of crystalloids over 10-15 minutes. Clinically, an improvement in a physiologic parameter after a fluid bolus may also be considered fluid responsive.

Dynamic Parameters of Fluid Responsiveness

Dynamic parameters of fluid responsiveness are assessed before and after a fluid bolus to measure the magnitude of response to the acute change in preload. Dynamic parameters are more accurate at predicting fluid responsiveness compared to static parameters, but they are generally not used to assess volemic status (Evans 2021). Dynamic parameters may include CO measurement, systolic pressure variation (SPV), pulse pressure variation (PPV), stroke volume variation (SVV), and plethysmographic variability index (PVI).

Cardiac Output: Veterinarians do not commonly measure CO in clinical patients, however, it is the gold standard for assessing fluid responsiveness. There have been many different techniques described for measuring CO including Fick O_2 consumption, thermodilution, lithium dilution, pulse contour analysis, transesophageal echocardiography, transthoracic bioimpedance, bioreactance, and ultrasound velocity dilution. Fick O_2 consumption ($CO = VO_2 / (CaO_2 – CvO_2)$) is based on the Fick principle which states that the amount of oxygen consumed must equal the difference in oxygen content between the arterial and venous circulation. The benefit of CO measurement using this technique is that it is not affected by arrhythmias, decreased CO, or valvular regurgitation, however, it does require measurement of VO_2.

Thermodilution is considered the clinical gold standard and relies on the measurement of temperature changes in blood from the right atrium to the pulmonary artery. The primary limitation to this technique is that it requires placement of a pulmonary arterial catheter, which comes with risks. Lithium dilution does not require a pulmonary arterial catheter, but it does require large volumes of blood to be aspirated from the patient across a lithium sensitive cathode and lithium can accumulate. Pulse contour analysis can provide a broad range of parameters and is reliable during hemodynamic instability. However, it requires frequent recalibration, an optimal arterial waveform, and it can be affected by arrhythmias.

Transesophageal echocardiogram uses aortic flow velocity and cross-sectional area to estimate SV. It has good correlation with thermodilution, but it requires anesthesia and training in the technique. Transthoracic bioimpedance and bioreactance have not been used as frequently in veterinary patients. Ultrasound velocity dilution uses an extracorporeal circuit, peristaltic pump, and ultrasound velocity sensors to measure cardiac output. It is less invasive than other techniques but does requires specialized equipment.

Systolic Pressure Variation, Pulse Pressure Variation, and Stroke Volume Variation: The measurement of SPV, PPV, and SVV require highly controlled conditions, so the patient must be on mechanical ventilation. Spontaneous respiratory effort, arrhythmias, right heart failure, and altered intra-abdominal pressure may affect these measurements. The measurement of these variables is based on the arterial pressure waveform and depend on the heart-lung interactions.

SPV = SP max – SP min

SP = systolic pressure

PPV = [(PP max – PP min) x 100] / [(PP max + PP min) / 2]

PP = pulse pressure

SVV = [(SV max – SV min) x 100] / [(SV max + SV min) / 2]

SV = stroke volume

The literature can be difficult to interpret due to lack of standardization of the fluid type used for the challenge, fluid volume, and ventilator settings. Overall, the larger the variation, the more likely the patient will be fluid responsive. Generally speaking, a change of > 10-15% may predict fluid responsiveness, regardless of the dynamic parameter used (Boysen 2021).

Plethysmographic Variability Index: PVI is determined by a pulse oximeter during a complete respiratory cycle. The pulse oximeter measures the perfusion index, which is a relative measure of tissue perfusion and blood flow.

PVI = [(PI max – PI min) x 100] / PI max

PI = plethysmographic index

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PATHOPHYSIOLOGY OF HEMORRHAGIC SHOCK

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Learning objectives:

- Describe the cellular and systemic physiological changes associated with hemorrhagic shock.

- Explain the mechanisms by which hypoperfusion and oxygen debt lead to organ dysfunction.

- Discuss the role of inflammatory and immune responses in the progression of shock.

- Apply insights from translational research to enhance clinical understanding and interventions in veterinary hemorrhagic shock.

Proceeding:

This advanced lecture will provide a deep dive into the cellular and systemic consequences of hemorrhagic shock, with a core focus on ischemia-reperfusion injury (IRI) as the pivotal mechanism driving organ dysfunction and poor outcomes in veterinary patients. Rather than centering on the broader concept of oxygen debt, the session will explore how restoring perfusion—while necessary—triggers a complex and often damaging cascade of molecular events.

We begin with an overview of how acute blood loss initiates systemic hypoperfusion, which compromises oxygen delivery and nutrient supply. However, the primary emphasis of this lecture lies in what happens after perfusion is restored—a paradoxical moment where tissues are rescued and injured. We'll explore how reperfusion initiates a surge in reactive oxygen species (ROS), calcium overload, and inflammation that overwhelms cellular defenses and drives mitochondrial dysfunction.

Mitochondria are a central focus throughout the lecture, as they are both targets and amplifiers of reperfusion injury. We'll unpack the molecular mechanisms by which mitochondrial membranes destabilize, triggering energy failure, apoptosis, and the release of danger signals. These events contribute to systemic inflammatory response syndrome (SIRS) and, ultimately, multi-organ dysfunction syndrome (MODS). Emphasis will be placed on endothelial injury and glycocalyx shedding as key contributors to vascular leak and coagulopathy.

Attendees will also gain insights into the interconnectedness between mitochondrial damage and systemic pathophysiology, including acute kidney injury, cardiac dysfunction, and hepatic stress. Understanding these pathways allows for a more comprehensive approach to shock resuscitation—beyond volume and perfusion targets—to one considering cellular bioenergetics and redox balance.

Finally, the lecture will touch on translational insights, particularly from military and experimental models of hemorrhagic shock. We will discuss how this growing understanding of IRI and mitochondrial biology shapes the development of novel therapeutics—ranging from antioxidants to mitochondria-targeted peptides—and what that means for future clinical care.

This session is designed for delegates interested in critical care and pathophysiology, providing a scientific foundation for advanced clinical reasoning and therapeutic innovation.

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ASSESSMENT AND MANAGEMENT OF HEMORRHAGIC SHOCK

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Learning objectives:

- Perform an effective clinical assessment of hemorrhagic shock in dogs and cats, integrating physical exam findings with point-of-care diagnostics.

- Utilize point-of-care ultrasound (POCUS) to support decision-making in shock resuscitation.

- Formulate and apply evidence-based strategies for fluid resuscitation and hemorrhage control.

- Tailor organ support interventions to the stage and severity of shock for optimal patient outcomes.

Proceeding:

This lecture focuses on the clinical assessment and intensive care management of hemorrhagic shock, targeting a wide range of emergency and critical care practitioners. It offers a detailed, practical guide for diagnosing, monitoring, and stabilizing veterinary patients experiencing life-threatening blood loss.

We will begin with a structured approach to shock recognition, combining physical examination with bedside tools and laboratory data. We'll discuss key indicators such as mentation, mucous membrane color, pulse quality, blood pressure, capillary refill time, lactate, and base excess. Special emphasis is placed on trending parameters over time and using shock indices to stratify risk and response to therapy.

A cornerstone of the lecture is the application of point-of-care ultrasound (POCUS). Participants will learn how to integrate POCUS in the emergency setting to identify free fluid, assess cardiac function, evaluate vascular volume status, and detect potential thoracic or abdominal hemorrhage causes. The role of serial ultrasound for monitoring resuscitation response will also be discussed.

Therapeutic strategies focus on goal-directed resuscitation, judiciously using crystalloids, and emphasizing early blood product administration when warranted. We will cover decision-making for component therapy and the importance of calcium supplementation in the context of citrate load and coagulopathy.

Notably, while surgical and interventional radiology approaches are beyond the scope of this lecture, we will discuss non-invasive and critical care-specific hemorrhage control tools—including the principles and potential use of resuscitative endovascular balloon occlusion of the aorta (REBOA). The indications, limitations, and logistical considerations of REBOA in veterinary patients will be presented based on emerging data and experimental work.

Finally, we will touch on organ support in the intensive care setting following stabilization—particularly strategies to protect kidneys, monitor for evolving coagulopathy, and manage ongoing metabolic disturbances.

This session is ideal for clinicians involved in direct resuscitation and monitoring shock patients. It offers a modern, evidence-informed approach rooted in practical application and advanced critical care techniques.

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EXPANDING OPTIONS FOR TRANSFUSIONS: INFUSION READY PLASMA AND STORED WHOLE BLOOD

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Learning objectives:

By the end of this lecture you will be able to:

- Recall indications for whole blood and plasma transfusion

- Describe the findings of research investigating the haemostatic capacity of stored whole blood in dogs

- Describe the findings of research investigating the haemostatic capacity of infusion ready plasma in dogs, including never-frozen and thawed refrigerated plasma, as well as lyophilised plasma.

Proceeding:

Blood product transfusions are indicated for a variety of situations including whole blood loss, anaemia, and coagulopathy. Veterinary blood banking is constantly evolving, and we are fortunate to have a plethora of recent transfusion medicine and blood banking research to inform that evolution. In this lecture, new data will be reviewed that provides insights into the role of novel transfusion products and broadens our understanding of the benefits of existing blood products. We fill focus on two novel transfusion products that I believe are practice changing; infusion ready plasma and stored whole blood (SWB).

Stored whole blood

In the setting of a high-volume emergency clinic, patients often present with life-threatening haemorrhagic shock, necessitating rapid and immediate transfusion of blood products. Transfusion medicine in both humans and companion animals is an evolving science, with ongoing research designed to ensure that our clinical practice best meets the needs of these patients. Optimising transfusion management in turn will help ensure the best outcomes for these patients, since haemorrhagic complications are a common reason for morbidity and mortality.

Historically, transfusions were provided as fresh whole blood (FWB), given immediately from the donor to the recipient. As transfusion medicine has developed, so too has the use of component therapy and our ability to store blood components for longer periods of time. Component therapy involves separating fresh whole blood into its components; most commonly packed red blood cells (pRBCs) that are stored refrigerated for up to 42 days, and plasma, generally fresh frozen plasma (FFP). Component therapy facilitates targeted transfusion therapy; patients that only require red cells can be administered pRBCs, and patients that only require plasma can be administered a plasma product. Not only does this facilitate

optimal use of a precious resource, it also reduces the risk of certain transfusion reactions, such as volume overload.

More recently however, there has been a renewed interest in the use of whole blood for transfusion to haemorrhaging patients, as it is not only more efficient to replace whole blood with whole blood, but potentially also more efficacious in controlling further haemorrhage. In fact, warm FWB has even been shown to improve survival for human patients with combat-related traumatic injuries.[1-3] While transfusion of multiple units of FWB in a civilian human or veterinary emergency setting remains impractical due to limited donor availability and the time taken to collect each transfusion, the potential to administer cold-stored whole blood (SWB) is a promising alternative. Interestingly, a recent survey of transfusion practices by our research group in Australia, documented that the majority of veterinarians that store blood products are using SWB, as they lack the capacity to prepare their own blood components.[4]

Cold-stored whole blood has not traditionally been considered a useful product because it was thought that platelets rapidly lost their activity, and that clotting factor activity was also short-lived in refrigerated blood.[5, 6] But in contrast to studies from the 1960s and 1970s, recent data in human medicine has documented that clotting factors and platelets actually do retain haemostatic activity in SWB for 14 days or longer,[7, 8] and that SWB is safe and effective for rapid resuscitation of bleeding human civilian trauma patients.[9, 10]

Recently, our group and others have demonstrated the stability of dog clotting factors or platelets in SWB, generating data that can inform the clinical use of this product.[11, 12] These studies using investigating traditional coagulation test results, viscoelastic test results, coagulation factor activities, and platelet aggregometry, suggest that canine SWB retains haemostatic capacity, albeit with deterioration over 3+ weeks of refrigerated storage. Clinical trials are indicated to determine if this hemostatic capacity is clinically relevant in bleeding dogs, but findings are indeed promising.

Infusion ready plasma

Plasma transfusion is primarily indicated for the treatment of coagulopathy. Specifically, we look for our patients to fulfil two criteria as indications for plasma transfusion. Firstly, the presence of a laboratory defined coagulopathy, and secondly either active bleeding, or for pre-emptive correction of a coagulopathy in a patient requiring a surgical or invasive procedure.

FFP and FP are generally our go-to plasma products for the majority of cases with coagulopathy, however since they are stored frozen, and the requirement for thawing prior to administration results in a delay between recognition of the need for plasma transfusion and commencement of the transfusion.

A variety of methods have been used to thaw plasma for transfusion in veterinary medicine. Perhaps the most common include warm water baths (either dedicated or “makeshift), and more recently dedicated plasma dry thawing devices. Time to thaw a unit of canine FFP varies amongst publications from 15 minutes to around 35 minutes.[13, 14] While thawing times may not be a problem in stable patients, this time delay could contribute to a poor patient outcome in very unstable patients. One possible solution to this problem is the use of microwave plasma defrosters or modified commercial microwave warmers,

however data has shown that clotting factor activity is decreased when dog plasma is thawed with these products.[15, 16] As such, investigators have explored three options **for infusion ready plasma**; never frozen refrigerated plasma, thawed refrigerated plasma, and lyophilized plasma.

Never frozen refrigerated plasma

Never frozen refrigerated plasma is that which is separated from pRBCs immediately (within 8 hours) after collection and stored refrigerated without ever having been frozen. In human medicine this is called "Liquid plasma", but I refer to it as never frozen refrigerated plasma, to differentiate it from thawed refrigerated plasma, described below. One study has evaluated the ex vivo coagulation factor stability in never frozen refrigerated plasma in dogs.[14] Whole blood was collected from 9 dogs and half kept as never frozen refrigerated plasma, while the other half was frozen for comparison. Samples were collected from the never frozen refrigerated plasma at baseline, day 1, 5, 7 and 14 for measurement of coagulation factor activity. The authors found that although the activity of all clotting factors (V, VII, VIII, IX, X and fibrinogen) decreased and clotting times (PT and aPTT) increased significantly over time, no values were outside the reference intervals. Additionally, bacterial cultures from the bags were negative.[14] Based on this study some hospitals started to stock never frozen refrigerated plasma in their blood banks.

A more recent study from my research group evaluated the stability of ex vivo coagulation factor activity in never-frozen and thawed refrigerated canine plasma stored for 42 days.[17] In this study we collected whole blood from 10 dogs, and separated the plasma, storing half as never frozen refrigerated plasma, and half as thawed refrigerated plasma. The thawed refrigerated plasma was frozen at -20°C for six months, after which it was transferred to the refrigerator (more information below). In the never frozen plasma we found that activity of FV, VII, IX and X decreased slowly over time but stayed within reference intervals for 42 days. The 95% confidence intervals around the estimated marginal means for FVIII, vWF and fibrinogen concentration dropped below the reference interval by day 35-39 days, such that we recommended an expiry date for never-frozen refrigerated plasma of 32 days.

Thawed refrigerated plasma

The findings were similar for thawed refrigerated plasma, in that activity of FV, VII, IX and X decreased slowly over time but stayed within reference intervals for 42 days.[17] The 95% confidence intervals around the estimated marginal means for FVIII, vWF and fibrinogen concentration dropped below the reference interval by day 32-35 days, such that we recommended an expiry date for never-frozen refrigerated plasma of 28 days.[17]

Lyophilised plasma

Desiccated, freeze-dried (lyophilised) plasma has most recently been produced by the company BodeVet in the United States but is not currently commercially available. Lyophilised plasma products are created by freezing pooled or individual donor plasma under a vacuum and removing the ice by sublimation.[18] Such products have been used in human medicine, including in combat situations dating back to World War II. Lyophilised plasma products have the advantage of being stable with room temperature storage (~20-22°C) for up to 2 years, and then are simply reconstituted within 5 minutes using sterile water. One

study of two different canine lyophilised plasma products suggested that it may be appropriate to use such products for up to 14 days after reconstitution, but with varied stability of different coagulation factors particularly after 14 days.[18] Another study has shown equivalent hemostatic capacity to FFP.[19]

Hopefully, if the cost of production is able to be reduced, this product will reach the market and be another source of infusion ready plasma, particularly for hospitals with a lower case load that can't justify stocking liquid plasma products.

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Nurse & Tech Stream, Saturday 7 June 2025

ANAESTHESIA FOR THE HIGH-RISK PATIENT

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Learning objectives:

- Gain understanding of the drug choices used in high-risk patients

- Provide the ability to make an appropriate anaesthetic plan based on the patient's needs

- Define the ASA grading system

- Demonstrate an understanding of monitoring tools and how to mitigate risks

Proceeding:

Identifying the high-risk patient

The critical care patient requiring anaesthesia comes with higher morbidity and mortality risks. Preoperative stabilisation can prove difficult in most of these high-risk patients, and often these patients may need to be anaesthetised without being fully stabilised. The ability to predict and address perioperative and post operative risks to these patients is crucial to their survival, with diligent monitoring and guided clinical interventions being the mainstays of the veterinary nurse's role. Within human medicine, the American Society of Anaesthesiologists (ASA) created a scoring system to assess and communicate patient co-morbidities. This scoring system has been adapted for animals and is based on the patient's overall health rather than the procedure being performed. The classifications are as follows:

ASA I: Minimal risk of a normal healthy patient with no underlying disease.

ASA II: Slight risk of a slight to mild systemic disease.

ASA III: Moderate risk, obvious systemic disease.

ASA IV: High risk with severe, systemic, life-threatening disease.

ASA V: Extreme risk, moribund; patient will probably die with or without surgery.

Class IV examples may include those with severe dehydration, shock, uraemia, toxaemia, pyrexia, unmanaged diseases such as diabetes, cardiac and pulmonary diseases. Those in class V will often have decompensated states of shock, congestive heart failure, liver or kidney failure, embolus and metastasis. Classifying a patient may be subjective, though the use of huddles between the whole surgical team can

mitigate this through the use of a shared mental model. Those identified as higher risk (i.e. class IV and V) may require more pre-operative testing, stabilisation and potentially experienced staff to create a tailored anaesthetic plan. In addition, patients who are identified as higher risk patients may prompt conversations with owners surrounding survival rates, and potential cardio-pulmonary resuscitation (CPR) wishes.

Making an anaesthetic plan for the high-risk patient

Many anaesthetic drugs can impair the physiologic function of vital organ systems, potentially leading to anaesthesia-related complications. Patient factors, including extremes of age (i.e., neonatal, geriatric), presence of disease, and extremes of body weight and size, can also contribute to complications during anaesthesia, as can duration of anaesthesia, surgical procedure, approach, and invasiveness, along with patient positioning, can exacerbate some anaesthesia-related complications. It is important to use the ASA grading system to highlight individual patient risks and create a personalised plan including anaesthetic drug and dosage selection, equipment preparation, contingency planning, intra and post operative patient support and physiologic monitoring. Appropriate use of analgesic and sedative drugs tends to decrease the necessary dose of induction and inhalant drugs, improving anaesthetic safety since most adverse effects of anaesthetic drugs are dose dependent. Depending on the degree and source of pain, analgesic considerations should include opioids, local anaesthetic drugs, as well as options such as ketamine infusions to help reduce cardiovascular depressant effects of inhalant anaesthetics. Excessive and inadequate depth of anaesthesia are some of the most common and dangerous complications. Excessive anaesthetic depth often is not recognised until it results in the other complications such as hypotension and hypoventilation, and can also result in the prolonged recovery of the patient. Monitoring of the cardiovascular system using pulse oximetry, direct or indirect blood pressure monitoring and capnography will alert the veterinary nurse to rapid changes in decline, enabling them to enact on these as well as any abnormal electrocardiograms. Placement of central multi-lumen catheters will provide the ability to run multiple infusions, often required with these patients, as well as hyperosmolar solutions to reduce phlebitis risk, and the ability to collect serial blood samples which may be necessary pre, intra and post operatively. Vascular access may be difficult in these patients without fluid boluses and component therapy, or even the use of vasopressors to maintain a mean arterial pressure of more than 60 mmHg. Oxygenation is extremely important in high-risk patients, due to increased myocardial oxygen demand and often the anaerobic metabolism seen in disease processes such as shock and sepsis. Pre-oxygenation during the period leading up to intubation is important to avoid decompensation, further to that ventilatory support such as positive-pressure ventilation, may be necessary due to respiratory depression seen with some opioids and ketamine. To reduce surgical time, the operating theatre should be completely prepared and the patient may even tolerate clipping and some surgical skin preparation prior to being anaesthetised; this will reduce the total anaesthetic time and reduce distractions from monitoring the patient. Placement of nasogastric tubes will allow for gastric emptying, reducing the risk of aspiration pneumonia or oesophagitis. In addition, anti-emetics may be administered as part of the pre medication to reduce the risk of regurgitation. Emergency drugs for CPR efforts should be calculated and well stocked, as well as smaller endotracheal tubes available for the recovery period.

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RIDING THE WAVES- INTRODUCTION INTO VENTILATOR WAVES FORMS

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Learning objectives:

Delegates will be able to:

- Recognise standard waveform shapes

- Recognised what waveforms look like in different types of scalars

- Identify what common problems may look like on our waveforms

- Recognise patient deterioration

Proceeding:

Knowledge of ventilator waveforms is important when working with patients who are being mechanically ventilated in the ICU. This ensures that any signs of ventilator complications or patient deterioration can be spotted quickly, to prevent lung injury or patient ventilator dyssynchrony (PVD).

On the ventilator monitor the patient's breaths are displayed graphically as a waveform know as scalars or loops.

Scalars

The y axis on the graph (scalar) can be independent on which ventilator mode and setting have been selected, this can be volume, pressure and flow. The x axis is always time, which is measured in seconds, a scalar is where a single parameter is plotted over time. There are six standard scalar wave form shapes square, ascending ramp, descending ramp, sine, exponential rise and exponential decay. These typical waveform shapes can change depending on what type of ventilator mode had been set and multiple scalars can be displayed depending on the type of ventilator set up you have.

Volume scalars show us the amount of gas delivered into the lungs over time, this is usually shown as a numerical value but seeing the waveform can show us if the patient is producing spontaneous breaths and measure the volume of those breaths. The inspiration is a steep slope upwards, and expiration is sloping the curve downwards.

Pressure scalars show us how compliance the patient airways are. During pressure control ventilation the waveform is square shaped as this a constant pressure being delivered. During volume control ventilation the waveform is exponential rise.

Flow scalars show us the flow of air between the patient and the ventilator, their waveforms can be repeatable or vary in shape subject to which ventilator mode has been selected. Inspiratory waveform is positive and the expiratory is negative. The shape characteristics of the inspiratory limb is dependent on the ventilator mode.

Loops

A “loop” is where two parameters are plotted against each other at the same time, there are two types of loops pressure volume (PV) loops and flow volume (FV) loops e.g. y axis volume and x axis pressure. Each loop has an inspiratory curve and expiratory curve, which allows for assessment of lung function.

PV loops are formed anticlockwise from the lower left corner where the graph originates with the start of inspiration then a steep rise carrying anticlockwise until expiration where the curve come back to the point of origin. A “normal” PV loop waveform should be rugby ball shaped, changes in this shape may correlate to changes in lung compliance. Patients triggering a breath may give a figure of eight appearance to the loop. The shape of the PV loop may widen due to increases in resistance in the airway or circuit, this could mean there is an obstruction in the endotracheal tube. Excessive tidal volumes may cause a “beaking” like effect on the loop, ventilator settings should be checked/adjusted to prevent volutrauma. A broken loop suggests a leak in the circuit.

FV loops are formed clockwise, with the inspiratory limb above the x axis and the expiratory limb below the x axis. FV loops can identify if there are any airway obstructions or leaks in the circuit, FV loops with a saw tooth appearance may be due to excessive secretions, scooping of the expiratory limb may be due to an airway obstruction and an incomplete FV loop may be due to a leak in the circuit.

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BREAKING UP WITH BREAKING DOWN - IMHA

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Learning objectives:

- Describe the process of red blood cell destruction in immune-mediated hemolytic anemia (IMHA)

- Categorize IMHA as associate or non-associative

- Identify key diagnostic tests utilized to confirm erythrocyte destruction and immune-mediated disease

- Explain the role of thromboprophylaxis in treatment

- Recognize the importance of critical patient monitoring and care in IMHA

Proceeding:

Immune-mediated hemolytic anemia (IMHA) is a life-threatening disease in small animals where the immune system inappropriately destroys red blood cells (RBCs). Aged RBCs are normally removed from circulation based on antigens found on the surface through extravascular hemolysis. The breakdown of RBCs results in a globulin and a heme molecule, which is further broken down into iron and bilirubin. In cases of IMHA, RBCs are inappropriately marked for removal by autoantibodies resulting in too many being removed from circulation. The presence of these autoantibodies also causes the RBCs to begin to adhere to each other or agglutinate, which increases the risk for thromboembolism.

Clinical signs are often consistent with anemia, including lethargy, tachycardia, and tachypnea. Extravascular hemolysis can lead to hyperbilirubinemia and hyperbilirubinuria causing icteric mucous membranes and skin seen on physical exam. Intravascular hemolysis can also occur resulting in an increase in free hemoglobin which can cause hemoglobinemia and hemoglobinuria. Alterations to mentation can vary depending on the severity of the disease.

IMHA can be classified as associative or non-associative. Associative IMHA indicates that a comorbidity was identified, whereas non-associative IMHA indicates the disease is idiopathic or has an unidentified comorbidity. Diagnosing non-associative IMHA requires signs of both immune-mediated disease and hemolysis, while ruling out other causes.

Various in-house diagnostic tests can be used to support the diagnosis of IMHA, though external testing may need to be utilized to confirm diagnosis or comorbidities. Complete blood cell count (CBC) can be

used to evaluate the degree of anemia and hemoglobin levels. Presence of spherocytes on blood smear prior to transfusion and a positive slide agglutination testing can indicate the disease is immune mediated. It is important to note that lack of spherocytes on blood smear or a negative slide agglutination test does not rule out IMHA.

In cases with a negative slide agglutination, Coomb's testing at an external lab can be used to test for the presence of autoantibodies. Chemistry panels can be used to evaluate bilirubin levels, which increase with extravascular hemolysis. Hemolytic anemia could result for a variety of reasons that are not immune-mediated, including infectious diseases, medications, toxicants, and vaccines. Additional diagnostic tests, such as tick-borne disease testing and radiographs, should be considered to rule out non-immune mediated causes.

Treatment of non-associative IMHA focuses on initial stabilization, immunosuppressive therapy, and thromboprophylaxis. Blood transfusions using fresh packed red blood cells may be necessary to stabilize patients that have developed clinical signs for anemia. Administration of blood products can help temporarily improve the patient's signs, but without immunosuppression the RBCs will continue to be destroyed. Glucocorticoids are used as a first line immunosuppressive, though additional agents may be added based on the patient's response to treatment. Thromboprophylaxis should be started early due to the increased risk of thromboembolism from agglutination.

While it does not replace the need for long term medications, therapeutic plasma exchange is being explored for its usefulness in the initial treatment of IMHA. Antibodies present in the blood are removed with the patient's plasma, which can help reduce the need for multiple transfusions and decrease the risk of thromboembolic events. Nursing care for IMHA patients requires close monitoring and a thorough understanding of the critical signs to observe in the event of worsening anemia or thromboembolism. While managing IMHA patients can be challenging, knowledgeable and skillful veterinary nurses can help ensure prompt interventions to help increase the patient's chances of survival.

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TRANSFUSION TROUBLESHOOTING: IS THIS A REACTION?

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Learning objectives:

- Identify transfusion reactions. Recognise key clinical signs of both acute and delayed transfusion reactions and differentiate between immunological and non-immunological causes.

- Identify and apply preventative strategies to enhance transfusion safety and contribute to effective transfusion protocols.

- To monitor key parameters during transfusions and understand their role in identifying reactions and minimising risks to patient safety.

- Develop response strategies for suspected transfusion reactions.

Proceeding:

Introduction

Blood transfusions are life-saving interventions in veterinary medicine, but their success relies on careful patient selection, appropriate product use, and vigilant monitoring. Despite best efforts, transfusion reactions can occur, requiring rapid identification and appropriate management to minimise risks to the patient.

Is this a reaction?

Identifying transfusion reactions is crucial for ensuring patient safety and an effective transfusion therapeutic response. These reactions are classified by their clinical signs, time frame, and etiology; they are either immunologic or non-immunologic in nature. Reactions to blood products can be acute or delayed, developing hours to days after the transfusion, with severity ranging from mild symptoms, such as fever or rash, to more severe life-threatening reactions, like anaphylaxis or haemolytic transfusion reactions. Clinical signs vary but may include fever, tachycardia, hypotension, dyspnoea, vomiting, and haemoglobinuria. It is essential to differentiate true transfusion reactions from reperfusion (a temporary rise in temperature due to improved perfusion), underlying patient conditions, fluid overload, and other complications.

Preventative measures

Preventing transfusion reactions begins with selecting the appropriate blood components and therapy, ensuring the patient is exposed only to the necessary portion of blood to minimise risk. Whole blood, packed red blood cells, plasma, and platelet-rich plasma should be chosen based on the patient's specific needs. Pre-transfusion testing, including blood typing and crossmatching of the recipient and donor, are crucial steps in reducing the risk of haemolytic reactions. Proper handling and administration techniques, such as using the correct filters, ensuring accurate infusion rates, employing compatible pumps and administration drivers, and minimising storage time, further reduce the risk of non-immunologic red blood cell damage.

Patient monitoring

Effective transfusion monitoring is essential for the early detection of complications. Veterinary nurses should conduct baseline assessments and regularly monitor vital signs, including temperature, heart rate, respiratory rate, and blood pressure, at specified intervals during the transfusion. These measurements should be recorded on a recognised transfusion monitoring chart. It is also vital to ensure that the intravenous catheter remains patent, and that the blood product is administered correctly and efficiently for safe delivery.

Response strategies

When a transfusion reaction is suspected, immediate intervention is essential. The transfusion should be halted immediately, and the clinician should be alerted to assess the situation. Appropriate care, including stabilisation with supportive measures such as oxygen therapy, intravenous fluids, or antihistamines, should be administered as prescribed. Severe reactions, such as anaphylaxis or haemolytic crises, require urgent emergency intervention and advanced supportive care. Post-reaction investigations should include repeat haematology, blood typing, and, if contamination is suspected, bacterial culture of the transfused product. Documenting the reaction and updating transfusion protocols based on the findings helps to enhance patient safety in future transfusions.

Conclusion

Transfusion reactions can pose significant risks to patient safety, and it is crucial for veterinary teams to recognise, monitor, and respond to them effectively. By selecting the appropriate blood products, implementing preventative strategies, and carefully monitoring the patient throughout the transfusion process, we can reduce the likelihood of complications. In the event of a suspected reaction, immediate intervention and thorough investigation are vital for ensuring the patient's well-being. Ongoing education, documentation, and protocol updates are essential for continuously improving transfusion safety and care. Through these measures, veterinary professionals can ensure safer and more successful transfusion practices for their patients.

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LIFE AFTER XENOTRANSFUSION

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Learning objectives:

- Provide a comprehensive overview of xenotransfusion, its historical evolution and relevance in emergency and ethical dilemmas.

- Delve into the medical applications and benefits of xenotransfusion as a life saving measure

- Examine patient welfare and ethical considerations and the practical challenges of its short-term efficacy and delayed reactions inherent in xenotransfusion practice.

- Explore current research

- Outline a systematic approach to post-xenotransfusion patient management.

Proceeding:

Introduction

Xenotransfusion provides a vital option, particularly in feline patients, in modern veterinary transfusion medicine. This lecture aims to provide a comprehensive overview of xenotransfusions history, the benefits in our feline patients, including responses and outcomes illustrated through case studies, discussing the welfare and ethical considerations as well as the challenges surrounding xenotransfusions. Additionally, it outlines strategies for effective post-xenotransfusion care providing veterinary professionals with essential tools for navigating this evolving practice.

History of xenotransfusion

A xenotransfusion is a transfusion from one species to another. First recorded in 1667 when a boy received lambs blood, xenotransfusions have since developed and the term xenotransfusions is now commonly used to describe canine blood products given to a feline recipient.

Why do we need xenotransfusions?

Sourcing a suitable feline blood donor can be challenging. Feline blood products are a limited resource, particularly in the UK. Rarer blood types such as B or AB add to this challenge. As cats have naturally occurring alloantibodies, transfusion of type A blood to a type B cat could be fatal. Crossmatching

between recipient and donor blood prior to transfusion is strongly recommended. If no suitable blood match is found, then xenotransfusion should be considered.

The maximum volume of a whole blood feline blood donation is approximately 60ml, if the recipient is requiring a larger volume due to haemorrhage, a xenotransfusion may be more appropriate.

Contraindications and risks

Xenotransfusions may be contraindicated and are not without risk. It is extremely important to note that a cat can only receive a xenotransfusions once in its lifetime. Once the cat has developed antibodies against canine blood, usually after 24 hours, a second exposure to canine blood will lead to a fatal transfusion reaction. It is therefore essential to determine if the patient has previously received a xenotransfusion.

Welfare and ethical concerns

In many cases, this is a lifesaving intervention that will then allow for further investigations, as well as locating a suitable feline blood donor if required.

Welfare and ethical considerations regarding the donor cats is essential. Ensuring their health is not compromised and the blood donation is carried out a stress free as possible. If a feline blood donation is unable to occur, a xenotransfusion should then be considered.

How to safely administer a xenotransfusion

A case series found that the median volume of canine packed red blood cells transfused to feline patients was 14.6 ml/kg (Le Gal at al 2020).

The rate of administration is dependent on the patient's condition. More critical cases may require a bolus of blood however it is recommended to begin a transfusion slowly, such as 1ml/kg/hr for the first 15 minutes. Blood products can only remain out of the fridge no more than four hours. If the patient requires a slow rate for the volume required, the blood may be drawn up into syringes.

Close monitoring of recipients during and after the transfusion for any signs of a reaction is extremely important.

Post xenotransfusion management

Le Gal et al 2023 reported that haemolysis of the canine red blood cells can occur between 1-6 days post xenotransfusion. It is essential to monitor for signs of haemolysis both pre and post transfusion, such as icteric mucous membrane and serum, increased bilirubin, or change in the recipients demeanour.

If the xenotransfusion recipient is non regenerative, or likely to require further blood products, sourcing a suitable feline donor is paramount.

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Resident Stream, Saturday 7 June 2025

CORE PRINCIPLES OF STATISTICS

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Learning objectives:

- Understand the various ways data can be presented based on its distribution and how this impacts statistical testing.

- Understand what a P-value represents and the how this impacts the clinical use of this.

- Apply sensitivity, specificity and predictive values to a patient population.

Proceeding:

Summarizing and presentation of data

How data is summarized and presented is based on the type of observation that is made to create the data set. Commonly data is viewed as categorical (nominal), ordinal or numerical. Nominal data can be both qualitative and quantitative in origin. The classic example of qualitative data in veterinary medical studies is sex distribution (e.g., neutered male, intact female, etc.). Data that is originally quantitative can be categorized to become nominal data for presentation (e.g., % of patients that are hyperlactatemic vs. not). Ordinal data are characterizations of data that have an inherent order. In its simplest form this could be abnormal laboratory values that are presented as number of mild, moderate, or severe abnormalities. Numerical data are observations for which the differences between numbers have meaning on a numerical scale are sometimes called quantitative observations because they measure the quantity of something. There are two types of numerical scales: continuous (interval or ratio) and discrete scales. A continuous scale has values on a continuum (e.g., age); a discrete scale has values equal to integers (e.g., number of fractures).

Categorical or ordinal data are traditionally presented in text as the number(n) in each group +/- the percentage of each grouping or they can also be presented graphically. Numerical data is classically presented as value of central tendency and a measure of the spread of this data. To decide what values to use, the data should be assessed to see if it is normally (Gaussian) distributed or not. This can be accomplished by a variety of methods. The simplest is to plot the variables as a histogram and visually inspect the data to ensure the distribution is similar to a “bell curve” shown below, or if the data appears to be skewed either with a positive or negative bias. An alternative visual inspection process is called a QQ plot. This stands for a quantile vs quantile plot where the data is plotted in theoretical vs actual quantiles of the data being analyzed. The final method for determining normality is through statistical testing (e.g., Shapiro-Wilk test for normality, D’Agostino-Pearson normality test, etc.) that is performed

with software. The result based on the P value generated for that test determines if that data fits a Gaussian distribution.

The central tendency of normally distributed data is typically reported as the mean value, which is the arithmetic average of the observations. Not normally distributed data is often reported with the median value, which is the middle observation (e.g., half the observations are larger, half are lower). The spread of data for a normally distributed dataset is usually presented as the standard deviation. When data is not normally distributed, the spread is often reported as either the 25-75th percentiles (interquartile range), the range, or both. The interquartile range is used when trying to display the central 50% of data regardless of shape, while the range is used when the purpose is to emphasize extreme values.

Hypothesis testing

For simple testing of one, two or more groups of data, a statistical hypothesis statement can be made as the probability that there is a difference between either the single group and expected values, or between the two or more groups that have been sampled. The null hypothesis is a statement claiming that there is no difference between either an assumed value or between groups. A very important principle in statistical is that the tests are determining the probability that the null hypothesis should be rejected (P value). As a scientific community, it has been decided that if the P value is less than 0.05, then those results are considered “significant”. This translates to accepting that there is a less than 5% chance that rejecting the null hypothesis was the correct decision. The means that the lower the P value, the less chance that rejecting a correct null hypothesis has occurred (e.g., P value of <0.001 means there is a less than 0.1% chance). Rejecting the null hypothesis when it is correct is called a Type 1 error.

Power is another important concept in hypothesis testing. Power is the probability of rejecting the null hypothesis when it is false also known as the ability to detect a true difference. ß is known as a Type II error and is related to the sample size of the values. Power is calculated as (1-ß) and commonly studies are powered to either 80% or 90% to determine then number of values needed in the study. This translates to either an 80% or 90% probability that the testing will detect a difference in the sample sets if there truly is one. This is an essential step when designing a study; however, in clinical veterinary medicine there is often a lack of data to generate this estimate, or lack of samples to achieve it. A summary of type I and II errors are represented in the table below.

Sensitivity and Specificity

Sensitivity and specificity are statistical measures used to evaluate the performance of a diagnostic test or a classification model. They provide valuable information about how well the test or model can correctly identify positive and negative instances within a population.

Sensitivity, also known as the true positive rate or recall, measures the proportion of true positive cases correctly identified by the test or model. It quantifies the ability of the test to detect individuals who have the condition or attribute being tested for. A high sensitivity indicates that the test has a low false negative rate, meaning it can effectively identify most of the true positive cases.

Specificity, on the other hand, measures the true negative rate of a test or model. It indicates the proportion of true negative cases correctly identified as negative. Specificity evaluates the ability of the

test to accurately exclude individuals who do not possess the condition or attribute being tested for. A high specificity suggests a low false positive rate, meaning the test can effectively rule out most of the true negative cases.

To calculate sensitivity and specificity, one needs to compare the test results against a reference standard or a gold standard. The reference standard is a reliable method or established criteria that determine the true status of individuals (positive or negative) for the condition in question.

Both sensitivity and specificity are expressed as percentages or proportions ranging from 0 to 1 (or 0% to 100%). In general, a higher sensitivity is desirable when the consequences of false negatives are severe, as it ensures that most true positive cases are correctly identified. Conversely, a higher specificity is desirable when the consequences of false positives are significant, as it minimizes the misclassification of true negative cases.

Predictive Values

Positive predictive value (PPV) and negative predictive value (NPV) are statistical measures used to assess the accuracy and reliability of a diagnostic test or screening tool. They help evaluate the likelihood of a positive or negative result accurately predicting the presence or absence of a particular condition or outcome.

The positive predictive value (PPV) is a measure of the probability that individuals with a positive test result truly have the condition of interest. In other words, it determines the proportion of true positives among all the individuals who tested positive. A high PPV indicates that a positive test result is highly indicative of the presence of the condition.

Mathematically, PPV is calculated as the number of true positive results divided by the sum of true positive and false positive results, multiplied by 100 to express it as a percentage.

A high PPV suggests that a positive test result is highly reliable and can be used to make accurate predictions regarding the presence of the condition.

The negative predictive value (NPV) is a measure of the probability that individuals with a negative test result truly do not have the condition of interest. It determines the proportion of true negatives among all the individuals who tested negative. A high NPV indicates that a negative test result is highly indicative of the absence of the condition.

Mathematically, NPV is calculated as the number of true negative results divided by the sum of true negative and false negative results, multiplied by 100 to express it as a percentage.

A high NPV suggests that a negative test result is highly reliable and can be used to make accurate predictions regarding the absence of the condition.

However, it's important to note that PPV and NPV are influenced by the prevalence of the condition in the population being tested. As the prevalence of the condition increases, the PPV tends to increase, while the NPV tends to decrease, and vice versa.

MITOCHONDRIAL INJURY IN CRITICAL ILLNESS: MECHANISMS AND MITIGATION STRATEGIES

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Learning objectives:

- Recognize the central role of mitochondria in the pathophysiology of ischemia-reperfusion injury.

- Describe key mechanisms of mitochondrial damage during critical illness.

- Identify current and emerging therapeutic strategies aimed at preserving or restoring mitochondrial function.

- Translate basic science findings into practical implications for managing critically ill veterinary patients.

Proceeding:

This lecture introduces the emerging field of mitochondrial pathobiology within critical illness, focusing on ischemia-reperfusion injury as seen in hemorrhagic and septic shock. The content is geared toward residents and seasoned clinicians interested in understanding how cellular bioenergetics impact clinical outcomes—and how this understanding can guide future therapies.

The session begins with a primer on mitochondrial structure and function, specifically their role in energy production, apoptosis regulation, and cellular signaling. The unique vulnerabilities of mitochondria during hypoxia and reperfusion—such as calcium overload, ROS generation, and mitochondrial permeability transition pore opening—will be discussed in relation to organ dysfunction.

A key concept explored is the "mitochondrial tipping point": the threshold beyond which mitochondrial damage triggers irreversible cellular injury. We will examine how mitochondrial dysfunction contributes to organ failure in various shock states, using animal models and human critical care literature examples.

The second half of the lecture focuses on therapeutic strategies aimed at preserving or restoring mitochondrial integrity. This includes established techniques—such as hypothermia, oxygen titration, and antioxidant therapy—and cutting-edge research into mitochondria-targeted agents. Particular attention will be given to peptides like GJA1-20k, which enhance mitochondrial trafficking and function, and agents like Elamipretide, which stabilize mitochondrial membranes.

Finally, we will discuss translating mitochondrial health into a clinically meaningful parameter. Could mitochondrial biomarkers or point-of-care technologies eventually guide resuscitation? What are the challenges in targeting mitochondria therapeutically in a real-world veterinary setting?

Attendees will leave with a foundational understanding of mitochondrial injury mechanisms and an appreciation for their relevance in veterinary critical care. The lecture aims to spark curiosity and support clinicians interested in bridging basic science with clinical innovation.

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PULMONARY DEAD SPACE

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Learning objectives:

- To understand what dead space ventilation is and how it can change with disease states

- To recognize how dead space ventilation will impact pulmonary gas exchange

- To understand how changes in pulmonary dead space will alter $PaCO_2$ and $ETCO_2$

- To review how mechanical ventilation strategies can alter the quantity of dead space and hence gas exchange in patients

Proceeding:

f – respiratory frequency per minute

V_E – Total minute ventilation

V_T – Tidal volume

V_D – Dead space volume

V_A – Alveolar minute ventilation

Pulmonary gas exchange

The relative degrees of ventilation and perfusion of each alveoli will determine the surface area and partial pressure gradient driving diffusion of oxygen and carbon dioxide.

Ventilation-perfusion (V/Q) matching can be classified by their ratio. Alvoelar dead space (zero ventilation, with some blood flow) has a ratio of 0. Optimal V/Q has a ratio of 1 and intrapulmonary shunt (ventilated alveoli but no blood flow) has a ratio of infinity.

The total volume of gas that enters and leaves the mouth and/or nose per minute is known as the total minute ventilation. This volume is not the same as the volume of gas that moves in and out of the alveoli per minute. The difference in these volumes is known as dead space. Dead space (V_D/V_T) is defined as the portion of the tidal volume that does not participate in gas exchange.

Total minute ventilation (V_E) = Tidal volume (V_T) x Respiratory rate (f)

Alveolar minute ventilation (V_A) = [Tidal volume – Dead space] x Respiratory rate

$= [V_T - V_D] \times f$

Anatomic dead space

In the normal animal there are two types of dead space, anatomic and alveolar. Anatomic dead space includes all the conducting airways including the nose and/or mouth, trachea, bronchi down to the terminal bronchiole. Beyond the terminal bronchiole gas exchange occurs. Anatomic dead space is considered functionally important as it provides warmth and humidity to inspired gas. It also reduces the ability of inhaled particulate matter to reach the alveoli.

Alveolar dead space

Ventilation of alveoli that are not perfused will constitute alveolar dead space. In healthy patients there is minimal to no alveolar dead space present. As there is greater blood flow to the dependent regions of the lung and there is less to the non-dependent regions, alveolar dead space commonly occurs in the non-dependent regions (West Zone 1). Alveolar dead space can be increased by low cardiac output as this will lead to less blood flow to the lung and a greater proportion of the non-dependent lung may not be perfused. Pulmonary thromboembolism (PTE) is another cause of increased alveolar dead space.

Apparatus dead space

When animals have an artificial airway we introduce apparatus (also known as mechanical) dead space. This is the volume of the breathing circuit from the nose/incisors of the animal to the source of fresh gas flow in the breathing circuit.

Physiological dead space

Physiological dead space is the sum of the alveolar and anatomic dead space (and any apparatus dead space between the measurement point of $ETCO_2$ and the patient). The Bohr equation allows determination of physiological dead space. The concentration of CO_2 in exhaled gas will be diluted by gas from dead space regions that contain zero CO_2. Alveolar PCO_2 can be assumed to equal $PaCO_2$ as carbon dioxide readily equilibrates. So the difference between alveolar CO_2 ($PaCO_2$) and mixed expired CO_2 represents the quantity of dead space present.

Modified Bohr equation for physiological dead space:

As there is minimal difference between $PECO_2$ and end tidal CO_2 (ETCO2) in the healthy animal the Bohr equation can be modified to:

$Vd/Vt = (PaCO_2 - ETCO_2)/PaCO_2$

It is important to note that this modified formula may not be accurate in all clinical situations.

Clinical significance of increased dead space

With increased dead space there has to be a decrease in effective alveolar ventilation (assuming no change in minute ventilation). This would cause in an increase $PaCO_2$ as a consequence. The following equation shows the inverse relationship $PaCO_2$ has with alveolar ventilation.

$PaCO_2 = VCO_2/V_A$

Where VCO_2 is carbon dioxide production. It is usually possible to counteract the effect of increased dead space with an increase in alveolar minute ventilation. In the awake, otherwise healthy animal, the respiratory center should respond to an increase in $PaCO_2$ with increased minute ventilation to target a normal $PaCO_2$. In the anesthetized animal this response maybe blunted or if the animal is on mechanical ventilation with a fixed minute ventilation, hypercapnia will develop. Increased dead space is not a mechanism of hypoxemia. The blood passing from the right side of the heart to the left side of the heart does not perfuse areas of dead space and as such, if blood is inadequately oxygenated – it cannot be due to dead space. Pulmonary parenchymal disease such as pneumonia, pulmonary edema or pulmonary hemorrhage are not expected to change alveolar dead space. These diseases create regions that have less ventilation than there is perfusion (low V/Q regions) or areas with no ventilation but maintained perfusion (No V/Q or physiologic shunt). As such these abnormalities are potent causes of hypoxemia.

Mechanical ventilation

Dead space is of major clinical relevance in the patient on mechanical ventilation. There is addition of apparatus dead space by virtue of connection to the breathing circuit but this is offset by the decrease in anatomical dead space from endotracheal or tracheostomy tubes. Overall apparatus dead space is not likely to be a significant issue in all but the very small patient. The application of positive end expiratory pressure (PEEP) can have variable effects on alveolar dead space. If PEEP is successful at recruitment and reducing physiologic shunt there may be a decrease in the dead space (increased alveolar ventilation so percent of tidal volume that is dead space will reduce). PEEP can also increase alveolar dead space. As described by West's zone 1, when alveolar pressure exceeds perfusion pressure of alveoli, there will be compression of alveolar blood vessels and alveolar dead space is created. This is most likely to occur in the non-dependent areas of the lung where perfusion pressure is the lowest. If PEEP reduces cardiac output, it is likely to further increase alveolar dead space. Measurement of V_D/V_T has been used as a strategy to determine optimal PEEP. Low tidal volume ventilation is major part of a lung protective ventilation strategy and will also impact dead space. Physiologic dead space is a relatively fixed volume. As tidal volume decreases, the percent of dead space has to increase and hence alveolar ventilation is reduced. For this reason, higher respiratory rates are commonly utilized with low tidal volume ventilation in an effort to maintain adequate alveolar ventilation. Despite this, permissive hypercapnia is commonly part of the lung protective strategy as it can be challenging to provide sufficient alveolar ventilation to maintain normocapnia with very low tidal volumes.

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BARORECEPTOR PHYSIOLOGY

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Learning objectives:

- Explain the baroreceptor reflex

- Apply an understanding of the baroreceptor reflex to explain the pathophysiology of shock states

Proceeding:

Perfusion of systemic capillary beds relies upon a pressure gradient between the arterial and venous blood, as stated in Ohm's law:

Flow = Pressure Gradient / Resistance, or Q = ΔP/R.

For the majority of tissues that are perfused throughout the cardiac cycle, the largest determinant of this pressure gradient is mean arterial blood pressure (MAP). This in turn depends on cardiac output and systemic vascular resistance. Given the importance of MAP, it is unsurprising that the body contains baroreceptors that measure MAP and initiate reflex activity in response to abnormalities.

The Baroreceptor Reflex

The baroreceptor reflex is a classic example of a homeostatic response that relies upon negative feedback loops to maintain a physiologic parameter, in this case MAP, within a tightly regulated range. This reflex pathway consists of sensors, afferent neural pathways, a coordinating centre, efferent neural pathways, and effectors.

The Baroreceptor Sensor and Afferent Pathways

Baroreceptors detect vessel wall stretch rather than pressure itself. High-pressure baroreceptors are located in highly compliant major arteries with mostly elastic fibres in their walls: the carotid sinuses and the aortic arch. In these locations, sensory nerve fibres are embedded within the arterial wall. The receptor terminals of these neurons contain stretch-sensitive ion channels linked to the actin cytoskeleton. Cell deformation alters the protein conformation and opens the ion channel, depolarizing the receptor terminal. The sensitivity of baroreceptors to stretch can be altered by many paracrine and autocrine mediators.

Depolarization of the receptor terminal results in increased frequency of action potentials along the neuronal axon. As pressure increases, additional neurons are recruited to augment the response. Signals from the carotid body are carried by the sinus nerve, a branch of the glossopharyngeal nerve (CN IX),

with cell bodies in the petrosal (or inferior) ganglion. The aortic arch signals are carried by the depressor branch of the vagus nerve (CN X), with cell bodies in the nodose (or inferior) ganglion.

The Coordinating Centre, Efferent Pathways and Effectors

Signals from peripheral baroreceptors are projected to the cardiovascular centre of the medulla, specifically the nucleus tractus solitarii (NTS). Interneurons project to both the vasomotor and cardioinhibitory areas within the medullary cardiovascular centre. The vasomotor area maintains tonic vasoconstriction. Interneurons from the NTS inhibit vasomotor activity, resulting in vasodilation in response to increased baroreceptor activity. Conversely, excitatory interneurons from the NTS increase the activity of the cardioinhibitory area, resulting in bradycardia in response to increased baroreceptor activity. A minor contribution to the bradycardic response to baroreceptor activity also results from inhibitory interneurons that project to the cardioacceleratory area in the dorsal medulla.

The efferent pathways of the baroreceptor reflex involve both the sympathetic and parasympathetic arms of the autonomic nervous system. An increase in baroreceptor firing results in inhibition of sympathetic and augmentation of parasympathetic transmission.

Sympathetic innervation is carried through the thoracolumbar sympathetic trunk, with post-synaptic neurons utilizing the neurotransmitter norepinephrine. Increased sympathetic activity leads to vasoconstriction of most vessels, mediated by α adrenergic receptors. Arterial vasoconstriction increases systemic vascular resistance. Some key vascular beds such as the cerebral, coronary, and skeletal muscle arteries vasodilate due to β_2 receptor stimulation, which ensures blood flow to those organs is maintained. Sympathetic innervation to the heart, mediated by β_1 receptors, causes increased heart rate, contractility, conduction, and relaxation. Sympathetic nerves also stimulate the adrenal medulla, which can release both epinephrine and norepinephrine into the circulation to function in an endocrine manner.

Parasympathetic innervation is carried in the vagus nerve, utilizing the neurotransmitter acetylcholine. Increased parasympathetic activity in the heart causes a decrease in heart rate. The parasympathetic contribution to vascular tone is minimal.

Overall Effects

The overall effects of the baroreceptor reflex are to attempt to return MAP to normal and maintain homeostasis. Hypotension causes decreased baroreceptor firing. Parasympathetic activity decreases and sympathetic activity increases. Vasoconstriction increases systemic vascular resistance, while increased heart rate and stroke volume increase cardiac output. The MAP returns towards normal and the baroreceptor firing decreases, constituting negative feedback. This is a vital component of compensation for circulatory shock and explains the common clinical signs of peripheral vasoconstriction and tachycardia. Conversely, hypertension causes increased baroreceptor firing. There is inhibition of the sympathetic nervous system, causing vasodilation. Increased parasympathetic activity causes bradycardia, frequently referred to as reflex bradycardia. The baroreceptor reflex is an essential cardiovascular compensatory mechanism. Impairment of baroreceptor reflex function can contribute to derangements of cardiovascular homeostasis in conditions such as hypertension and heart failure.

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ADVANCED RENAL PHYSIOLOGY AND RELEVANCE TO FLUID THERAPY DECISION MAKING; ACID BASE ANALYSIS AND DIURETICS USAGE

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Learning objectives:

Review ion transport throughout the renal nephron: the proximal convoluted tubule, loop of Henle, distal convoluted tubule and collecting duct, including principal cells and type A, B and non-α, non-β intercalated cells.

Describe the effects of anti-diuretic hormone (ADH) and aldosterone on the reabsorption of water. Review how the kidney contributes to acid base balance. Review the use, administration and complications of diuretics

Proceeding:

The renal nephron is comprised of the proximal convoluted tubule (PCT), the loop of Henle (LOH), distal convoluted tubule (DCT) and collecting duct (CD).

Sodium ion transport is critical for body water homeostasis.

- The PCT is the primary site of sodium reabsorption. Passive luminal absorption is established by basolateral Na^+K^+ATPase pumps. Sodium is usually co-transported with other solutes. Approximately 65-80% of water reabsorption occurs isosmotically with sodium via aquaporin (AQ)1.

- In the impermeable thick ascending limb (TAL) of the LOH, sodium absorption via the Na-K-2Cl cotransporter (NKCC) establishes a medullary concentration gradient. Water is reabsorbed across the descending limb by apical and basolateral AQ1.

- Apical sodium chloride cotransporters (NCC) reabsorb sodium in the early DCT and epithelial sodium transporter (ENaC) reabsorbs most sodium in the late DCT and CD

- In the CD, antidiuretic hormone (ADH) binds to basolateral V2 receptors in principal cells leading to increased AQ2 synthesis and insertion into the apical membrane. Water is then absorbed down a concentration gradient into the hyperosmotic interstitium. Urea is concentrated in the CD until it is reabsorbed by urea transporters A1 and A3 into the medullary interstitium.

During hypovolaemia, the renin angiotensin aldosterone system (RAAS) is activated by:

Decreased sodium and chloride absorption by the macula densa cells in the early DCT which triggers renin release from juxtaglomerular cells (JGC).

B-1adrengeric and baroreceptor stimulation of JGC

Angiotensin II promotes sodium reabsorption (primarily via the PCT apical NaH+ antiporter) and stimulates secretion of aldosterone. Aldosterone stimulates ENaC.

Potassium homeostasis is vital for numerous cell functions. Approximately 75% of potassium is reabsorbed in the PCT (passive and paracellular) and 25% via the NKCC in the LOH. Passive and active potassium secretion occurs in the LOH, DCT and CD. Renal excretion in the CD involves uptake at basolateral Na^+K^+ATPase then passive exit via renal outer medullary K (ROMK) and big potassium (BK) channels. Hypokalaemia occurs secondary to aldosterone's action on ENaC, increased distal sodium delivery (leading to increased potassium leaching via ROMK and BK to maintain electroneutrality) and acid base status.

Both active (driven by Na^+/H^+) and passive (1. late PCT due to early PCT impermeability and 2. Solvent drag)) chloride absorption occurs in the PCT. It is also actively absorbed in the LOH via the NKCC transporter and via the NCC in the DCT. Chloride can be secreted or reabsorbed in the CD: paracellular absorption is drive by ENaC, apical absorption is driven by a chloride bicarbonate exchanger (Pendrin) and apical excretion by sodium dependent chloride bicarbonate exchanger.

Renal contribution to acid-base balance in the PCT requires sodium reabsorption in exchange for H^+ (which combines with e.g. filtered hydrogen phosphate). Hydrogen secretion stimulates the formation of bicarbonate. Angiotensin II stimulates the Na^+-H^+ antiporter and the basolateral sodium bicarbonate cotransporter. In the CD, the hydrogen is actively secreted by a H^+ATPase and there is a basolateral chloride/bicarbonate exchanger. These are stimulated by aldosterone. Hypokalaemia perpetuates acid retention by stimulating H^+K^+ATPase and reducing pendrin activity.

Most diuretics e.g. loop, thiazides, carbonic anhydrase and those acting via ENac, decrease renal sodium reabsorption to induce diuresis. Blocking specific channels or maintaining electroneutrality results in complications such as hypocalcaemia (blocking NaCC pump), hypokalaemia and metabolic alkalosis.

Oral Abstracts, Original Study, Thursday 5 June 2025

PREDICTIVE UTILITY OF PLATELET RATIOS IN DIFFERENTIATING IMMUNE AND NON-IMMUNE THROMBOCYTOPENIA IN DOGS: A RETROSPECTIVE ANALYSIS

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Introduction:
Early differentiation between primary immune-mediated thrombocytopenia (ITP) and thrombocytopenia of other causes is crucial for timely and appropriate treatment. Various conditions may affect both white blood cell (WBC) and platelet counts, mimicking hematologic alterations observed in ITP. This study evaluated whether platelet ratios with leukocyte subpopulations can distinguish ITP from non-immune-mediated thrombocytopenia (Non-ITP).

Methods:
Retrospective data from an academic teaching hospital between January 2020 and December 2024 were analyzed to assess platelet ratios in dogs using two-sample t-tests, the Kruskal-Wallis Rank Sum Test, and multinomial regression to evaluate associations with ITP or Non-ITP. AUROC analysis was additionally performed. Inclusion criteria required a manual platelet count below 100,000/µL from a complete blood count (CBC) within 24 hours of hospitalization. Cases with missing data, prior steroid administration, recent trauma, or a diagnosis of infectious or hematologic disease were excluded.

Results:
A total of 196 dogs were included: 94 ITP and 102 Non-ITP. The median (25th, 75th percentiles) of the following ratios was higher in the Non-ITP than the ITP group: platelet-to-neutrophil (PNR) 8.036 (3.224, 16.155) vs. 1.850 (0.490, 5.291) ($p = 0.001$); platelet-to-lymphocyte (PLR) 44.315 (23.416, 77.612) vs. 13.208 (4.177, 40.698) ($p = 0.001$); platelet-to-monocyte (PMR) 90.833 (36.370, 256.360) vs. 16.506 (4.831, 57.692) ($p = 0.001$); platelet-to-eosinophil (PER) 258.807 (137.730, 791.683) vs. 112.500 (21.407, 700.000) ($p = 0.001$); platelet-to-basophil (PBR) 26000.000 (6010.714, 55750.000) vs. 7000.000 (2000.000, 34250.000) ($p = 0.003$); and platelet-to-WBC (PWR) 5.118 (2.335, 9.328) vs. 1.082 (0.374, 3.735) ($p = 0.001$). AUROC analysis determined the optimal cutoff for PNR in distinguishing ITP from Non-ITP. The threshold to maximize sensitivity and specificity was 1.5687, with a PNR ≥1.5687 classifying Non-ITP cases with 93.1% sensitivity and 49.5% specificity.

Conclusions:

This study highlights the potential utility of platelet ratios in differentiating thrombocytopenia etiologies. PNR, PLR, PMR, PER, and PBR may serve as useful diagnostic markers in distinguishing ITP from Non-ITP. In particular, a PNR cutoff of 1.5687 demonstrated high sensitivity in identifying Non-ITP cases. Prospective studies are warranted to validate these findings and refine diagnostic thresholds.

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SELECTIVE REMOVAL OF PLASMA PROTEINS BY DOUBLE FILTRATION PLASMAPHERESIS IN CANINE BLOOD: AN EX-VIVO STUDY

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Introduction:
Double filtration plasmapheresis (DFPP) is a recently developed plasma exchange modality that allows selective clearance of high molecular weight proteins including immunoglobulin, immune complexes and toxins, minimizing the loss of albumin and the need for substitution. Due to its advantages over conventional therapeutic plasma exchange, DFPP has been increasingly used in human medicine to treat different immune-mediated, hematological, neurological and metabolic conditions. Reports concerning DFPP use in dogs are scarce. This study evaluates the quantitative net loss of different plasma proteins fractions in canine blood processed via DFPP.

Methods:
The commercially available extracorporeal circuit for INUSpheresis® with a standard plasma separator and a TKM58® as plasma fractionator were used to perform DFPP using a canine blood reservoir bag (RB). During treatment 1.5, 2 and 3 plasma volumes (PV) were processed over 180 minutes. Plasma proteins fractions were measured in the RB at baseline (pre-treatment) and in the effluent bag at the end of each target PV exchanged to calculate the net loss of selected plasma proteins.

Results:
At 1.5 PV, net γ-globulin and albumin loss was 50 and 25%, respectively. At 3 PV, net γ-globulin and albumin loss was 57 and 40%, respectively. Net fibrinogen loss ranged from 14 to 28% at 1.5-3 PV. Notably, fibrinogen concentration was unmeasurable low in the RB after processing 1.5 PV.

Conclusion:
INUSpheresis® allows selective plasma proteins removal, with sparing effect on albumin, with the maximum selectivity encountered at 1.5 PV and progressively reduced at incremental plasma volume. The albumin sparing reduces the requirement for replacement solutions, minimizing costs and the potential for plasma-transfusion-related complications. Fibrinogen, a large-size intravascular protein, was extensively cleared during DFPP treatment. Since net fibrinogen loss due to filtration was low, additional mechanisms including adsorption by the TKM58® and dilution should be considered.

DFPP seems to be an interesting and promising technique, that deserves further investigations in veterinary patients.

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ASSESSING THE EFFICACY OF INTRAOSSEOUS CATHETER PLACEMENT AT VARIOUS SITES DURING ACTIVE CARDIOPULMONARY RESUSCITATION

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Introduction:
Intraosseous (IO) catheterization provides rapid vascular access during cardiopulmonary resuscitation (CPR). Humeral IO injections reach the heart faster than tibial IO, but placement may be more challenging and prone to dislodgement during CPR. This study compares IO placement time and dislodgement rates between humeral and tibial sites during active CPR and non-CPR (NCPR) conditions. We hypothesize that CPR would prolong IO placement time, tibial IO would be faster than humeral during CPR, and humeral IO would have a higher displacement rate after 10 minutes of CPR.

Methods:
A randomized crossover study using 14 canine cadavers evaluated IO catheter placement in CPR and NCPR conditions. The order of IO placement (tibia or humerus first) and CPR status were randomized. CPR followed RECOVER guidelines with dogs in right lateral recumbency, endotracheal intubation, positive pressure ventilation, and chest compressions. Chest compressions were paused for radiographic confirmation of IO placement before resuming for 10 minutes. NCPR placements were performed in left lateral recumbency. A single DACVECC used a spring-loaded SAM-IO drivers were used for catheter placement. In both groups, following IO placement, a 10 mL saline flush was followed by 2 mL of iohexol IO, with lateral radiographs confirming placement. Dislodgement was assessed with a second radiograph after 10 minutes and 3 ml of iohexol IO. Statistical analysis included D'Agostino Pearson for normality, paired t-test/Wilcoxon test for time comparison and Fisher's exact test for success rate. Outliers were excluded using the ROUT method with Q = 1%.

Results:
Humeral IO placement had a 100% success rate, while tibial IO was 92.8% (p = 0.49). Overall, humeral IO was placed faster (median: 50 sec, IQR: 20) than tibial IO (median: 68 sec, IQR: 20) (p = 0.001). During CPR, humeral IO placement was significantly faster (mean: 38 sec +/- 9) than tibial IO placement (mean: 57.6 sec, +/- 22, p=0.011). No catheters dislodged after 10 minutes.

Conclusion:
Humeral IO placement was faster than tibial IO in both CPR and NCPR conditions, with no difference in dislodgement rates. These findings support the use of humeral IO for rapid vascular access during CPR.

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PREVALENCE AND CHARACTERIZATION OF EXTENDED SPECTRUM BETA-LACTAMASE PRODUCING ENTEROBACTERIACEAE (ESBL-PE) FROM FECAL SAMPLES OF VETERINARY STAFF AND THEIR PET DOGS AT A VETERINARY REFERRAL TEACHING HOSPITAL

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Introduction:
Antimicrobial resistance poses a global health crisis. Extended spectrum β-lactamases producing Enterobacteriaceae (ESBL-PE) exhibit a variety of resistance patterns to antibiotics carrying β-lactam rings, including penicillins, cephalosporins and monobactams, limiting their therapeutic efficacy. This study aimed to identify the prevalence of ESBL-PE bacteria isolated from fecal samples of veterinary staff and their pet dogs.

Methods:
This was a prospective, cross-sectional observational study. Following written consent, staff members were asked to provide paired fecal samples from themselves and their dogs, up to 72 hours apart. Samples were transported to the Microbiology laboratory for agar plating and bacterial analysis using MALDI-TOF. For positive pairs, antibiotic susceptibility was determined by VITEK microbial identification and susceptibility testing. Participants filled a questionnaire regarding their lifestyle.

Results:
A total of 85 humans and 96 dogs participated in the study (35 veterinarians, 25 nurses, 19 students and 6 administrative staff). Of the study population, 31/85 humans (36%) and 25/96 dogs (26%) were ESBL-PE positive. In 13 of the households (15%), at least one dog and its owner were ESBL-PE positive. Nine paired dog-human samples, sharing the same household, grew identical ESBL-PE strains [Escherichia coli (n=8), and Klebsiella pneumoniae (n=1)]. Two human participants had two different types of ESBL-PE. Among ESBL-PE negative owners, 79.6% had a negative dog, while only 20.4% owned one positive dog or more. In contrast, among ESBL-PE positive owners, 58.1% had a negative dog, while 41.9% owned a positive dog (P= 0.034). The odds of being an ESBL-PE positive human, when living in a household with an ESBL-PE positive dog, were 2.82 times higher compared to living in a household with an ESBL-PE negative dog (95% confidence interval 1.1-7.7). No difference was found in the prevalence ESBL-PE positive samples in staff members working in 24/7 vs. daytime departments, nor between any of the other risk factors examined.

Conclusions:
This study found a high prevalence of ESBL-PE co-carriage among veterinary staff and their household dogs, highlighting the potential for cross-species bacterial transmission within shared living

environments. These findings emphasize the importance of One Health initiatives to mitigate risks of transmission and antibiotic resistance.

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RISK FACTORS FOR ACUTE POSTOPERATIVE HEMORRHAGE FOLLOWING MITRAL VALVE REPAIR: A RESTROSPECTIVE STUDY

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Introduction:
In people, numerous risk factors for post-operative bleeding following cardiac surgery have been documented including sex, bodyweight, degree of hemodilution, cardiopulmonary bypass duration, preoperative platelet count and body temperature at ICU admission. The aim of this study was to assess these risk factors for post-operative hemorrhage in dogs undergoing mitral valve repair.

Methods:
Dogs undergoing mitral valve repair at a single hospital between September 2010 and January 2020 which survived the first 24 hours post-operatively were included. Signalment, clinicopathological data and patient outcomes were collected. The volume of pleural hemorrhage (VPH) was calculated for the first 24 hours post-operatively (ml/kg/hr). Degree of hemodilution was calculated as the difference between pre- and post-operative total solids (g/l). Pre-operative total solids and platelets were measured within 28d preceding surgery on routine pre-operative bloodwork and post-operative total solids were measured within 1 hour of admission to the ICU. Univariate linear regression was used to assess risk factors for VPH followed by multivariate analysis for those with $P < 0.2$. Robust methods (bootstrapping) were used.

Results:
Sixty-seven dogs met the inclusion criteria with sufficient data for univariate analysis. Median age was 9.25 years (range 0.67 – 14 years) and median bodyweight was 6.7kg (range 2.5-23.4 kg). Cavalier King Charles spaniels (n = 19) and Chihuahuas (n = 14) were the most common breeds. Forty-three dogs were male (10 entire, 33 neutered) and 24 were females (5 entire, 19 neutered). Five risk factors had $P < 0.2$ in univariate analysis: sex ($P = 0.17$), bodyweight ($P < 0.001$), degree of hemodilution ($P < 0.001$), CPB duration ($P = 0.19$) and ICU admission temperature ($P = 0.04$). Thirty-three dogs had sufficient data for multivariate analysis and no risk factor retained significance. Three dogs required revision surgery, the indication in two was ongoing pleural hemorrhage. Fifty-eight dogs (86.6%) survived to hospital discharge. There was no significant difference in VPH between survivors (median 1.06, range 0.18-4.08 ml/kg/hr) and non-survivors (median 1.82, range 0.31-3.63 ml/kg/hr) ($P = 0.06$).

Conclusion:
No reliable risk factor was documented for VPH in multivariate regression however missing data impacted the analysis.

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FIXED VS. ROTATING RESCUER VENTILATION: ADHERENCE TO RECOVER GUIDELINES IN VETERINARY CPR

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Introduction:
Ventilation is a key component of cardiopulmonary resuscitation (CPR). In veterinary medicine, resuscitation teams are small, often requiring the rescuer ventilator to rotate and perform chest compressions to avoid fatigue. This differs from human medicine, where teams often have designated CPR roles. This study investigated whether using a single rescue ventilator improves adherence to RECOVER ventilation guidelines compared to rotating rescue ventilators.

Methods:
A 3-day randomized cross-over study was conducted using third-year DVM students undergoing RECOVER certification. Students were randomly divided into groups of four to perform RECOVER scenarios (excluding mega codes), using a single ventilator (SV) or rotating ventilators (RV). Overhead camera footage was reviewed to collect data on breaths per minute (BPM). Scenarios were excluded if the ambu-bag was not visible or if the scenario was incomplete.

Comparisons between SV and RV groups were performed. Ventilator adherence rates were analyzed within and between groups and compared to expected rates from the human literature. Normalcy was assessed via D'Agostino Pearson. Statistical significance was assessed using unpaired t-tests, Mann-Whitney U tests, and one-way ANOVAs based on normalcy ($P \leq 0.05$ considered significant).

Results:
Eighteen scenarios were analyzed. There was no significant difference in BPM between groups (SV 8.468, SD 1.734) (RV 8.456, SD 1.943, P=0.9709). Within groups, BPM remained consistent over time. Adherence to guidelines was poor for both SV (16.13%) and RV (11.69%) groups, with no significant differences between groups. Overall adherence rates (14.6%) were significantly lower than reported in the human literature (27-65%, $P<0.05$). Adherence rates decreased over time in SV group, with higher adherence during minutes 1–4 compared to minutes 5–8 ($P<0.01$).

Conclusions:
Rotating rescue ventilators does not appear to have an impact on adherence to BPM guidelines in RECOVER simulations, which was generally poor. Although there was no difference in adherence rates between SV and RV groups, SV adherence decreased over time, suggesting a possible benefit to rotating the rescue ventilator, although larger studies are needed to confirm these findings. Further studies are needed to assess whether auditory aids improve adherence.

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RETROSPECTIVE EVALUATION OF APPROPRIATE EMPIRIC ANTIMICROBIAL CHOICE ON SURVIVAL IN DOGS WITH SEPTIC PERITONITIS

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Introduction:
Appropriate empiric antimicrobial use has been associated with improved survival in people with sepsis of various causes. In contrast, a previous study (Dickinson et al., 2015) did not document this association in dogs with septic peritonitis. The primary aim of this study was to review the association between appropriate empiric antimicrobial treatment and survival in a larger sample of dogs with septic peritonitis. Secondary aims were to evaluate the effect of antimicrobial use in the preceding 90 days on the appropriateness of empiric antimicrobial choice.

Methods:
Dogs with septic peritonitis between January 2015 and December 2023 were eligible for inclusion. Cases required cytologic diagnosis and a positive bacterial culture with culture and sensitivity for inclusion. Cases euthanized at the time of diagnosis were excluded. Empiric antimicrobial therapy was considered appropriate if all cultured organisms were sensitive to the antimicrobial choice prescribed. Signalment, medical history, clinicopathologic data, antimicrobial susceptibility data and outcomes were recorded. Dogs were stratified into illness groups: No organ dysfunction, presence of organ dysfunction, and septic shock. Baseline characteristics between survivors and non-survivors were analyzed to enable adjusted odds ratios to be calculated.

Results:
The final study population comprised 175 dogs. Overall survival to discharge was 64.6% (113/175 dogs). Baseline characteristics age ($P = 0.035$) and illness group ($P < 0.001$) were significantly different between survivors and non-survivors. Use of appropriate empiric antimicrobials was significantly positively associated with survival ($P = 0.002$, unadjusted OR 2.8, 95% CI 1.5 – 5.3). When adjusted for age and illness group, the use of appropriate empiric antimicrobials had an even greater significant positive association with survival ($P < 0.001$, adjusted OR 4.5, 95% CI 1.9 – 11.0). Preceding 90 day antimicrobial history was available for 139 dogs. Of these, most dogs (n = 84, 60%) received antimicrobials. Presence of antimicrobial use in the preceding 90 days was significantly negatively associated with appropriate empiric antimicrobial use ($P < 0.001$).

Conclusions:
Appropriate empirical antimicrobial choice is associated with survival in dogs with septic peritonitis. Prior antimicrobial use (90 days) had a significant negative association on the appropriateness of empiric antimicrobial choice.

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PLASMA DISTRIBUTION OF XENOBIOTICS AND CLINICAL OUTCOMES AFTER INTRAVENOUS LIPID EMULSION THERAPY IN VETERINARY INTOXICATIONS: A PROSPECTIVE MULTICENTER STUDY

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Introduction:
Intravenous lipid emulsion (ILE) therapy is increasingly utilized for managing neurotoxic intoxications in veterinary medicine. However, its application often lacks evidence-based support due to the limited literature on its clinical efficacy in specific intoxications and the general physiochemical behavior of a toxin following ILE administration in a clinical setting. This scarcity of data complicates the formulation of general recommendations for the clinical use of ILE therapy in intoxications. This prospective, multicenter study aimed to investigate the plasma distribution of xenobiotics following ILE therapy in intoxicated dogs and cats and to assess associated clinical outcomes.

Methods:
Client-owned cats and dogs with acute neurological signs were included if intoxication was suspected or confirmed, no contraindications to ILE therapy existed, and the attending clinician predicted ILE effectiveness based on current guidelines. Conducted across eight European veterinary referral centers, standardized ILE therapy was administered. Blood samples were collected before (T0) and directly after (T1) ILE therapy. Plasma was separated into lipid and aqueous fractions via ultracentrifugation. Xenobiotics were identified and quantified using spectrometric analysis. Clinical outcomes were assessed by two blinded clinicians. Statistical analyses included Wilcoxon Signed-Ranked test, Spearman's correlation, and linear regression to evaluate differences between plasma fractions, relationships with the octanol-water partition coefficient (log P), and associations with clinical outcomes.

Results:
Forty-two animals (9 cats, 33 dogs) met final inclusion criteria with quantifiable xenobiotics. A total of 23 xenobiotics were identified, predominantly permethrin and tetrahydrocannabinol (THC). In 36 of 42 animals, xenobiotic concentrations were higher in the lipid fraction at T1. Additionally, 28 animals had xenobiotics with log P ≥ 1. No significant relationships were found between lipid/aqueous fraction differences and log P, nor with clinical outcomes.

Conclusion:
The study suggests that while generally the studied xenobiotics tend to distribute to the created lipid fraction following ILE therapy in dogs and cats, log P does not reliably predict this distribution, and no general predictors for clinical outcomes were identified. Further research is needed to explore more reliable physiochemical predictors, such as log D, and other factors influencing ILE therapy effectiveness in intoxications

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EVALUATION OF THE VETERINARY RAPID ULTRASOUND IN SHOCK (VETRUSH) PROTOCOL TO IDENTIFY THE UNDERLYING CAUSE OF SHOCK IN DOGS WITH UNDIFFERENTIATED CARDIOVASCULAR INSTABILITY

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Introduction:
The RUSH protocol is a systematic point-of-care ultrasound protocol that evaluates the heart, inferior vena cava, lungs, and abdomen in humans presenting with shock and hypotension. Our study adapted the human protocol to compare its diagnostic accuracy against a clinical reference standard established by clinical experts.

Methods:
Twenty-one dogs presenting with cardiovascular instability were prospectively enrolled from July 2022 to December 2024 at a Veterinary Teaching Hospital Emergency Service. Cardiovascular instability was defined as systolic blood pressure (BPsyst) <90 mmHg plus ≥1 sign of global hypoperfusion (altered mentation, pale/pale-pink mucous membranes (MM), weak/absent femoral pulses, body temperature <37.5 °C, shock index >1, or lactate >2.5 mmol/L). Normotensive dogs were included if ≥3 hypoperfusion signs were present. Cineloops were recorded per the vetRUSH protocol for later analysis. Three reviewers evaluated each case. Reviewer 1 used only signalment, presenting complaints, and triage findings such as heart rate, body temperature, respiratory rate, MM/CRT, and BP (TRIAGE). Reviewer 2 assessed the vetRUSH cineloops without clinical information (vetRUSH). Reviewer 3 combined TRIAGE findings with vetRUSH results (COMBO). Each reviewer assigned a shock subtype from eight predefined categories (obstructive - cardiac tamponade, obstructive - pulmonary thromboembolism, obstructive - tension pneumothorax, cardiogenic, distributive, hypovolemic, hypovolemic/distributive, and mixed). The final diagnosis of shock subtype was determined by a panel of three independent, board-certified specialists (two criticalists and one cardiologist). Diagnostic accuracy was calculated by comparing each method to the expert consensus. The diagnostic agreement for each comparison was analyzed using McNemar's Exact test.

Results:
TRIAGE identified the correct shock subtype in 6 of 21 cases (28.6%; 95% CI: 9.24%–47.9%). VetRUSH was concordant in 11 cases (52.4%; 95% CI: 31.0%–73.7%). COMBO demonstrated the highest accuracy, agreeing with the final diagnosis in 17 cases (81.0%; 95% CI: 64.2%–97.7%). The difference between TRIAGE and COMBO was statistically significant (P = 0.0034), while comparisons between TRIAGE and

vetRUSH (P = 0.27) and vetRUSH and COMBO (P = 0.11) were not.

Conclusions:
The combination of vetRUSH with triage findings significantly improves diagnostic accuracy compared to TRIAGE or VetRUSH alone. These results support the utility of an integrated diagnostic approach and using the vetRUSH protocol to identify the cause of cardiovascular instability in dogs.

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COMPARISON OF OXYGEN RESERVE INDEX MEASUREMENTS FROM DIFFERENT ANATOMICAL SITES IN DOGS

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Introduction:
The oxygen reserve index (ORi) is a non-invasive parameter measured by pulse CO-oximetry. Unlike pulse oximetry (SpO_2), which reflects arterial hemoglobin saturation, ORi also accounts for venous oxygen saturation, which can vary by anatomical site. It estimates mild hyperoxemia, defined as arterial partial pressure of oxygen (PaO_2) between 100 and 200 mmHg. In dogs, ORi has been assessed using a tongue-placed probe, showing moderate correlation with PaO_2. This study examines the CO-pulse oximeter's ability to measure ORi at alternative sites.

Methods:
This prospective observational study included 16 adult anesthetized dogs undergoing elective procedures with a fraction of inspired oxygen between 0.21 and 0.50. Two multi-wavelength CO-oximeters measured ORist on the tongue and ORifoot or ORitail on the foot or tail. The Pearson correlation coefficient (r) was calculated between ORist and ORifoot or ORitail. Trending ability was assessed using a 4-quadrant plot for proportional ORist changes. Bland–Altman analysis assessed the agreement between ORist and ORifoot or ORitail measurements.

Results:
A total of 64 ORi measurements (18 ORifoot and 46 ORitail) in 14 dogs were collected. In two animals, ORitail was not detected, likely due to pigmented skin. The mean thickness of the foot and tail where the probe was applied was 13.9±1.0 mm and 13.3±2.0 mm, respectively. A very strong correlation was observed between ORist and ORifoot (r=0.95), and between ORist and ORitail (r=0.89). The trending ability of ORifoot was poor (58.3%), and that of ORitail moderate (73.9%). Bland–Altman analysis showed a mean bias of −0.04 (95% limits of agreement: −0.27 to 0.19) between ORist and ORifoot, and 0.06 (95% limits of agreement: −0.27 to 0.40) between ORist and ORitail.

Conclusions:
The CO-pulse oximeter's ability to measure ORi at alternative sites such as the foot or tail may provide a non-invasive tool for oxygenation monitoring in dogs, expanding ORi's use beyond the tongue and potentially enabling monitoring in non-anesthetized dogs. The tail appears to be a better site than the foot for evaluating oxygenation trends. However, further studies are needed to measure PaO_2 and assess the reliability of these measurements. Additionally, pigmented skin may interfere with detection.

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USE OF A TRAINING SIMULATOR FOR DETECTION OF PNEUMOPERITONEUM BY NOVICE SONOGRAPHERS USING POCUS.

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Introduction:
Based on human clinical and experimental canine studies, reverberation artifact and the enhanced peritoneal stripe sign (EPSS) are sensitive and specific point-of-care ultrasound (POCUS) indicators of pneumoperitoneum, though they may be confusing for novice sonographers. Simulation-based training effectively enhances ultrasound competency in human medicine and is increasingly used in veterinary education. We hypothesize that simulator-based training will improve detection of the EPSS and reverberation artifact, leading to more accurate pneumoperitoneum detection by novice sonographers.

Methods:
Animal and human ethics approval were obtained for this prospective study. Twenty-one 2nd and 3rd-year DVM students were enrolled. Students completed a pre- and post-training assessment on POCUS diagnosis of pneumoperitoneum and an online training module before random assignment to one of two groups: simulator model (online and simulator training) or control (online training only). The simulator group practiced identifying the EPSS/reverberation on ballistic gel pneumoperitoneum models with instructor guidance. Both groups were assessed on canine cadavers, blinded to pneumoperitoneum status, through assessor-led stations. Using an Objective Structured Clinical Examination style questionnaire, students' ability to identify the EPSS, reverberation artifact origin (free or intestinal air), and diagnose pneumoperitoneum was recorded. D'Agostino-Pearson was used for normalcy. Sensitivity (Se), specificity (Sp), accuracy, positive predictive value (PPV), negative predictive value (NPV), Chi-squared, and Fisher's exact tests compared groups ($p \leq 0.05$ considered significant).

Results:
There was no difference between pre (57.1%) and post online training (75.5%) assessment scores between groups (p=0.2517). Following training, the simulator group was more successful than the control group in correctly identifying pneumoperitoneum (p=0.0014), EPSS (p=<0.0001), and the origin of reverberation artifact (p=0.0005) in cadavers. Overall Se/Sp, accuracy, PPV/NPV of the EPSS for diagnosis pneumoperitoneum was 81.3%, 87%, 83.9%, 88.1% and 79.8% respectively, and varied between the control and simulator groups (control: Se=70.3%, Sp=82.4%, Accuracy=75% PPV=86.4%, NPV=63.64%, simulator group: Se=90%, Sp=97.5%, accuracy=93.75%, PPV=97.3%, NPV=90.7%).

Conclusion:
Use of a simulated pneumoperitoneum model significantly improves identification of pneumoperiteoneum compared to online modules alone. Preliminary results suggest novice

sonographers can identify sonographic findings supportive of pneumoperitoneum in cadavers, although application on live dogs requires further investigation.

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A COMPARISON OF HYPERECHOIC VERTICAL ARTIFACT CHARACTERISTICS IN LUNG ULTRASOUND PERFORMED WITH A MICROCONVEX, PHASED ARRAY, AND LINEAR TRANSDUCER

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Introduction:
This study compared hyperechoic-vertical artifact (HVA) characteristics using three transducer types (microconvex, phased array (PA), linear) in dogs with pulmonary disease. We hypothesize there is high-level reviewer agreement in assessing HVA image quality and characteristics, and image quality/characteristics differ between the three transducers.

Methods:
Owner consent was obtained prior to diagnostic and/or therapeutic ultrasound examination. Dogs with HVAs and sonographic absence of lung consolidations, pleural effusion, and/or pneumothorax were enrolled. Cine-loops (5-second) containing HVAs were retrospectively and independently reviewed by two reviewers blinded to case details but not transducer type. The study had 2 phases: 1) reviewers agreed on HVA characteristics to assess, 2) reviewers assessed cine-loops for the following: do HVA's meet B-line criteria (i.e. originate from pleural line, extend to far field, move with lung sliding), ease of counting HVAs (clarity of edges, blending of HVAs, graininess, fast movement, variability of width/echogenicity), and overall image quality (Lickert scale, 0-100). Paired cine-loops from the same patient using different transducers were then compared for HVA quality. Interrater concordance was determined using the Kappa coefficient, Kendall's tau, and Pearson's tau; characteristics were compared with chi-square and Kruskal-Wallis tests (level of significance; $\alpha = 0.05$).

Results:
Twenty-four cine-loops were assessed from 8 dogs (3 from each). Overall image quality concordance was good (Pearsons's coefficient=0.82). The PA scored lower for image quality ($p<0.001$), HVA blending ($p=0.014$), graininess ($p<0.001$), and clarity of edges ($p<0.001$) compared to microconvex and linear transducers. The identification of B-line criteria differed between transducers ($p=0.024$). More HVAs failed to reach the far field with the linear (10/16, 62,5%) compared to the microconvex (8/16, 50%) and PA transducers (3/16, 18,5%). The linear scored higher than the microconvex and PA transducers for ability to count B-lines ($p<0.001$). The PA had more uncountable HVAs (31,5% vs 12,5% microconvex, and 0% linear). The PA scored lowest in comparing paired cine-loops for image and HVA quality ($p<0.001$).

Conclusions:

Sonographic characteristics of HVAs can be consistently and reliably assessed. Transducer type significantly impacts the characteristics of HVAs, which could affect clinical findings. The PA transducer created lower-quality images than the microconvex and linear transducers.

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CASE REPORT OF METALDEHYDE INTOXICATION MANAGED WITH RENAL REPLACEMENT THERAPY AND MECHANICAL VENTILATION

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Introduction:

Metaldehyde is a molluscicide responsible for neurointoxication in companion animals. Metaldehyde poisoning can cause mild signs (ataxia, salivation or vomiting) to respiratory failure and seizures, necessitating intensive nursing care.

Synopsis:

A 3-year-old sterilized male Australian shepherd dog presented to the ICU for deteriorating respiratory distress and seizures secondary to metaldehyde ingestion. The animal presented stuporous in lateral recumbency with ptyalism, cyanosis, tonic-clonic status epilepticus, hyperthermia, dyspnea and tachycardia.

Anticonvulsant drugs (midazolam 0,2 mg/kg intranasal and then IV and levetiracetam at 40 mg/kg IV) were administered and endotracheal intubation was performed with propofol induction. Cooling measures were implemented. Decontamination by gastric and rectal lavage removed large quantities of blue granules consistent with metaldehyde. However, respiratory distress persisted needing oxygen supplementation via endotracheal tube at 50 ml/kg/min.

Continuous monitoring was set up. A urinary catheter was placed for comfort and hygiene. Every 4 hours we performed: oral care with dilute aqueous chlorhexidine lavage, ocular lubrication to prevent ulcers, and range-of-motion physical therapy. Due to frequent obstruction of the upper airways, we performed repeated suctioning with aseptic technique. The ETT required several urgent changes due to obstruction.

The generalized tremors persisted despite anticonvulsant treatments (midazolam, levetiracetam, methocarbamol, dexmedetomidine CRI and propofol CRI). A central venous catheter was placed for hemodialysis. This was performed for 4 hours and stopping the tremors. Despite this, respiratory signs worsened and mechanical ventilation was started. The next morning, the dog arrested. We suspected worsening pneumonia or multiple organ dysfunction syndrome.

The metaldehyde poisoning mechanism is poorly understood but it is theorized that decreases in the concentrations of central nervous system neurotransmitters may increase neuronal excitation causing seizure activity. Recent reports show dialysis is effective in treating metaldehyde poisoning in dogs. In such severe cases, intensive nursing care and close monitoring are required because they are at high risk of severe complications.

Conclusion:

Metaldehyde poisoning causes severe effects as seen with this dog. Seizure management is essential and dialysis allows patient stabilization and could be considered as a first-line treatment in similar cases. This report highlights the nursing care of a dog under long-term general anesthesia for seizures.

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Poster Abstracts

Original Study

RELATION BETWEEN SEVERE ANEMIA AND HYPERLACTATEMIA IN CATS

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Introduction:
Hyperlactatemia Type A and B has been identified in cats. Type A hyperlactatemia results from an imbalance between tissue oxygen supply and tissue oxygen demand. Anemia may lead to decreased tissue oxygen supply and type A hyperlactatemia.

The goal of the study presented here was to investigate the correlation between severe anemia and type A hyperlactatemia in cats. In addition, we hypothesized that there is a correlation between the degree of anemia and the severity of type A hyperlactatemia as has been previously reported in dogs and horses.

Methods:
Cats with severe anemia (HCT ≤13%) presenting to the emergency service of the Veterinary Teaching Hospital were identified retrospectively. Hematocrit and lactate were measured on admission before any intervention. Cats were included if the medical records were complete. Cats were excluded if any other causes for hyperlactatemia (type A or B) apart from anemia were identified or suspected based among others on history, physical examination, blood pressure measurement, laboratory analysis and abdominal and/or thoracic POCUS.

Results were analyzed using R software. A linear and logarithmic univariant regression of lactatemia as a function of microhematocrit was applied to our model. A P value of 0.05 was considered significant.

Results:
Sixty-one cats with severe anemia (HCT ≤13%) were identified initially. Thirty-eight were excluded for the following reasons: corticosteroid therapy within 6 weeks of consultation (n=24), total protein < 52 g/L (n=4), hypotension (n=4), positive abdominal and/or thoracic POCUS (n=3), suspected trauma (n=1), incomplete medical records (n=2).

Mean microhematocrit of the remaining 23 cats was 9.36 ± 2.59%, and mean lactate concentration 8.86 ± 5.33 mmol/L.

Anemia and hyperlactatemia were not significantly correlated (P = 0.8192). Pearsons correlation coefficient did not show a correlation between the degree of anemia and that of hyperlactatemia (R^2 = 0.05355).

Conclusions:

The results of this study did not show a correlation between anemia and hyperlactatemia in cats with severe anemia. In addition, no direct relationship between severity of anemia and severity of hyperlactatemia was identified as previously reported in dogs and horses. Further studies to investigate lactate in cats with different degrees of anemia may be indicated.

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COMPARISON OF TWO DIFFERENT ADSORBER COLUMNS ON IMMUNOGLOBULIN CONCENTRATION IN DOGS TREATED WITH IMMUNOADSORPTION

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Introduction:
Immunoadsorption (IA) reduces immunoglobulin concentration in humans with severe immune-mediated diseases. This study evaluates the immunoglobulin reduction in dogs treated with IA using two adsorbers.

Methods:
In this prospective clinical trial immunoglobulin levels in dogs with immune-mediated diseases treated with IA over five years were evaluated. IA was performed in 13 dogs with the Ligasorb adsorber (n=8; 2019–2023) and, as the Ligasorb was not available from 2023, with the IgOmni 1 (n=5; 2023–2024). With Ligasorb, all patients had 1 treatment and, with IgOmni 1, one dog was treated once and 4 twice at a 2-days interval.

Immunoglobulin (Ig) G and IgM concentrations were analyzed before, after, and 12–24 hours after IA. IgM was analyzed with turbidimetry and IgG with electrophoresis. Data were analyzed using a t-test, repeated-measures-ANOVA with post hoc Bonferroni's Multiple Comparison test. P-values <0.05 were considered significant.

Results:
Seventeen treatments (Ligasorb n=8; IgOmni 1 n=9) were included. Underlying diseases included immune-mediated hemolytic anemia (6), leishmaniosis-induced glomerulonephritis (2), myasthenia gravis (2), hepatopathy of unknown origin (1), immune-mediated glomerulonephritis (1) and chronic steroid responsive meningitis-arteritis (1). Treatment time (105; 70–270 min; 110; 60–286 min) and processed plasma volume (91.3±27.5 ml/kg; 113.4±18.2 ml/kg) were not different between adsorbers (p=0.595; p=0.066).

IgG decreased with Ligasorb from 701±248 mg/dl to 339±145 mg/dl and did not differ to 12–24 hours (460±162 mg/dl; p<0.001), and with IgOmni1 from 840±638 mg/dl to 436±357 mg/dl and increased to 12–24 hours (691±511 mg/dl; p=0.002). IgM decreased with Ligasorb from 129±63 mg/dl to 66±39 mg/dl and did not differ to 12–24 hours (83±42 mg/dl; p<0.001), and with IgOmni1 from 100±53 mg/dl to 59±41 mg/dl and increased to 12–24 hours (72±46 mg/dl; p<0.001). IgG values were lower at 12–24 hours compared to pre-values for both adsorber and IgM for Ligasorb but nor for IgOmni1. Mean IgG and IgM values did not differ between adsorbers at any time point

Conclusions:
IA is a promising tool for reducing immunoglobulin levels in dogs with severe, unresponsive cases of immune-mediated diseases. A rebound of both IgG and IgM was observed.

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RETROSPECTIVE EVALUATION OF PERIOPERATIVE POTASSIUM CHANGES IN DOGS AND CATS UNDERGOING CRANIOTOMY FOR TUMOR REMOVAL

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Introduction:
Cranial surgery has become standard of care for many intracranial tumors in veterinary medicine, but data on the physiological changes during surgery is limited. In humans, transient hyperkalemia requiring treatment can occur during craniotomy and has been linked to tumor handling or intraoperative administration of hyperosmolar agents causing transcellular potassium shifts. Whether relevant hyperkalemia occurs during intracranial tumor resection in dogs and cats is unclear.

Methods:
Medical records of dogs and cats undergoing craniotomy for tumor resection at the Veterinary Medical Teaching Hospital, University of California, Davis between 2009-2023 were retrospectively reviewed for point-of-care electrolyte variables. Potassium concentration ($[K^+]$) was assessed at three time points for all included cases: preoperatively, intraoperatively during tumor resection or at peak $[K^+]$ (where multiple blood tests were recorded), and immediately postoperatively. Medications and intravenous fluids administered were recorded, including dosages. $[K^+]$ was assessed for normality using the D'Agostino-Pearson test. Changes in $[K^+]$ over time and among patients that did and did not receive hyperosmolar therapy were compared using repeated-measures two-way ANOVA and Tukey's multiple comparisons test. Adjusted $p<0.05$ were considered significant.

Results:
Sixty-one dogs and fourteen cats were included. Meningioma was the most common tumor type, in 42/75 (56%) of cases, 51% in dogs and 79% in cats. Mean (±SD) preoperative $[K^+]$ (mmol/L) was 3.75 (±0.45), increasing to 3.91 (±0.46) intraoperatively, and decreased postoperatively to 3.74 (±0.46). Three (2.7%) patients developed mild intraoperative hyperkalemia, defined as $[K^+]$ <5.4 mmol/L. Mild elevations in $[K^+]$ were observed in 3/75 (4%) patients preoperatively, and 3/75 (4%) postoperatively. A single dose of mannitol was administered to 24 (39%) dogs and 5 (36%) cats, and hypertonic saline to 1 dog (2%). Intraoperative $[K^+]$ was significantly higher than postoperative ($p=0.0005$), while the administration of hyperosmolar therapy did not affect $[K^+]$ ($p=0.56$).

Conclusions:
Mild, transient intraoperative increases in blood potassium occur during craniotomies of dogs and cats. While significantly higher than post-operative concentrations, their extent is unlikely to be of clinical significance. Hyperosmolar therapy did not notably impact $[K^+]$. Until further prospective research is

available, intraoperative point of care bloodwork is still advised, especially in patients receiving multiple doses of hyperosmolar therapies.

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PROSPECTIVE EVALUATION OF THE EFFECT OF DESMOPRESSIN ON PRIMARY HEMOSTATIC DYSFUNCTION IN DOGS WITH ACUTE KIDNEY INJURY USING WHOLE BLOOD IMPEDANCE PLATELET AGGREGOMETRY

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Introduction:
Bleeding tendencies associated with decreased platelet aggregation can occur in dogs with acute kidney injury (AKI).

Hypothesis/Objectives:
To evaluate the effect of intravenous desmopressin (IV DDAVP) in dogs with decreased platelet aggregation due to AKI using impedance platelet aggregometry (IPA). Our hypothesis was that the use of IV DDAVP may improve platelet function, potentially decreasing bleeding complications derived from invasive procedures in these patients.

Animals – 12 client-owned dogs with AKI and primary hemostatic dysfunction.

Methods:
Pre-post study. Dogs with an International Renal Interest Society (IRIS) AKI grade III (blood creatinine 2.6-5.0 mg/dl) or above and documented platelet dysfunction were included. Blood samples were collected in hirudin-coated tubes for IPA analysis before and 2 hours after the administration of IV DDAVP 1 mgr/Kg. Platelet function was assessed evaluating the area under the curve (AUC), aggregation, and velocity for arachidonic acid (ASPI), adenosine diphosphate (ADP), and collagen (COL) as agonists, both pre- and post-DDAVP administration. Pre and post DDAVP values were compared using the paired t test or Wilcoxson signed rank test as appropriate.

Results:
Baseline AUC, aggregation and velocity were: 49.83 ± 36.4, 96.72 ± 65.32, and 11.15 ± 8.09 using ADP as agonist; 19.0 (3-87), 69.63 ± 55.45, and 4.95 (1.8-20.6)] using ASPI; and 10 (1-21), 48.83 ± 37.05, 10.07 ± 7.26 using COL. After administering DDAVP, AUC, aggregation and velocity were: 52.63 ± 26.6, 95.5 ± 32.05, and 9.55 ± 3.19 using ADP; 29.0 (5-97), 62.44 ± 41.50, and 5.6 (1.6-13.2) using ASPI; and 10.50 (1-64), 76.08 ± 65.25, and 10.08 ± 6.72 using COL. The baseline and post-treatment analyses revealed no

statistically significant differences for any agonist. However, despite the lack of significant changes, collagen-induced aggregation showed clinical improvement in 8 of 12 dogs.

Conclusions:
Although our results did not reach statistical significance, most dogs showed improved collagen-induced platelet aggregation following DDAVP administration.

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BROMETHALIN EXPOSURE IN DOGS AND CATS: A 14-YEAR RETROSPECTIVE STUDY (2010-2023) FROM THE CALIFORNIA ANIMAL HEALTH AND FOOD SAFETY LABORATORY SYSTEM

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Introduction:
Bromethalin is a rodenticide widely used to control rodent populations. Restrictions on other rodenticides in the US have increased its usage. Bromethalin is a neurotoxin that disrupts oxidative phosphorylation, causing neuronal damage. Diagnosis is challenging and typically relies on clinical history and supportive diagnostic tests.

Methods:
A retrospective study was conducted to characterize bromethalin exposure in samples from dogs and cats submitted to the California Animal Health and Food Safety Laboratory System from 2010-2023. Data on demographics, clinical signs, diagnostic tests, and autopsy findings were collected and analyzed. Bromethalin exposure was confirmed by detecting its metabolite, desmethylbromethalin (DMB), in tissues using liquid chromatography-mass spectrometry (LC-MS/MS).

Results:
A total of 223 cases were included, comprising 123 dogs and 100 cats. The number of cases increased 2.8-fold from 59 (2010-2016) to 164 (2017-2023). Cats were significantly younger (median 24 months, IQR: 41.5) than dogs (36 months, IQR: 60.0) (P=0.016) and more likely to have confirmed DMB exposure (60% vs. 25%, P<0.0001). Adipose tissue (37%), liver (20%), and brain (19%) were the most common samples analyzed. Clinical signs included neurological (e.g. seizures, tremors) and myelopathic symptoms (e.g. weakness, paralysis). Bromethalin traces were found in the milk of a lactating bitch. Samples from 14 kittens and 3 puppies, all ≤2 months old, revealed 14 DMB-positive cases and 3 with trace levels, suggesting bromethalin transfer into milk and potential risk to nursing litters. Magnetic resonance imaging (MRI) scan findings in 17 cases were consistent with bromethalin intoxication in 77% of cases. Autopsy findings (33 cases) revealed CNS lesions consistent with bromethalin toxicosis in 2/8 dogs and 24/25 cats and revealed diffuse white matter spongiosis, edema, and vacuolar myelopathy in the brain and spinal cord.

Conclusions:
Bromethalin exposure is becoming increasingly common in pets. Adipose tissue remains the most reliable diagnostic sample, with cats more likely to test positive for DMB and exhibit compatible autopsy

findings. This study provides the first evidence suggesting that bromethalin can transfer into milk. Additionally, MRI findings may support diagnosis. These results may enhance the understanding and management of bromethalin intoxication in pets.

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THE INFLUENCE OF PET BLOOD DONATION DISSEMINATION ON COLLEGE STUDENTS' KNOWLEDGE, ATTITUDES, AND INTENTIONS

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Introduction:
Companion animals need a reliable blood supply, but veterinary transfusion lacks regulations. This study, conducted by the Veterinary Transfusion Medicine Center (VTMC) at National Pingtung University of Science and Technology (NPUST) in Taiwan, evaluates the impact of dissemination on students' knowledge, attitudes, and intentions to enhance donor recruitment. The center ensures blood safety and donor welfare through screening, health checks, and pathogen testing.

Methods:
This study applies the Knowledge-Attitude-Behavior (KAB) model to assess the impact of pet blood donation dissemination on college students. Veterinary and non-veterinary students at NPUST attended a 30-minute lecture session by a veterinary professor, using PowerPoint and video, covering the importance of pet blood donation, blood types, procedures, and related news. Pre- and post-surveys were conducted to measure changes in knowledge, attitudes, and intentions. Data from 207 surveys were analyzed using SPSS with paired sample tests to assess changes in knowledge, attitudes, and intentions.

Results:
The results showed that after the dissemination, college students' blood donation knowledge, attitudes, and intentions significantly improved. The mean differences, t-values and p-values for each variable are shown below: knowledge (1.37, 22.93, $p<0.001$), attitudes (0.21, 5.12, $p<0.001$), and intentions (0.25, 3.97, $p<0.001$).

Conclusions:
The results indicate that before dissemination, college students had limited knowledge of pet blood donation intervals, eligibility criteria, and procedures. The dissemination effectively enhanced their knowledge while also significantly improving their attitudes and intentions toward pet blood donation, confirming its effectiveness and providing insights for future donor recruitment strategies.

Keywords: canine and feline blood donation, blood donation dissemination, blood donation intention

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SODIUM TO POTASSIUM RATIO IN DOGS WITH ACUTE KIDNEY INJURY VERSUS HYPOADRENOCORTICISM: A RETROSPECTIVE CASE CONTROL STUDY

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Introduction:
Electrolyte disturbances are common in acute kidney injury (AKI) and hypoadrenocorticism (HOAC). A sodium (Na^+) to potassium (K^+) ratio <27 is often used to support a diagnosis of HOAC, however lower ratios have also been observed in dogs with kidney disease. The primary aims of this study were to describe Na:K ratio of dogs presenting as an emergency, subsequently diagnosed with HOAC or AKI; determine the frequency of Na:K <27 in dogs with AKI, and identify point-of-care parameters that may help differentiate AKI from HOAC.

Methods:
Retrospective case-control study of dogs presenting as an emergency subsequently diagnosed with AKI or HOAC based on International Renal Interest Society guidelines and/or result of an ACTH stimulation test. Na^+, K^+, Na:K, serum creatinine (SCr), packed cell volume (PCV), total solids (TS) and systolic blood pressure (SBP) were compared between the groups. Continuous variables are expressed as mean ± standard deviation if normally distributed, or median (range) if not. Fisher's exact test was used to compare categorical data. T-test or Mann Whitney were used to compare continuous data. $P<0.05$ was considered significant.

Results:
Eighty-eight dogs with AKI, and forty-five dogs with HOAC were included. Median Na^+ concentrations were 133mmol/L (121-151, HOAC) and 145mmol/L (129-164, AKI; $p<0.0001$). Median K^+ concentrations were 5.7mmol/L (3.2-9.5, HOAC), and 4.9mmol/L (1.8-8.6, AKI; $p=0.0018$). Median Na:K ratios were 24.1 (13.7-45.4, HOAC) and 29.3 (16.7-80.7, AKI; $p<0.0001$). 31/88 (35%) dogs with AKI had Na:K <27. SBP and SCr were significantly higher in the AKI group (162mmHg ± 45.4 versus 94mmHg ± 32.0, $p<0.0001$; 533µmol/L, 27-1652, versus 166µmol/L, 41-654, $p<0.0001$). PCV was significantly lower in the AKI group (37% ± 12 versus 50% ± 12, $p <0.0001$). TS was not significantly different between the groups (66g/L ± 16, AKI, versus 62g/L ± 17, HOAC; $p=0.87$).

Conclusion:
Whilst Na^+ and K^+ concentrations, and Na:K ratios differed between the AKI and HOAC groups, Na:K <27 was not uncommon in dogs with AKI. The presence of hypotension and/or hemoconcentration, and severity of azotemia may help differentiate the two conditions in the emergency setting.

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LABORATORY COAGULATION ABNORMALITIES ASSOCIATED WITH VENOM-INDUCED CONSUMPTIVE COAGULOPATHY IN DOGS FOLLOWING EASTERN BROWN SNAKE ENVENOMATION

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Introduction:
Venom-induced consumptive coagulopathy (VICC) occurs rapidly following Eastern brown snake envenomation (EBSE). No publications describe viscoelastic test results following EBSE in dogs. This study aimed to prospectively describe coagulation disturbances over time in dogs following EBSE using standard point-of-care (POC) and laboratory coagulation tests, and thromboelastrometry (ClotPro®) and compare the frequency of abnormal results of these tests.

Methods:
Dogs with EBSE were tested on presentation, with those presented within two hours of observed envenomation eligible to also be tested at 8, 16, and 24 hours after presentation. Testing included point-of-care and Stago STA R Max prothrombin times and activated partial thromboplastin times (POC PT, STA R Max PT, POC APTT, STA R Max APTT), activated clotting time (ACT); Stago STA R Max fibrinogen concentration; ClotPro FIB-test, EX-test and IN-test; and brown snake serum venom levels. The frequency of abnormal results for each test was calculated with Wilson binomial confidence intervals and compared with McNemar's tests.

Results:
Seventeen dogs were enrolled, with six also included in the longitudinal study. All dogs had prolonged ACT, POC PT and STA R Max PT and decreased fibrinogen, and most dogs also had no clot formation on FIB-test (16/17) and hypocoagulable FIB-test (17/17), EX-test (15/17) and IN-test (14/17) tracings at presentation.

Across all time-points and samples (n=35), reduced fibrinogen concentrations (31/35) and prolonged FIB-test CT (32/35) were more frequently observed compared to prolonged POC PT (25/35; p = 0.035, p= 0.026 respectively) and prolonged POC aPTT (25/35; p = 0.035, p= 0.026 respectively). The POC PT was also prolonged less frequently (25/35) than the STA R Max PT results (34/35; p = 0.013). All dogs (6/6) had normalization of POC PT and aPTT over 24 hours. Only limited cases had normalization of MAX-ACT (2/6), STA R Max PT (1/6), aPTT (5/6), fibrinogen (3/6) and ClotPro tracings (2/6) during the study period.

Conclusions:
More severe coagulation abnormalities were recorded in dogs with an observed envenomation. Viscoelastic tests but not POC PT and PTT showed persistence of mild coagulation disturbances after 24 hours.

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COMPARISON OF INTRAVENOUS MIXED MICELLE PHYTOMENADIONE WITH TRADITIONAL STANDARD TREATMENT WITH EXOGENOUS COAGULATION FACTORS TRANSFUSION FOR RODENTICIDE TOXICOSIS IN DOGS AND CATS: A RETROSPECTIVE STUDY

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Introduction:
Ingestion of anticoagulant rodenticides causes severe coagulopathy due to a deficiency of Vitamin K-dependent clotting factors. The current recommended emergency treatment is to administer plasma-containing blood products. Administration of parenteral Vitamin K (phytomenadione) alone is discouraged as it takes up to 48 hours to reverse coagulopathy, even when given intravenously. Moreover, anaphylactoid reactions have been reported following intravenous administration of phytomenadione. Our objectives were to describe the incidence of adverse reactions to a mixed micelle formulation of phytomenadione (MMP) administered intravenously, its effect on coagulopathy, and to compare outcomes between patients treated with intravenous MMP and standard treatment.

Methods:
Dogs and cats treated for clinical rodenticide toxicosis between July 2021 and July 2024 were retrospectively reviewed. Patients were assigned to the "MMP" group if they received IV MMP, and into the "control" group if they didn't. Adverse reactions, timing and results of post-treatment coagulation testing, and outcomes (survival, length of hospitalisation, costs and transfusion requirements) were compared between the two groups.

Results:
Twenty-nine patients were included in the MMP Group (26 dogs, 3 cats) and 44 in the control group (all dogs). Patients in the control group received FFP (n=40), WB (n=1) or vitamin K via PO, SQ or IM route as first line treatment while those in the MMP group received IV MMP. There were no reported adverse reactions to MMP. Median post-treatment PT for the MMP and control group was 15 and 16 seconds respectively. There was no significant difference in survival ($p=0.28$) or red blood cell transfusion requirements ($p=1$). Patients in the MMP group had significantly shorter length of hospitalisation ($p=0.01$) and lower cost of treatment ($p<0.01$). None of the patients in the MMP group received a plasma transfusion. Within this group 22 patients had a PT rechecked after MMP prior to any other therapy that could affect the result; median time to normalisation of PT was 2 and 2.3 hours post-MMP in dogs and cats, respectively.

Conclusions:
Intravenous MMP was found to be safe and rapidly reversed coagulopathy. Outcomes for patients

receiving MMP and traditional therapies were similar. MMP can be used as a substitute for plasma transfusion in dogs and cats with anticoagulant rodenticide toxicosis.

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RETROSPECTIVE EVALUATION OF THE RELATIONSHIP BETWEEN BLOOD AMMONIA AND ACID-BASE OR BIOCHEMICAL PARAMETERS IN AZOTEMIC CATS

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Introduction:
Renal ammoniagenesis has been linked to acidosis and hypokalemia in people. Our objective was to evaluate a possible association between blood ammonia and acid-base parameters or electrolytes in cats with renal azotemia. We hypothesized that the degree of hyperammonemia in this population is associated with the magnitude of acid-base and electrolyte abnormalities.

Methods:
Medical records from our institution were reviewed to include cats with renal azotemia (serum creatinine ≥ 141 umol/L or 1.6 mg/dL) where blood ammonia was measured. Recorded data included demographic variables, blood ammonia, sodium, potassium, creatinine, urea, pH, bicarbonate, venous CO2 and base excess. Exclusion criteria were liver dysfunction, portosystemic shunt, UTIs by urease-producing bacteria and hypocobalaminemia. Cats were divided into two groups based on the presence of hyperammonemia (NH3⩾65 mmol/L). A Pearson correlation test was performed to evaluate the associations between blood ammonia with the various biochemical parameters. A mixed linear model (LMM) was performed with blood ammonia as the response variable, biochemical parameters and demographic variables as fixed effects and the patient as the random effect to account for the repeated measures.

Results:
Thirty-four cats were included in the study. There were 39 paired measurements of blood ammonia with laboratory parameters. Laboratory values were measured twice in four cats, three times in one and once in the remaining 29 cats. Nine cats (26.5%) had hyperammonemia (median 124 mmol/L, range 71-230) whereas 25 cats (73,5%) had blood ammonia within reference intervals (median 27.5 mmol/L, range 0-61). Hyperammonemic cats had significantly lower body weight compared to non-hyperammonemic cats ($p < 0.001$). They also had significantly higher BUN ($p < 0.001$) and creatinine ($p = 0.001$).

Statistically significant correlations were found between blood ammonia and pH ($r = -0.533$, $p < 0.001$), venous bicarbonate ($r = -0.388$, $p = 0.015$), base excess ($r = -0.438$, $p = 0.005$), and potassium ($r = 0.328$, $p = 0.042$). The LMM showed that higher creatinine [beta coefficient (BC)=4.391, p=0.045), higher CO2 (BC=12.857, p<0.01) and lower bicarbonate (BC=-17.492, p=0.006) were associated with blood ammonia.

Conclusions:
The degree of azotemia and metabolic acidosis are associated with higher blood ammonia in cats with renal impairment.

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RETROSPECTIVE EVALUATION OF THROMBELASTOGRAPHY FOR ASSESSING BLEEDING RISK AND TRANSFUSION REQUIREMENTS IN DOGS WITH IMMUNE-MEDIATED THROMBOCYTOPENIA

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Introduction:
Dogs with immune-mediated thrombocytopenia (IMTP) have variable bleeding tendencies that are inconsistently correlated with platelet count (PLT). Thrombelastography (TEG) is a valuable tool for assessing coagulation and has been used to predict bleeding tendencies. This study evaluates the utility of TEG in determining bleeding risk and blood transfusions requirements in dogs diagnosed with IMTP.

Methods:
A retrospective study was conducted, reviewing medical records from April 2019 to January 2025 for dogs with severe thrombocytopenia (<50.000/µL). Cases with sufficient follow-up to establish the diagnosis of confirmed or presumptive non-associative or associative IMTP were included. Dogs already treated with immunosuppressive therapy for more than 48 hours, or those who received blood transfusions or vincristine prior to initial laboratory analyses were excluded.

Hematocrit, PLT, fibrinogen, and TEG parameters (R time, K time, α-angle, maximum amplitude [MA]) were evaluated for differences between outcome groups: bleeding vs. non-bleeding and transfusion required vs. not required. Data were analyzed using Mann-Whitney-U test for non-normally distributed variables and Student's t-test for normally distributed variables. Effect sizes were calculated to quantify the differences (Cohen's d for t-test and Pearson correlation coefficient (r) for Mann-Whitney-U). Statistical significance was set at $p \leq .05$.

Results:
Fifteen cases were included in statistical evaluation. Median PLT count was 10.000/µL (range: 0–45.000/µL). Four dogs had non-associative IMTP, and eleven associative IMTP. Nine dogs had a bleeding event, and three dogs required transfusions.

PLT counts were significantly lower in dogs with bleeding events (median: 4.000/µL, range: 0–34.000/µL vs median: 20.000/µL, range: 7.000–45.000/µL, p=.04, r=-.55). No cutoff value was identified. No difference was found for blood transfusion requirement (p=.22). MA was significantly higher in non-bleeding dogs (median: 57.3 mm, range: 47.5–77.9 mm, p=.01, r=-.64 vs median: 38 mm, range: 12.1–58.2 mm). MA appeared lower in dogs needing blood transfusion, but this trend was not significant (p=.10) with a medium effect size (r=-.45). No significant differences were found for other variables (hematocrit, fibrinogen, R time, K time and α-angle).

Conclusion:
Maximum amplitude in TEG showed promising results to evaluate bleeding risk in dogs with IMTP and might be associated with transfusion requirements

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A RETROSPECTIVE STUDY ON INDICATIONS, APPLIED TECHNIQUES AND OUTCOME AFTER EXTRACORPOREAL THERAPIES IN COMPANION ANIMALS

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Introduction:
Extracorporeal therapies (ECT) are indicated for blood purification in endogenous substances accumulation (ESA) such as acute kidney injury (AKI), immune-mediated diseases (IMD) or severe intoxications applying either hemodialysis (HD), hemoperfusion (HP), or membrane-based therapeutic plasma exchange (mTPE). Studies describing ECT indications, complications, and outcomes based on the underlying cause are limited.

The objective of this retrospective study were to describe the signalment, indications, applied ECT, complications and patient outcome. Our hypotheses were that complication rates and outcome differed according to the indication, underlying cause of AKI and the applied ECT.

Methods:
Medical records of patients treated with ECT from November 18th 2023 to December 31st 2024 were retrospectively reviewed. Signalment, indication, applied ECT, complications, and outcome were recorded. One-month survival in AKI patients with confirmed leptospirosis was compared to those without. Survival was analyzed using Fisher's and Kruskal-Wallis tests. A generalized linear mixed model assessed complications by applied ECT.

Results:
Forty-one animals underwent 103 ECT sessions (37 dogs, 4 cats). Indications were ESA (28/41, 27 for AKI and 1 hepatic encephalopathy), intoxication (10), and IMD (3). Complication rate was not significantly different regarding applied ECT (HD 44%, HP 71,4%, mTPE 64%, p-value 0,32). The complications most commonly encountered during ECT were circuit clotting (15/103, 15%), hypocalcemia (10/103, 10%), bleeding (9/103, 9%), and hemodynamic instability (9/103, 9%). Sample size did not allow to assess whether a specific complication was associated with the applied ECT. However, none of the complications was associated with case fatality. Twenty-five animals were discharged (61%), 20 (80%) thereof were alive one month after discharge. Animals with intoxication had significantly higher survival rates (100%) compared to ESA (50%) or IMD (33%) patients (p-value 0,033). Dogs with leptospirosis had a better one-month survival prognosis than dogs with AKI without leptospirosis (100% vs 20%, p-value 0,0050).

Conclusions:
Complication rate associated with ECT was high regardless of the technique, but not associated with case fatality. Animals undergoing ECT for intoxication had an excellent outcome. Patients with AKI and confirmed leptospirosis had a significantly better one-month survival compared with AKI patients without evidence of leptospirosis.

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ETIOLOGY AND OUTCOME OF HYPOGLYCEMIA IN DOGS PRESENTING TO AN EMERGENCY ROOM

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Introduction:
Although there are many descriptions of common causes of hypoglycemia in dogs, the epidemiology of hypoglycemia has not been well studied to date. The aims of this study are to document period prevalence, underlying etiology and associated mortality of hypoglycemia in dogs presenting to an Emergency room.

Methods:
Over a 10-year period, dogs with a blood glucose concentration measured on a blood gas analyzer within 6 hours of presentation to the emergency room were identified. Medical records of dogs with hypoglycemia (blood glucose < 70 mg/dL) were reviewed further. Hypoglycemia was further categorized as mild (60-69 mg/dL), moderate (50-59 mg/dL) and severe (<50 mg/dL).

Results:
6,621 dogs met the inclusion criteria, and 255 dogs (3.8%) were hypoglycemic. Mild hypoglycemia was reported in 95/255 (37.3%), moderate in 50/255 (19.6%) and severe in 110/255 (43.1%) of dogs. Sepsis 65/255 (25.5%) and being an adult toy breed (<5kg) 70/255 (27%) were the most commonly identified causes of hypoglycemia.

Median blood glucose was significantly lower in dogs that died or were euthanized (median 48 mg/dl, IQR 35 – 61 mg/dL) compared to survivors (median 56.5 mg/dL, IQR 45.3 – 66 mg/dL) (P=0.004). Mortality was significantly higher in patients with severe hypoglycemia 71/110 (65%) compared to mild 45/95 (47%) (P=0.02) or moderate 23/50 (46%) (P=0.04) hypoglycemia.

Conclusion:
Hypoglycemia was uncommon in this patient population but was associated with a high mortality rate. Evaluation of blood glucose concentration is important in dogs in at-risk categories such as suspected sepsis and toy breeds. Further investigations on the prognostic significance and potential benefits of early resolution of hypoglycemia is warranted.

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CLINICAL PRESENTATION AND OUTCOMES OF DOGS UNDERGOING A NEGATIVE EXPLORATORY LAPAROTOMY: 45 CASES (2015-2023)

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Objective:
To describe the presentation, management and end diagnoses amongst dogs undergoing negative exploratory laparotomies.

Materials and Methods:
Clinical records were retrospectively reviewed for cases presenting to a university teaching hospital following a negative exploratory laparotomy between January 2015 and December 2023. A negative exploratory laparotomy was defined as a surgery which did not involve treatment of the underlying condition or when performed on suspicion of an obstruction or foreign body which was not identified, irrespective of whether a different diagnosis was made at surgery. Dogs were included if they had undergone a negative exploratory laparotomy within seven days following onset of gastrointestinal clinical signs or witnessed foreign body ingestion. Patient signalment, presenting clinical signs, diagnostic tests performed before surgery, reason for exploratory laparotomy (where available), surgical findings, whether biopsies were obtained, end diagnoses and presence of surgery-specific complications were recorded.

Results:
45 dogs were included in the study. 40 of 45 dogs (89%) presented with vomiting and 24 of 45 dogs (53%) presented with diarrhoea. Dogs underwent the following abdominal imaging: radiography only (n=19), radiography and ultrasonography (n=24) and ultrasonography only (n=2). The most common reported radiographic finding was intraluminal gas in the stomach, small intestine, or cranial abdomen in 11 dogs. The most frequent reasons for the exploratory laparotomy were: suspicion of a foreign body (n=32), no improvement in supportive care with surgery being considered the next diagnostic step (n=9) and suspicion of an intussusception (n=1). The most frequent surgical findings were inflamed small intestines (n=15). Biopsies were performed in 16 of 45 dogs. Of 9 dogs in which small intestinal biopsies were performed, 3 developed surgical site dehiscence and septic peritonitis. Thirty-nine dogs survived to discharge.

Conclusions:
In this population of dogs presenting with acute gastrointestinal signs and undergoing negative exploratory laparotomy procedures, foreign body obstructions were frequently suspected. Diagnostic

imaging was performed pre-surgically in all cases. Further data on the implications of negative exploratory laparotomies is required.

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MICROBIOLOGICAL STUDY OF RECIRCULATED SALINE IN HEMOFILTERS

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Introduction:
It is a standard procedure for humans undergoing CRRT to reconnect the patient to a recirculated saline circuit, from which they were disconnected, in order to perform diagnostic test. This strategy could be an option in veterinary patients. The purpose of this study is to investigate the sterility of recirculated saline in continuous renal replacement therapy (CRRT) platforms.

Methods:
Prior to starting extracorporeal renal therapy, a blood sample was taken from the patient`s vascular access as part of the AKI diagnostic procedure. After CRRT was completed, the circuit was exposed to five rinses using a one-liter bottle of SSF 0.9% and platform (Aquarius Nikkiso® and Aquarius Nikkiso Plus®) was placed in recirculation mode. After cleaning the sampling port of the access line with alcohol, a recirculated saline sample was drawn at 24 and 48 hours recirculation. All samples were place in culture bottles (Signal blood culture system Oxioid ®) and incubated. Positive samples were cultured for organism identification and susceptibility testing. No cartridge was reused. Categories with $n< 5$ were compared by Fisher´s exact test. Significance was set at $p < 0.05$.

Results:
In this prospective study, 42 samples were cultured [14 from blood and 28 from recirculating saline samples (14 at 24h and 14 at 48h)]. Bacterial growth was obtained from 1 blood sample (7.1%) and 7 saline samples (28.5 % at 24h and 21.4% at 48h). Bacteria of the genus Streptococcus (n=1), Pseudomona (n=2), Enterococcus (n=1) and Serratia (n=2) were isolated. The only positive blood sample (Klesiella pneumoniae) resulted in a positive growth at 24 and 48 h. No association was observed between positive blood and cartridge cultures (blood vs 24 h, $p= 0.286$; blood vs 48h, $p= 0.214$; 24h vs 48h, $p= 0.176$).

Conclusions:
This study demonstrated bacterial growth in saline after 24 and 48 h of recirculation. By acting as a culture medium, blood kept in the circuit can enhance the growth of germs. These bacteria may come from patients or from contamination of the circuit during handling. Based on our results, we cannot recommend reusing lines and hemofilters in patients undergoing extracorporeal purification techniques.

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KNOWLEDGE OF VETERINARY STUDENTS AND VETERINARIANS REGARDING PROGNOSIS AND PROGNOSTIC FACTORS OF COMMON CANINE EMERGENCY CONDITIONS AND THEIR ASSOCIATED COMMUNICATION STYLE

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Introduction:
Owner decisions in critical patients are influenced by financial and ethical concerns. Knowledge of prognosis and prognostic factors, and a balanced communication are pivotal to allow owners to take informed decisions. This study aimed to assess the knowledge of Belgian veterinary students and veterinarians regarding prognosis and prognostic factors of common canine emergency conditions and their associated communication style. Our hypotheses were that knowledge of prognosis, prognostic factors and communication style are different between veterinary students and veterinarians. We further hypothesize that the communication style is correlated with the perceived prognosis.

Methods:
A survey was sent to students and veterinarians, assessing knowledge regarding prognosis and prognostic factors reported in recent literature for septic peritonitis (SP), diabetic ketoacidosis (DKA), traumatic pneumothorax (PT), leptospirosis associated acute kidney disease (AKDL) and gastric dilatation-volvulus (GDV). Participants selected their preferred communication amongst 8 predefined (positive to negative) suggestions. Students were grouped in 5th with 6th year, veterinarians according to experience (more or less than 10 years). For statistical analysis, comparisons between groups were performed using the Chi-squared test.

Results:
One hundred and fifty-six (76 5th year, 80 6th year) students and 308 (161 <10-year, and 147 > 10-year experience) veterinarians responded anonymously. Over half of veterinarians and students missed the reported prognosis of PT, AKDL and GDV by at least 10%, although veterinarians scored better for PT (p=0,0001), and students for AKDL (p<0,0001). Knowledge of prognostic factors was limited, with in total 7 prognostic factors significatively more often identified by veterinarians, versus 3 by students. Of twenty-three prognostic factors over all conditions, sixth-year students identified 10 prognostic factors significantly more often than fifth-year students, and less experienced veterinarians identified 3 more often than more experienced veterinarians. The communication of students was significantly more

positive for SP (p=0,0001), AKDL (p=0,0019), PT (p<0,0001) and GDV (p=0,0072), veterinarians were more positive for DKA (p=0,027).

Conclusion:
Veterinarians and students have insufficient knowledge regarding prognosis of common emergency conditions. Veterinarians scored better for prognostic factors, students score better in their final year. The students' preferred communication style was more positive despite similar knowledge regarding prognosis.

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EFFECT OF CLINICAL CASE PRESENTATION AND TRIAGE FINDINGS ON THE PERCEIVED SURVIVAL ODDS AND COMMUNICATION STYLE OF VETERINARY STUDENTS AND VETERINARIANS

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Introduction:
Treatment decisions in critical care are based on factors such as the patient's prognosis and suffering. The impression of patient suffering, based on visual perception and triage findings, may influence the veterinarian's subjective estimate of survival probability, as well as the communication style used when discussing therapeutic options. This study investigated the effect of visualization of a patient and triage findings on a subjective estimate of survival probability and communication style of veterinary students and veterinarians. We hypothesized patient visualization and triage findings induces a change between the perceived prognosis for the condition and the subjective estimate of survival probability for the patient; additionally we hypothesize this change is associated with a changed communication style.

Methods:
An anonymous survey was sent to veterinary students and veterinarians. The survey assessed their perception regarding prognosis of septic peritonitis (SP), diabetic ketoacidosis (DKA), pneumothorax (PT), leptospirosis associated acute kidney disease (AKDL) and gastric dilatation-volvulus (GDV). Participants selected their preferred communication style from eight predefined options for each scenario. For each, a video of a patient at presentation was displayed, as well as the patient's triage findings, after which the subjective estimate of survival probability and the preferred communication style for the patient were asked. For statistics, McNemar and Wilcoxon signed-rank tests were used to study the evolution of responses.

Results:
Three hundred and eight veterinarians and 156 students participated. The presentation of the patient with SP, AKDL and GDV did not change the subjective estimate of survival probability of the patient compared to the perceived prognosis. However, the subjective estimate of survival probability of the patients with DKA ($p<0,0001$) and PT ($p<0,0001$) were significantly lower than the perceived prognosis for the conditions. Accordingly, the selected communication style did not significantly change before and after seeing the patients with SP, AKDL and GDV, whilst the selected communication style became significantly more negative after seeing the patients with DKA ($p<0,0001$) and PT ($p<0,0001$).

Conclusion:
Visual patient presentation and triage findings can significantly affect the subjective estimate of survival probability, and change the preferred communication style of veterinarians and veterinary students.

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CLINICAL RELEVANCE OF GLOBAL POCUS (CARDIAC, THORACIC AND ABDOMINAL) IN CONVULSING DOGS AND CATS UPON ADMISSION TO THE EMERGENCY DEPARTMENT

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Introduction:
Point-of-care ultrasound (POCUS) is widely used in emergency settings but remains underexplored in convulsive patients. This study aimed to assess the clinical utility of POCUS in these patients and its relevance in critical care management.

Methods:
A retrospective study was conducted from 2021 to 2024. Dogs and cats presenting with seizures and undergoing global POCUS (cardiac, thoracic, and abdominal) were compared to a control group without POCUS. Exclusions included cases unrelated to seizures, incomplete medical records, or neurological conditions other than seizures. Data on patient characteristics, seizure patterns, diagnoses, clinical signs, POCUS results, and management changes were collected and analysed using χ^2 tests ($p < 0.05$). Adjusted models assessed factors influencing patient management.

Results:
The study included 304 animals in the POCUS group (197 dogs, 107 cats) and 228 in the control group. A definitive neurological diagnosis was established in 37.8% (n = 115) of cases. Positive POCUS findings were observed in 38.8% (n = 118) of cases (25.0% thoracic, 19.4% abdominal, 8.2% cardiac). No significant association was found between species and POCUS results ($p = 0.53$). Older age significantly increased the likelihood of positive POCUS findings ($p < 0.01$). Vomiting and clinical abnormalities were also significantly associated with positive POCUS results ($p < 0.01$ for both).

Patient management was more frequently adjusted in the POCUS group (78.4% vs. 21.6%, $p < 0.01$). Among patients who had both global POCUS and care adjustments, 67.9% had positive POCUS findings. POCUS results and physical examination findings were both significantly associated with care adjustments ($p < 0.01$ for both). Patients with global POCUS had clinical abnormalities in 56.9% of cases, with no statistical difference from the group without POCUS ($p = 0.06$). In the POCUS group, 29% of patients without clinical abnormalities still had positive POCUS findings. Among these, in the subgroup with care adjustments, 95.8% had positive POCUS findings ($p<0.01$).

Conclusion:
Although seizure type did not significantly influence POCUS results, clinical abnormalities increased the likelihood of positive findings. Global POCUS appears to be a valuable tool for managing convulsive patients, influencing treatment, and guiding vital diagnostic decisions.

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THE EFFECT OF A CPR LECTURE AND LABORATORY SESSION ON KNOWLEDGE, PERCEIVED KNOWLEDGE, AND PERCEIVED COMFORT ON A COHORT OF SECOND-YEAR VETERINARY STUDENTS

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Introduction:
Veterinary CPR success rates are extremely low, with only 6-8% of small animals surviving after cardiopulmonary arrest. In 2011 (and updated in 2024), The Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative created evidence-based CPR guidelines based on the human and veterinary literature, with emphasis on the importance of proper training of rescuer teams performing CPR. As veterinary students represent a key part of the care team at veterinary teaching hospitals and the future of the profession, it is imperative that they are proficient in CPR. The present study aimed to evaluate how a lecture and interactive simulation session on veterinary CPR impacted knowledge, perceived knowledge, and perceived comfort in a cohort of second-year veterinary students.

Methods:
Second-year veterinary students participated in a CPR curriculum composed of a 2-hour lecture and interactive, hands-on, tournament bracket-style CPR simulation scenarios. The students completed a survey assessing knowledge, perceived knowledge, and perceived comfort with CPR before the curriculum, immediately post, and 1-month following the curriculum. Institutional Review Board (IRB) approval was obtained before the initiation of the study.

Results:
One hundred second-year veterinary students participated in this study, with 25 students lost in the post-survey and 76 students lost in the 1-month post-survey. Knowledge scores significantly improved from pre-curriculum to post curriculum ($p = <0.0001$). No significant changes in knowledge scores were found between the immediate post and 1-month post surveys ($p=0.17$). Similarly, students had increased perceived knowledge and perceived comfort scores between pre-curriculum and immediately following the curriculum. The only significant independent variable affecting pre-knowledge scores in this study was previous RECOVER training ($p=<0.001$).

Discussion:
This is the first study to evaluate second-year veterinary students' knowledge, perceived knowledge, and perceived comfort before, immediately after, and 1-month after a CPR lecture and interactive simulation session. As anticipated, students had a significant improvement in their knowledge, perceived knowledge, and perceived comfort scores immediately after the curriculum. This study also highlights the effectiveness of a combined lecture and simulation RECOVER CPR training. Further studies evaluating veterinary student knowledge retention, CPR performance, and perceived comfort in performing veterinary CPR are warranted.

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COMPARISON OF MEDICAL AND SURGICAL TREATMENT OF PYOTHORAX: A RETROSPECTIVE STUDY OF 105 CATS

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Introduction:
While most cats with pyothorax respond favorably to medical treatment, surgery is required in refractory cases. This retrospective study compared the outcome of medically and surgically treated cats and recorded thoracic computed tomography (CT) findings and complications of surgically treated pyothorax cats. We hypothesize prognosis of medically and surgically treated cases is not significantly different.

Methods:
Medical records from September 2014 to January 2025 were reviewed, including cats with pleural effusion and intracellular bacteria on cytology. Signalment, surgical or medical treatment, CT findings, type of surgical intervention and complications were recorded. A Chi-square test was performed to assess statistical differences between medically and surgically treated cats.

Results:
We included 105 cats, 88 medically treated, and 18 surgically (medically refractory) treated cats. Survival rate was 67.6% (71/105) overall and was not significantly different between medically (61/87; 70%) and surgically (10/18; 56%) treated ($p > 0.05$) cats. Prior to surgery, CT identified lymphadenopathy (13), atelectasis (15), abscess (10), and pleural thickening (8). Seventeen cats underwent median sternotomy, one a thoracotomy. Abscess debridement was performed in 15 (83%), lobectomy in 5 (27%). Intraoperative complication rate was 33% (6/18): fatal cardio-respiratory arrest (4), air leak (2), and hemorrhage (1). Postoperative complication rate was 78% (14/18): anemia (4), pain (4), pneumothorax (2), drain-related complications (2), and hypotension (2). Median preoperative hospitalization time was 7 days, median overall hospitalization time 8.5 days (range 3-21 days) for surgically treated cats. Chest tubes remained in place for a median of 5 days postoperatively for cases in which it was recorded (9/18). Upon drain removal, cytology revealed polymorphonuclear leukocytes (PMN) without evidence of bacteria. Outcome was not significantly different between surgically and medically treated cats ($p = 0,230$; OR = 1.87). No other recorded parameters (surgical approach, adhesions intraoperatively, postoperative complications) were associated with survival in surgically treated cats ($p > 0.05$).

Conclusion:
Prognosis was not significantly worse for cats requiring surgical treatment in this study, although this may be due to the low number of cats refractory to medical therapy. Surgical treatment was associated with a guarded prognosis, with significant peri-anesthetic mortality and a high complication rate.

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PHARMACOKINETICS OF AMPICILLIN IN DOGS WITH OLIGOANURIC ACUTE KIDNEY INJURY OF SUSPECTED INFECTIOUS ORIGIN UNDERGOING RENAL REPLACEMENT THERAPY - THE CONCENTRATE STUDY PART 1

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Introduction:
The objective of the study was to determine the impact of RRT on ampicillin pharmacokinetics in dogs with oligoanuric AKI. We hypothesized that these dogs would have a reduced clearance and increased volume of distribution of ampicillin.

Methods:
This single-centre, prospective, observational study included dogs with stage IRIS 4, oligoanuric (urine output < 1ml/kg/h) AKI of suspected/confirmed infectious origin in the ICU of VetAgro-Sup (SIAMU; France) and receiving 4-6hours sessions of low efficiency hemodiafiltration.

Institutional ethics approval was obtained (no. 2396). Ampicillin/sulbactam (22mg/kg IV) injections were administered via a calibrated syringe pump over three minutes and timed to ensure administration of a dose 30min post-RRT and every 8 hours then.

One millilitre of EDTA blood was sampled from the central venous catheter at seven time points over 24 hours: at study inclusion, immediately pre-RRT, then immediately before and after the three ampicillin/sulbactam injections post-RRT (no sample taken after the third injection). Samples were immediately centrifuged at 3500 rpm for 5 minutes. Separated plasma was immediately stored in Eppendorf tubes at -20°Celsius for up to 90 days. Plasma ampicillin concentrations were measured using ultra-performance liquid chromatography-tandem mass spectrometry. Pharmacokinetic modelling was performed (Lixoft, Monolix 2023R1 software) and results were compared with internal unpublished data of Ampicillin pharmacokinetics in healthy dog data from École Nationale Vétérinaire d'Alfort.

Results:
Ten dogs (5 females (3 neutered, 2 intact), 5 males (2 neutered, 3 intact)) were included. Median age was 3.5 years (range 0.5-10.5). Final diagnosis was leptospirosis (4), protein losing nephropathy (1), pyelonephritis (1), suspected poisoning (2) and unknown origin (2). Median creatinine at inclusion was 1154 µmol/L (883-2576) and median urine output during the sampling period was 0.022 mL/kg/hr (0-1.4). Three of ten dogs survived to discharge.

Oligoanuric dogs had significantly lower clearance (33.90 (28.25) vs 252.0 (99) mL/kg/h; P < .0005) and higher volume of distribution (605.0 (466.8) vs 125.0 (43.03) mL/kg; P <.001) than healthy dogs.

Conclusion:
Oligoanuric AKI dogs had markedly reduced clearance and increased volume of distribution of ampicillin. Ampicillin dosing in oligoanuric dogs should be adjusted accordingly.

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RETROSPECTIVE ANALYSIS OF THE ASSOCIATION OF APPLEFAST SCORE, QSOFA SCORE AND CLINICOPATHOLOGICAL DATA WITH OUTCOMES IN FELINE PYOTHORAX

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Introduction:
Feline pyothorax is a common emergency condition, however, research investigating factors associated with outcomes is limited. Illness severity scores such as the feline Acute Patient Physiologic and Laboratory Evaluation (APPLE) fast score and Quick Sepsis-related Organ Failure Assessment (qSOFA) have been found to be predictive of outcome in veterinary patients with sepsis, but these scores have not been explored in feline pyothorax.

Methods:
Electronic clinical records were searched for cats diagnosed with pyothorax. Environmental data, signalment, clinicopathological data and outcome data was collected. When possible, APPLEfast and qSOFA scores were calculated and whether cats met SIRS criteria was recorded. Logistic regression was used to explore the association between illness severity scores and 28-day mortality.

Results:
Fifty-six cats were included. The most common breeds were Domestic Shorthair (n=27), Maine Coon (n=10) and Ragdoll (n=4). Median age was 4 years 1 month (range 5 months to 12 years 4 months). Thirty-two cats received medical management whereas surgical management was pursued in 24 cats. Survival to discharge was 73.2% (41/56) and 28-day survival was 60% (24/40). There was no difference in baseline characteristics between treatment strategies or between survivors and non-survivors. There was no difference in markers of illness severity between cats undergoing surgical compared to medical management. Survival to discharge rates were lower among cats that met the SIRS criteria ($p = .003$), had creatinine levels over 139 umol/L ($p < .001$), or had albumin levels below 20 g/L ($p = .003$). Fewer cats who met SIRS criteria were 28-day survivors ($p = .001$). Logistic regression revealed no association between APPLEfast or qSOFA scores and 28-day mortality. Cats that met SIRS criteria on admission had an increased risk of 28-day mortality (odds ratio [OR] 36, 95% confidence interval [CI] 4.80 – 777.87, p=0.003). When this model was adjusted for confounders (duration of clinical signs and presence of comorbidities), the association between meeting SIRS criteria and 28-day mortality remained statistically significant (OR 51.14, 95% CI 4.59-1952.47, p=.007).

Conclusions:
Out of commonly used illness severity scores, only SIRS criteria was found to be associated with increased risk 28-day mortality in cats with pyothorax.

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Poster Abstracts

Case Reports

NON-TRAUMATIC RUPTURE OF A URACHAL REMNANT IN AN ADULT DOG, LEADING TO A UROABDOMEN

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Background:
The urachus is an allantoic remnant which allows urine to pass between the urinary bladder and the placenta. Normally the urachus undergoes complete atrophy by the time of birth and should be a nonfunctional fibrous connective tissue remnant. Sometimes this process fails (in humans as well as in animals); regression is incomplete and different types of urachal anomalies occur. Urachal anomalies can be symptomatic as well as asymptomatic. Rarely, severe complications later in life, caused by urachal anomalies have been described in some species, but not jet in dogs.

Case presentation:
An 8-year-old, female neutered Cesky Fousek (29 kg) was presented with abdominal pain and lethargy after jumping in the car. On clinical examination, red mucous membranes, an elevated heart rate and abdominal pain were found. Ultrasonographic examination revealed peritoneal effusion. The urinary bladder was completely empty, which hindered a detailed ultrasonographic assessment of the wall. Cranial along the wall, echogenic tissue and hyperechoic fat were present. Creatinine in the abdominal fluid (1785 micromol/L) was more than two times the creatinine level in the peripheral blood (513 micromol/L), consistent with a uroabdomen. With positive contrast cystography, leakage of contrast from the bladder into the peritoneal cavity was confirmed. The dog was treated with IV fluids, paracetamol, methadone, maropitant and amoxicilline/clavulanic acid. After stabilization the dog went to surgery; a large rupture of the bladder apex with an excentric mass was discovered. The abnormal part of the bladder wall was resected. Histopathology showed epithelial structures originating from a persistent urachus, there was no evidence of neoplastic tissue. The dog did well and went home 2 days after surgery.

Unique information:
As far as the author knows, this is the only known case of a urachal remnant in an adult dog leading to a uroabdomen. In emergency and critical care, veterinarians should be aware of the potential of ruptured urachal anomalies and a consequent uroabdomen in patients with abdominal pain (even without a history of obvious trauma). More research is necessary to determine which remnants are more likely to lead to clinical disease and when to surgically intervene.

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SEVERE ANAEMIA AND PALATINE HAEMORRHAGE SECONDARY TO MENRATH'S ULCERS IN A CAT, TREATED WITH MULTIPLE BLOOD TRANSFUSIONS AND COBLATION TECHNIQUE: A CASE REPORT

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Background:
Menrath's ulcers are well-demarcated palatal erosions localized on the rostral hard palate caudomedial to the canine tooth. These lesions of the oral cavity, most often caused by compulsive licking are rarely reported in cats. However, they may lead to haemorrhagic shock due to massive oral bleeding.

Case presentation:
A 7-year-old male neutered domestic shorthair cat was presented with acute collapse and hematemesis. Clinical examination revealed hypothermia, bradycardia, pale mucous membranes, and heavy flea infestation. Presence of blood in the oral cavity was observed originating from two ulcerative lesions of the hard palate.

Complete blood cell count revealed severe normocytic normochromic highly regenerative anaemia, as well as mild eosinophilia and basophilia, suggestive of parasite infestation. Blood smear examination showed abnormalities compatible with iron deficiency. Serum iron concentration was low. Biochemistry profile revealed moderate hypoproteinemia. Coagulation profile showed mild hypofibrinogenemia but was otherwise unremarkable. Findings on abdominal ultrasound were suggestive of severe parasite infestation while upper gastrointestinal endoscopy did not reveal any abnormalities. We postulated that the flea infestation led to compulsive licking resulting in palatal erosions and severe oral haemorrhage. The underlying cause for the iron deficiency was unclear however chronic blood loss from Menrath's ulcers, flea infestation or intestinal parasite infestation was most likely.

The cat's cardiovascular status and haematocrit stabilized after multiple blood transfusions. The oral ulcers were treated with surgical debridement and use of Coblation ND technique. Additional treatment consisted of iron supplementation, broad-spectrum antibiotic therapy, antiparasitic treatment and esophagostomy tube placement.

Recovery was uneventful. At 3 months follow-up the ulcers had healed completely and the anaemia had resolved.

New information:
Ulcers of the hard palate can result in severe haemorrhage and have to be part of the differential diagnosis in cats with anaemia highlighting the importance of performing a thorough examination of the oral cavity. Surgical management with aggressive supportive therapy maybe be necessary, and underlying causes such as flea infestation have to be identified and treated concurrently.

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ENDOSCOPIC RETRIEVAL OF A METALLIC ZIPPER FOREIGN BODY ENTRAPPED WITHIN THE ESOPHAGEAL MUCOSA OF A DOG

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Background:
Foreign body ingestion is a common clinical problem in dogs, with esophageal foreign bodies presenting as acute emergencies. Metallic foreign bodies, like zippers, are rare, and their entrapment in the esophageal mucosa is even less common. Timely diagnosis and removal are critical to avoid complications like obstruction, perforation, and necrosis. Endoscopy is the gold standard for retrieval, with 88% of foreign bodies successfully removed endoscopically.

Case Presentation:
A 1-year-old male Staffordshire Bull Terrier presented with acute lethargy and trembling. Initial examination showed the dog was bright, alert, and responsive, with normal vital signs. Radiographs revealed a metallic foreign body lodged in the esophagus at the cardiac base. The primary veterinarian attempted to remove the object using a plastic tube, but this was unsuccessful. Endoscopy confirmed the foreign body was a zipper, zipped to one side of the esophageal mucosa. A basket catheter was used to engage the top of the zipper, enabling it to slide caudally and facilitating safe removal by unzipping, with minimal mucosal trauma. The dog was discharged with acetaminophen for pain management.

New/Unique Information:
This case is unique due to the uncommon nature of the foreign body and the unusual clinical signs. While typical signs of esophageal foreign bodies include hypersalivation, gagging, and regurgitation, this dog presented with lethargy and trembling, likely due to discomfort from mucosal entrapment. Although zipper entrapment in the esophagus is rare, delayed removal can lead to complications such as necrosis or perforation. One previous report describes a case of zipper entrapment where the foreign body was attached to two portions of the esophageal mucosa, requiring forceps for removal and subsequent gastrostomy. This case, however, demonstrates the first successful retrieval of a zipper from a single mucosal attachment using a basket catheter, underscoring the effectiveness of endoscopic techniques and the importance of timely intervention to prevent further complications.

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SUCCESSFUL MANAGEMENT OF SEVERE COAGULOPATHY IN A DOG FOLLOWING CERASTES GASPERETTII (ARABIAN HORNED VIPER) ENVENOMATION: FIRST CLINICAL CASE DESCRIPTION

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Background:
Cerastes gasperettii (C. gasperettii) is a venomous snake endemic to the Middle East. While envenomations have been reported, detailed clinical descriptions are lacking. This case report aims to describe the clinical presentation, diagnostic findings, and treatment of C. gasperettii envenomation in a dog.

Case Presentation:
A 5-year-old female dog presented with ptyalism and submandibular swelling following a witnessed C. gasperettii envenomation. Clinical examination revealed progressive lethargy and excessive bleeding at venipuncture sites. Laboratory evaluation identified severe coagulopathy including prolonged clotting times, hypofibrinogenemia, and thrombocytopenia observed in both conventional coagulation tests and thromboelastometry. Despite the unavailability of species-specific antivenom, the dog responded positively to supportive care. Treatment included fluid therapy and fresh frozen plasma, resulting in the normalization of coagulation parameters and full recovery without residual deficits

New/Unique Information:
This is the first documented case of a naturally occurring C. gasperettii envenomation in a dog. and the first to describe specific clinical findings in any species. The case highlights the potential for severe coagulopathy associated with C. gasperettii envenomations and underscores the critical role of timely supportive care, including hemostatic support, even when specific antivenom is unavailable. This report contributes to the limited clinical data on C. gasperettii envenomations and emphasizes the need for further research to better understand the clinical manifestations and optimize treatment strategies.

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CLINICAL USE OF VENO-VENOUS BYPASS FOR EXTENSIVE HEPATOCELLULAR CARCINOMA RESECTION IN A DOG: A CASE REPORT

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Background:
Hepatocellular carcinoma (HCC) is the most common primary liver tumor in dogs, with a favorable prognosis when fully resected. However, massive or poorly positioned HCC poses significant challenges due to hemorrhage risks and surgical complexity, especially when tumors involve critical vascular structures like the caudal vena cava.

In human medicine, veno-venous bypass (VVB) is widely used during hepatic surgeries to maintain hemodynamic stability, allowing continuous venous return and reducing intraoperative bleeding. In veterinary medicine, VVB has only been reported in experimental models, and its clinical application in dogs remains undocumented. Additionally, the Cavitron Ultrasonic Surgical Aspirator (CUSA), which uses ultrasonic waves to selectively fragment liver parenchyma while sparing vessels and bile ducts, enhances surgical precision and reduces blood loss.

This report describes the first clinical use of VVB in a canine patient undergoing extensive hepatic resection for HCC, demonstrating its potential in complex liver surgeries.

Case Presentation:
A 10-year-old spayed female Beagle presented with a 10 × 8 cm HCC involving the left medial, lateral, and quadrate lobes, compressing the left hepatic vein and displacing the caudal vena cava. Three-dimensional reconstruction confirmed these findings. Given the high hemorrhage risk, VVB was implemented. Blood was diverted from the caudal vena cava to the right atrium using a dialysis machine (300 mL/min) via central venous access in the right jugular and femoral veins.

Rumel tourniquets were applied to the caudal vena cava and portal vein for intermittent Pringle maneuvers. A 50-minute hepatectomy, including the affected lobes, was performed using CUSA. Minimal bleeding occurred, and no transfusions were required. Two hypotensive episodes resolved with crystalloid boluses.

Postoperatively, the dog developed acute pancreatitis on day 2, likely due to Pringle maneuvers, which was treated successfully. By day 10, the dog fully recovered, with normal appetite and resolved abdominal pain. Histopathology confirmed well-differentiated HCC with clean margins. The dog remains healthy at a two-year follow-up.

New/Unique Information:
This first clinical use of VVB in a dog highlights its potential to improve outcomes in complex hepatic surgeries. Concurrent portal vein bypass may mitigate pancreatic hypoperfusion and reduce complications in similar cases.

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AN UNUSUAL CASE OF MIGRATION OF AN INGESTED SEWING NEEDLE FOREIGN BODY THROUGH THE RECTUM AND INTO THE HINDLIMB OF A YOUNG CAT

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Background:
Gastrointestinal sewing needle foreign bodies in cats are well reported in the veterinary literature. Case reports predominantly document perforation and migration from the upper gastrointestinal tract. This report highlights an unusual case of migration of an ingested sewing needle from the lower gastrointestinal tract.

Case presentation:
A 6 month old, male entire DSH cat presented with a two day history of vomiting, lethargy and straining to defecate. Physical examination showed moderate dehydration, pyrexia and caudal abdominal pain. Bloods showed mild neutropenia and hypochloraemia. Abdominal ultrasound showed a small volume of free fluid caudal abdomen. Sedated abdominal radiographs showed a sewing needle foreign body perpendicular to the rectum, suspected to be migrating through the pelvic obturator foramen into the left hind limb. On rectal palpation the needle was extraluminal.

The patient was stabilised with intravenous fluids and intravenous maropitant and cefuroxime. An exploratory laparotomy was undertaken, however the needle was not able to be retrieved. An inflamed mucosal lesion on the colon was omentalized and the abdomen flushed. Radiographs showed further migration of the needle down the limb. The needle was later removed surgically via the hindlimb. The patient made a full recovery.

New/ Unique information:
It is widely accepted that needle foreign bodies in the upper gastrointestinal tract should be removed endoscopically to prevent perforation. Opinions are divided as to the best approach to intestinal needle foreign bodies beyond the scope of endoscopy. Human medicine guidelines recommend repeated radiography and monitoring as it is accepted that a needle reaching the stomach will likely pass without issue. Surgical removal of lower intestinal needle foreign bodies can be controversial due to potential complications of enterotomy.

This case is an example of large intestinal perforation and subsequent peritonitis which would support removal of needle foreign bodies in the lower gastrointestinal tract via enterotomy if identified prior to perforation. In this case patient size in relation to foreign body was likely a contributing factor to the subsequent perforation. Further prospective studies comparing surgical and conservative management of lower gastrointestinal tract needle foreign bodies in cats are required to guide management of these cases.

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TRACHEAL INTUSSUSCEPTION IN AN 11-YEAR-OLD YORKSHIRE TERRIER: DIAGNOSIS AND SURGICAL MANAGEMENT

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Background:
Tracheal intussusception is an extremely rare cause of respiratory distress in dogs and has not been documented in humans. To date, only two cases of tracheal intussusception have been reported in the veterinary literature, both in dogs. One followed trauma, and the other had an unknown etiology. Of these, one case was managed conservatively, while the other was treated surgically with segmental resection and anastomosis.

Case Presentation:
An 11-year-old male Yorkshire Terrier presented with a chronic history of wheezing and an acute onset of dyspnea. Clinical examination revealed a cyanotic tongue and marked inspiratory effort. Hematology indicated a chronic inflammatory leukogram, while biochemistry showed mild elevations in blood urea nitrogen, liver enzymes, and cholesterol. Echocardiography revealed no cardiac abnormalities and no changes compatible with pulmonary hypertension. A CT scan of the neck and thorax showed a small 'step' in the tracheal luminal diameter at the C5-C6 level and mild dorsoventral narrowing at the thoracic inlet, raising suspicion for tracheal intussusception. A subsequent tracheoscopy confirmed the diagnosis, revealing a focal circular narrowing of the trachea. Surgery was offered to address the tracheal defect. A midline approach to the ventral neck was used. Fibrosis surrounding the trachea at the level of the intussusception was surgically dissected, and the intussuscepted tracheal ring was reduced. A sterile, medical-grade, polyvinyl chloride suction tubing splint was sutured to the ventral trachea to stabilize the airway and prevent recurrence. Post-operative bronchoscopy confirmed successful reduction and proper placement of sutures. The patient was discharged 96 hours after surgery with oral meloxicam, amoxicillin-clavulanate, and trazodone for two weeks. Ten months post-surgery, the dog was still alive.

New/Unique Information:
This case underscores the importance of considering tracheal intussusception as a differential diagnosis in dogs presenting with dyspnea. The use of advanced diagnostic imaging, combined with a novel surgical technique involving a rubber splint, offers valuable insights into the management and successful treatment of this rare condition.

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HYPERFIBRINOLYSIS, HYPOCOAGULABILITY AND HEMOABDOMEN SECONDARY TO LIVER LOBE TORSION IN A PUPPY

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Background:
Liver lobe torsion has been reported in dogs of diverse breeds and ages. However, coagulation abnormalities associated with this condition have yet to be described.

Case presentation:
A 6-month-old intact female Bernese Mountain dog, was presented for acute lethargy and vomiting, with no history of trauma or toxic exposure. On physical examination, pale mucous membranes and tachycardia were noted. She was diagnosed with hemoabdomen (PCV effusion 33%), and was referred to the veterinary teaching hospital's emergency service. On presentation, generalized weakness, pale mucous membranes, prolonged CRT, tachycardia (120 bpm) and tachypnea (60 brpm) were noted. Complete blood count (CBC) showed mild hypochromic normocytic regenerative anemia (Hct 33%) and mild neutrophilic (9920/uL) and lymphocytic (4210/uL) leukocytosis (15 030/uL). Serum biochemistry revealed marked elevation in ALT (3233 U/L) and panhypoproteinemia (albumin 22 g/L, globulins 15 g/L). A bed-side viscoelastic coagulation monitor (VCM) showed hyperfibrinolysis,(LI45 89%, RI 98-100) which was treated with tranexamic acid (10 mg/kg IV q8h). Serial VCM showed resolution of hyperfibrinolysis (LI45 100%) but progressive hypocoagulability (CFT 793 s, RI 104-266 s; Alpha 21º, RI 43-64; MCF 17, RI 29-44). An abdominal CT revealed left lateral liver lobe torsion with rupture of the hepatic capsule. Treatments included fluid resuscitation with crystalloids, blood product transfusions (20 mL/kg packed RBC, 10 mL/kg fresh frozen plasma) and autotransfusion (17.5 mL/kg whole blood, 8.7 mL/kg packed RBC). The patient underwent exploratory laparotomy and left lateral liver lobectomy. Histopathology of the liver lobe was consistent with infarction (subacute diffuse hepatic necrosis with severe congestion). Post-operatively, there was significant improvement in clinical signs and normalization of the VCM profile (CFT 187 s; Alpha 53º; MCF 37). However, the dog developed aspiration pneumonia, which prompted the initiation of antibiotic therapy (amoxicillin-clavulanate 15 mg/kg PO q12h) The dog was discharged 5 days after presentation. At 10-day follow-up, the dog's clinical signs had completely resolved. On recheck CBC and biochemistry, anemia had resolved (Hct 42%) and ALT elevation had improved (639 U/L).

New/Unique Information:
This is the first case describing hyperfibrinolysis and hypocoagulability in a dog with hemoabdomen secondary to liver lobe torsion.

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SEVERE HYPERCALCEMIA IN A DOG WITH FULMINANT IATROGENIC CALCINOSIS CUTIS

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Background:
This case report presents a fatal instance of severe iatrogenic calcinosis cutis, which developed sever hypercalcemia during treatment with topical therapy with dimethyl sulfoxide (DMSO) and tapering of prednisolone.

Case presentation:
A 6-year-old male pug diagnosed with canine atopic dermatitis was presented with severe calcinosis cutis, attributed to prolonged prednisolone therapy. The dog received 1 mg/kg/day prednisolone, 8 mg/kg/day cyclosporine, and 10 mg/kg/day clindamycin. Topical treatment consisted of chlorhexidine shampoo every other day and 50% DMSO-spray once daily. The dog developed temporalis muscle atrophy, palpably enlarged liver, truncal obesity, and extensive purulent calcinosis cutis affecting 40-50% of the skin, complicated by secondary bacterial infections. Prednisolone was gradually tapered over the following weeks.

Twenty-three days later, the dog was presented to the emergency department with anorexia and lethargy. Blood gas analysis revealed severe ionized hypercalcemia (iCa) (1.88mmol/L). The dog was treated with pamidronate (1 mg/kg i.v.) and isotonic saline infusion (3 ml/kg/h). Nevertheless, iCa levels increased to 2.0 mmol/L the following day, prompting the administration of a furosemide bolus (2 mg/kg i.v.) and an increase in fluid rate to 4 ml/kg/h. Over the next hours severe deterioration occurred with progressive hypovolemia and systemic hypotension (60-80 mmHg), iCa further increased to 2.14 mmol/L and severe hypernatremia (187mmol/L) occurred. Free water deficit was calculated, and the following treatment protocol was initiated: bolus of buffered, isotonic balanced electrolyte solution (20 ml/kg), followed by a constant rate infusion (CRI) of a crystalloid solution with 5% glucose and reduced sodium content (53.7mmol/L) (8.5 ml/kg/h), 5% glucose solution (2.5 ml/kg/h), furosemide CRI (0.25 mg/kg/h), noradrenaline-CRI (0.1 mg/kg/min), and calcitonin (50 IU s.c.).

Eighteen hours later the sodium (154mmol/L) and calcium levels (1.69mmol/L) had decreased. The infusion was changed to an isotonic balanced electrolyte solution. Despite normalization of electrolytes, the dog developed progressive respiratory distress, leading to respiratory arrest. Postmortem computed tomography revealed extensive metastatic mineralization in various tissues.

New/Unique Information:
The application of DMSO in fulminant calcinosis cutis cases may result in life-threatening hypercalcemia, necessitating close monitoring of calcium levels. Elevated ionized calcium in those patients may require prompt and intensive countermeasures to mitigate potential complications.

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DELAYED LEUKOENCEPHALOMALACIA FOLLOWING CARBON MONOXIDE TOXICOSIS IN A DOG

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Background:
Carbon monoxide (CO) toxicosis caused by inhalation of incomplete hydrocarbon combustion products leads to the formation of carboxyhemoglobin causing hypoxemia and subsequent tissue hypoxia. Delayed post-hypoxic leukoencephalopathy (DPHL), characterized by periventricular white matter damage is well-documented in human medicine but underreported in veterinary literature. This case report describes the clinical progression, neuroimaging findings and histopathological changes in a dog with CO toxicosis shedding light on this rare condition in veterinary practice.

Case Presentation:
A 2-year-old intact male American Staffordshire Terrier was presented with acute respiratory distress and altered mentation following CO inhalation from a butane heater.

On admission, the patient had cyanotic mucous membranes, expiratory dyspnea and a temperature of 38.8°C. Neurologically, the patient was stuporous and non-ambulatory with cranial and spinal reflexes but absent bilateral menace response. Bloodwork revealed neutrophilic leukocytosis, elevated alkaline phosphatase and transient hyperlactatemia. Carboxyhemoglobin levels could not be measured.

Initial stabilization included transtracheal intubation and mechanical ventilation using a volume-controlled mode with an initial FiO2 of 100%. The patient's mental state and respiratory signs allowed extubation after two hours. After extubation, the patient exhibited disorientation, vocalization, and agitation. He was administered a continuous infusion of dexmedetomidine and butorphanol during the first 24 hours. Neurological monitoring using the Glasgow Coma Scale (GCS) revealed a score of 8 on day one. On days two and three, the patient showed slight neurological improvement, with GCS scores of 13 and required boluses of dexmedetomidine for sedation at specific intervals. However, on day four, the patient’s condition worsened dramatically. Neurological deterioration was confirmed with a GCS score of 9 and a Magnetic Resonance Imaging (MRI) revealed bilateral symmetrical T2W/FLAIR hyperintensities in the periventricular white matter consistent with leukoencephalopathy.

Based on the clinical deterioration and MRI findings, euthanasia was performed with the owner's consent. Postmortem examination confirmed axonal degeneration, gliosis, endothelial hyperplasia and cortical laminar necrosis.

New/Unique information:
This case highlights the clinical variability of delayed post-hypoxic leukoencephalopathy in CO toxicosis,

emphasizing the value of advanced imaging in similar cases. Despite prompt oxygen therapy and sedation, cerebral damage progressed, underscoring the unpredictable nature of CO toxicosis.

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CONGENITAL PERITONEOPERICARDIAL DIAPHRAGMATIC HERNIA (PPDH) IN A DOG WITH A THORACIC CAVITY WALL, PERICARDIUM, AND DUCTAL PLATE MALFORMATION

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Background:
This case report describes the clinical presentation, investigation findings, interventions, and outcome of a peritoneopericardial diaphragmatic hernia (PPDH) in a dog with a thoracic cavity wall, pericardium, and ductal plate malformation

Case presentation:
A one-and-a-half-year-old female neutered Labradoodle was presented to a referral practice for acute distension of the abdomen and vomiting. Tachycardia with a heart rate of 150 beats per minute, intermittent panting, borborygmi noted on chest auscultation, and distended and tense cranial abdomen were identified, while the rest of the physical examination was unremarkable. The investigations showed mild haemoconcentration with a PCV of 60%, total solids 85 g/L, marked metabolic alkalosis (pH 7.594 [RI 7.35-7.45], pCO2 38.2 mmHg [RI 26-36], [BE 13.7 mmol/L [RI -8--2]) with moderate hypochloremia (98 mmol/L [116-126]), moderate hypokalemia (2.6 mmol/L [RI 3.5-4.8]), mild increase in serum ALT (142 U/L, RI 10 – 125), mild lymphocytosis, and mild monocytosis. Thoracic and abdominal X-rays confirmed herniation of gas-filled intestinal loops into the chest, enlarged cardiac silhouette, elevated trachea and caudal vena cava, dorsal lung displacement/ retraction from left to right, cranial displacement of abdominal viscera, absent sternal segments, and cervical ribs. The initial therapy consisted of fluid resuscitation, potassium supplementation, antiemetics, and analgesia administration. The blood tests were rechecked 14 hours later and the results were unremarkable. A nasogastric tube was placed to remove 350 ml of gastric residual. Surgery was performed to reposition the organs and suture the diaphragm. During this procedure, an incomplete pericardial sack was noted, therefore sternotomy was also effectuated to detach the pericardium edges from the loose mediastinum. A liver biopsy was taken and the results revealed a ductal plate malformation, which was suspected due to the previously reported association with the PPDH. The surgery was unremarkable and the dog responded well to antiemetics, analgesia, and intravenous fluids, therefore it was discharged 4 days later with no complications. Three months later, the dog was reported back to its normal self.

New/Unique information:
To the author's knowledge, this is the first case report of a congenital PPDH in a dog with all the reported changes.

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RIGHT PULMONARY ARTERIAL THROMBOEMBOLISM DURING TRANEXAMIC ACID THERAPY FOR SEVERE HYPERFIBRINOLYSIS IN A DOG WITH ANGIOSTRONGYLOSIS

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Background:
Treatment with Tranexamic acid (TXA), an antifibrinolytic, has been described for hyperfibrinolysis associated with angiostrongylosis. In human medicine, TXA treatment is associated with increased risk of thromboembolic disease. This case describes right pulmonary arterial thromboembolism during TXA therapy for hyperfibrinolysis in a dog with angiostrongylosis.

Case Presentation:
A 3-year-old male neutered Beagle presented for acute bilateral miosis, right-sided circling and head turn. Blood work demonstrated eosinophilia (1,470/µL [6 – 1,230]), thrombocytopenia (106,000/µL [148,000-484,00]), and elevated C-reactive protein concentrations (52mg/L [<10]). The dog developed dyspnea (SpO2: 88% [>95%]), obtundation, and seizures overnight. Point-of-care ultrasound (POCUS) identified diffuse B-lines and shred signs. A positive antigen test diagnosed angiostrongylosis and treatment with oxygen (nasal canula 150ml/kg/min), levetiracetam (30mg/kg TID), topical imidacloprid (10mg/kg)/moxidectin (2.5mg/kg), prednisolone (0.5mg/kg BID), and TXA (10mg/kg TID) was initiated.

Viscoelastography (VCM-Vet) was performed for suspected coagulopathy, and demonstrated hypocoagulability (Clot Formation Time (CFT) 45.8m [1.7-4.4]) and hyperfibrinolysis (Lysis Index at 30 minutes (LI30): 61% - Lysis Index at 45 minutes (LI45) : 59% [99-100]). Fresh frozen plasma (10ml/kg) normalized CFT but hyperfibrinolysis (LI 30: 37%, LI 45: 37%) persisted. A constant rate infusion (CRI) of TXA (10-20mg/kg/h) normalized the viscoelastographic profile within 8 hours.

After gradual improvement the dog experienced a syncope with sudden cyanosis on day 12. POCUS identified a subjectively increased number of caudodorsal B-lines and high-flow oxygen support (1l/kg/min) was initiated. Thoracic radiography suggested pulmonary thromboembolism and echocardiography identified a right pulmonary artery thromboembolism and right heart modifications compatible with pressure overload. Viscoelastography indicated a hypercoagulable state (Alpha 69° [42-64], Maximum Clot Firmness (MCF) 48 [29-44]), leading to discontinuation of TXA and administration of enoxaparin (0.8 mg/kg SC QID) and clopidogrel (2 mg/kg SID).

By day 20, the arterial thrombus resolved, but right heart changes persisted. The dog was discharged on clopidogrel (2 mg/kg SID). All treatments were discontinued 1 week after, with the resolution of echocardiographic changes.

New Information:
Right pulmonary arterial thromboembolism, observed in a dog receiving TXA for hyperfibrinolysis secondary to angiostrongylosis, was successfully treated with enoxaparin and clopidogrel. Whether TXA is associated with increased risk of thromboembolism remains to be investigated.

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UNINTENTIONAL INTRAVENOUS ADMINISTRATION OF ENTERAL NUTRITION IN A DOG: A CASE REPORT ON MEDICAL ERROR AND RECOVERY

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Background:
Medical errors, the majority being medication errors, occur in approximately 0.5% of veterinary consultations. Accidental intravenous administration of enteral nutrition in human medicine leads to sepsis, multiorgan dysfunction, coagulation disorders, long-term neurological deficits, and often death. This case describes a medical error with a favorable outcome.

Case Presentation:
A 1-year-old spayed female Australian Shepherd presented in status epilepticus, already treated with phenobarbital (2.5mg/kg BID) and continuous rate infusion (CRI) of midazolam (0.2mg/kg/h). Initial treatment included hypertonic saline (4mL/kg), levetiracetam (60mg/kg, then 30mg/kg TID), midazolam CRI (0.2mg/kg/h), phenobarbital (5mg/kg, then 2.5mg/kg BID), and maropitant (1mg/kg SID). Blood work, magnetic Resonance Imaging and cerebrospinal fluid analysis were unremarkable. The dog developed hypoventilation (PvCO2: 80mmHg), requiring mechanical ventilation (MV). Nystagmus, tachycardia, and transient hyperthermia initially persisted during MV, and were managed with ketamine (5mg/kg) and propofol CRI (0.2mg/kg/min). Metoclopramide CRI (0,2mg/kg/day) and enteral nutrition (20% Resting Energy Requirement [RER]) via a nasogastric tube were initiated. The dog recovered and was weaned off MV, propofol, and midazolam by day 4, with enteral nutrition gradually increased to 150% of RER. On day 8, approximately 50mL (estimated from time of syringe connection to onset of clinical deterioration) of low-fat gastrointestinal enteral nutrition was inadvertently administered intravenously. The dog became hyperthermic (40.1°C), lethargic, laterally recumbent, tachycardic (140 beats per minute), with a systolic blood pressure of 90mmHg. Amoxicillin-clavulanic acid (20 mg/kg QID) and marbofloxacin (4 mg/kg SID) were administered. Daily performed blood work, urinalysis, vascular and cardiac ultrasounds, revealed no significant abnormalities. Urine and blood culture were negative. Hyperthermia resolved after 24 hours, although the dog remained obtunded in lateral recumbency until day 13, walked with mild ataxia on day 16, and fully recovered by day 20, when she was discharged. Antibiotics were discontinued two weeks later, when clinical examination and blood work had remained unremarkable.

New information:
Catheter misconnections are rarely reported in veterinary medicine. We describe the clinical consequences, medical management, and outcome after inadvertent intravenous food administration to a dog. The low-fat content and hydrolyzed proteins of the administered food may have reduced the likelihood of fat embolism, infection, and hypersensitivity reactions.

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PERICARDIAL HEMORRHAGE, TAMPONADE AND ARRHYTHMIA IN A DOG AFTER CARDIOPULMONARY RESUSCITATION

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Background:
Point-of-Care ultrasound (POCUS) in veterinary practice rapidly identifies life-threatening conditions. We describe a dog with pericardial hemorrhage and tamponade identified on POCUS, as well as an arrythmia, after cardiopulmonary resuscitation (CPR) for cardiopulmonary arrest (CPA).

Case presentation:
A 1-year-old female spayed crossbreed dog was presented one week after elective ovariohysterectomy for hyperthermia and hyporexia. At registration the dog collapsed and was bradycardic, hypoglycemic (2mmol/L) and hyperlactatemic (15mmol/L) on triage. The dog experienced CPA before administration of a bolus of glucose and isotonic crystalloids. CPR was rapidly initiated, together with administration of glucose, crystalloids and adrenaline (an erroneous overdose of 0.5mg/kg), leading to return of spontaneous circulation (ROSC) within 5 minutes with sinus tachycardia (180bpm).

Muffled heart sounds were identified after ROSC, and thoracic POCUS was performed to rule out pleural/pericardial effusion. Pericardial effusion, tamponade and a large pericardial thrombus were identified. Pericardiocentesis was performed under electrocardiographic monitoring. Post-centesis the dog was fine with a rapid sinus rhythm (140bpm). Complementary examinations, including general blood work, a coagulation panel, a blood culture and abdominal ultrasound failed to identify an underlying etiology of CPA. The patient was hospitalized and received intravenous glucose (5%, 2mL/kg/h), amoxicillin clavulanic acid (20mg/kg TID), buprenorphine (10µg/kg TID), maropitant (1mg/kg SID), metoclopramide (0,5mg/kg TID) and a nasogastric feeding tube. The patient recovered uneventfully, regaining appetite after 1 day. By day 3 the pericardial effusion and thrombus had resolved on echocardiography. An electrocardiogram identified an atrioventricular block grade II Mobitz type I and the dog was discharged with amoxicillin clavulanic acid (22 mg/kg BID) and theophylline (9 mg/kg BID). On day 9 the dog remained clinically stable without reported hyperthermia or arrhythmia. Theophylline was discontinued 1 week thereafter.

New/Unique information:
POCUS is performed in human CPR, diagnosing tamponade, hypovolemia, pulmonary embolism, or assessing cardiac wall motion, which correlates with survival. Our case displayed a pericardial thrombus and tamponade. Pericardial thrombi are reported secondary to trauma (e.g. atrial rupture, invasive cardiac procedures) . As the patient had not experienced trauma or invasive cardiac procedure before CPR, the hemopericardium, thrombus and arrhythmia are speculated to have been the consequence of CPR.

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DROPPING HEAD SYNDROME (DHS) AS AN ATYPICAL MANIFESTATION OF PITUITARY MACROADENOMA AND SECONDARY MULTIENDOCRINOPATHY IN A DOG

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Background:
DHS is a rarely reported pathological entity in veterinary medicine, characterized by severe weakness of the cervical paravertebral extensor musculature [1,2]. This condition has been associated with muscular, neurological, or neuromuscular disorders [1,3,4]. In human medicine, its association with myopathies secondary to hypothyroidism or hyperadrenocorticism has been proposed; however, this has not been reported in veterinary [5]. This case report describes the main findings of this disorder in a dog, presumably associated with a polyendocrinopathy.

Case presentation:
A 6-year-old mixed-breed female dog was brought to the clinic with a history of apathy, weakness, hyporexia, polyuria/polydipsia, and marked ventroflexion of the neck. The general physical examination revealed moderate dehydration (5%), fever (40.7°C), and tachycardia (140 bpm), with a normal neurological examination and no other relevant clinical findings. The complete blood count showed lymphopenia (0.3 K/µL, Range: 1.05-5.10), while serum biochemistry indicated hypokalemia (2.8 mmol/L, Range: 3.5-5.8), ALT (454 U/L, Range: 10-125), ALKP (395 U/L, Range: 23-212), bilirubin (3.9 mg/dL, Range: 0-0.9), and creatine-kinase (518 U/L, Range: 10-200). The urine culture was negative. Hormonal profile revealed: basal cortisol 27.7 µg/dL (Range: 0.5-5.5), ACTH 583 pg/mL (Range: 9-67.7), renin 3.87 ng/mL/hour (Range: 0.3-2.6), aldosterone 54.15 pg/mL (Range: 15-102), thyroxine <0.500 µg/dL (Range: 1.3-2.9), and TSH 0.090 ng/mL (Range: 0-0.5). Thiamine deficiency (44 µg/dL, Range: 30-95) and infectious diseases were excluded using rapid tests (dirofilariasis, Lyme disease, ehrlichiosis, anaplasmosis, and leptospirosis). The initial hydroelectrolytic treatment resulted in partial improvement, with resolution of generalized weakness but persistent neck ventroflexion. MRI and CT revealed a pituitary macroadenoma and increased signal in the cervical and frontal paravertebral musculature. After 48 hours and clinical deterioration, owners opted for euthanasia. Necropsy confirmed a functional corticotropic adenoma with apoplexy, cortical hyperplasia, and nonspecific cervical myopathy with muscle fiber swelling.

New/Unique information:
This case describes an unusual presentation of DHS in veterinary, presumably linked to a polyendocrinopathy after ruling out other causes. It emphasizes the need to assess hormonal function in emergency patients with suspected myopathy, even if limited to the neck. To the authors' knowledge, this is the first reported of DHS with a potential hormonal cause in veterinary.

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URETHRAL OBSTRUCTION CAUSED BY OBSTIPATION IN CATS: A RETROSPECTIVE CASE SERIES

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Background:
Urethral obstruction (UO) is a common and potentially life-threatening condition in cats, typically associated with urolithiasis or idiopathic cystitis. However, the role of constipation or obstipation as a contributing factor is mostly unknown. This retrospective case series describes the clinical features, diagnostic findings, treatment, and outcomes of nine cats with suspected UO secondary to obstipation.

Case-series presentation:
The cohort included nine cats [7 males (6 castrated, 1 intact) and 2 spayed females], with a median age of 9 years (range: 0.125–14 years), primarily mixed breed. Three cats presented with classic UO signs, four with constipation later diagnosed with UO, and two with absence of both urination and defecation. Median creatinine was 0.221 mmol/L (range: 0.124–2.095 mmol/L). Obstipation was diagnosed at presentation in 7/9 cats and within 3–8 days in the remainder. Imaging (radiographs in all, ultrasonography in 4/9, urethrogram in 1/9) confirmed colonic dilation and fecal impaction, with no evidence of uroliths or other causes of UO.

Urethral catheterization was required in 8/9 cats and maintained until obstipation resolved, typically within 24–48 hours. Catheters were removed at the time of enema administration. Enema followed by laxative therapy successfully resolved UO in all cats, with spontaneous urination restored and no need for further urinary intervention. Post-treatment creatinine levels decreased or remained stable (median: 0.115 mmol/L, range: 0.053–0.283 mmol/L). No recurrence of UO was observed during follow-up (median 37.5 days, range 14–120 days).

New/Unique Information:
This case series highlights the importance of considering obstipation as a potential cause of UO. Abdominal palpation imaging should be integral to the diagnostic workup in obstructed cats. Prompt identification and treatment of constipation may resolve secondary UO, improve outcomes, and reduce recurrence. Urination in cats with megacolon or obstipation should be closely monitored, as partial or complete UO may develop. Although obstipation was likely the cause in these cases, incidental association cannot be fully excluded.

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SUCCESSFUL SURGICAL TREATMENT OF A SEPTIC PYOTHORAX DUE TO PNEUMONITIS DURING A DIROFILARIASIS INFECTION IN A DOG

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Background:
Dirofilaria immitis is a vector-borne nematode becoming increasingly prevalent in northern Europe due to the rising importation of pets. Adult parasites primarily inhabit the pulmonary artery and right ventricular outflow tract, leading to pulmonary hypertension and respiratory distress.

Case presentation:
A five-year-old female neutered Cavalier King Charles Spaniel, imported from Hungary seven months prior, was presented with exercise intolerance. Echocardiography showed pulmonary hypertension and moderate heartworm infestation in the pulmonary artery, with one worm extending toward the tricuspid valve. The dog received its first melarsormine injection following the American Heartworm Society guidelines (2.5mg/kg into lumbar muscles), along with additional therapy (prednisolone 0.5mg/kg PO q12h for three days, pimobendan 0.23mg/kg PO q12h, enoxaparin 1mg/kg SQ q8h).

Six weeks later, the dog was presented as an emergency with moderate tachypnea. Thoracic radiographs revealed pleural effusion, and 100 ml of purulent septic effusion was evacuated. Computed tomography showed severe bronchopneumopathy, infectious consolidation of the left cranial lung lobe and thrombosis in the pulmonary arteries. A partial lobectomy was performed, and a chest tube was placed. During surgery, a dead adult worm was found in the thoracic cavity. Histopathology confirmed hemorrhagic necrotizing pneumonia with Dirofilaria immitis remnants.

Postoperatively, the dog developed anemia and hypoalbuminemia, requiring a whole blood transfusion as well as frozen plasma. Arterial blood gas analysis was measured twice daily to monitor oxygenation, and thoracic lavage was performed over six days. The dog was treated with amoxicillin/clavulanic acid (20mg/kg IV q8h) and marbofloxacin (2mg/kg IV q12h). Initially, a continuous rate infusion of fentanyl (2.5µg/kg/h) and ketamine (0.3mg/kg/h) provided analgesia. Moderate pulmonary hypertension was managed with sildenafil (1.2mg/kg PO q8h), and thrombosis was addressed with enoxaparin (1mg/kg SQ q8h). After ten days of intensive care, the dog was discharged in good general health. Follow-up examinations one and four weeks later revealed clinical improvement, with normalizing inflammatory markers. The macrofilaricidal therapy with melarsormine was continued.

New/unique information:
This is the first veterinary case report describing pyothorax secondary to Dirofilaria immitis infection and its successful treatment. Previously, only one case report has been published describing pneumothorax associated with dirofilariasis.

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"EYE OF THE BLAST": OCULAR TRAUMA FROM EXPLOSIVE AMMUNITION IN FOUR DOGS

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Background:
Explosive ammunition releases energy that generates blast waves, resulting in primary injuries from the kinetic force of the blast wave and secondary injuries from penetrating fragments. Released fragments may include components of the explosive device, as well as environmental debris (e.g., sand and pebbles). Blast injuries related to explosive ammunition are rare in small animal practice and there is a paucity of literature describing the specific injury patterns or providing evidence-based treatment recommendations.

Case presentation:
Between October 2023 and January 2024, four dogs—three military working dogs and one companion dog—were treated for ocular blast injuries. Three dogs sustained injuries from hand grenade explosions, while a mortar explosion injured one. The blast trauma resulted in a wide range of injuries. Two dogs underwent full-body computed tomography (CT) scans, while the other two had thoracic radiographs. Ocular ultrasonography was performed in two cases. All four dogs sustained ocular injuries, with intraocular foreign fragments detected in each case. Additionally, three dogs had shrapnel embedded in vital organs, and two suffered traumatic head injuries. One dog presented with a complete globe avulsion in one eye and a corneal laceration in the contralateral eye; the laceration was managed with a third eyelid flap. The second dog exhibited bilateral corneal lacerations; with one eye treated using a third eyelid flap. The third dog sustained a corneal laceration in one eye and received ultraviolet-C (UVC) light treatment followed by keratoplasty. The fourth dog presented with a corneal laceration in one eye, which was managed with a conjunctival flap. All four dogs survived to discharge; however, the dog with the globe avulsion lost vision in both eyes, and two dogs developed unilateral blindness.

New/Unique Information: This is the first documented case series describing ocular blast injuries resulting from explosive ammunition in dogs. The findings underscore the critical importance of early ophthalmologic evaluation and timely intervention to optimize clinical outcomes.

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SEVERE MUSHROOM INTOXICATION IN A YOUNG SIBERIAN HUSKY CAUSED BY INOCYBE SP. AND HEBELOMA CRUSTULINIFORME INGESTION

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Background:
Mushroom ingestion is relatively common in juvenile dogs, however there are only a few reports with confirmed species (Seljetun et Krogh 2017, Seljetun et Kragstad 2023). The genus Inocybe contains muscarine causing systemic parasympathomimethic crisis (Bejamin 1995). The genus Hebeloma causes gastrointestinal irritation (Puschner 2011). Majority of dogs ingest mushrooms in gardens and the clinical signs develop within two hours (Seljetun et Kragstad 2023).

Case presentation:
One year old female Siberian Husky was found collapsed 60 minutes after the evening feed. Salivation, urination, and inability to walk were the early signs.

On physical exam the dog was obtunded with nonambulatory tetraparesis, locked jaw and hypersalivation. Mucous membranes were hyperemic with capillary refill time of one second. Cardiothoracic auscultation revealed a heart rate of 162 beats per minute with no murmur or arrythmia. The respiratory rate was 10 breaths per minute with a very shallow breathing pattern. Abdomen was tense and rectal temperature 38.2°C.

Clinically significant findings were hypertension (systolic blood pressure 167mmHg) and hypoventilation (oxygen saturation SpO_2 92%, hypercapnia and respiratory acidosis). Point of care abdominal ultrasound and radiographs revealed distended stomach.

Suspicion of intoxication or neurological disorder was raised.

The treatment was initiated with oxygen and fluid therapy. Intravenous lipid emulsion was administered. Atropine was not prescribed due to persistent tachycardia and hypertension. Induction of emesis with apomorphine intramuscularly and ropinirol transconjuctivally were unsuccessful. Gastric lavage under general anesthesia was therefore performed. Large amount of mushrooms were detected in the stomach content. Charcoal was administered afterwards.

The dog recovered gradually within 12 hours at intensive care unit. The only detected complication was macroscopic hematuria lasting 24 hours.

Stomach content was assessed by a mycologist. Genera Inocybe sp. and Hebeloma crustuliniforme were identified.

New information:
To the authors' knowledge this is the first reported case of a dog with mushroom intoxication, where two different mushrooms were ingested. This patient was presented with tachycardia and hypertension that persisted despite adequate fluid therapy and analgesia, and do not correlate with muscarine signs in the literature (Waser 1961). The novel sign was hematuria, which could have resulted from muscarine excretion via kidneys.

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UNRECOGNIZED BITE WOUND LEADING TO CHRONIC INTRACRANIAL EMPYEMA IN A STRAY KITTEN

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Background:
Intracranial empyema or abscessation in cats are rare conditions requiring medical and/or surgical intervention. Stray and shelter cats often have incomplete medical histories, complicating diagnosis and treatment. This case illustrates the challenges of managing a stray kitten with neurologic episodes caused by an undiagnosed intracranial infection due to an old bite wound.

Case Presentation:
A 5-month-old male stray kitten was admitted with a history of recurrent epileptic seizures of varying frequency and visual impairment over a one-month period. The referring veterinarian had previously noted a scab on its head, initially thought to be seizure-related trauma.

On admission, the general clinical examination was unremarkable. The head wound had healed completely. The neurologic examination revealed a severely obtunded mentation and bilaterally absent menace response. Bloodwork revealed persistent leukocytosis and mildly elevated CPK activity. Serum toxoplasma IgG antibodies were elevated, while other infectious disease panels (FIV, FeLV, FIP, panleukopenia) were negative. As MRI was not available in-house, a full-body CT scan was performed, revealing chronic remodeling of left neurocranial fractures, focal brain herniation, left intra-axial hemorrhagic lesion and severe left-sided cerebral edema, likely from past trauma. Intracranial empyema secondary to a bite wound was suspected.

Therapy was started with ampicillin, and after intracranial infection and edema were confirmed, ceftriaxone and dexamethasone were introduced. During hospitalization, the kitten developed cluster seizures, initially managed with midazolam boluses and a phenobarbital loading dose, followed by phenobarbital maintenance therapy and continuous midazolam infusion.

Due to neurological deterioration, left rostrotentorial craniectomy was performed. Surgical exploration revealed lacerated dura with gas bubbles underneath and a hematoma with yellow granulomatous inclusions at the skull base. Bacteriologic and mycologic examinations of the dura mater and brain abscess samples were negative.

The kitten was discharged two days post-surgery with normalized leukocyte counts and no recurrent seizures. Two brief seizures occurred over the next month. Follow-up information was not available due to international adoption.

New/Unique Information:
This case highlights the complexities of managing a shelter cat with incomplete history and seizures from an undiagnosed old bite wound leading to an intracranial infection. Medical and surgical treatment resulted in a positive short-term outcome.

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HEMOABDOMEN SECONDARY TO AN INTESTINAL MURAL HEMATOMA IN A SPHYNX CAT WITH HEMOPHILIA

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Introduction:
Hemophilia in cats is a rare, inherited coagulation disorder characterized by a deficiency in clotting factors VIII or IX. While mild to moderate deficiencies often result in subcutaneous hematoma, more severe complications, such as internal bleeding events, are less common. This case report aims to describe a feline patient with mild hemophilia who developed a hemoabdomen secondary to an intestinal intramural hematoma, highlighting the diagnostic and therapeutic challenges associated with this rare clinical presentation.

Case presentation:
A 3-year-old neutered male Sphynx cat was presented with a two-month history of chronic, non-hemorrhagic diarrhea, vomiting, anorexia, and lethargy. On physical examination, the cat showed signs of hypovolemic shock, including pale mucous membranes, bradycardia, hypotension, and hypothermia. Diagnostic tests revealed a normocytic, normochromic anemia (27%), leukocytosis with neutrophilia, and a prolonged activated partial thromboplastin time (aPTT) (>300 seconds). Abdominal ultrasound revealed free fluid and a lesion in the transverse colon consistent with an intramural hematoma. Abdominocentesis confirmed the presence of an active hemoabdomen. The cat had been diagnosed with hemophilia after presenting episodes of subcutaneous hematomas and factor VII and IX activities of 60% and 58%, respectively, two years earlier. An abnormal thromboelastography (TEG), which showed a hypocoagulable state, confirmed impaired clot formation.

The patient was treated with fluid therapy, vitamin K, tranexamic acid, desmopressin and blood and plasma transfusions. Despite initial clinical improvement, the cat was readmitted three days later with increased abdominal distention, worsening anemia, and a significantly enlarged abdominal hematoma. Given the poor prognosis and the need for additional transfusions, euthanasia was elected. A post-mortem examination confirmed a large mural hematoma in the intestinal wall, with muscle expansion, peripheral fibrovascular tissue, and hemorrhagic areas.

Unique Information:
While intestinal hematomas have been documented in human medicine, this is the first case that describes hemoabdomen secondary to intramural intestinal hematoma in a cat with hemophilia. This case highlights that even mild-to-moderate deficiencies in coagulation factors can predispose animals to severe internal hemorrhage, underscoring the need for early diagnosis and careful management of such patients. Additionally, it emphasizes the challenges of managing recurrent and severe bleeding episodes, even with intensive therapeutic interventions.

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PERIOPERATIVE STABILIZATION OF HYPOKALEMIA IN A CAT WITH PRIMARY HYPERALDOSTERONISM

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Background:
Most cases of feline primary hyperaldosteronism are caused by an adrenocortical tumor that secretes aldosterone, resulting in excessive potassium excretion and sodium retention. It most commonly affects middle-aged and geriatric cats, although its actual incidence is unknown.

Case presentation:
An 8-year-old neutered male cat was presented for lethargy and apathy. During clinical exam, the abdomen was tense, and an epigastric mass was palpated. Serum biochemistry revealed hypokalemia (2.9 mmol/L), which responded poorly to intravenous potassium supplementation. Abdominal radiographs and bi-cavitary CT scan identified a suspected left adrenal mass, measuring 5.9 x 3.8 x 3.8 cm. Mild pleural and pericardial effusion were also noted and followed by echocardiography, which was unremarkable. Serum aldosterone was markedly elevated (140 ng/dL).

The cat was scheduled for unilateral adrenalectomy. On the day of surgery, marked hypokalemia was present (2.6 mmol/L), accompanied by mild metabolic alkalosis (pH 7.468), arterial hypertension (SAP 182 mmHg) and inappetence. The retinal exam revealed no signs of chronic systemic hypertension. Surgery was postponed, and a nasoesophageal feeding tube was placed. A continuous rate infusion (CRI) of liquid veterinary food was administered over 10 hours. After 24 hours of intravenous potassium supplementation and nutritional support, potassium increased to 3.4 mmol/L, allowing surgery to proceed. Anesthesia was stable, the mass was removed without complications and histopathology confirmed a neuroendocrine secreting tumor.

Postoperatively, intravenous potassium supplementation and CRI feeding were continued. Vitals, arterial blood pressure and serum potassium values were measured every 4 hours. The cat was discharged three days after surgery, with serum potassium of 3.9 mmol/L and SAP at 118 mmHg. A follow-up after 7 days confirmed complete resolution of clinical signs, normotension and physiologic potassium levels.

New/Unique Information:
Most guidelines suggest preoperative stabilization with oral potassium, aldosterone receptor blockers and antihypertensives. However, due to inappetence, tumor size, and poor prior response to potassium supplementation, surgical treatment was prioritized. Patient stabilization prior to surgery was needed and ensured by intravenous potassium supplementation and continuous dietary potassium intake. This approach quickly corrected hypokalemia, enabling safe anesthesia for surgery, without the use of oral medication in an inappetent cat.

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SUCCESSFUL TREATMENT OF A DOG WITH IATROGENIC POLYETHYLENE GLYCOL ELECTROLYTE SOLUTION ASPIRATION PNEUMONITIS UNDERGOING BRONCHOALVEOLAR LAVAGE AND MECHANICAL VENTILATION

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Background:
PEG electrolyte solution is considered a safe treatment for constipation in dogs and cats. Due to its relatively easy use, it is frequently administered without noticeable problems. However, both in humans and dogs inadvertent PEG aspiration has been described, sometimes with fatal consequences. The objective of this case report is to describe successful intensive care management of a dog with inadvertent administration of polyethylene glycol (PEG) electrolyte solution causing aspiration pneumonitis, using bronchoscopic airway lavage (BAL) and positive pressure mechanical ventilation.

Case presentation:
In the current case, tracheal placement of the feeding tube led to aspiration of PEG and active charcoal solution in a dog. The dog, a 3-year-old female intact Chihuahua-Pomeranian crossbreed, initially presented at the referral clinic with abdominal pain after eating the contents of a garbage bin containing diapers and spare ribs. The dog was sedated for the placement of a nasoesophageal tube after which the PEG and active charcoal solution was started. The tracheal placement led to an aspiration pneumonitis causing dyspnea and coughing. Initial treatment involved BAL and medical support such as antibiotics. Due to the development of severe dyspnea, mechanical ventilation was needed. During mechanical ventilation two complications occurred on the first ventilator day. The first complication was hyperkalemia of unknown origin which was treated with glucose, calcium gluconate, insulin and terbutaline. Secondly, severe clogging of the heat-moisture exchanger filters with the PEG electrolyte solution occurred, resulting in significant hypercapnia. After 4 days the dog could be weaned from the ventilator and eventually made a full recovery.

New or unique information provided:
To the authors' knowledge, this is the first case report describing successful treatment of inadvertent administration of PEG electrolyte solution involving BAL and mechanical ventilation in a dog.

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