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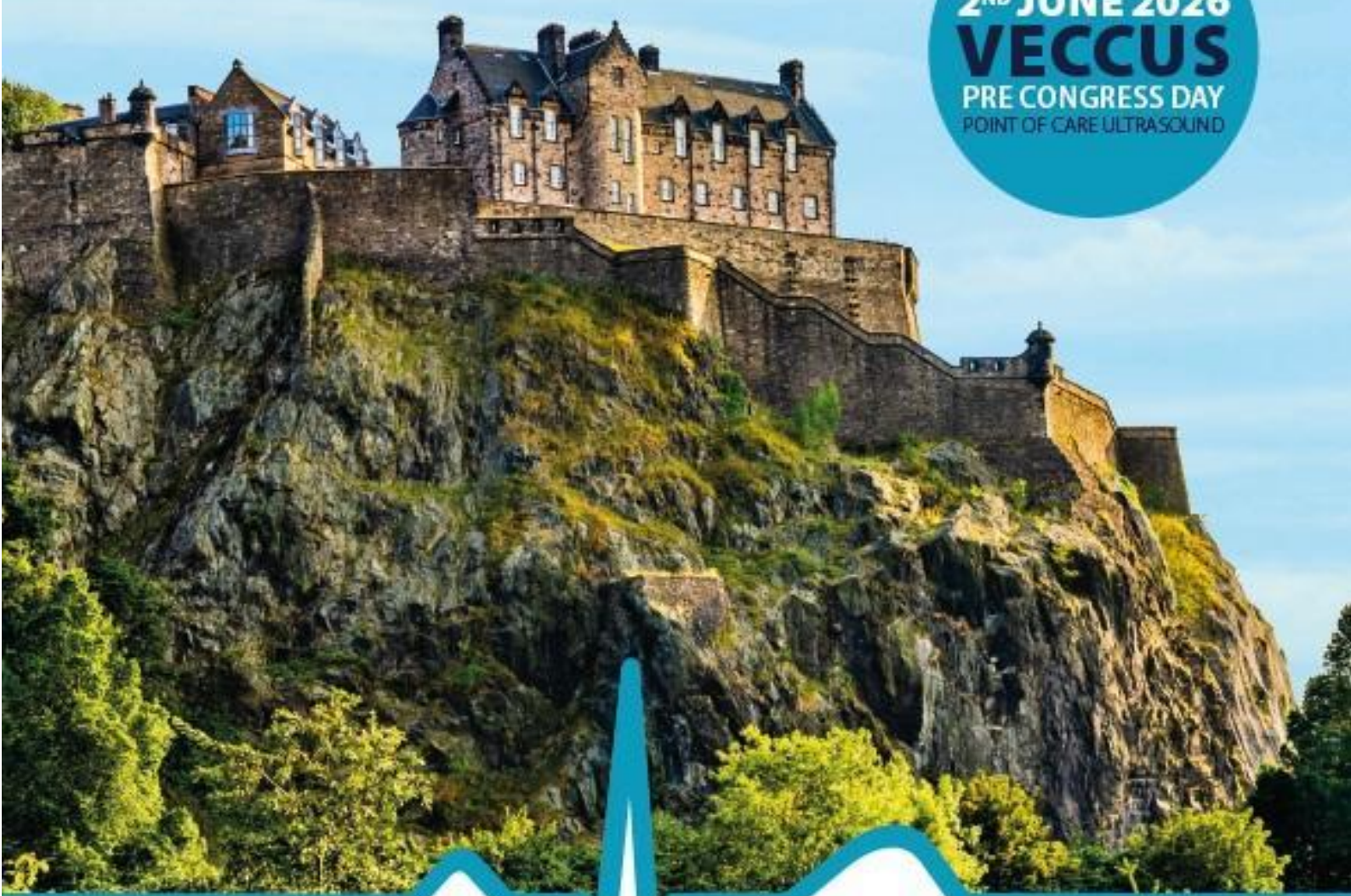

2ND JUNE 2026
VECCUS
PRE CONGRESS DAY
POINT OF CARE ULTRASOUND

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EUROPEAN VETERINARY EMERGENCY AND CRITICAL CARE CONGRESS

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Inhoud

VECCUS Symposium – Main Stream, Wednesday 3 June 2026

AI ASSISTED IMAGING: IS THERE A PLACE IN POCUS YET?

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Learning objectives:

By the end of this lecture, participants will be able to:

- Describe the current clinical applications of AI-assisted imaging in point-of-care ultrasound (POCUS), including acquisition guidance, automated quantification, and decision support.
- Summarize the available human and veterinary evidence supporting AI integration in cardiopulmonary POCUS.
- Critically evaluate the limitations of AI in POCUS, including image quality dependence, dataset bias, and lack of outcome-based validation.
- Interpret AI-generated outputs (e.g., automated B-line quantification) within the appropriate clinical context.
- Define the appropriate current role of AI in POCUS as a complementary decision-support tool rather than a replacement for clinician expertise.

Proceeding:

Introduction

Point-of-care ultrasound (POCUS) has become a core component of emergency and critical care medicine. Its strengths—bedside availability, repeatability, and real-time decision support—are counterbalanced by a well-known limitation: operator dependency. Image acquisition, interpretation accuracy, and inter-observer variability remain significant sources of inconsistency. Artificial intelligence (AI) has emerged as a potential solution to these limitations. The central question is no longer whether AI can be applied to ultrasound, but whether it currently has a meaningful and safe role within clinical POCUS.

Current Applications of AI in POCUS

Recent literature demonstrates rapid development of AI tools in acute care ultrasound. Applications can be broadly divided into four domains:

1. Acquisition Guidance – AI-assisted systems provide real-time feedback to help users obtain standardized views, particularly in focused cardiac and lung ultrasound. This may reduce the learning curve for novice operators.
2. Image Quality Assessment – Algorithms evaluate image adequacy and prompt users when acquisition is insufficient for reliable interpretation, potentially reducing diagnostic errors linked to poor image quality.

3. Automated Quantification – AI can automatically calculate ventricular function parameters, chamber dimensions, or quantify B-lines in lung ultrasound. Automation improves reproducibility and reduces inter-operator variability.
4. Pattern Recognition and Decision Support – Deep learning models have demonstrated promising performance in detecting reduced left ventricular systolic function, pleural effusion, pneumothorax, and interstitial syndromes.

Scoping reviews in human emergency medicine confirm growing adoption of AI-assisted POCUS, particularly in cardiopulmonary imaging. However, most studies remain validation-based rather than outcome-driven, and clinical impact on patient-centered outcomes is still under investigation.

Evidence from Veterinary Emergency and Critical Care

While AI development has primarily occurred in human medicine, veterinary applications are emerging. In our recent SMARTDOG study evaluating AI-assisted lung POCUS in dogs, automated B-line quantification demonstrated excellent agreement with expert assessment (intraclass correlation coefficient approximately 0.88). Diagnostic accuracy ranged between approximately 84–86% compared with experienced clinicians.

Importantly, the study also highlighted key limitations: AI performance declined in suboptimal image acquisitions, and a proportion of cine loops could not be interpreted by the algorithm. These findings reinforce a fundamental concept—AI cannot compensate for inadequate image acquisition. Instead, it functions best when integrated into structured scanning protocols with appropriate user training.

The veterinary data therefore align closely with findings from human literature: AI is a promising adjunct, particularly for quantifiable tasks such as B-line counting, but remains dependent on operator technique and appropriate clinical integration.

Limitations and Barriers

Despite promising performance metrics, several challenges limit widespread implementation:

- Image Quality Dependence – AI systems are only as reliable as the images provided. Poor probe positioning or inadequate windows significantly reduce algorithm performance.
- Generalizability – Models trained on specific datasets may not perform consistently across different machines, species, or clinical environments.
- Clinical Validation – Few prospective trials demonstrate improved clinical outcomes attributable directly to AI assistance.
- Risk of Overreliance – There is potential for automation bias, where clinicians may overtrust AI output without sufficient clinical correlation.

These limitations suggest that AI integration must be cautious, supervised, and embedded within existing clinical reasoning frameworks.

Where Does AI Currently Fit in POCUS?

At present, AI-assisted imaging in POCUS appears most appropriate in:

- Structured cardiopulmonary applications
- Repetitive quantification tasks (e.g., B-line scoring)
- Training environments to support novice acquisition
- Situations where reproducibility and standardization are critical

AI does not replace clinician expertise. Instead, it enhances consistency, reduces variability, and provides an additional layer of decision support.

Conclusion

AI-assisted imaging in POCUS has moved beyond theoretical development and now demonstrates measurable clinical utility in focused applications. Evidence from both human acute care literature and our veterinary SMARTDOG study supports its role as a complementary tool. However, limitations related to image quality, generalizability, and outcome validation remain significant.

There is a place for AI in POCUS—but currently as an adjunct, not an autonomous diagnostic system. The future of AI in bedside ultrasound will depend not only on algorithm performance, but on responsible clinical integration, continued validation, and preservation of strong foundational ultrasound training.

SOFT TISSUE ULTRASONOGRAPHY IN EMERGENT AND CRITICALLY ILL VETERINARY PATIENTS

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Learning objectives:

- Identify appropriate soft tissue and musculoskeletal ultrasound applications within point-of-care ultrasound (POCUS) for emergency and critical care patients
- Understand how to perform targeted image acquisition and recognise normal and abnormal ultrasonographic findings of soft tissue structures in acute presentations
- Select high-yield clinical indications for the use of soft tissue and musculoskeletal POCUS in emergency settings
- Integrate soft tissue ultrasound findings into timely diagnostic and therapeutic decision-making
- Recognise the limitations of POCUS when utilised to assess soft tissue and musculoskeletal investigations in emergency scenarios.

Proceeding:

Musculoskeletal point-of-care ultrasound (MSK POCUS) of superficial soft tissues is an emerging yet underutilized extension of the physical examination in small animal emergency and critical care. While abdominal and thoracic POCUS applications are well established in dogs and cats, MSK POCUS has received comparatively less attention despite its practical value. In patients presenting with trauma, acute lameness, focal swelling, fever of unknown origin, or unexplained pain, bedside ultrasound can rapidly answer targeted clinical questions, guide interventions, and inform decision-making when advanced imaging is unavailable or patient instability limits transport. As portable ultrasound units become increasingly accessible, incorporation of musculoskeletal applications into routine POCUS practice represents a logical progression in veterinary critical care.

Effective MSK POCUS relies on understanding normal sonographic anatomy and good technique. High-frequency linear transducers provide optimal resolution for superficial structures. Evaluation in both longitudinal and transverse planes, with comparison to a contralateral structure, when possible, improves diagnostic confidence. Normal skeletal muscle appears relatively hypoechoic with linear hyperechoic striations, while tendons and ligaments demonstrate a parallel fibrillar echogenic pattern when visualised transversely. Failure to maintain probe alignment can result in anisotropy and false hypoechoic regions. Cortical bone produces a sharply defined hyperechoic interface with distal acoustic shadowing; disruption of this margin is a key indicator of surface pathology.

One of the most common emergency applications is differentiation between cellulitis and abscessation. Physical examination alone may not reliably distinguish diffuse inflammation from a drainable purulent collection. Sonographically, cellulitis typically appears as diffuse subcutaneous thickening with increased echogenicity and a “cobblestone” pattern reflecting edema. In contrast, abscesses are visualized as encapsulated hypoechoic to anechoic fluid that may contain echogenic

debris, or gas artifacts. Ultrasound-guided aspiration improves diagnostic accuracy, and serial examinations allow monitoring of treatment in septic or critically ill patients.

Ultrasound is also valuable for detecting migrating foreign bodies such as grass awns and porcupine quills, which may be radiographically occult. These structures typically appear as linear hyperechoic interfaces with variable acoustic shadowing and are often surrounded by hypoechoic inflammatory tracts. Early localization can refine surgical planning and reduce morbidity associated with blind exploration. Bite wounds and penetrating trauma can likewise be assessed for retained debris, hematoma formation, and fascial plane disruption.

In trauma patients, MSK POCUS complements thoracic and abdominal POCUS examinations. Muscle tears appear as focal disruption of normal fiber architecture with hypoechoic or heterogeneous regions consistent with hemorrhage or edema. Fascial plane separation may be visible as abnormal fluid tracking between muscle groups. Although radiography remains the standard for fracture assessment, ultrasound can identify cortical discontinuity in accessible long bones and ribs. Rib fractures are particularly amenable to sonographic detection and may appear as interruptions or step defects in the hyperechoic cortical interface. Recognition of these injuries in unstable patients may influence analgesic and respiratory management when radiographic positioning is not feasible. While intramedullary bone cannot be evaluated, ultrasound is sensitive to cortical surface abnormalities such as osteomyelitis or aggressive bone neoplasia, which may manifest as irregular cortical margins and altered acoustic shadowing. In suspected discospondylitis, ventral vertebral endplate irregularity and surrounding hypoechoic soft tissue changes may be detected, prompting expedited confirmatory imaging.

Joint assessment represents another important application. Even small effusions can be detected as anechoic or mildly echogenic distension of the joint capsule. In patients with suspected septic arthritis or immune-mediated polyarthritis, rapid confirmation of effusion and ultrasound-guided arthrocentesis can significantly shorten time to diagnosis. Ultrasound guidance improves accuracy in small or anatomically challenging joints. Preliminary clinical observations suggest that periarticular soft tissue thickening may be present in some cases of immune-mediated polyarthritis, even when effusion is limited.

In recumbent or non-ambulatory patients, MSK POCUS assists in evaluation of asymmetrical swelling, suspected pressure-related muscle injury, or evolving hematomas. Assessment of the cervical region may also aid in cases of upper respiratory distress by identifying masses, fluid accumulations, or structural abnormalities impinging on the larynx. Beyond diagnosis, MSK POCUS enhances procedural safety by improving accuracy for fine needle aspiration, abscess drainage, drain placement, and vascular access through edematous or altered tissues.

Despite its versatility, musculoskeletal POCUS remains operator dependent and subject to artifacts. It is designed to answer focused clinical questions rather than replace radiography, computed tomography, or magnetic resonance imaging. Peer-reviewed veterinary literature specific to small animal MSK POCUS in emergency settings remains limited, although existing studies support its utility in detecting various soft tissue abnormalities. With appropriate training and integration into structured POCUS curricula, musculoskeletal ultrasound has the potential to become a standard component of small animal emergency and critical care practice.

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CORE ULTRASOUND GUIDED LOCOREGIONAL ANAESTHESIA BLOCKS

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Learning objectives:

- Understand the role of ultrasound-guided locoregional anaesthesia in improving analgesia and physiological stability in emergency and critical care patients.
- Identify key sonoanatomical landmarks for commonly used core UG-LRA blocks in dogs and cats.
- Select appropriate locoregional blocks based on patient presentation, injury pattern, and clinical priorities.
- Recognize potential complications and safety considerations associated with UG-LRA in critically ill patients.

Proceeding:

Ultrasound-guided regional anaesthesia (UGRA) is a valuable tool in veterinary emergency and critical care, providing effective, site-specific analgesia while reducing reliance on systemic drugs and their associated adverse effects. This presentation reviews core UGRA techniques most applicable in emergency and ICU settings, focusing on indications, technical considerations, safety, and clinical decision-making through selected case examples.

Case 1. A 2-year-old male Chihuahua was presented following a road traffic accident with polytrauma, including severe respiratory distress due to pulmonary contusions, thoracic and abdominal effusion, and a femoral fracture. Initial treatment prioritized respiratory stabilization using high-flow nasal cannula oxygen therapy. Despite ongoing respiratory compromise, fracture analgesia and immobilization were required to allow handling and bandage application. Escalation of systemic sedatives or opioids was considered undesirable. An ultrasound-guided sciatic nerve block was therefore performed using ropivacaine 0.5% (2 mg/kg). The block provided effective analgesia, allowing safe fracture management without worsening respiratory function.

Case2. A 6-year-old, 43-kg Labrador Retriever was hospitalized for acute pancreatitis, presenting with severe abdominal pain and reduced gastrointestinal motility. Opioids at standard doses provided insufficient analgesia. A lidocaine constant rate infusion was avoided due to concerns about worsening ileus and vomiting, while increased opioid dosing risked excessive sedation and impaired ambulation. An ultrasound-guided transversus abdominis plane (TAP) block was therefore performed under light α_2-agonist sedation using ropivacaine 0.5% (3 mg/kg total dose). The block resulted in marked pain relief when combined with low-dose opioids and a dexmedetomidine infusion, preserving gastrointestinal function and mobility.

Case3. An 8-year-old Maltese underwent emergency surgery for gallbladder rupture with bile peritonitis. A preoperative ultrasound-guided quadratus lumborum (QL) block was included in the analgesic plan. Approximately 10 hours later, pain scores increased despite fentanyl, lidocaine, and

ketamine infusions. Repeating the QL block was technically challenging due to postoperative intrabdominal free air impairing ultrasound visualization. An ultrasound-guided erector spinae plane (ESP) block was instead performed using bupivacaine 0.4% (2 mg/kg). Within 30 minutes, pain improved significantly, allowing reduction of fentanyl and ketamine infusions; lidocaine was discontinued prior to block placement. The dog became comfortable and ambulatory. The ESP block was repeated three times during hospitalization in response to pain recurrence approximately 7–10 hours after each block, providing consistent analgesia.

Case 4. A 2-year-old male cat with polytrauma and bilateral hindlimb fractures received a bilateral ultrasound-guided sciatic nerve block for analgesia and wound management. Bupivacaine 0.5% was administered at a calculated dose of 3 mg/kg based on owner-reported body weight. Although analgesia was adequate, the cat developed arrhythmia and cardiovascular instability shortly after injection, consistent with local anaesthetic systemic toxicity (LAST). Intravenous intralipid therapy was promptly administered, resulting in full recovery. Subsequent weighing revealed the cat's actual body weight was 4.1 kg rather than the reported 6.5 kg, resulting in an unintended dose of 4.7 mg/kg. This case highlights the importance of accurate weight measurement and awareness of bupivacaine toxicity, particularly in unstable patients.

Case 5. An 8-year-old female Jack Russell Terrier with chronic gastrointestinal and pancreatic disease was presented for acute abdominal pain and anorexia. Abdominal POCUS and radiographs showed no acute surgical disease. An ultrasound-guided ESP block was performed using ropivacaine 0.5% (3 mg/kg), combined with fentanyl and dexmedetomidine infusions. Thirty minutes later, the dog was comfortable and ambulatory. Two hours after block placement, the dog ate but subsequently developed unilateral pelvic limb neurological deficits. Initially, unintended local anaesthetic spread to the lumbar plexus was suspected. Within three hours, the condition progressed to bilateral pelvic limb paresis with reduced reflexes. Emergency MRI revealed a spinal cord neoplasia not detected on radiographs. The owner later reported subtle neurological signs in the days preceding admission. It was presumed that effective analgesia and muscle relaxation unmasked the underlying disease. Due to poor prognosis, the dog was euthanized. This case underscores the importance of careful neurological assessment and history-taking prior to UGRA and consideration of pre-existing neurological disease when postoperative deficits occur.

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KEYNOTE LECTURE: POCUS IN HUMAN INTENSIVE CARE: COMPETENCE, CONFIDENCE, AND CLINICAL CONSEQUENCES

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Learning objectives:

- Develop an undersatnding ofthe scope of point of care ultrasound in adult human intensive care settings
- Gain knowledge of the benfits limitations and challenges of accredited training pathways in point of care ultrasound in adult human intensive care
- Describe limitations of diagnosis using point of care ultrasound in adult human intensive care

Proceeding:

This lecture will take a case based approach to descibe indications and usage of point of care ultrasound (PoCUS) in adult human intensive care medicine. I will summarise commonly used ultrasound examinations and then using real life clinical cases demonstrate the challnges associated with safe and effective use of PoCUS in adult human intensive care. These include design and delivery of training pathways, awareness and acceptance of limitations of utility of point of care ultrasound, ongoing accreditation and development and clinical and information governance. I will also discuss how human fallibilty can lead to incorrect diagnosis and treatment decisions due to over reliance on point of care ultrasound-dervied findings.

Point of care ultrasound may be ued for both procedural and diagnostic indications. Procedural use ill will not be covered in this talk, but is used to faciltate placement of peripheral and central vascular access devices and for nerve blocks, used as part of multi-modal analgesia strategies in post-surgical nad trauma patients. Diagnostic point of care ultrasound can be used to assess the cardiovascular, respiratory, gastrointestinal, renal, neurological and musculoskeletal systems. In the United Kingdom point of care ultrasound in intensive care is most commonly used to assess the cardiovascular and respiratory systems.

In UK adult human ICU, accrediation in point of care ultrasound is not mandatory to complete training to become a consultant physician in intensive care medicine. This is likely due to a lack of trainers. There are also significant challneges in terms of accessing training time and opportunities faced by doctors working in intensive care medicine. There are several pathways availble to physicians (and other ICU staff such as physiotherapists and advanced critical care practitioners) administered by the Intensive Care Society and the British Society of Echocardiogrpahy. Basic (Level 0 and Level 1) accrediation pathways do not require re-assessment of demontration of yearly minimum scan numbers, or evidence of ongoing engagment with Continuous Professional Development activities.

Guidelines for the Provision of Intensive Care Services (GPICS) is a guideline that applies to provision of intensive care services in England, but its recommendations are considered persuasive for all UK

ICUS. It recommends every ICU has a lead clinician for ultrasound provision with protected time in their job plan to provide education, training and support. It also recommends that all scans are recorded and uploaded to the hospital storage system to allow incorporation into the medical record of the patient, alongside a structured report. Given the large numbers of unaccredited physicians undertaking point of care ultrasound the concept of 'ghost scanning' will be discussed.

Two real life scenarios will be discussed in which use of point of care ultrasound led to delyed diagnosis and near-miss inappropriate treatment, The challnges with using point of care ultrasound in high pressure situations and the danger of overionterpreting images will be discussed

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ADVANCES IN POCUS TRAINING - PHANTOMS, CADAVERS AND AUGMENTED REALITY

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Learning objectives:

- By the end of this session, learners will be able to:
- Describe the role of phantoms, cadavers, and virtual augmented reality (VAR) in POCUS training.
- Compare the advantages and limitations of phantoms, cadaver-based training, and VAR for acquiring POCUS skills.
- Identify appropriate educational contexts in which each training modality is most effective.
- Apply knowledge of advanced POCUS training tools to improve skill acquisition, procedural accuracy, and learner confidence.

Proceeding:

Point-of-care ultrasound (POCUS) is a cornerstone bedside tool for veterinary emergency practice. Confidence in POCUS amongst veterinary practionners is often reported to be low and they express a desire for more training opportunities (MacDonald, 2023; Valcke, 2024). Training on live dogs is often time and resource heavy and limited by tolerance of the animal. There therefore is a need for alternative teaching methodologies to teach this important skill. Complementary methods including training phantoms, cadavers, and augmented/virtual reality platforms have the potential to develop POCUS psychomotor skills and clinical integration.

Phantoms have long been the workhorse of ultrasound skill acquisition. Home-made phantoms can be constructed cheaply using pots and agar, natural gelatin or psyllium husk to replicate tissue, and the addition of fruits, balloons filled with water to reproduce organs. These products are particularly effective to teach basic probe movements, knobology and to visualise landmarks. They are also useful to teach probe/ hand coordination. Gelatine based often fail to replace the exact density and texture of animal flesh and therefore animal-derived products may be preferred due to their resemblance to animal tissue haptic and echogenicity (Selame, 2021; Chakroun-Walha, 2023). Home-made phantoms using food products such as chicken, spam and tofu can be utilised to practice ultrasound guided vascular access. A pork rib bucket model has been described to teach thoracocentesis in humans and is also used in veterinary medicine. A slab of pork rib is inserted into a Ziploc bag. A second Ziploc bag is filled with water and a cellulose sponge. The pork rib Ziploc bag is taped above the water filled sponge Ziploc bag and over a upside down bucket. It replicates pleural effusion and has been used to teach ultrasound guided thoracocentesis and small-bore guide wire thoracostomy tube placement (Gillett 2025). These models are also useful for practicing interventional ultrasound; -centesis, fine needle aspiration and ultrasound guided vascular access (Phillips, 2023). The major limitation is their short-life span and need to make new ones for each training session. The use of ballistic gel allows for more durable training product, with longer shelf

life. The growing use of 3D printing allows to create more durable and realistic models. Recently a 3d printed pericardiocentesis using ballistic gel, 3D printed rib cage and a balloon filled with a tennis ball and water has been described in veterinary medicine (Hillstead, 2026). Commercial ultrasound phantoms are available and are typically constructed from tissue-mimicking materials to replicate acoustic and mechanical properties of skin. These various phantom models are beneficial for procedural training (fine needle aspiration, centesis and ultrasound guided vascular access), however they are less effective for teaching clinical decision-making involved interpreting POCUS.

With appropriate preservation cadaver-based POCUS training restores the complexity of three-dimensional anatomy. Furthermore, canine and feline cadavers can be intubated and positive pressure breath can be given using an ambubag, replicating breathing movement. Frozen thawed intubated cadavers have been used to teach pleural and lung ultrasound (PLUS), (Finch 2026). Additionally if not already present in frozen thawed cadavers, pathologies can be created including pleural effusion, pneumothorax, pneumoperitoneum. Fresh-frozen specimens retain tissue pliability and sonographic characteristics allowing learners to reconcile two-dimensional sonographic planes with the relationship of fascial planes, organs and vessels. Clinical decision-making involved interpreting POCUS can be layered in the training, depending on the learner's skill and knowledge. For instance, novices will learn how to perform PLUS and recognize pathology on frozen thawed cadavers, whereas more advanced learners will be ask to interpret and treat a simulated patient represented by the frozen thawed cadaver in front of him. Fresh-frozen cadavers can be used but freezing spoils their texture. They are using a single day only use for POCUS teaching purposes. Fresh cadavers are more realistic but only last a few days. They are harder to source and bodies can degrade quickly leading to possible health hazards. An alternative is light embalming, a technique through which specialised solution (Thiol formula) is injected in the cadaver arterial's system and then stored in refrigerated settings. This model can preserve the cadaver in a life-like manner which results in a more realistic training model that lasts months-years. These embalmed cadavers are particularly used for anatomical POCUS. Embalmed cadavers have been used successfully in training undergraduate veterinary students in radiography and abdominal ultrasound (Thanaboonnipat, 2021) and abdominal POCUS (unpublished). The limitations of cadavers are that they are expensive, require speciated storage and a substantial amount of preparation time.

Immersive virtual reality (IVR) is an emerging technology with significant potential in ultrasound education. Immersive VR (IVR) involves use of a head-mounted device (HMD) that allows the user to observe and move around in a simulated, virtual 3-D environment, while controllers or hand-tracking allow the user to interact with the environment. Instructors can visualize the virtual 3D environment via streaming on a tablet and give feedback and instruction. The use of a mannequin allows learners to have the touch sensation similar to POCUS on a live dog or cat and refine probe placement. Mannequins can be soft toys, commercial teaching mannequins or created from large buckets with glued yarn and fabric to replicate a dog's chest and abdomen. Commercial veterinary virtual reality software are available with or without mannequins (Deepscop,veterinary Ldt). IVR simulator allow recognition of normal 3D canine anatomy and present pathologies with the potential to immerse students in life-like clinical scenarios. This enables the learner to learn how to perform POCUS at their own pace, without the need for live dogs or cadavers. In addition, it also allows encounter the pathology without risk to the patient. Immersive virtual reality has shown to be non-inferior to an instructor-led basic point-of-care ultrasound training with associated lower costs and increased accessibility if instructor numbers are limited. It also allows learners to practice without the need of

an instructor, live dog or cadaver. It could be an equivalent alternative to instructor-led lessons courses, as well as increase the scalability of training opportunities (Andersen, 2023). There is a relatively steep learning curve associated IVR and few studies have reported discomfort wearing the VR glasses and few cases of cybersickness (Saliba, 2025). The initial cost for the software and head mounted device is also not negligible.

These different POCUS training tools have their own benefits and limitations. A training programme that includes all training tools to improve proficiency is likely to be the most beneficial training for teaching POCUS. Phantoms and VR lower the barrier to entry to POCUS and cadavers offer anatomic depth and tactile realism. Together they form a continuum to allow learners tentative probe handlers to confident clinicians who feel confident in utilising POCUS as an extension of their physical exam.

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TIPS FOR USING DOPPLER ULTRASOUND IN OUR CASES

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Learning objectives:

- Understand the principles, uses, and common pitfalls of colour and spectral Doppler, including pulse wave and continuous wave Doppler, in clinical echocardiography.
- Select appropriate Doppler modalities and echocardiographic views to answer specific clinical questions efficiently, particularly in the ECC setting.
- Use Doppler echocardiography to identify and interpret common cardiac diseases, including MMVD, CHF, HCM with SAM, pulmonary hypertension, and obstructive or shunt lesions.
- Differentiate clinically useful Doppler findings from less impactful measurements, prioritizing rapid, decision-making–focused assessments.

Proceeding:

Doppler ultrasound is a powerful adjunct to point-of-care echocardiography in the emergency and critical care (ECC) setting. It provides rapid, clinically relevant information regarding blood flow direction, velocity, and haemodynamic consequences of cardiac disease. Understanding the strengths and limitations of colour and spectral Doppler is essential to avoid misinterpretation and to maximise diagnostic utility in time-pressured environments.

Colour Doppler

Colour Doppler is mostly used to assess flow direction, turbulence, and the presence of abnormal jets. Flow towards the transducer is displayed in red and flow away in blue. The appearance of colour variance, often green, indicates turbulent or high-velocity flow that exceeds the Nyquist limit and results in aliasing. This is useful for identifying valve regurgitation, high-velocity jets, and obstructions to flow within the heart. The velocity range displayed is determined by the Nyquist limit and should be adjusted based on the expected flow velocities. Increasing or decreasing the scale can significantly alter the appearance of colour jets and turbulence. Gain should be adjusted carefully to the level just before random background noise appears; excessive gain can artefactually enlarge flow areas, while insufficient gain may mask clinically relevant flow. Jet area alone is a poor indicator of regurgitant severity, as it is heavily influenced by gain and scale settings. More accurate quantification, such as calculation of effective regurgitant orifice area using the proximal isovelocity surface area (PISA) method, is preferred but is rarely practical in an ECC environment. Colour Doppler can also be used to identify vessels and assess tissue perfusion in other parts of the body.

Spectral Doppler

Accurate spectral Doppler assessment relies heavily on optimal beam alignment, and the direction of the ultrasound beam should be as parallel to blood flow direction as possible. Pulse wave Doppler allows measurement of blood flow velocities from a specific intracardiac location between the

sample gates, enabling precise localisation of flow profiles. However, it is limited by aliasing when velocities exceed the Nyquist limit. Continuous wave Doppler, in contrast, samples the entire length of the ultrasound beam and can measure very high velocities, making it ideal for stenotic lesions and severe regurgitation, though localisation of flow origin is sacrificed. When measuring continuous wave Doppler profiles, attention should be paid to selecting a signal with a clear, well-defined edge—measuring the "chin" rather than the "beard." Spectral profiles can help differentiate fixed versus dynamic obstructions and estimate regurgitant severity based on signal density and diastolic decay. Pressure gradients can be estimated using the modified Bernoulli equation. Spectral Doppler can be used to estimate cardiac output, shunt ratios, and stenosis severity, this requires optimal image acquisition, multiple measurements, and complex calculations. As such, they are generally not suitable for routine use in the ECC setting.

Specific Disease Applications

In myxomatous mitral valve disease (MMVD), colour Doppler is useful for identifying mitral regurgitation, while E-wave velocity on spectral Doppler can provide an estimation of left atrial pressures. In hypertrophic obstructive cardiomyopathy and systolic anterior motion (SAM), combined colour and spectral Doppler allow identification and quantification of left ventricular outflow tract obstruction. In suspected pulmonary hypertension, tricuspid and pulmonic regurgitant velocities can be measured and used to estimate pulmonary artery pressures. For pulmonic and aortic stenosis, outflow velocities exceed 2m/s, though breed variations exist. Colour Doppler assists in localising the stenosis as subvalvular, valvular, or supravalvular. Patent ductus arteriosus can be identified using colour Doppler to localise the shunt, while spectral Doppler demonstrates continuous flow across the ductus and allows estimation of pressure gradients. Intracardiac shunts may also be detected using Doppler techniques, but this requires advanced anatomical knowledge and careful evaluation, and the ECC environment is often not conducive to such detailed assessment.

Conclusion

Doppler ultrasound is a valuable tool in ECC when used with an understanding of its technical principles and limitations. Optimising settings and focusing on clinically relevant questions allow colour and spectral Doppler to enhance rapid decision-making without overcomplicating assessment. When integrated thoughtfully into point-of-care echocardiography, Doppler ultrasound can significantly improve haemodynamic evaluation and patient management in the emergency setting.

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VECCUS Symposium – Foundations Stream, Wednesday 3 June 2026

ABDOMINAL POCUS MADE SIMPLE: HIGH-VALUE SCANS YOU CAN USE TOMORROW

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Learning objectives:

- Describe the role of abdominal point-of-care ultrasound (POCUS) as a clinically driven adjunct to history and physical examination in emergency and critical care patients.
- Identify appropriate indications for abdominal POCUS in both trauma and non-trauma presentations, including cardiorespiratory instability and acute collapse.
- Select appropriate ultrasound equipment, probes, and machine settings to optimize abdominal POCUS image acquisition in the ER and ICU.
- Perform a systematic abdominal POCUS examination, including correct patient positioning, probe handling, and identification of the five standard scanning sites.
- Recognize common abdominal POCUS findings, such as free fluid, gall bladder wall edema, urinary bladder distension, and gastrointestinal motility abnormalities, and integrate these findings into rapid clinical decision-making and patient monitoring.

Proceeding:

Indications

Abdominal POCUS initially described in assessment and management of small animal trauma cases. However, subsequently it has been found to be useful in non-trauma cardiorespiratory unstable patients and can help expediate diagnosis and management of these cases. Abdominal POCUS can be performed alongside other diagnostics and intervention such as oxygen supplementation, intravenous catheterization and fluid resuscitation.

Ultrasound machine and functions

Abdominal POCUS can be performed on any machine, however, the smaller and more portable the ultrasound machine the better. Various handheld machines are now available making abdominal POCUS more accessible. POCUS relies on three key machine settings: depth, gain, and frequency. The depth (depth of ultrasound penetration and the displayed image) and gain (overall image brightness) should be adjusted when scanning to optimize image quality. Frequency (resolution of image) is often a product of the ultrasound probe; linear probe has higher frequency (6-12mHz) than a microconvex probe (2.5-7.5mHz) but most probes allow frequency adjustments. The higher frequency linear probe provides better resolution but does not have the depth of penetration compared to the microconvex probe. A microconvex probe is recommended for abdominal POCUS as this probe has a good balance between frequency (resolution of image) and depth of penetration. Perfect image quality is not essential for interpreting abdominal POCUS and therefore the use of factory-defined pre-set programmes available on the ultrasound machine can be useful in the

emergency setting as once the pre-set has been selected the scan can be started immediately. Depth will need to be adjusted depending on the scanning site.

Technique

Positioning and patient preparation: The original abdominal POCUS technique (AFAST™) was performed in lateral recumbency. POCUS in standing and lateral recumbency have been compared and demonstrate good -excellent correlation for detection of peritoneal effusion in these different positions. Abdominal POCUS should be performed in whatever position they are most comfortable and it is particularly important to avoid moving a cardiorespiratory unstable patient. Patients should not be placed in dorsal recumbency to avoid compromising their cardiovascular function, which can lead to increased work of breathing and decreased venous return. Abdominal POCUS rarely requires sedation but analgesia should prioritized over POCUS.

Shaving is typically unnecessary unless the patient has a thick fur coat that impedes image resolution, such as Huskies and Northern breeds with dense undercoats. Alcohol is used for skin preparation, but it's important to part the fur before applying alcohol. Alcohol is noxious and flammable and therefore it is recommended to not over-saturate the patient with alcohol and wipe away any excess alcohol. To improve image quality gel may be used, and often required when using a probe with a large footprint. The gel should be applied directly to the skin or probe and not directly to the fur.

Protocol: During the examination, the ultrasound probe is systematically placed on five regions of the abdomen (figure 1). At each site, the probe is fanned and rocked through a 45° angle in both long in long axis (figure 2), and then should time permit rotate the probe 90 degrees and fan and rock the probe in short axis view). Moving the probe 1 inch in cranial, caudal, left, and right directions helps increase the area assessed at each site. This technique of fanning and rocking enhances the likelihood of detecting abdominal fluid.

Scanning sites

Subxiphoid site: The subxiphoid site, located just caudal to the xiphoid process. Scanning this site allows visualization of the diaphragm, liver, gallbladder, and caudal vena cava and with increased depth the pleural space, pericardium and caudal lungs. To visualize this site effectively, palpate the "V" at the xiphoid region and place the probe in a long axis to the body. Then, rock the probe until the diaphragm is visible, adjusting the depth until the entire diaphragm encircling the liver is observed. Note that individual liver lobes cannot be detected in a normal patient.

Urinary bladder site: This site facilitates the visualization of the bladder and the colon, great vessels and uterus may be identified. To begin, position the probe in a long axis to the body between the pelvic limbs. Upon locating the bladder, it is crucial to adjust the depth to visualize both the dorsal and ventral walls of the bladder. Subsequently, slide the probe to locate the apex of the bladder. Once positioned at the apex, employing techniques such as fanning, rocking, and rotating the probe to a short axis, followed by fanning and rocking in the short axis, will enable the visualization of abnormal effusion. Furthermore, the probe can be shifted to either side of the bladder and fanned to detect fluid in deeper gravity-dependent sites at the body wall

Right paralumbar site: This site enables the visualization of the liver, right kidney, body wall, and intestines. Obtaining this view can be challenging as it often requires maneuvering between ribs to visualize the normal structures. In some cases, it may be necessary to begin in the short axis to the body so that the probe can be positioned between ribs. In smaller dogs and cats, the probe can be placed in the long axis to the body caudal to the 13th and final rib. Once the liver is visualized, the probe can be moved caudally to locate the kidney. It's important to position the probe quite laterally from midline to locate these organs.

Left paralumbar site: This site allows visualization of the spleen and left kidney. The probe must be placed quite laterally to midline to visualize both organs. Initially, the probe is positioned in the long axis to the body, often mid-abdomen and lateral to start. It is usually easier to locate the spleen first and then slide the probe caudally until the left kidney is found. Utilizing fanning and rocking techniques with the probe aids in locating the organs of interest.

The umbilical site: The probe is placed at the umbilicus (ventral midline) angled at approximately 45 degrees, with the head of the probe oriented toward the tabletop. This site enables localization of gravity-dependent abdominal effusion. The probe should be gently rocked and fanned to optimize visualization.

Clinical uses

Free fluid can be readily detected at all scanning sites by novice sonographers. Free fluid often appears anechoic or hypoechoic, however highly cellular fluid, often seen with acute haemorhage is more echogenic and fanning and rocking can help differentiate from soft tissue. At the subxiphoid site fluid is usually localised between the diaphragm and liver or between liver lobes, at the paralumbar sites fluid is often localised to the poles of the kidneys and at the urinary bladder view fluid usually accumulates at the apex of the bladder. Attention should be given to position of the animal when scanning for free fluid, as free fluid often accumulates at the most gravity dependent site.

In animals presenting with trauma fluid scoring of a total of 4 for presence of fluid in subxiphoid, left paralumbar, right paralumbar and urinary bladder; 1 point for each positive site helps predict complications and monitor progression or resolution of peritoneal effusion. The presence of free fluid, confirmed as blood has been shown to be useful in predicting the need for transfusion in cats and in dogs higher fluid score (3/4) have been associated with PCV drop and need for transfusion. The frequency of scanning should be dictated by the clinical state of the animal, and this can be within minutes if the animal's clinical status deteriorates and a minimum of four hours in a stable animal.

Aside from trauma cases, free fluid is much readily detected in animals presenting to the emergency room that are cardiorespiratory unstable. Detection and subsequent sampling of the fluid can help detect life threatening conditions that need immediate intervention such as septic peritonitis. Furthermore, patients that have no major body system abnormalities can also free fluid and its detection can help guide decision making and therapy. Ultrasound can assist sampling safely small volume effusions. In addition to detection of free fluid abdominal POCUS can help detect major abnormalities that may help guide immediate management. Anaphylaxis is a life-threatening condition that causes acute collapse, cardiovascular compromise with cutaneous signs and/or gastrointestinal signs. The presence of a gall bladder "halo sign," which represents gall bladder

oedema, alongside a marked increase in the hepatocellular parameter ALT is highly suggestive of anaphylaxis. It is important to note that the halo sign is not specific to anaphylaxis. Other causes, include right sided congestive heart failure, hypoalbuminemia, fluid overload and cholangitis. It is therefore important to interpret your findings in light of the clinical presentation.

Abdominal POCUS can be integrated into the daily assessment of the ICU patient. Monitoring urine output is a key part of critical care. Assessment of the bladder and estimation of bladder volume can easily be performed on abdominal POCUS. Various algorithms for volume assessment are available on many commercial ultrasound, alternatively the following formula can be used; π x 0.3 x Length x Width x Height. Length and height are measured in the long axis and width is measured in the short axis.

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MASTERING THE THORAX: FOUNDATIONAL CARDIAC, LUNG & PLEURAL POCUS EVERY VET SHOULD KNOW

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Learning objectives:

- Explain how patient positioning affects pleural pathology (gas rises, fluid falls).
- Identify the five sonographically defined pleural and lung borders.
- Recognize and interpret the Bat sign, the pleural line, and lung sliding and define normal vs abnormal B-lines and understand the limitations of “wet lung” vs “dry lung” terminology.
- Apply pleural and lung ultrasound (PLUS) to clinical cases.

Proceeding:

The PLUS Approach

PLUS allows veterinarians to answer urgent bedside binary questions in dyspneic or unstable patients:

Is there pleural effusion?

Is there pneumothorax?

Is lung surface pathology present (increased B-lines or consolidation)?

Is cardiac disease contributing to respiratory distress?

PLUS is targeted, situational, and patient-centered. It prioritizes the most pressing clinical question while accounting for patient stability and positioning.

PLUS is dynamic and context-driven.

PLUS is binary and clinically driven.

Key binary questions:

Pleural effusion: yes/no

Pneumothorax: yes/no

Abnormal lung surface (B-lines or consolidation): yes/no

Clinical reasoning directs the sequence. For example:

Dyspneic Doberman with murmur → Are B-lines present?

If yes → Is the left atrium enlarged?

PLUS findings must always be interpreted within the full clinical picture.

Sonographically Defined Lung Borders

Understanding lung borders is essential and should not rely on external landmarks.

Five borders:

Caudal border – Abdominal curtain sign

Dorsal border – Hypaxial/sublumbar muscles

Cranial border – Limited by thoracic limb musculature

Ventral pleural border – Sternal/pectoral muscles

Ventral lung border

Everything between borders is lung.

The Six Key Lines and Signs of PLUS

Pleural line

Bat sign

Lung sliding

A-lines

B-lines

Abdominal curtain sign

Additional useful concepts include lung pulse, ski jump sign, PD window, Z-lines, I-lines, and defined lung borders.

*Pleural Line & Bat Sign*When the probe is placed transverse to ribs:

Rib heads and shadows form the “wings”

Pleural line forms the “body”

This creates the Bat sign.

Mnemonic: ultrasound does not traverse Bone or Air when Transverse.

The pleural line is the first white line below rib heads and represents the lung–chest wall interface.

*Lung Sliding (Glide Sign)*Lung sliding is a shimmering motion along the pleural line during respiration.

Two requirements:

Visceral and parietal pleura must be in contact.

The patient must breathe.

Lung sliding confirms lung–chest wall contact.

Adjusting gain to make the pleural line slightly “grainy” can improve detection.

*A-Lines*Horizontal reverberation artifacts equidistant from the pleural line.

“A” stands for air.

Seen in normal lung and pneumothorax.

If lung sliding is present with A-lines only → lung surface is normal at that site.

*B-Lines*Vertical, laser-like hyperechoic lines with 5 defining features:

Vertical

Originate at pleural line

Move with lung sliding

Extend to far field

Obscure A-lines

B-lines represent decreased peripheral lung aeration.

Normal vs Abnormal B-Lines

Small numbers may be normal:

≤3 B-lines at a single site can be normal.

1–2 B-lines common.

Occasionally up to 3 still normal.

Abnormal findings:

3 B-lines at a single site.

2 positive PLUS sites per hemithorax.

Always assess the entire hemithorax before concluding abnormality.

<u>Causes of Increased B-Lines</u>

“Wet” B-lines (increased extravascular lung water):

CHF

ARDS

Noncardiogenic pulmonary edema

Hemorrhage/contusions

Pneumonia

“Dry” B-lines (decreased air, increased cellularity):

Atelectasis

Fibrosis

Neoplasia

Therapy depends on cause; thus, avoid simplistic “wet lung/dry lung” labeling.

5. *Normal Lung Surface (Previously “Dry Lung”)*

Diagnosis requires:

Presence of lung sliding (aka; the glide sign).

≤3 B-lines at probe site.

A-lines alone can suggest normal lung, but absence of B-lines in the presence of lung sliding is more reliable.

Lung must be in contact with chest wall before assessing lung aeration (presence of B-lines).

Abdominal Curtain Sign Marks caudal lung border.

Created when air-filled lung overlies abdominal soft tissue.

Appears as sharp vertical edge artifact.

Important distinctions:

Do not confuse with diaphragm.

Curtain movement ≠ lung sliding.

Present in both healthy patients and pneumothorax (appearance differs).

Patient Positioning:

Gas Rises, Fluid Falls

Understanding gravity is critical.

Pneumothorax accumulates non-gravity dependent.

Pleural effusion accumulates gravity dependent.

Scan strategy must adapt to:

Lateral recumbency

Sternal recumbency

Standing patients

Stability and urgency

Example: Aspiration pneumonia often distributes cranial-ventral in upright patients following vomiting.

Integration with Cardiac POCUS

Thoracic POCUS often leads to cardiac questions.

Common binary cardiac question:

Is the left atrium enlarged?

In dyspneic patients:

Increased B-lines + enlarged LA → likely CHF.

B-lines without LA enlargement → consider non-cardiac causes.

Cardiac POCUS complements PLUS and improves differentiation of cardiac vs respiratory disease.

Recognizing artifacts prevents misdiagnosis.

Practical Pearls

Always identify the Bat sign first.

Confirm lung sliding before assessing aeration.

Estimate B-lines (<3,>3) per site and number of sites (<2,>2) per hemithorax.

Adapt scanning based on position and clinical suspicion.

Interpret findings within entire clinical picture.

PLUS is not a full echocardiogram or comprehensive thoracic ultrasound—it is a focused extension of physical examination.

Key Takeaways

PLUS is binary, targeted, and patient-centered.

Lung sliding (in the absence of a lung point) confirms pleural contact.

A-lines alone do not equal pneumothorax.

B-line thresholds matter.

“Wet lung”/"dry lung" terminology is overly simplistic.

Sonographic borders guide success.

Positioning determines where pathology accumulates.

Cardiac POCUS complements lung findings.

When applied systematically, thoracic POCUS allows rapid bedside differentiation of:

CHF vs pneumonia

Pleural effusion vs pneumothorax

Cardiac vs primary pulmonary disease

It enhances clinical decision-making while minimizing patient stress.

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BEYOND LUNG SLIDING: KEY CONCEPTS AND PARADIGM SHIFTS FOR LUNG & PLEURAL PATHOLOGY, AND AN INTRO TO CARDIAC POCUS

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Learning objectives:

- Describe PLUS findings used to assess pleural space pathology and understand how patient positioning affects pneumothorax and pleural effusion detection.
- Classify subpleural lesions/lung consolidations and explain why B-lines vs. consolidation occur.
- Describe key cardiovascular findings assessed with cardiac POCUS.
- Interpret cardiac POCUS findings to determine left atrial enlargement and guide furosemide decisions.

Proceeding:

Main Clinical Questions

Point-of-care ultrasound (POCUS) helps answer urgent bedside questions:

Is the collapsed patient in shock and fluid responsive?

Does the dyspneic patient need furosemide (CHF) or alternative therapy (e.g., asthma, pneumonia)?

Is fluid pleural or pericardial?

Is there pneumothorax?

Is left atrial enlargement present?

Is cardiac function reduced?

Pleural and lung ultrasound (PLUS)

Increased B-lines (Alveolar-Interstitial Syndrome, AIS)

B-lines indicate reduced peripheral lung aeration (outer ~3 mm), regardless of cause.

Key concepts:

More B-lines = less aerated lung.

Numerous B-lines may coalesce.

Distribution of B-lines helps determine cause but is not diagnostic alone.

Differentials mirror interstitial-alveolar radiographic patterns.

Prognosis depends on underlying cause (CHF, contusion, pneumonia, ARDS, etc.).

Lung ultrasound detects only peripheral disease (fortunately most clinically relevant diseases reach the surface).

Common B-line Pitfalls

Z-Lines:

Originate from parietal pleura.

Do NOT move with lung sliding.

Do NOT erase A-lines.

Ill-defined, fade within 2-5 cm.

Present in >80% of healthy animals.

E-Lines

Caused by subcutaneous emphysema.

Originate superficial to pleural line.

Pass through and obliterate pleural line.

Do not move with respiration.

Scanning over stomach or curtain sign may mimic B-lines or consolidation.

Pleural Effusion

Appears as hypoechoic fluid between thoracic wall and lung.

Differentiation of intra-thoracic fluid at the pericardio-diaphragmatic (PD) window

Pleural effusion: fills costophrenic recess, tracks diaphragm (mediastinal triangle is lost).

Pericardial effusion: follows contour of the heart (mediastinal triangle preserved).

The presence of lung sliding indicates the pleura are in contact; excludes effusion at that site.

Patient Position Matters

Lateral recumbency: fluid accumulates dependently (often pericardial window).

Sternal recumbency (preferred in dyspnea): fluid accumulates ventrally below pericardial window.

Protocols must be modified accordingly.

Turn the probe parallel to the ribs in the ventral thorax for detection of small volume pleural effusion.

"Ski Jump" sign: pleura in contact = no effusion.

"Sail" sign: triangular fluid pocket between lung and sternum that changes with respiration = pleural effusion.

Pneumothorax

Air rises to non-dependent regions. Positioning matters.

Four Key Findings

Exclusion Criteria - in the absence of a lung point:

Presence of lung sliding (or lung pulse) rules out pneumothorax at that site.

Presence of B-lines or tissue signs excludes pneumothorax at that site.

Inclusion Criteria

Lung point (pathognomonic).

Abnormal curtain signs (double and asynchronous).

Lung Point

Defined as transition between:

Area without lung sliding

Area with lung sliding

Move probe toward lung hilus until sliding reappears.

Not seen in massive pneumothorax (no re-contact).

Lung Consolidation (Tissue-Like Pattern)

Tissue like patterns occur when there is less than 10% air at the lung periphery regardless of the cause:

Atelectasis

Pneumonia

Contusion

Thromboembolism

Neoplasia

ARDS

Diagnostic Criteria of Tissue Signs:

Located in thorax (not liver/spleen).

Arises from pleural line.

Tissue-like (hepatization pattern).

Defined boundaries.

Classification:

Partial consolidation (contacts air):

Irregular air interface = Shred sign

Smooth circular interface = Nodule sign

Translobar consolidation:

Entire lung width involved

Tissue sign/hepatization

Air bronchograms appear as hyperechoic dots/lines within consolidation.

Interpret findings in context of history and distribution.

Cardiac POCUS: Binary Questions

Pericardial effusion present?

Decreased contractility?

Volume depletion or overload?

Left atrial enlargement (LA:Ao ratio)?

Essential Views (Beginner CPOCUS Framework)

Most questions answered with:

Right parasternal short-axis "mushroom" view

Right parasternal LA:Ao view ("Mercedes and whale")

Subxiphoid view

4-chamber long axis (may be more challenging to aquire accurately)

Pericardial Effusion

Easily identified from subxiphoid view.

In dogs: heart normally contacts diaphragm.

In cats: heart may not contact diaphragm unless effusion present.

If LV free wall blends with diaphragm = no pericardial effusion.

If anechoic fluid separates LV free wall from diaphragm:

Fluid arching around cardiac apex = pericardial effusion.

Fluid tracking diaphragm and filling costophrenic recess = pleural effusion.

Always confirm in multiple windows.

Left Atrial Enlargement (LA:Ao Ratio)

Goal: Differentiate left sided congestive heart failure from primary pulmonary disease or pleural effusion.

Left atrial enlargement supports CHF diagnosis in dyspneic patients and informs furosemide decisions.

Right parasternal short-axis; "Mercedes & Whale" View

Normal LA:Ao < 1.5 (cats and dogs).

Gray zone up to ~1.6 (especially cats).

2:1 strongly suggests significant LA enlargement.

Subjective Aortic fit rule:

Normal: 2.5 aortas fit within the LA.

IF 4 aortas fit in LA = severe enlargement.

Key Paradigm Shifts

B-lines do not equal "wet lung" alone - represent loss of aeration from multiple causes.

In the absence of a lung point, lung sliding and B-lines rule out pneumothorax at that site.

The lung point (identified when B-lines and lung sliding become visible) confirms pneumothorax.

Patien positioning dramatically affects pleural space diagnosis.

Pleural vs pericardial effusion is distinguished at key locations.

LA:Ao measurement helps differentiate cardiac vs respiratory dyspnea.

Don't need all windows to answer key CPOCUS questions.

Clinical Integration

POCUS should not replace clinical reasoning but enhance it. Findings must be interpreted in light of:

History

Signalment

Physical exam

Radiographs (when available)

Hemodynamic status

When used systematically, lung and cardiac POCUS allow rapid, bedside differentiation of:

CHF vs pneumonia vs asthma

Pleural vs pericardial effusion

Pneumothorax

Shock states

Volume depletion vs overload

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HEART IN HAND: PRACTICAL CARDIOVASCULAR POCUS TO GUIDE ACUTE CLINICAL DECISIONS

Laura Cole [1], Julie Menard [2]

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Learning objectives:

- Define cardiac point-of-care ultrasound (POCUS) and describe its role as an adjunct to physical examination in emergency and critical care patients.
- Identify appropriate clinical indications for cardiac POCUS in dogs and cats, particularly in dyspnoeic or hemodynamically unstable patients.
- Describe suitable ultrasound equipment, probe selection, and machine settings for performing cardiac POCUS efficiently in the ER and ICU.
- Perform and recognize key cardiac POCUS views and understand their diagnostic relevance.
- Use cardiac POCUS findings to support rapid clinical decision-making, guide emergency procedures, and monitor hospitalized patients, while recognizing its limitations.

Proceeding:

Focused cardiac ultrasound is defined by the American Society of Echocardiography as a focused examination of the cardiovascular system performed by an appropriately trained clinician using ultrasound as an adjunct to the physical examination. The focused assessment, alongside the physical examination helps expediate clinical decision-making. It should be clinically driven to answer key clinical questions to guide subsequent management. Cardiac point-of-care ultrasound is not a replacement for a good physical examination nor a replacement for echocardiography.

Indications

Cardiac POCUS has been described in assessment and management of cardiorespiratory unstable cats and dogs as well as cats with asymptomatic cardiac disease. It is most useful clinically in the emergency setting when utilised with clinical questions in mind, for example does the dyspnoeic cat with a gallop rhythm have congestive heart failure? Does the collapsed large breed dog have a pericardial effusion or dilated cardiomyopathy? Does my cat on high-rate fluids have occult heart disease? With these questions in mind cardiac POCUS can help expediate management of unstable patients in the emergency room. Focused cardiac ultrasound can also assist with life-saving emergency procedures such as pericardiocentesis. Cardiac POCUS, alongside pleural space, lung and abdominal POCUS can be incorporated into the primary survey of any animal presenting to as an emergency as well as a monitoring tool in hospitalized animals. Cardiac POCUS is non-invasive and can be performed safely on most cardiorespiratory unstable patients alongside other diagnostics and intervention such as oxygen supplementation, blood pressure measurement and intravenous catheterization.

Ultrasound machine and functions

Cardiac POCUS can be performed on any machine, however, the smaller and more portable the ultrasound machine the better. This is particularly important in the dyspneic patient, often you want to avoid moving the patient. With a portable machine in may be even possible to scan whilst the animal remains in an oxygen kennel. Various handheld machines are now available making cardiac POCUS more accessible. Echocardiography is performed a high frequency probe with a small footprint, the phased array probe. However, a curvilinear microconvex probe, used for pleural space, lung and abdominal POCUS is perfectly suitable for cardiac POCUS and prevents the need for changing probe between scanning sites, thereby expediating diagnosis and emergency management. The cardiac pre-set available on the ultrasound machine provides a high contrast image of the cardiac chambers and should be selected where possible. However, again for ease and streamlining the complete POCUS examination often the abdominal POCUS pre-set, used for abdominal and lung and pleural space can also be used cardiac assessment. What is important to note is the cardiac preset inverts the image left-to-right, so the indicator marker on the probe that corresponds to the marker on the ultrasound screen is often on opposite sides of the screen dependent on which pre-set you are on. It is important for image interpretation, particularly chamber recognition to recognize if the indicator marker is pointing cranially or caudally when you are scanning and what this corresponds to on the screen. Depth should be adjusted to visualize the entire heart in the majority of the view.

Technique

Positioning and patient preparation: Although cardiac POCUS may be performed in lateral recumbency, standing or sternal recumbency are often safer in dyspneic animals. Often animals tolerate the scan with minimal restraint. Addition of anxolytic, such as 0.2mg/kg butorphanol IM/IV can help facilitate the assessment. Shaving is typically unnecessary unless the patient has a thick fur coat that impedes image resolution. Alcohol is used for skin preparation, but it's important to part the fur before applying alcohol. Alcohol is noxious and flammable and therefore it is recommended to not over-saturate the patient with alcohol and wipe away any excess alcohol. To improve image quality gel may be used but should be applied directly to skin or probe and not the fur as air trapping between the fur can reduce the image quality.

Protocol: Most cardiac POCUS is performed transthoracically. The transducer is placed perpendicular to the ribs at the level of the point-of-maximal intensity of the heartbeat (around intercostal space 5) on the right side of the hemithorax. This site often identifies the parasternal long axis 4 chamber view. The transducer is then rotated clockwise to identify the parasternal short axis at the level of the ventricles identified as a symmetrical short axis of the heart, and then fanned dorsally through several short axis cardiac planes to visualise different chambers; including the right parasternal short axis at the ventricle level (“Mushroom view) and right parasternal short axis at the heart base (left atrium:aorta view) In large dogs the subxiphoid view may be useful to visualise the pericardium and pleural space. The pericardiodiaphragmatic scanning site on lung and pleural space may improve visualisation of the pericardium.

Cardiac POCUS views

Right parasternal long axis four chamber review: This view allows a global assessment of the pericardium, the left and right atria and left and right ventricle. Subjective assessment of contractility could be performed in this view as well as assessment of atrial diameter and assessment of pericardium for detection of a pericardial effusion.

Right parasternal short axis ventricular level "Mushroom view": In this view the left and right ventricle can be evaluated. It is termed the "mushroom" view as the lumen of the left ventricle appears as a mushroom. The primary purpose of this view is assessment of left ventricular contractility and/or wall thickness. Fractional shortening can be calculated using M mode. Once the loop is frozen the left ventricular internal dimension at end diastole (LVIDd) and end systole (LVIDs) are measured. Fractional shortening is calculated using the following equation; FS (%) (LVIDd-LVIDs)/LVIDd x100. FS is often <20% in animals with dilated cardiomyopathy. However, accurate measurement of FS is difficult, particularly on POCUS, and subjective assessment is preferred in the emergency setting. Dogs with DCM the left is often ventricle severely dilated, spherical and markedly hypodynamic.

Right parasternal short axis view at the heart base: In this view the left atrium and aorta can be assessed and the left atrial size evaluated relative to the aorta. The left atrial area can be subjectively assessed by an subjective area assessment; estimating how many "aortas" may fit in the left atrium or linear method; measuring and comparing the diameter of the left artrium:aorta. A left atrium:aorta ratio in a clinical patient >2:1 is highly suggestive of congestive heart failure.

Subxiphoid scanning site: This abdominal POCUS scanning site with sufficient depth allows cardiac long axis 4 chamber view and is useful for assessment of the heart and pericardium particularly in large breed dogs.

Clinical uses

Supporting a diagnosis of congestive heart failure is one the most clinical useful utilities of cardiac POCUS. Cardiac scanning is very sensitive to probe orientation, often the perfect "views" are not achieved during POCUS and therefore the commonly cited left atrium: aorta cut-off of 1:1.6 for diagnosis of left atrial enlargement in cats and dogs cannot be applied to cardiac POCUS in the emergency setting. Usually on cardiac POCUS left atria size is limited to subjective assessment. A good rule of thumb is if mutiple "aortas" can fill the left atria or the left atrial diameter is more then double the diameter of the aorta then animal is likely to have congestive heart failure. Cardiac POCUS can be used alongside lung and pleural space POCUS to further support a diagnosis of congestive heart failure. Lung and pleural space POCUS findings including > 3 B lines in mutiple scanning sites, the presence of pleural effusion and the presence of pericardial effusion can help support the diagnosis of congestive heart failure as a cause of dyspnoea. Cardiac POCUS alongside lung and pleural space ultrasound has a particular useful role at helping expediate emergency diagnosis and treatment of cats with congestive heart failure in cats. However, it should not be used as a replacement for a good history or physical examination. Sometimes history and physical examination, such as exercise intolerance, presence of a murmur/gallop can be enough information to initiate diuretic therapy in a very unstable patient who won't tolerate handling and POCUS can be delayed. Serial assessment of the left atrial/aorta ratio and lung and pleural space POCUS may help in early detection of fluid overload in a hospitalised patient.

The right parasternal long axis view and subxiphoid cardiac long axis view are particularly useful for detection of pericardial effusion and cardiac tamponade. Pericardial effusion is identified as anechoic fluid encircling the heart. Observing the heart from mutiple angles will help differentiate pericardial and pleural effusion. Adjunctive findings at the subxiphoid scanning site include the gall bladder halo sign, distended vena cava and hepatic veins, and abdominal free fluid may further support a diagnosis of pericardial effusion. Neoplastic masses may be visualised on the short or long axis view at the level of the heart base. Alongside its diagnostic utility cardiac POCUS may have a role in assisting pericardiocentesis. Ultrasound could be used to identify the optimal site for pericardiocentesis (ultrasound-assisted) or guide the pericardiocentesis in real time (ultrasound guided).

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NEEDLES WITH VISION: INTERVENTIONAL POCUS

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Learning objectives:

At the end of this 45-minute fundamental lecture, participants will be able to:

- Describe the basic ultrasound principles used to identify veins and arteries for vascular access in veterinary patients.
- Differentiate veins from arteries using sonographic characteristics including compressibility and wall appearance.
- Identify appropriate clinical indications for ultrasound-guided vascular access and arterial cannulation.
- Explain the principles, advantages, and limitations of in-plane and out-of-plane needle guidance techniques.
- Select an appropriate needle guidance approach based on vessel characteristics and clinical context.

Proceeding:

Vascular access and arterial cannulation are core procedures in veterinary medicine, performed daily in emergency, critical care, anesthesia, and internal medicine. Despite their routine nature, these techniques can be technically challenging, particularly in patients that are hypotensive, hypovolemic, obese, very small, or critically ill. In such situations, traditional landmark-based approaches may result in multiple failed attempts, delayed treatment, and an increased risk of complications such as hematoma formation, accidental arterial puncture, thrombosis, or infection.

Point-of-care ultrasound (POCUS) has become an increasingly accessible and valuable tool in veterinary practice. When applied to procedural guidance, interventional POCUS allows real-time visualization of vascular structures and needle advancement, converting blind techniques into controlled, image-guided interventions. This approach improves first-attempt success rates and enhances procedural safety, especially in high-risk or technically difficult patients.

This 45-minute fundamental lecture provides an introduction to the principles of interventional POCUS for ultrasound-guided vascular access and arterial cannulation in veterinary patients. The session begins with a review of basic ultrasound concepts required for vessel identification, including recognition of veins and arteries based on wall appearance, compressibility, and flow characteristics. Practical strategies to avoid common misidentification errors will be discussed, with emphasis on simple, reproducible techniques suitable for everyday clinical use.

Participants will then be introduced to the two primary needle guidance techniques used in interventional ultrasound: the **in-plane** and **out-of-plane** approaches. The lecture will explain how each technique is performed, their respective advantages and limitations, and how to choose the

most appropriate approach based on vessel size, depth, and clinical context. Special attention will be given to maintaining needle tip visualization and understanding common pitfalls that may lead to procedural failure or complications.

The session also addresses the use of ultrasound guidance for arterial cannulation, a procedure increasingly employed for invasive blood pressure monitoring and arterial blood sampling. Participants will learn how ultrasound can assist in identifying arteries, confirming flow, and improving accuracy during catheter placement, even in patients with reduced pulsatility.

Designed for veterinarians with limited prior experience in interventional ultrasound, this lecture focuses on building a strong conceptual foundation rather than advanced technical proficiency. By the end of the session, participants will have a clear understanding of when and why ultrasound guidance should be considered, how to recognize vascular structures, and how interventional POCUS can be safely integrated into routine clinical practice to improve patient care and procedural confidence.

FROM SYMPTOM TO SCAN: CASE BASED POCUS

Laura Cole [1]

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Learning objectives:

By the end of this lecture, participants should be able to:

- Describe appropriate equipment and patient positioning for POCUS
- Select POCUS scanning sites based on suspected aetiology
- Recognise key pleural, lung, cardiac, and abdominal POCUS findings
- Integrate POCUS results with clinical assessment to guide emergency decision-making

Proceeding:

Point-of-care ultrasound can be performed on any ultrasound machine, with any transducer. However, the more portable and durable the ultrasound machine the better as it needs to be moved to the patient and withstand regular usage. Hand-held devices that attach to a tablet or mobile phone are now available which improves the accessibility of POCUS. Out of the three main ultrasound probes available, curvilinear, linear and phased array, the curvilinear probe is the most suited for POCUS of all regions (pleural space, lung,cardiac and abdomen). This probe has a good balance between frequency (resolution of the image) and penetration depth. Furthermore, the footprint of the transducer is small so it can fit intercostally. POCUS is particularly useful in patients presenting cardiorespiratory unstable.

The original POCUS techniques were performed in lateral recumbency. However, POCUS should be performed in whatever position the animal is comfortable in. In a patient with respiratory distress this is usually sternal or standing. Dorsal recumbency should be avoided, The fur does not, and often should not be, clipped if it would cause stress to the patient. Instead, the fur is parted over the site of interest, alcohol applied directly to the skin and coupling gel applied to the transducer. Often a disposable ultrasound probe cover is placed over the transducer to protect it from alcohol damage.

Prior to performing POCUS priority should be given to initial emergency stabilisation. In a cardiorespiratory unstable patient this would often be oxygen supplementation and analgesia or anxiolytic. If the animal is severely dyspnoeic and there is concern for a large volume pneumothorax or pleural effusion diagnostic and therapeutic thoracocentesis should be performed prior to POCUS. However, most often, POCUS can be performed safely in patients receiving oxygen supplementation whilst other interventions are being performed, such as intravenous catheterization, crystalloid fluid bolus and oxygen supplementation.

POCUS (pleural space, lung, cardiac and abdominal) can be incorporated into a primary survey of any animal presenting to as an emergency as well as a monitoring tool in hospitalised animals

POCUS should be clinically driven and used alongside history and physical examination. Having a clinical question in mind is useful, for example is this tachycardic dog post road traffic accident have

evidence of internal haemorrhage, Does the post-operative regurgitating dog have ileus? Is the azotaemic cat producing urine? Does the dyspnoeic dog with a murmur in congestive heatt failure? Has the previously vomiting dog that is now dyspnoeic aspirated?

The priority of POCUS region (abdominal, pleural space and lung or cardiac) should be driven by the clinical question as well as the POCUS technique. There are various lung and pleural space POCUS protocols, including the TFAST™, VetBLUE™, ABCDE, Vertical sliding, PLUS protocol which have between 4-19 scanning sites. It is useful to perform a standardised scanning protocol for repeatability and record keeping but number of scanning sites may vary depending on patient size, stability and suspected aetiology. For example, if pleural effusion is clinically suspected the transducer must be placed ventrally where fluid is likely to accumulate. It is particularly important to streamline scanning technique in a cardiorespiratory unstable patient.

POCUS interpretation

Findings on POCUS should be interpreted in light of the history and physical examination as well as other diagnostic tests. It is particularly important to interpret pleural space and lung POCUS considering the complete clinical picture as the majority of normal and abnormal sonographic finding seen on pleural space and lung POCUS are artefacts because of the ultrasound beam hitting air or an air-soft tissue interface. B lines, are hyperechoic laser-beam artefacts that extend from the pleural line to the far periphery obliterating the normal A-line artefact and move with respiration. A B line represents an area of decreased aeration at the lung periphery (1-3mm), which is often a consequence of infiltration of the alveoli and/or interstitium with fluid or cells (inflammatory or neoplastic) but may also occur secondary to atelectasis. The differential diagnosis for B lines is based the clinical picture. In a patient with respiratory distress and concern for congestive heart failure they may represent cardiogenic pulmonary oedema. A greater number of B lines (total B-line score ≥ 10 and presence of ≥ 2 sites strongly positive for B lines) increases the likelihood of cardiogenic pulmonary oedema. B-lines have been reported in 10-30% of healthy animals, but these are often limited to 1-2 B lines in few scanning sites. Other tissue-like patterns such as shred, nodule and wedge sign may be identified.

Subjective assessment of left atrium/aorta ratio alongside other cardiac and lung and pleural space POCUS findings including > 3 B lines in multiple scanning sites, the presence of pleural effusion and the presence of pericardial effusion can help support the diagnosis of congestive heart failure as a cause of dyspnoea alongside history (e.g exercise intolerance) and clinical findings (e.g presence of murmur/gallop in the emergency setting. Cardiac POCUS has a particular useful role at helping expediate emergency diagnosis and treatment of CHF in cats and may be an adjunctive test in dogs but does not replace good history or physical examination.

Cardiac POCUS findings identified at parasternal short axis view alongside focused abdominal scanning at the subxiphoid site can be helpful in the diagnosis of moderate-severe pulmonary hypertension. Right heart changes, including right ventricular hypertrophy or enlargement of the right atria and ventricle, intraventricular septal flattening, abdominal effusion and a distended non-compliant caudal vena cava should prompt consideration for pulmonary hypertension contributing to the clinical signs.

Cardiac, PLUS and abdominal POCUS are invaluable techniques in the emergency room. The chosen technique and interpretation require integration based on history, triage assessment and should always be clinically driven.

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Main Stream, Thursday 4 June 2026

CORNEAL AND EYELID EMERGENCIES

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Learning objectives:

- Identify true corneal emergencies on presentation
- Differentiate painful vs vision-threatening disease
- Initiate appropriate immediate treatment for corneal injury, eyelid trauma, and globe proptosis
- Know when delay causes permanent harm

Proceeding:

Introduction

Corneal and eyelid disease represent some of the most time-critical ophthalmic presentations encountered in emergency and critical care practice. While many cases initially present with similar clinical signs (e.g. ocular pain, blepharospasm, discharge, corneal opacity), the consequences of delayed or inappropriate management vary dramatically. This lecture focuses on rapid recognition of true corneal and periocular emergencies, early stabilisation, and the practical steps that can be taken in the emergency setting to preserve vision and patient comfort.

Corneal ulcers

Corneal ulceration is among the most common ophthalmic emergencies, yet the severity of disease is variable and may be underestimated. Superficial ulcers may be painful but are rarely vision-threatening, whereas deep stromal ulcers, descemetocoeles, and corneal perforations represent genuine emergencies requiring immediate action. Identification of a descemetocoele is critical, as these lesions carry a high risk of imminent rupture. Fluorescein staining remains a cornerstone of assessment, with a focal area of non-uptake surrounded by fluorescein uptake indicating exposure of Descemet's membrane. In these cases, referral for surgical intervention should not be delayed.

Keratomalacia

Melting keratitis is another rapidly progressive condition in which early intervention can significantly influence outcome. Proteolytic degradation of the corneal stroma may lead to globe rupture within hours. Frequent topical antiprotease therapy, combined with appropriate antimicrobial coverage and analgesia, is essential to slow stromal loss prior to referral. Systemic non-steroidal anti-inflammatory drugs may improve comfort.

Chemical injuries

Chemical injuries to the eye are often dramatic and deceptively challenging. Alkali burns penetrate ocular tissues rapidly and cause ongoing damage long after initial exposure. Immediate and copious irrigation is the most important intervention and should be initiated as soon as possible, even before

complete examination. Delay in decontamination markedly worsens prognosis, highlighting the need for decisive action in the emergency setting.

Eyelid trauma

Eyelid trauma, including lacerations and avulsions, may appear minor but can have significant functional consequences if improperly managed. Accurate apposition of eyelid margins is essential to prevent chronic exposure keratopathy and corneal ulceration. Temporary measures, including lubrication and protection of the ocular surface, are critical until definitive repair can be performed.

Proptosis

Globe proptosis represents one of the more visually intimidating ocular emergencies. Prognosis is influenced by several factors, but avulsion of multiple extraocular muscles is strongly associated with poor visual outcome. Rapid assessment of globe viability, lubrication to prevent corneal desiccation, and prompt replacement or referral are essential. Even when vision cannot be salvaged, timely intervention can save the globe and significantly improve patient comfort.

Analgesia

Across all corneal and eyelid emergencies, effective analgesia is both a welfare and a diagnostic priority. Ocular pain can be severe, and systemic opioids are often required. Topical anaesthetics may facilitate examination but should never be dispensed for ongoing use due to their deleterious effects on corneal healing.

Summary

By focusing on early recognition, appropriate first-line therapy, and timely referral, emergency clinicians can play a pivotal role in preventing irreversible vision loss. Understanding which cases demand immediate action, and which can be stabilised safely, is a core competency in emergency ophthalmic care.

INTRAOCULAR EMERGENCIES

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Learning objectives:

- Identify clinical features suggestive of intraocular emergencies
- Distinguish between common causes of red eye and acute vision loss
- Initiate appropriate early management for uveitis, glaucoma, and intraocular haemorrhage
- Avoid common diagnostic and therapeutic errors in the emergency setting

Proceeding:

Introduction

Intraocular disease may be under-recognised in emergency patients. Many intraocular emergencies present with nonspecific signs such as red eye, corneal oedema, or reduced vision, potentially leading to misdiagnosis or delayed treatment. This lecture reviews the recognition and initial management of common intraocular emergencies, with an emphasis on practical triage and early decision-making.

Glaucoma or uveitis?

Acute glaucoma and anterior uveitis are often confused, yet their management priorities differ substantially. Ideally any “red eye” should have the intraocular pressure measured. While both conditions tend to present with a painful, hyperaemic eye, a patient with a mid-dilated pupil with corneal oedema favours a diagnosis of acute glaucoma and should prompt immediate efforts to reduce intraocular pressure. In contrast, uveitis is typically associated with miosis, aqueous flare, and lower intraocular pressure. Failure to differentiate between these conditions can result in inappropriate therapy and rapid vision loss. In reality, glaucoma and uveitis frequently present together, presenting a significant diagnostic and therapeutic challenge.

Hyphaema

Hyphaema may arise from ocular trauma, systemic disease, or coagulopathy. Identifying the underlying cause is essential; bilateral involvement or concurrent retinal haemorrhages should raise concern for a systemic aetiology. In these cases, management extends beyond the eye, and systemic stabilisation and investigation take precedence.

Lens luxation

Lens luxation represents a true ophthalmic emergency, particularly when the lens is displaced into the anterior chamber. Elevated intraocular pressure, corneal oedema, and acute pain may develop rapidly, with the cornea oedema sometimes making diagnosis challenging. An ocular ultrasound can be useful to show an anteriorly displaced lens. The use of topical prostaglandin analogues in these

cases is contraindicated, as pupillary constriction may trap the lens in the anterior chamber, exacerbating obstruction of aqueous outflow. Early recognition and referral are therefore critical.

Retinal detachment

Not all intraocular emergencies present with marked pain. Retinal detachment, for example, may result in profound vision loss with surprisingly minimal external ocular discomfort, underscoring the importance of assessing visual function and fundic appearance.

Trauma

Sharp or blunt-force trauma may result in serious consequences such as globe rupture, lens rupture, retinal detachment or intraocular haemorrhage. Excessive manipulation and delays in treatment may worsen outcome. Emergency management should focus on preventing further damage (e.g. lubrication, buster collar, sedation), analgesia, and swift referral.

Summary

Ultimately, the emergency clinician's role is not to definitively manage every intraocular condition, but to recognise when time is critical, initiate appropriate early therapy, and avoid common pitfalls. Prompt intervention, combined with timely referral, can make the difference between reversible and permanent vision loss.

PRACTICAL WOUND CARE AND TRICKY WOUND CLOSURE TECHNIQUES

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Learning objectives:

- Detail initial triage and stabilsation of patients with traumatic wounds
- Describe how to initially manage wounds including lavage and debridement
- Know the advantages/ disadvantages as well as how to apply dressings
- Know key factors deciding when surgical management is appropriate
- Key tips for optimizing surgical management

Proceeding:

This lecture outlines a structured approach to the assessment and management of trauma patients with wounds, emphasising prioritisation of life-threatening problems before definitive wound care. Initial triage of all acute trauma patients should follow a major body systems approach, ensuring airway patency, adequate breathing, and effective circulation. If any component is absent, stabilisation is required.

Cardiovascular assessment includes heart rate, mucous membrane colour, capillary refill time, pulse quality, and blood pressure measurement, ideally using Doppler sphygmomanometry. Trauma patients commonly develop hypovolaemic shock due to blood loss, presenting with tachycardia, pallor, and weak pulses, though cats may show bradycardia in specific circumstances. Initial fluid resuscitation with isotonic crystalloids (5–10 ml/kg over 10–15 minutes) is recommended, followed by reassessment. Concurrently, the underlying cause of shock should be investigated, with point-of-care ultrasound playing a key role in detecting haemothorax or haemoperitoneum.

Respiratory assessment can be challenging, as stress may cause tachypnoea or open-mouth breathing. Flow-by oxygen should be administered early as it is unlikely to cause harm. Upper airway noise may occur with facial trauma but is not always clinically significant. More serious conditions such as pulmonary contusions, diaphragmatic rupture, and pneumothorax can lead to hypoxaemia and respiratory compromise. These conditions may be identified by physical examination and ultrasound findings, and while oxygen therapy is useful initially, prompt recognition and treatment of pneumothorax with thoracocentesis is essential.

Neurological assessment is often brief, focusing on mentation and ambulation. If abnormalities are detected, further evaluation is required. Traumatic brain injury is relatively common, particularly in cats, and the Modified Glasgow Coma Scale (MGCS) is used to assess and monitor severity. Although not perfect, it allows consistency between clinicians and can guide decision-making. Hyperosmolar therapy should be considered in cases of suspected raised intracranial pressure, particularly with severe neurological impairment or deterioration, after perfusion, oxygenation, and basic blood parameters have been optimised. Either hypertonic saline or mannitol may be used, alongside supportive measures such as head elevation and avoidance of jugular venepuncture.

Once the patient is stabilised, the focus can be on wound management. Initially, wounds should be covered to prevent further contamination, and definitive treatment delayed until the patient is stable. Although prompt and appropriate analgesia is important, wound lavage and assessment should be performed as soon as possible under sedation. If appropriate, general anaesthesia can allow more thorough assessment and debridement. If primary closure is not possible, due to concerns over skin viability, contamination or infection, open wound management should be performed with the goal of establishing a healthy granulation tissue bed.

Assessment of tissue viability is critical. Necrotic skin appears pale, grey, or black and may feel thin or form a firm eschar. Limb wounds require assessment of distal perfusion, using Doppler examination or evaluation of bleeding from pads or nails. Shock, hypothermia, or vascular injury can complicate this assessment. Certain orthopaedic injuries may compromise blood supply and require urgent reduction. Skin avulsions are particularly challenging, as necrosis may take several days to become apparent, necessitating careful monitoring and owner communication.

Surgical debridement follows clear principles: non-viable skin, fat, and muscle should be removed, while nerves and vessels should be preserved whenever possible. Muscle viability is assessed by colour, texture, and contractility. Actively bleeding vessels should be ligated, and joints or tendons should be lavaged, repaired, and immobilised as appropriate. When tissue viability is uncertain, staged daily debridement is preferred to avoid unnecessary tissue loss.

Dressings are selected based on wound stage and exudate level. Initially, non-selective debridement dressings such as wet-to-dry or dry-to-dry are commonly used for heavily contaminated wounds, the disadvantages being increased patient discomfort and need for frequent changes under sedation or anaesthesia. An alternative would be a hydrocolloid gel, however, this may be less efficient at debridement. As the wound improves and granulation tissue forms, more advanced dressings such as foams or hydrocolloid gels are used to maintain a moist environment and reduce dressing frequency. Negative pressure wound therapy is an option for highly exudative or infected wounds following adequate debridement.

Wound lavage is an essential component of care. While tap water may be used initially for gross contamination, sterile isotonic fluids are preferred thereafter. Modern antimicrobial solutions such as PHMB (tradename Prontosan) are increasingly used due to their efficacy and minimal impact on healing tissues.

Surgical closure is considered once the wound is clean, viable, infection-free, and has sufficient healthy tissue, or when function would be compromised by contraction or scarring. Throughout management, clear communication with owners is vital, as wound care is time-consuming, costly, and requires frequent reassessment, but can ultimately yield excellent outcomes.

SURGICAL MANAGEMENT OF UPPER AIRWAY CRISES

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Learning objectives:

- Recognise an upper respiratory tract crisis
- Key steps to stabilising patients with URT crisis
- Surgical procedures for BOAS and laryngeal paralysis
- Post-operative considerations for patients after URT surgery
- Placement and management of temporary tracheostomy tubes

Proceeding:

Recognising an Upper Respiratory Tract (URT) Crisis

A URT crisis occurs when airflow is critically obstructed at or above the larynx, leading to rapid respiratory distress and potential respiratory arrest. Early recognition is vital. Clinical signs typically include inspiratory dyspnoea, stridor, increased respiratory effort, extended head and neck posture, cyanosis, and anxiety or agitation. Affected animals may pant excessively, collapse, or show worsening distress with handling or stress.

Common causes include brachycephalic obstructive airway syndrome (BOAS), laryngeal paralysis, upper airway trauma, foreign bodies, neoplasia, inflammation, or post-extubation laryngeal oedema. Progression can be rapid, with hypoxia, hypercapnia, and secondary pulmonary oedema developing if obstruction is not relieved promptly.

Key Steps to Stabilising Patients with a URT Crisis

Stabilisation focuses on minimising stress, maintaining oxygenation, and relieving airway obstruction. Patients should be handled gently and kept calm; excessive restraint can precipitate complete airway collapse. Supplemental oxygen should be provided immediately, ideally via flow-by or oxygen cage to reduce stress.

If anxiety or agitation worsens respiratory effort, light sedation (e.g. opioids, low-dose acepromazine or dexmedetomidine in selected cases) can significantly improve airflow by reducing panic and upper airway resistance. Corticosteroids may be administered to reduce laryngeal or pharyngeal oedema are suspected (e.g. BOAS crisis).

Active cooling may be necessary in hyperthermic patients, particularly brachycephalics. If the patient cannot maintain adequate ventilation or deteriorates despite conservative measures, advanced airway control is required. This may include endotracheal intubation, temporary tracheostomy, or emergency surgical airway access. Intubation can be challenging due to distorted anatomy or swelling, and preparation for immediate tracheostomy is essential in high-risk cases.

Surgical Procedures for BOAS and Laryngeal Paralysis

Surgical intervention is often required to provide definitive airway relief.

BOAS surgery aims to reduce airway resistance at multiple levels. Procedures commonly include:

- Rhinoplasty to widen stenotic nares
- Staphylectomy to shorten an elongated soft palate
- Sacculectomy to remove everted laryngeal saccules

These procedures improve airflow and reduce the negative pressure contributing to progressive airway collapse. Surgery is ideally performed early to prevent irreversible changes such as laryngeal collapse.

Laryngeal paralysis is typically treated with unilateral arytenoid lateralisation (tie-back). This procedure permanently abducts one arytenoid cartilage to enlarge the rima glottidis, improving airflow while minimising aspiration risk. Bilateral procedures are avoided due to a high risk of aspiration pneumonia.

Post-Operative Considerations After URT Surgery

Post-operative management is critical, as many complications occur during recovery. Airway swelling, laryngospasm, haemorrhage, and aspiration pneumonia are key concerns. Patients should be closely monitored in a quiet, oxygen-rich environment with minimal stimulation.

Extubation should be delayed until the patient is fully awake and able to protect its airway. Some patients may require temporary re-intubation or tracheostomy if post-operative swelling compromises airflow. Corticosteroids, analgesia, and continued sedation may be necessary to reduce inflammation and stress.

Feeding is often delayed post-operatively, particularly after laryngeal surgery, to reduce aspiration risk. Long-term management includes weight control, exercise moderation, and avoidance of heat or stress. Owners should be counselled that surgery improves quality of life but may not completely resolve clinical signs.

Placement and Management of Temporary Tracheostomy Tubes

Temporary tracheostomy is a life-saving procedure for patients with severe URT obstruction unresponsive to medical management. Placement involves creating a surgical opening between tracheal rings, typically in the mid-cervical region, and inserting an appropriately sized tube.

Post-placement care is labour-intensive and essential for success. Tubes must be kept patent, as mucus plugging is the most common and life-threatening complication. This requires frequent suctioning, humidification, and regular cleaning or tube changes. The stoma site should be monitored for infection, swelling, or dislodgement.

Patients with tracheostomy tubes require continuous observation, particularly in the first 24–48 hours. Sedation may be necessary to prevent tube removal. Once upper airway swelling resolves or definitive surgery has been performed, the tube can be removed, and the stoma allowed to heal by second intention.

In summary, URT crises are true emergencies requiring rapid recognition, calm stabilisation, and timely intervention. Successful outcomes depend on early airway support, appropriate surgical

management when indicated, meticulous post-operative care, and diligent tracheostomy management when required.

IMPROVING TRAUMA PATIENT OUTCOMES: THE VETATLS

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Learning objectives:

- Apply the VetATLS framework for approaching veterinary trauma patients
- Identify and apply resources that can be leveraged to reduce cognitive load during trauma patient stabilization
- Describe how hospital preparation facilitates rapid resuscitation of trauma patients
- Explain the principles of the primary survey and interventions that may be immediately required
- Identify the components of a secondary survey, including adjuncts that may be appropriate during its performance and pitfalls

Proceeding:

Hospital readiness

A theme throughout trauma care is the term “resources”. This refers to both physical supplies (e.g., catheters, fluids, surgical equipment) and people (veterinary nurses, Emergency doctors, specialists, support staff). While it is not feasible (nor necessary) for all veterinary facilities to have all resources a trauma patient may ultimately require (e.g., CT, surgeon, 24-hour care), it is important that all veterinary facilities are prepared for the initial assessment and stabilization of any small animal trauma patient. Traumatic injury is very common in dogs and cats, and a subset sustain severe injuries that require a prepared team to assess and intervene immediately to prevent additional morbidity and mortality. One recommendation is to have a physical “trauma bay” or “resuscitation area” established in all facilities that includes supplies necessary in high acuity situations to include a table or other surface for the patient with room for team members to access the patient from multiple angles in addition to a crash cart, toolbox or other container that is restocked after each use with tools necessary for stabilization of any high acuity case. While there is no one definitive list of supplies, an example created by the Veterinary Committee on Trauma (VetCOT) Trauma Support Team Subcommittee can be found here: https://vetcot.org/category/trauma-support-best-practices/.

A second component of trauma care that has a well-documented impact on patient outcome in human trauma literature is the effectiveness of the team utilizing the resources via effective communication, teamwork, and application of locally derived processes. Team development interventions (TDIs) are researched in various settings, including medical teams, and show effectiveness in minimizing errors and improving outcomes. The RECOVER initiative’s online and scenario/simulation-based course is a great model for including concepts of situational awareness, cross-monitoring and leadership skills interwoven into knowledge and skills training for performing cardiopulmonary resuscitation (CPR). The Veterinary Advanced Trauma Life Support (VetATLS) will

similarly model elevating communication, teamwork, and leadership skills, and provide resources that teams can "take with them" to continue enhancing their teams' effectiveness in high acuity situations. Additionally, the VetCOT is working to establish a series of "Clinical Practice Guidelines" (CPGs) for veterinary practitioners on various clinical scenarios with respect to trauma patients, modeled after both the Eastern Association for the Surgery of Trauma Practice Management Guidelines (https://www.east.org/education-career-development/practice-management-guidelines) and the military's working dog guidelines intended for non-veterinary medical providers (https://jts.amedd.army.mil/assets/docs/cpgs/Military_Working_Dog_CPG_12_Dec_2018_ID16.pdf). The intent is that the VetCOT created CPGs can be adapted for various resourced facilities and used as a tool to help the veterinary team's preparation for assessing and addressing high acuity trauma patients.

A third component of preparation is identifying the network of other veterinary facilities in the region that may provide additional resources if not available to the team triaging the acutely injured patient. Anecdotally, it seems the consequences of higher caseloads and lower staffing seen in conjunction with COVID resulted in many regions taking the proactive steps to work together to match patients with resources (rather than competing for patients) – highlighting, again, that resources include physical tools (e.g., cage space) and people (e.g., nursing care team). Identifying under which circumstances a patient may need to be transported to a facility with additional resources and having an established system for transferring patients is critical for the patient, clients, and teams at both ends of the care continuum.

Primary survey

Leveraging the concept of improving outcome with checklists, Advanced Trauma Life Support (ATLS) methodology, and seeking common language across the medical community, the VetATLS utilized the XABCDE as the "checklist" approach to the acutely injured patient. Influenced both by the military experience ((M)MARCH) and European trauma networks, prioritizing attenuation of pulsatile, life-threatening hemorrhage as first step, then moving on to the more traditional ABCDE is recommended. It should be noted that while the "steps" are intended to imply priority order, the reality of many clinical situations is a team concurrently addressing multiple steps (e.g., thoracocentesis and intravenous (IV) catheter placement at the same time by separate team members). Again, the goal with the acronym is to have a mental checklist, in order of life-threatening priority, as the patient is evaluated, and any time the patient's status changes for the worse during (and after) the resuscitation period. Getting through the whole checklist assessment (and interventions) may take as little as 15 seconds with a minorly injured patient, and as long as 3-5 minutes in a patient that requires interventions at one or more of the steps.

X (eXsanguination): This step is primarily 3 components: visualization, hand sweep and verbalization acknowledging "yes/no" regarding pulsatile bleeding identified. If frank, red, pulsatile blood is identified, one team member addresses (e.g., hemostats, bandage, tourniquet if distal limb, etc.) while another teammate moves onto the next step.

A (Airway): The central assessment goal is to determine if the patient has a patent airway. Abnormal findings could be a result of direct trauma to or obstruction of the large airways preventing movement of air. The assessment is achieved primarily by visual and auditory exam +/- gentle palpation. Based on findings, intervention may include obtaining an airway (endotracheal intubation, tracheotomy, cricothyroidotomy) and oxygen supplementation.

B (Breathing): The primary goal here is to determine the patient's ability to move air through the airway (assessed in "A"). If a patient is nonresponsive with a patent airway and is not breathing, the RECOVER initiative recommends initiating Basic Life Support (BLS) with high quality chest compressions and intubation and ventilation, followed by Advanced Life Support (ALS). It's important to note that volume replacement (ideally blood product) is recommended in cardiopulmonary resuscitation (CPR) secondary to exsanguination. If the patient is breathing, but has inadequate respiratory effort, auscultation and thoracic point of care ultrasound (TFAST) can be leveraged to help identify the cause, and interventions may include thoracocentesis, chest tube placement, oxygen supplementation and/or sedation, intubation and ventilation.

C (Circulatory): Evaluation of mucous membrane color, capillary refill time (CRT), pulse (rate and quality), distal extremity "warmth" and mentation are all part of assessing for evidence of circulatory shock. It is during this step that abnormalities or concern for shock are addressed by obtaining intravenous access, and typically obtaining a blood sample to measure packed cell volume and total protein, lactate, and venous blood gas (if available). Identification of internal bleeding will be assessed here, typically via point of care ultrasound (AFAST/TFAST/GlobalFAST).

D (Disability): The primary goals with this step are to quantify neurologic capacity, ideally utilizing the mGCS score, and an assessment for spinal injury by palpation along the spinal column and assessing peripheral limb sensation. A target is to administer pain relief *after* the patient's sensory and central nervous system has been assessed. Interventions may include alternate fluid choices for suspected traumatic brain injury (TBI), head elevation above the heart, oxygen supplementation and possibly backboard restraint if spinal trauma, fracture, or luxation is suspected.

E (Exposure/Environment): The goals for this step are to temporarily address any open wounds or exposures (preventing further contamination by covering with sterile material), minimize ongoing heat loss, and addressing any patient distress.

Secondary survey

The secondary survey begins only *after* the primary survey is completed and any related interventions have been initiated or completed. The recommendation is to sequentially and thoroughly evaluate the patient's head, thoracic and cervical region, abdomen, pelvis and perineum, neurologic system, and limbs for additional injuries that may require intervention. Adjuncts to perform this assessment include the veterinarian's eyes, hands, ears (physical exam) and possibly point of care ultrasound (AFAST, TFAST, GlobalFAST), radiographs, CT, reflex hammer, otoscope, laryngoscope, ophthalmoscope and/or otoscope. Interventions are based on findings, and may include abdomino- or thoracocentesis, additional pain relief, additional fluids, definitive repair of soft tissue or orthopedic injuries, etc. It is at this point that the Animal Trauma Triage (ATT) score is calculated to help assess severity of injury and inform likelihood of additional interventions beyond the initial triage period.

Reassessment and Disposition

Any time a patient's status changes after initial resuscitation, restarting the evaluation process with the primary survey is warranted. Trauma patients, particularly with polytrauma and/or crushing injury, can be dynamic and require additional interventions to (re)stabilize even after an initial stabilization has occurred. As noted above, there may be a subset of patients assessed by the trauma team that require resources that are unavailable at the facility where the patient has arrived. This is where the efforts to establish a clinic/hospital network pays off for the team and patient. It is also recognized the client resources may impact ability to pursue additional care and management for advanced injuries.

Acknowledgements

The Veterinary Advanced Trauma Life Support (VetATLS) will be fully released in 2026. The creation of the course is a result of the contributions of many, all of whom will be listed and acknowledged in the final VetATLS program. I would like to thank the Module 1 contributors, whose joint efforts I've leveraged here, for the incredible collaboration in developing this important work to enhance confidence and competence in all veterinary trauma care providers.

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RESUSCITATION OF THE ACUTELY HAEMORRHAGING PATIENT

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Learning objectives:

By the end of the session, the learner will be able to:

- Describe pre-clinical and human research-based recommendations for resuscitating acutely hemorrhaging small animal patients, with a focus on dogs, and some on cats
- Identify potential barriers to implementation, as well as opportunities for barrier reduction regarding resuscitation recommendations for the acutely hemorrhaging patient
- Identify clinical markers for determination of need for hemostatic resuscitation in dogs

Proceeding:

CASE PRESENTATION

An 8-year-old, male neutered German Shepherd dog, *Joust*, arrives to your clinic for acute "lethargy". He was missing for a few hours and returns home right after the UPS truck dropped a package off. He "seems off" and "wobbly" to his owners, so they bring him straight to your clinic (they are long time clients with a multi-pet household and donated $3 million to your new hospital wing). Joust was diagnosed with immune-mediated thrombocytopenia 2 years ago (weaned off medications 12 months ago), has access to anti-coagulant rodenticide (lives on an old farm), and loves chasing the horses on the property.

On primary survey, he has no evidence of external red pulsatile bleeding (X), he has no evidence of abnormal airway sounds (A), and he has a rapid shallow breathing pattern with normal lung sound (B). His mucous membranes are pale pink/capillary refill time is 2-3 seconds; he is tachycardic (HR 160) with weak femoral pulses (C). You verbalize to your team that *Joust* is in shock and request your team to place an intravenous catheter (IVC) and to start an intravenous fluid bolus of…*** while you move on to the rest of your primary survey (D, E: dull mentation, toe sensation x 4; no evidence of open wounds, rectal temperature=99°F/37.2°C). On secondary survey, abdominal Point of Care Ultrasound (POCUS) reveals an abdominal fluid score (AFS) of 3, and no evidence of pericardial effusion through the DH view. Fluid obtained on abdominocentesis appears to be frank blood (PCV and protein levels – pending). Joust collapses as the IVC is finished being secured by your team.

***What fluid is chosen first? Next? Why?

***What bedside tests might help inform your choice?

INTRODUCTION

Hemorrhagic shock in small animal patients (dogs/cats) can loosely be divided into three mechanisms: traumatic (non-coagulopathic or coagulopathic), spontaneous non-traumatic (non-

coagulopathic or coagulopathic), or primary hemostatic disorders.[1] While identification of the mechanism for acute hemorrhage is important for definitive management of the patient, the approach to volume resuscitation to address acute blood-loss is similar across all three mechanisms: cardiovascularly stabilize the patient first. Much of the research available regarding resuscitation strategy in a patient in hemorrhagic shock is through the lens of traumatic injury, which in most cases is a result of both blood loss and tissue injury. Historically, crystalloids and synthetic colloids were developed to aid packed red blood cells (pRBCs) in volume expansion in hemorrhagic shock; however, more recent literature identifies complications associated with large volume resuscitation with these fluids including endothelial/glycocalyx injury, dilutional coagulopathy, coagulation and platelet function impairment, and renal impairment. This presentation will explore current evidence and recommendations for the stabilization of the acutely hemorrhaging small animal patient, with a focus on dogs.

HUMAN CLINICAL RESEARCH

Hemostatic resuscitation is the term used in human medicine for stabilization of the acutely hemorrhaging patient – typically associated with traumatic injury (formally coined, "Damage Control Resuscitation" or DCR). This involves triage (identification of the acutely bleeding patient), temporary hemorrhage control, massive transfusion, permissive hypotension (except if concurrent traumatic brain injury), avoidance of fluids/dilutional coagulopathy, treating established coagulopathies, and definitive hemorrhage control.[2] Current recommendations regarding volume resuscitation of the acutely hemorrhaging patient are heavily informed by pragmatic studies from US military experiences in the 1990s and early 2000s. A significant survival advantage was identified in soldiers that required massive transfusion when the ratio of plasma:pRBCs was < 1:2.[3] These findings were evaluated in a series of multi-center civilian clinical trials that verified findings (PROMMTT, PROPPR).[4,5] For years, resuscitation recommendations in the Advanced Trauma Life Support manual recommended 2L of 0.9% NaCl for pre-hospital resuscitation of bleeding trauma patients. Based on hospital-level shifts in resuscitation recommendations, 2 subsequent studies evaluated administration of prehospital plasma during transport (PAMPR, COMBAT).[6,7] Both studies investigated pre-hospital administration 2 U of plasma compared with crystalloid-based resuscitation, and in post-hoc analysis, it was determined that plasma transfusion reduced 28-day mortality (compared to crystalloid-based) when transport times were > 20 minutes.

Identification of the patient population that would benefit from transfusion resuscitation (or massive transfusion) is understood to be critical, including timing of administration. For example, the single center COMBAT study results indicated no benefit to plasma resuscitation in regions where transport to definitive care would be < 20 minutes (as in many urban areas) and discouraged supplying plasma to transport vehicles acknowledging the resulting additional need to manage resources and potential negative financial implications.[7,8] Inclusion criteria for these various trials are a good indicator for criteria evaluated to determine transfusion need. These criteria include systolic blood pressure, perfusion parameters (e.g., heart rate, pulse quality, capillary refill time), shock index, lactate, base excess, and urine output. Regarding prognostic indicators, the "triad of death" (hypothermia, coagulopathy, acidosis) in severe trauma has been expanded to the "diamond of death" to include ionized calcium.[9]

VETERINARY EVIDENCE

A multicenter clinical trial evaluating lyophilized platelets and plasma vs. crystalloid resuscitation in acute canine hemorrhagic shock patients (LoVLETR) is completing enrollment in 2024 – results pending.[10]There are a number of single center retrospective studies evaluating transfusion practices in trauma and acute hemorrhage, but no randomized trials that evaluate composition or timing of various resuscitation strategies (to the authors' knowledge – and please inform me if I've missed!).[11] There are a number of pre-clinical trials leveraging induced canine hemorrhage models that help inform clinical recommendations including a series of articles evaluating renal, inflammatory and coagulation system effects of fresh whole blood (20 ml/kg) vs. crystalloid (80 ml/kg) vs. HES (20 ml/kg) vs. 4% succinylated gelatin (20 ml/kg).[12]

Regarding identification of acutely hemorrhaging dogs, there are five single center veterinary studies that have evaluated point of care diagnostic tests with respect to prediction of transfusion in hemorrhaging patients. These studies specifically looked at base excess (BE), ionized calcium (iCa), abdominal fluid score (AFS), packed cell volume (PCV), total solids (TS), and lactate. A BE < -6.6 mmol/L, iCa <1.25 mmol/L, Abdominal Fluid Score (AFS) ≥3, PCV < 39%, T/S < 4.5 g/dL, and Lactate >5 mmol/L have all been identified as independent predictors of receipt of transfusion in hemorrhaging dogs with greater than 89% specificity.[11, 13-16] The LoVLETR clinical trial is using shock index as an inclusion criteria, as well.

CURRENT RECOMMENDATIONS AND BARRIERS

Two (very similar) proposed resuscitation strategy algorithms for acutely hemorrhaging (trauma) dogs and cats have been proposed in recent peer-reviewed literature.[1,17] It is recognized that these recommendations will continue to shift as application, pragmatic studies, and current research efforts are analyzed regarding outcomes in dogs and cats to inform best-practices. While it is acknowledged that leveraging judicious crystalloid resuscitation as a bridge to blood product use/availability is a reality in even well-resourced veterinary practices, replacement of lost blood via fresh whole blood (FWB) or plasma:pRBC:+/-platelets in a 1:1:+/-1 ratio are recommended early in the resuscitation phase. Regarding FWB administration as a top choice: veterinary medicine has led the way as many veterinary practices have donor animals available on site. Regarding 1:1(:1) ratio resuscitation: blood bank access is growing in the USA (local and regional), although challenges and barriers exist in that space, too.[18] When FWB or component therapy are not available, making "next best" choices for a patient becomes resource dependent (e.g., autotransfusion, blood products available, etc.).

FUTURE DIRECTIONS

Continued efforts regarding development and availability of freeze-dried products (plasma, platelets) would increase the ability across the veterinary care spectrum to administer plasma or platelet equivalent product due to shelf stability and shelf life.[19] Improving referral networking capabilities across the veterinary spectrum to match patient in need with resources is an ongoing opportunity in the veterinary profession.[20] Published results from multiple pre-clinical and clinical hemorrhagic shock studies will be available in the next year (or so) and may help further refine recommendations.

CASE WRAP-UP

The clinical course of *Joust* will be revisited throughout the presentation with opportunity for audience input/interaction.

Note: these proceeding notes were leveraged from proceedings from a similar presentation by the author (KHall) at a similar ACVIM presentation.

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ARRHYTHMIAS: WHAT'S YOUR DIAGNOSIS AND HOW WILL YOU TREAT?

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Learning objectives:

- Develop a structured approach to basic ECG interpretation, including heart rate assessment, rhythm identification, and verification of lead placement errors.
- Apply ECG interpretation principles to the ECC setting, prioritising rapid diagnosis and clinically relevant decision-making.
- Differentiate common bradyarrhythmias and tachyarrhythmias encountered in ECC patients using ECG features.
- Formulate appropriate first-line treatment plans.
- Recognise when additional diagnostics are required (e.g. electrolytes, echocardiography) to identify reversible or contributing causes of arrhythmias.

Proceeding:

A systematic approach to electrocardiography (ECG) interpretation is essential in the emergency and critical care (ECC) setting. Initial assessment should include confirmation of correct limb lead placement, followed by evaluation of heart rate, rhythm (regular, irregular, or regularly irregular), and identification of P waves, QRS complexes, and T waves as well as their association. Early differentiation between supraventricular rhythms (typically narrow QRS complexes) and ventricular rhythms (wide, bizarre QRS complexes) is critical. The haemodynamic impact of an arrhythmia depends on the heart rate, rhythm, duration, abrupt changes in heart rate, origin of the rhythm (supraventricular vs ventricular), and the presence of underlying cardiac disease. Immediate effects include reduced blood pressure and stroke volume which clinical signs such as weakness and syncope, while sustained arrhythmias may lead to neurohormonal activation, altered peripheral resistance, cardiac remodelling and even congestive heart failure.

Types of bradyarrhythmias that may be encountered include sinus bradycardia, sinus node dysfunction, sinoatrial block, sinus arrest, and atrioventricular (AV) block. Sinus bradycardia is often the result of systemic changes such as high vagal tone, electrolyte abnormalities (notably hyperkalaemia), drugs (opioids, antiarrhythmics, α2-agonists) and extracardiac disease. Absence of visible P waves with bradycardia warrants consideration of atrial fibrillation with third-degree AV block, sinoatrial block, sinus standstill, atrial standstill, or a sinoventricular rhythm (hyperkalaemia). The atropine response test may assist diagnosis but carries risks including paradoxical AV block, severe tachycardia, hypertension, and pro-arrhythmic effects. Treatment options include medical management with sympathomimetics or methylxanthines versus permanent pacemaker placement in refractory or symptomatic cases.

Tachyarrhythmias are categorised as supraventricular tachycardia (SVT) or ventricular tachycardia (VT). The origin and complexity of arrhythmias are important to stratify patient risk, for example a

dog presenting with sustained polymorphic ventricular tachycardia poses a much higher risk for sudden cardiac death compared to a dog with intermittent focal atrial tachycardia. An SVT commonly encountered is atrial fibrillation which is characterised by absent P waves, an undulating baseline, and an irregular ventricular response with accompanying pulse deficits. Therapy usually involves the use of diltiazem, digoxin, or combination therapy. Other SVTs include atrial flutter, atrial tachycardias, junctional tachycardia, and reciprocating tachycardias. These will have variable presentations, clinical implications, and treatment strategies depending on the underlying arrhythmogenic mechanism, rate, and overall arrhythmogenic burden. Emergency room treatment options include vagal manoeuvres, intravenous diltiazem at 0.25 mg/kg IV slowly, repeatable up to four doses over 60 minutes, or esmolol 500 µg/kg IV bolus. If refractory, electrical cardioversion should be considered in consultation with a cardiologist.

Wide complex tachycardias should be presumed ventricular tachycardia until proven otherwise. Findings consistent with ventricular tachycardias include AV dissociation, regular R-R intervals, and the presence of capture or fusion beats. Treatment is indicated for haemodynamically significant rhythms, frequent non-sustained (<30 s), sustained (>30 s), or very rapid ventricular tachycardia (instantaneous heart rate of >260 bpm is associated with increased risk of sudden cardiac death), or frequent R-on-T phenomena. Management prioritises ensuring electrolyte correction, followed by administration of a lidocaine 2 mg/kg IV bolus, repeatable up to three times over 5 minutes, then a continuous rate infusion of 40–80 µg/kg/min. Additional options include magnesium sulphate, procainamide, esmolol, oral sotalol, intravenous amiodarone, or electrical cardioversion.

Conclusion

Effective management of arrhythmias in the ECC setting relies on rapid, structured ECG interpretation combined with an understanding of haemodynamic consequences to guide decision making for treatment. Early differentiation between supraventricular and ventricular rhythms, assessment of patient stability, and timely initiation of appropriate therapy can significantly improve outcomes. Identifying reversible or extra-cardiac causes of arrhythmias and recognising when advanced interventions such as pacemaker placement or electrical cardioversion are required are components of evidence-based arrhythmia management in critically ill patients.

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CLINICAL CARDIOLOGY CASES – WHAT WOULD YOU DO?

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Learning objectives:

- Apply point-of-care diagnostics (POCUS, ECG, blood pressure) to rapidly assess and triage emergency cardiology cases in the ECC setting.
- Differentiate cardiac and non-cardiac causes of respiratory distress, including pericardial effusion, pleural effusion, and pulmonary oedema.
- Select appropriate initial stabilisation strategies based on pathophysiology rather than default treatments.
- Recognise when cardiac disease may mimic non-cardiac presentations, such as seizures or altered mentation.
- Identify patients who may not benefit from standard therapies, including supplemental oxygen, and understand the underlying physiological reasons.

Proceeding:

Emergency cardiology cases often present with non-specific or misleading clinical signs, requiring rapid assessment and targeted stabilisation. Point-of-care diagnostics, including focused physical examination, ECG, blood pressure measurement, and ultrasound, are essential tools for differentiating cardiac from non-cardiac disease and guiding appropriate therapy.

Presentation and Initial Stabilisation

Initial stabilisation focuses on minimising stress and addressing life-threatening abnormalities. Initial stabilisation involves provision of supplemental oxygen and stress reduction (sedation with butorphanol 0.2 mg/kg). Perform a thorough physical examination and blood pressure measurement to guide decision making. Basic laboratory evaluation, including electrolytes or blood gas analysis where feasible, provides valuable information. An ECG should be applied early to assess rhythm disturbances and allow continuous monitoring during stabilisation. Point-of-care ultrasound of the thorax, abdomen, and heart should be performed if tolerated, with cardiac assessment often providing the most critical diagnostic information. In severely stressed or unstable patients, staged evaluation is preferable to prolonged diagnostic procedures.

Case Examples

Aortic Thromboembolism (ATE)

ATE is often suspected based on history, clinical signs, and physical examination. A brief point-of-care echocardiogram demonstrating left atrial enlargement supports a cardiac origin. Management focuses on analgesia and prevention of further thrombus formation using antithrombotic therapy. Thrombolysis may be considered in acute cases (<6 hours). Patients should be monitored closely for congestive heart failure, acute kidney injury, and reperfusion-associated hyperkalaemia.

Pericardial Effusion

Pericardial effusion may present with acute lethargy, collapse, tachypnoea, tachycardia, weak pulses, and pulsus paradoxus. Abdominal distension may be present in more chronic cases. Diagnosis is confirmed with point-of-care echocardiography. Treatment involves pericardiocentesis, with or without placement of an indwelling drain, typically performed under sedation. Pericardial fluid should always be analysed, as neoplastic and septic effusions may appear grossly haemorrhagic. Once stabilised, a comprehensive echocardiogram is indicated to investigate underlying causes.

Left Atrial Tear

Left atrial rupture is an uncommon cause of pericardial effusion and is usually associated with advanced myxomatous mitral valve disease. Echocardiographic findings include pericardial effusion, possible hyperechoic clot formation, and reduction in left atrial size following the tear. Pericardiocentesis is generally avoided unless there is clear evidence of tamponade. Management is largely supportive and may include oxygen therapy, cautious diuretic use depending on left atrial pressures, crystalloid support acutely, inotropes such as pimobendan or dobutamine, afterload reduction with ACE inhibitors or amlodipine.

Acute Cardiogenic Pulmonary Oedema

Acute cardiogenic pulmonary oedema is typically characterised by increased respiratory rate and effort with echocardiographic evidence of left atrial enlargement and B-lines present on thoracic ultrasound. The cornerstone of treatment is diuresis with intravenous furosemide. Bolus dosing is essential before transitioning to a continuous rate infusion because CRI alone will delay achieving therapeutic plasma concentrations. Stress reduction, oxygen supplementation, and possibly inotropic support are important. Preload reduction with sodium nitroprusside may be used in severe cases; however, without concurrent diuresis, pulmonary oedema will recur once the infusion is discontinued. Mechanical ventilation should be considered in patients with refractory distress or respiratory fatigue.

Cardiogenic Pleural Effusion in Cats

Cats with pleural effusion often present with increased respiratory effort and rate. Thoracic POCUS readily identifies effusion, while echocardiography demonstrating left atrial enlargement or right-sided cardiomegaly supports a cardiogenic cause. NT-proBNP measurement in serum or pleural fluid can aid differentiation. Thoracocentesis is the primary treatment in compromised patients because resolution of the effusion would be prolonged with diuretic use alone. Fluid analysis is essential to exclude alternative diagnoses.

Cardiac Mimics and Unusual Presentations

Paroxysmal third-degree AV block in cats may present as seizure-like activity, highlighting the importance of ECG evaluation in neurological presentations. Right-to-left shunting congenital defects may cause systemic hypoxaemia or hyperviscosity syndrome. In hypoxaemic patients, oxygen therapy provides limited benefit due to shunt physiology. Management focuses on anxiolysis and improving pulmonary blood flow using phosphodiesterase 5 inhibitors such as sildenafil. Patients with erythrocytosis-related clinical signs require phlebotomy with concurrent crystalloid replacement.

Conclusion

Emergency cardiology cases require rapid, structured assessment and stabilisation guided by understanding the underlying pathophysiology. Integrating POCUS, ECG, and targeted diagnostics allows clinicians to identify life-threatening cardiac conditions, recognise atypical presentations, and treat appropriately.

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HEART-LUNG INTERACTIONS

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Learning objectives:

- Define heart-lung interactions and explain their mechanisms.
- Explain the cardiopulmonary differences between spontaneous (negative pressure) and positive pressure breaths.
- Understand the role of heart-lung interactions in selected diseases.
- Consider how we may use heart-lung interactions to guide clinical decisions.

Proceeding:

The heart and lungs are interconnected.[1,2,3,4,5] The heart delivers blood to the lungs, and the lungs oxygenate blood and regulate its chemical composition. Mechanically, the lungs function as a negative-pressure (suction) pump, while the heart is a positive-pressure pump. Both reside within the thorax: intrathoracic pressures influence pulmonary and cardiac function, and cardiac and pulmonary pressures reciprocally affect each other – these are the foundations of heart-lung interactions.

Spontaneous breathing (negative pressure ventilation)

During normal spontaneous breathing, negative intrathoracic pressure results in lung inflation *and* alters cardiac filling and emptying.[1,2] As the heart is contained within the thorax, when the diaphragm contracts to decrease the pressure in the pleural space, thus expanding the lungs, this negative pleural pressure is transmitted to the heart.[5] Concurrently, the descent of the diaphragm into the abdomen increases intraabdominal pressure.[1] The gradient for venous return is defined by the difference in upstream venous pressure and the downstream right atrial pressure.[4] With the decline in intrathoracic pressure, the right atrial pressure decreases; with the increase in intraabdominal pressure the upstream venous pressure increases.[1,2] Thus, the gradient for venous return increases and right heart preload increases.

As the right ventricular preload increases, the interventricular septum is shifted to the left: as the cardiac volume is limited (by the pericardium), the left ventricular preload is necessarily decreased if the right ventricular volume increases.[1,3] This phenomenon is *(parallel) ventricular interdependence.* Concurrently, the decrease in intrathoracic pressure increases left ventricular afterload.[1,3] Afterload is the pressure that needs to be overcome to eject blood out of the heart and into the systemic arterial system. As the pleural pressure becomes more negative, it provides a pulling-like force on the left ventricle, essentially pulling it open. In order for the left ventricle to contract to the necessary pressure to eject blood into the aorta (≈ 120 mmHg), the left ventricle must overcome this pulling force generated by the negative pleural pressure.[3,5] With the combination of decreased preload and increased afterload, the left ventricular stroke volume decreases.

Previously termed the “respiratory pump”, the effects of spontaneous breathing are beneficial for the exercising patient with a healthy heart. However, in patients with failing hearts, the hemodynamic consequences can be deleterious.

Mechanical ventilation (positive pressure ventilation)

With the application of positive pressures to the respiratory system, the same rules apply but in reverse. Now the inflation of the lungs is driven by positive pressure forcing air into the lungs: this increases intrathoracic pressure. The increase in intrathoracic pressure is transmitted to the right atrium, which is the downstream pressure for venous return.[1] As a consequence, the gradient for venous return decreases and right heart preload decreases. As the right ventricle fills to a lower volume, the left ventricle is free to fill to a greater volume (ventricular interdependence). Additionally, as the pulmonary pressure and lung volume increase, blood within the lung is displaced forward into the left atrium, thus the left heart preload increases.[1,3]Moreover, as the intrathoracic pressure is now positive on inspiration, this pressure is transmitted to the left ventricle and *decreases* left ventricular afterload, which can improve the performance of the failing heart, as has been demonstrated in dogs.[4] Initially, the left ventricular stroke volume increases and, providing that the reduction in right ventricular preload is not marked, the cardiac output can increase.[1,4]

Positive pressure inflates the lungs. The application of positive pressure and the increase in lung volume can collapse pulmonary vessels, leading to an increase in pulmonary vascular resistance.[1,3,5] This increases right ventricular afterload. In the case of the volume underloaded patient or the failing right ventricle, this increase in pulmonary vascular resistance can be disastrous. However, with the volume overloaded, left-sided cardiac failure patient, the reduction in right ventricular preload and reduction in left ventricular afterload can be beneficial, whilst the volume-loaded state of the patient can lessen the increase in pulmonary vascular resistance.[1]

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MANAGEMENT OF MULTIPLE ORGAN DYSFUNCTION SYNDROME

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Learning objectives:

- Understand the pathophysiology of MODS
- Identify patients at high risk for MODS
- Diagnose MODS in a clinical patient
- Manage patients with MODS to maximize recovery and survival

Proceeding:

Introduction

Multiple organ dysfunction syndrome (MODS) is a serious and frequently fatal condition characterized by dysfunction and/or failure of two or more major organ systems secondary to a dysregulated immune response to septic or non-septic etiologies. Progressive organ deterioration often leads to death or euthanasia, so early recognition and rapid, appropriate treatment are vital for success.

Pathophysiology

A dysregulated immune response to infectious or non-infectious insults results in an imbalance between a proinflammatory systemic inflammatory response syndrome (SIRS) and a compensatory anti-inflammatory response syndrome (CARS). Widespread endothelial activation, microcirculatory dysregulation, and impaired oxygen delivery and utilization commonly ensue. Many of the exact mechanisms are complicated and poorly understood.

One of the primary drivers of organ injury, dysfunction, and failure is damage to the microvasculature. This results in dysregulation of vascular tone, with excessive and heterogeneous vasodilation, increased capillary leakiness, microthrombi-induced ischemia, and mitochondrial derangements that lead to cellular energy failure (cytopathic hypoxia). These deleterious changes to the microcirculation may lead to organ dysfunction despite mild alterations in macrocirculatory parameters.

Organs commonly affected include the gastrointestinal tract, liver, kidneys, coagulation system, cardiovascular system, brain, and lungs. Neuroendocrine dysfunction and metabolic derangements can exacerbate organ dysfunction and failure.

Recognition and Diagnosis

Animals at risk for MODS include those with SIRS of any origin. Common conditions that may predispose to MODS in dogs and cats include sepsis, trauma, heatstroke, pancreatitis, toxins, immune-mediated diseases, burn injury, and envenomation. Changes in temperature, heart rate, respiratory rate, and white blood cell differentials are used to diagnose a systemic inflammatory

response syndrome (SIRS). Dogs typically present with signs of a hyperdynamic cardiovascular system, including red mucous membranes, tachycardia, and fever, while cats are often hypodynamic, with pale mucous membranes, bradycardia, and hypothermia.

A complete blood count and biochemical profile, coagulation testing, blood gas and lactate, urinalysis (+/-culture), thoracic and abdominal imaging, fluid analysis (and culture) from any diseased effusive organs or cavities, and blood cultures, if indicated, are commonly performed on presentation and as needed in patients with MODS. Additional testing, such as echocardiogram, joint, spine, or CNS imaging/cytology, should be done based on clinical suspicion of the underlying disease process or additional organ involvement. The recognition of MODS is primarily based on the underlying disease(s) and the specific organs that are affected. Intensive monitoring and treatment of all body systems is essential and often dynamic.

Management

The primary goal is the treatment of the underlying cause of MODS, including antibiotics for control of bacterial sepsis (and surgical source control if indicated), cardiovascular and pulmonary support, organ-targeted medical therapy, correction of coagulation disturbances, acid-base imbalances, and metabolic and electrolyte abnormalities. Normalization of oxygen delivery and prevention of further organ injury are also vital.

Management of cardiovascular dysfunction must address vasodilatory hypotension; this typically involves the administration of vasoconstricting agents, such as catecholamines (e.g., norepinephrine, epinephrine, dopamine), vasopressin, or a combination of the two. Positive inotropes (e.g., dobutamine, pimobendane) may be useful in patients with decreased contractility, and antiarrhythmics are used for severe arrhythmias.

Treatment of acute kidney injury generally involves maintaining euvolemia (and avoiding excessive volume administration), monitoring and addressing changes in urine output (e.g., giving furosemide for oligoanuria), and ensuring adequate renal perfusion and oxygen delivery to the kidneys.

Management of hepatic dysfunction focuses on supplementing albumin and, if indicated, supporting coagulation. Early nutritional support is also important.

Acute respiratory distress syndrome (ARDS) often requires intubation and lung-protective ventilation strategies. For less severely affected animals, high-flow nasal oxygen may be adequate.

Gastrointestinal dysfunction is typically managed with supportive medications, including antiemetics, promotility agents, and antacid therapy, as indicated, along with early enteral nutrition.

Treatment of coagulation dysfunction includes anticoagulation for hypercoagulable states or blood products for hypocoagulable patients who are bleeding or require an invasive procedure.

Management of central nervous system dysfunction includes close monitoring of neurological status, hypertonic solutions, and/or antiepileptics if indicated, and general supportive care to maintain oxygen delivery to the brain.

Adrenal insufficiency, often referred to as critical illness-related corticosteroid insufficiency (CIRCI), is commonly treated with physiologic doses of corticosteroids. Metabolic abnormalities such as hypo- or hyperglycemia should be treated with dextrose supplementation or insulin, respectively.

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OCULAR MANIFESTATIONS OF SYSTEMIC DISEASE

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Learning objectives:

- Recognise ocular signs associated with significant systemic disease
- Identify ophthalmic findings suggestive of hypertension, neurologic disease, and diabetes mellitus
- Integrate ocular examination findings into emergency diagnostic and triage decisions
- Avoid misdiagnosing systemic illness as isolated primary ocular disease

Proceeding:

Introduction

The eye provides a unique window into systemic health, and ocular abnormalities may be the first, or most visible, sign of serious systemic disease in emergency and critical care patients. This lecture explores how ophthalmic findings can reflect underlying vascular, neurologic, metabolic, infectious, and inflammatory disorders, and how these signs can inform diagnostic prioritisation and case management.

Systemic hypertension

Systemic hypertension is seen relatively frequently in critically ill patients. While transient increases in blood pressure may occur due to stress or pain, certain ocular findings are more indicative of chronic disease. Bilateral retinal detachment, retinal haemorrhage, and vascular tortuosity suggest sustained hypertension and warrant immediate systemic evaluation and blood pressure control where appropriate.

SARDs vs optic neuritis?

Acute blindness with minimal fundic change presents a diagnostic challenge. Sudden acquired retinal degeneration syndrome (SARDs) should be considered in dogs presenting with symmetrical acute vision loss, absent menace response, sluggish pupillary light reflexes, and a history of polyuria and polydipsia, even when fundoscopic examination appears normal. Failure to recognise this condition may lead to unnecessary investigations and delayed owner counselling. The major differential is optic neuritis, which often presents with concurrent neurological signs, or asymmetrical ocular signs. An ERG (electroretinogram), +/- MRI scan and CSF analysis may be required for definitive diagnosis.

Diabetes

Diabetes mellitus is another systemic disease with characteristic ocular manifestations. Rapid cataract formation in diabetic dogs results from intralenticular sorbitol accumulation creating an osmotic gradient and influx of water which disrupts and expands the lens fibres. Recognition of this

mechanism is essential, as early intervention may prevent lens-induced uveitis and secondary glaucoma.

Infections/inflammatory/neoplastic disease

Infectious, inflammatory, and neoplastic systemic diseases may first manifest with ocular signs, through a variety of mechanisms. Bilateral anterior uveitis in a patient with concurrent systemic signs (e.g. pyrexia, lymphadenomegaly) should raise suspicion of systemic disease rather than isolated ocular pathology. In such cases, treating the eye alone is insufficient, and systemic investigation is required.

Neurological disease

Neurologic disease may be reflected in abnormal pupillary responses, altered menace reaction, or changes in fundic appearance. In unstable ECC patients, fundic examination offers real-time insight into vascular integrity and neurologic status without the need for advanced imaging.

Summary

Misdiagnosing systemic illness as primary ocular disease represents a significant clinical risk. Integrating ophthalmic findings into the broader clinical picture allows emergency clinicians to identify red flags for life-threatening disease, prioritise diagnostics appropriately, and improve overall patient outcomes. By recognising the eye as an extension of the systemic examination, clinicians can enhance both diagnostic accuracy and patient care in the emergency setting.

WET OR DRY-HOW NOT TO DIE

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Learning objectives:

- Understand the physiology of fluids within the body and distribution following intravenous fluid administration
- Assess volume status in a critically ill patient and understand the goals of resuscitation
- Formulate a fluid plan to follow resuscitation and prevent volume overload
- Accelerate removal and discontinuation of fluid therapy appropriately.

Proceeding:

Introduction

Fluids are drugs and must be prescribed with a thorough understanding of their indications, anticipated distribution in the body, and potential complications. There are numerous fluid types available, so a thorough understanding of the types and uses in different clinical scenarios is important.

Pathophysiology

Living mammals maintain volume homeostasis by consuming the fluids they need daily, driven by thirst and hunger. When clinicians give fluids by parenteral routes, they are associated with great risks but also several anticipated benefits that can be lifesaving when used appropriately. Critically ill patients pose a unique challenge due to the predisposing factors that cause their altered fluid distribution and increased fluid losses. Dynamic changes in fluid needs necessitate frequent reassessment and adjustments to the fluid therapy plan. The veterinary criticalist must have a thorough understanding of fluid distribution and homeostasis, and their impact on hemodynamics and oxygen delivery to the tissues. Judicious fluid therapy is typically recommended, especially as our understanding of the adverse effects of volume overload has evolved. For example, fluid overload is associated with increased mortality and length of hospitalization in both humans and small animals.

Fluid Therapy Strategies

There are numerous strategies used in human medicine to address fluid needs in critically ill patients, often referred to as “fluid stewardship.” Early goal-directed fluid management, late conservative fluid management, and late goal-directed fluid removal are often discussed. The “four D’s” of fluid therapy refer to the drug, dosing, duration, and de-escalation phases. Another popular approach uses the mnemonic “ROSE” which refers to Resuscitation, Optimization, Stabilization, and Evacuation. Each of these stages has a distinct goal, monitoring needs, and therapeutic strategies.

Fluid **resuscitation** addresses life-threatening, non-cardiogenic circulatory shock. This typically includes the first 3-6 hours following the initial assessment using an early, adequate, goal-directed

approach. But how early is too early, and how fast is too fast? Does all resuscitation need to be protocolized? Or is personalized therapy better?

During fluid resuscitation, there is inevitably some degree of a “second hit” phenomenon as ischemic tissues are reperfused. During this process, endothelial damage and capillary hyperpermeability often ensue. Continued administration of high volumes of fluids is likely to be deleterious, leading to interstitial edema and organ dysfunction. Fluid **optimization** is key following resuscitation; further administration should be guided by ongoing losses and fine-tuning of deficits rather than urgent or aggressive replacement. Animals with distributive shock might benefit from earlier vasopressor and/or inotropic support rather than excessive fluid volumes. Maintaining euvolemia and homeostasis are ideal, while fluid overload should be avoided at all costs!

Stabilization is the third phase of fluid therapy and typically ensues over the next few days of hospitalization. During this time, the threat of shock is generally resolved, and the clinician’s focus shifts to supporting organ function and maintaining a steady state while the underlying disease continues to resolve. Generally, ongoing maintenance rates are delivered to account for sensible and insensible losses. Close attention should be paid to “extra” fluids delivered in the form of diluted medication solutions, enteral or parenteral nutrition, or transfusions.

Many animals enter the “flow” phase of spontaneous fluid **evacuation** after recovering from the second hit. However, some critically ill patients remain in a “no-flow” state, followed by a third hit, usually resulting from a diffuse capillary leak syndrome. Regardless, patients should enter a phase of “de-resuscitation,” often referred to as late goal-directed fluid removal and late conservative fluid management. These involve active fluid removal (usually with diuretics) and a moderate fluid therapy strategy. In human medicine, two consecutive days of negative fluid balance within the first week in the ICU is a strong and independent predictor of survival.

Although there is a dearth of literature to guide prescriptive fluid therapy in critically ill dogs and cats, the evidence from human medicine and the smaller, but convincing, studies in veterinary medicine suggest that fluid stewardship is a cornerstone of better outcomes for most critically ill animals.

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ARDS IN VETERINARY MEDICINE: UPDATED ARDSVET DEFINITIONS AND PRACTICAL CLINICAL APPLICATION

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Learning objectives:

By the end of this session, learners will be able to:

- **List the five required diagnostic criteria** for ARDSVet and describe how they differ from prior veterinary ARDS definitions.
- **Identify common probable and possible risk factors** for ARDS in small and large animal patients.
- **Explain how SpO_2/FiO_2 ratios and PaO_2/FiO_2 ratios** are used to define and stratify hypoxemia in ARDSVet.
- **Describe the role of thoracic POCUS** in diagnosing ARDS and excluding cardiogenic pulmonary edema.
- **Differentiate non-intubated ARDS, IMV-ARDS, and patients at risk for ARDS**, and recognize that severity may evolve over time.

Proceeding:

Acute Respiratory Distress Syndrome (ARDS) is a life-threatening cause of acute hypoxemic respiratory failure in veterinary patients, but historically it has been inconsistently recognized across species and practice environments. The updated ARDSVet definitions were developed via an international, evidence-based consensus process to modernize diagnosis while remaining usable in resource-variable settings. Key changes include allowing SpO_2/FiO_2 as an alternative oxygenation metric (under specific conditions), accepting thoracic point-of-care ultrasound (POCUS) as a primary imaging modality, and expanding classification to include non-intubated ARDS, IMV-ARDS, and patients at risk for ARDS, without requiring histopathology or mandatory mechanical ventilation. These changes align with updates made to the human ARDS definitions as outlined in the recently published Global ARDS Definitions in people.

1) What changed: From "VetARDS/VetALI" to ARDSVet

Earlier veterinary frameworks were modeled after the AECC human definitions and commonly leaned on arterial blood gases and thoracic radiographic or CT-based criteria. The ARDSVet working group explicitly acknowledged that these requirements can create diagnostic barriers in real-world veterinary care (cost, logistics, euthanasia before escalation, limited diagnostics), and that veterinary emergency/critical care has evolved in the years following the original definitions (wider use of high flow nasal oxygen, mechanical ventilation sophistication and use of newer modalities such as airway pressure release ventilation, and most widespread of all- point of care ultrasound expansion).

Major updates in the ARDSVet framework include:

Clinical diagnosis: ARDS is a *clinical construct*; histopathology is not required.

Expanded oxygenation assessment: SpO_2/FiO_2 can substitute for PaO_2/FiO_2 *when* $SpO_2 \leq 97\%$ (because values above this flatten on the oxyhemoglobin dissociation curve and become less informative).

Imaging requirements: Thoracic POCUS is accepted as a primary tool to identify diffuse pulmonary infiltrates and support ruling out cardiogenic edema/volume overload in addition to thoracic radiographs or CT.

Severity as a moving target: Patients may move between severity categories as disease evolves, and definitions explicitly accommodate both non-intubated and mechanically ventilated patients. This underlines the reality that ARDS is a continuum of illness, and patients may meet the requisite diagnostic criteria, even if mechanical ventilation is not feasible as a means of respiratory support.

2) The five required diagnostic criteria for ARDSVet

ARDSVet is anchored on **five required criteria** (plus optional supportive data), designed to be applicable across variable resource settings:

* **Risk factor / predisposing insult** (known or suspected)

* **Timing**: new or worsening respiratory distress within 1 week of the insult

* **Origin of edema**: pulmonary edema not fully explained by left-sided CHF or fluid overload (must be *ruled out*, using whatever tools are available)

* **Thoracic imaging** showing diffuse pulmonary infiltrates (radiographs, CT, or thoracic ultrasound/POCUS)

* **Impaired oxygenation** assessed by PaO_2/FiO_2 *or* SpO_2/FiO_2 (when valid)

* **Optional supporting criterion** (helpful but not required): inflammatory airway fluid (neutrophilic inflammation/high protein via tracheal wash/BAL), especially for extrapulmonary triggers.

<u>Key updates from prior veterinary ARDS definitions:</u>

POCUS accepted (not just radiographs/CT)

SpO_2/FiO_2 accepted (with SpO_2 ≤97%)

Formal category for **"at risk"** patients before full criteria are met.

3) Risk factors: "probable" vs "possible"

ARDSVet intentionally groups triggers into **probable** and **possible** risk factors, reflecting the strength and consistency of evidence across species and reports. This is meant to improve diagnostic clarity while staying flexible enough for real clinical complexity (many patients have >1 insult).

Examples featured in the working group materials include:

* **Probable** (commonly supported): aspiration injury, SIRS, sepsis, acute pancreatitis, toxin inhalation, drowning, trauma, smoke inhalation; and for equids, Rhodococcus equi and equine influenza

* **Possible** (less consistent/limited evidence): transfusion of blood products, ventilator-induced lung injury, and selected infectious triggers in horses.

In practice, this framing helps veterinarians rapidly connect the dots between an inciting disease process and evolving respiratory failure—especially in fast-moving ER/ICU timelines—without claiming causality where the evidence base is weak.

4) Oxygenation: using either PaO_2/FiO_2 or SpO_2/FiO_2

PaO_2/FiO_2: PaO_2/FiO_2 remains preferred when arterial blood gas analysis is available, but ARDSVet intentionally reduces dependence on ABGs to keep the definition usable broadly.

SpO_2/FiO_2: The ARDSVet definitions accept SpO_2/FiO_2 when **SpO_2 ≤97%**. This is an explicit attempt to address settings where ABG sampling is difficult/unavailable (small patient size, staffing, cost, equipment access), while acknowledging limitations of pulse oximetry and FiO_2 estimation with some oxygen delivery methods.

Implications for clinical workflow:

If you're using SpO_2/FiO_2, you must be confident in **(a)** the SpO_2 signal quality and **(b)** the delivered FiO_2 (known FiO_2 environments like oxygen kennel setpoints, HFNO, or intubated ventilation are most defensible). Use SpO_2/FiO_2 strategically to enable earlier classification, trend severity dynamically, and support standardization—especially when ABG access is intermittent.

5) Thoracic POCUS: role in diagnosing ARDS and excluding cardiogenic edema

ARDSVet requires thoracic imaging evidence of diffuse pulmonary infiltrates but makes changes to what counts as acceptable imaging: radiographs, CT, or thoracic ultrasound (POCUS).

Why POCUS is included in the updated definition:

It can be performed cage-side/stall-side in unstable patients where transport to radiology is unsafe. It supports both parts of the diagnostic problem: demonstrating diffuse pulmonary infiltrates (e.g., B-lines, consolidations, pleural line abnormalities) and contributing evidence to rule out L-CHF/volume overload, particularly when paired with focused cardiac views (e.g., LA:Ao assessment).

Importantly, ARDSVet does not require echocardiography or invasive wedge pressures; instead, it asks clinicians to rule out cardiogenic causes using available tools in a reasoned, multimodal way (history/PE + imaging + ultrasound when available), which reflects the practical realities often encountered in veterinary ECC.

6) The three ARDSVet categories and the "severity can evolve" concept

The ARDSVet definitions stratify patients into:

Non-intubated ARDS

IMV-ARDS (invasive mechanical ventilation)

Patients at risk for ARDS (early identification before full criteria met)

This structure is designed to match real clinical trajectories, where patients may present early (risk factor + hypoxemia) and later develop diffuse infiltrates, or where escalation to HFNO/IMV happens after initial categorization. Severity can (and often does) shift over time, so the ARDSVet definitions account for patient reassessment rather than a single static label.

Clinical takeaway from both vignettes: The updated ARDSVet definitions are designed to (1) standardize diagnosis and documentation, (2) work even when ABG/CT/IMV and other expensive diagnostic and treatment modalities are not available or feasible in resource-variable veterinary settings, and (3) align classification with what clinicians actually assess in real time-patient progression, reassessment, and variability in respiratory support.

8) Implementation tips for ECC teams: A practical approach to using ARDSVet in real time:

*** Start with the timeline**: Is there a plausible risk factor AND is distress new/worsening within **1 week**?* **Choose the "best available oxygenation metric"**: prefer PaO_2/FiO_2 when feasible, use SpO_2/FiO_2 when SpO_2 ≤97% and FiO_2 is known/defensible.

*** Use POCUS early when unstable:** document diffuse infiltrates + focused evidence against cardiogenic edema/volume overload.

*** Label the category** (at risk vs non-intubated vs IMV-ARDS), then **reassess** as support changes (oxygen cage/nasal oxygen→ HFNO → IMV) and as imaging evolves.

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LITERATURE - VETCOT STUDIES - WHAT HAVE WE LEARNT AND HOW TO BE INVOLVED

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Learning objectives:

- By the end of the session, the learner will have a better understanding of the VetCOT Registry's contributions to better understanding the following regarding veterinary trauma care:
- Trauma epidemiology and characteristics of injury patterns
- Trauma severity scoring and physiologic predictors of care interventions
- Similarities and differences between cats and dogs sustaining traumatic injury
- Impact of the registry on broader translational trauma efforts

Proceeding:

Insights from the VetCOT Registry

Trauma is a leading cause of morbidity and mortality in small animal patients. Improving outcomes for injured dogs and cats necessitates a coordinated approach to care, data collection, and research. The Veterinary Committee on Trauma (VetCOT) was established with the goal of creating a network of lead hospitals to develop trauma systems, define high standards of care, and disseminate information. A cornerstone of the VetCOT initiative is the **VetCOT trauma registry**, a multicenter, prospective clinical research database.

The VetCOT registry is designed to inform the improvement of trauma patient care, aid in the design of clinical trials, and enhance and promote research collaborations. Data is collected and managed using the REDCap electronic data capture tool supported by awards from the National Institutes of Health (NIH). As of the most recent version of the registry (version 3.0, Jan 2022), environmental injuries are also included in the registry.

Since its inception in 2013 with nine inaugural Veterinary Trauma Centers (VTCs), the registry has grown significantly, amassing over 70,000 cases by December 2024. The registry captures detailed information on trauma characteristics, physiological parameters, diagnostic findings, treatments, and outcomes. Data validation efforts are ongoing to ensure accuracy, completeness, and consistency, including the development of tools like RVetQual. The large, multi-institutional dataset generated by the VetCOT registry provides an invaluable resource for understanding the epidemiology of veterinary trauma and identifying factors associated with outcome.

Trauma Epidemiology

Analysis of the extensive data within the VetCOT registry has yielded significant findings regarding the types of trauma encountered and patient factors influencing outcomes. The type of trauma sustained is a critical factor associated with patient outcome. Penetrating trauma appears to be

slightly more common in dogs (53.3%) compared to blunt trauma (41.2%), while blunt trauma is more prevalent in cats (58%) than penetrating trauma (35%). Critically, the survival rates differ based on trauma type. Dogs sustaining only penetrating trauma have demonstrated a higher survival rate (96.5%) compared to those with blunt trauma (89.5%) or combined blunt and penetrating trauma (86.3%). Similarly, cats with penetrating trauma exhibit a higher survival rate (90%) when compared to those with blunt trauma (80%) or combined trauma (68%). Dogs and cats that suffer combined blunt and penetrating trauma are more likely to present with moderate to severe injuries and consequently have lower survival rates. In cats, the combined blunt and penetrating trauma category had the highest proportion of severely injured patients (26%), significantly higher than those with only penetrating trauma (6%). This highlights the complex and often more devastating nature of injuries resulting from multiple trauma mechanisms. Certain injury locations carry a poorer prognosis. The presence of head trauma and spinal trauma are specifically associated with increased mortality risk. VTCs, particularly Level I centers, tend to manage a higher caseload of head trauma patients. Increasing age is associated with a higher case fatality rate across trauma types. Geriatric status has been identified as an independent risk factor for death in dogs with moderate to severe trauma. In cats, studies have shown that increased body weight is associated with a decreased odds of non-survival, while older age increased the odds of non-survival. Analysis has also indicated that male dogs may have a lower odds of survival compared to female dogs following trauma.

Trauma Severity Scoring and Physiological Predictors

Illness severity scoring systems are vital tools in trauma management for triage, benchmarking performance, guiding resource allocation, and facilitating research. The VetCOT registry has been instrumental in the external validation of widely used veterinary trauma scores. The Animal Trauma Triage (ATT) Score is a composite score (0-18) based on evaluating six body systems: perfusion, cardiac, respiratory, eye/muscle/skin, skeletal, and neurologic. VetCOT registry data has consistently validated that a higher ATT score is associated with worse outcomes, including decreased survival and an increased likelihood of euthanasia due to grave prognosis. Specific ATT score ranges correlate with injury severity in dogs: 0-3 for mild injury (mortality <10%), 4-6 for moderate injury (mortality 10%-50%), 7-10 for severe injury (mortality >50%), and 11-18 for very severe injury (mortality ≥90%). An ATT score ≥5 has been found to have high sensitivity and specificity for predicting non-survival in dogs. For cats, an ATT score >3 can serve as a predictive cut-off for non-survival. Analysis of the ATT subscores within the registry data revealed differences in their predictive performance. The neurological, perfusion, and respiratory subscores were identified as the three most predictive components of outcome, while the eye/muscle/integument subscore was the least predictive. This suggests that a more parsimonious score based only on these key components could potentially offer equivalent predictive performance.

The Modified Glasgow Coma Scale (mGCS, 3-18) specifically assesses neurological function by evaluating brain stem reflexes, motor activity, and level of consciousness. Lower mGCS scores indicate more severe neurological impairment and are associated with decreased survival. While the mGCS is valuable, particularly in head trauma cases, the motor subscore of the mGCS was found to be the most predictive component. The median mGCS score in the overall trauma populations documented in VetCOT is often 18, suggesting that severe neurological dysfunction is less common across all trauma types compared to specific studies focusing on head trauma. However, in cats, lower mGCS scores were significantly associated with non-survival. Both the ATT and mGCS are

significantly associated with outcome. In studies using VetCOT data, the full ATT score demonstrated better discriminatory performance for predicting mortality than the mGCS score in the overall trauma population. Even in the subset of patients with head injury, the ATT score performed better than the mGCS.

Beyond these composite scores, several physiological parameters measured upon presentation serve as important independent predictors of outcome. Elevated plasma lactate and a more negative base excess (BE) are consistently associated with increased mortality risk in trauma patients. In a study of cats, those with a positive abdominal fluid score (pAFS) had more negative BE. Lower PCV and TS values on admission are associated with decreased survival. Ionized calcium is also identified as a significant physiological predictor of outcome. The findings from Point of Care Ultrasound (POCUS) examinations, specifically the abdominal fluid score (AFS) derived from AFAST, are predictive of outcome and resource utilization. A recent study utilizing VetCOT data found that cats with a positive AFS upon presentation were more likely to require hospitalization and receive blood transfusions than those with a negative AFS.

Interventions and Informatics

The VetCOT registry also collects data on interventions and aspects of patient care, enabling analysis of their impact on outcome and providing insights into current practices. Data from the registry indicates that undergoing surgery (both operating room and emergency room procedures) and receiving blood products are associated with improved survival outcomes in trauma patients. This finding supports the importance of timely surgical intervention and resuscitation with blood products for appropriate trauma cases. In dogs, operating room surgeries, particularly general surgery and neurology, were associated with increased length of hospitalization, cost, and ATT scores. Blunt trauma was more commonly addressed by orthopedic, neurology, and dental surgeries, while penetrating injuries were more often managed by general and ophthalmology surgery. Cats requiring orthopedic surgery were more likely to receive blood products than those undergoing other surgical specialties.

The registry captures data on whether pre-hospital care was provided and by whom. While evidence in the veterinary pre-hospital setting is limited compared to human medicine, the VetCOT Prehospital Committee has developed best practice recommendations based on available human and veterinary literature and expert consensus. These guidelines cover scenarios such as hemorrhage control, respiratory distress, fluid therapy, basic and advanced life support, neurological trauma, and management of penetrating trauma.

The VetCOT registry is a prime example of leveraging veterinary informatics for large-scale clinical research. Collecting data from multiple VTCs relies heavily on information extracted from Electronic Medical Records (EMRs) and Practice Information Management Systems (PIMS). However, challenges persist due to a lack of standardization in data formatting and nomenclature across different systems, making data extraction and analysis complex. Initiatives like the development of the RVetQual quality control assessment tool for the VetCOT database aim to address these challenges by systematically evaluating data accuracy, completeness, and consistency. Continued efforts are focused on refining the registry's data dictionary and implementing enhanced quality assurance/quality control processes (versions 2.0 and 3.0) to strengthen the dataset.

Future Directions

The VetCOT and its multicenter registry represent a significant advancement in veterinary trauma care and research. By systematically collecting and analyzing data from a large population of injured dogs and cats across numerous VTCs, the VetCOT initiative has generated crucial scientific findings regarding trauma epidemiology, the performance of severity scoring systems, and the predictive value of physiological parameters. These insights are vital for improving patient outcomes, benchmarking hospital performance, and guiding future research efforts. Continued collaboration and data quality improvement are essential for maximizing the impact of this invaluable resource.

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PANEL - SURGICAL TIMING (PYOTHORAX, SEPTIC PERITONITIS, DIAPHRAGMATIC HERNIA, GDV)

Angela Briganti [1] + Lynda Rutherford [2]

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Learning objectives:

- Differentiate conditions requiring immediate surgery
- Integrate physiologic optimization and anesthetic/surgical planning
- Anticipate surgery and anesthesia-specific risks and complications

Proceeding:

Optimal surgical timing in critically ill patients requires balancing the urgency of source control with the patient's physiological reserve. The decision to proceed to surgery hinges on whether the patient can tolerate anesthesia and surgical stress without precipitating or worsening organ dysfunction.

This panel will explore how **physiologic stabilization, risk stratification, and anesthetic planning** influence timing of surgery, highlighting when **immediate intervention is life-saving** versus when **delayed or staged surgery** improves outcome.

Key anesthetic considerations include optimization of oxygen delivery, hemodynamic support, acid–base and electrolyte correction, ventilation strategies, and anticipation of peri-operative complications specific to each disease process. Close collaboration between anesthetist and surgeon is emphasized to ensure that surgical timing is aligned with both **source control priorities and anesthetic safety**.

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Nurse & Tech Stream, Thursday 4 June 2026

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Learning objectives:

- Become confident defining and classifying jaundice
- Analyse the pathophysiology of bilirubin
- Recognise the clinical signs of hyperbilirubinaemia
- Overview the common causes of hyperbilirubinaemia
- Recognise the clinical consequences and appropriate prioritisation of care

Proceeding:

Jaundice can be defined as the yellow abnormal discolouration of mucous membranes, skin and sclera, caused by a hyperbilirubinaemia that causes pigmentation. Hyperbilirubinaemia is defined as an increased serum bilirubin level above the normal reference range that causes jaundice. Mild increases usually do not cause discolouration in the form of jaundice but can often be seen as an icteric colour of serum blood samples. Jaundice normally becomes clinically apparent when serum bilirubin >2.5mg/Dl (>43micromol/L). It's important to remember that jaundice is not a disease or diagnosis, but a clinical manifestation of various primary diseases. To classify the causes of jaundice, we must first understand how bilirubin is metabolised and excreted in the body. Bilirubin is a normal byproduct of erythrocyte metabolism and breakdown of haemoglobin through a cascade of metabolic pathways which eventuate with bilirubin diffusing or being actively transported into hepatocytes of the liver where it is conjugated into a water-soluble version of bilirubin and excreted through the bile canaliculi. A small amount will be recycled but eventually come back to the hepatocytes to be excreted into bile. The bile canaliculi carry bilirubin to the gallbladder where it is temporarily stored before bile ducts carry it to the duodenum for enterohepatic recirculation, excretion in faeces or a small amount is metabolised and excreted by the kidneys. Jaundice takes days to clear as it stains the skin.

Classifications

Because bilirubin travels throughout the body on its path from production to excretion, it is important to localise the cause of the hyperbilirubinaemia to identify which condition is causing it. These causes can be divided into the following three conditions: pre-hepatic, hepatic and post-hepatic.

Pre-hepatic jaundice: Usually moderate to severe in nature, pre-hepatic jaundice is the result of excessive extravascular or intravascular haemolysis. This excessive production of bilirubin can overwhelm the system, leading to accumulation of bilirubin in the blood, causing discolouration throughout the body. Classic diseases that cause this type of jaundice include immune-mediated

haemolytic anaemia (IMHA), infectious diseases (e.g. Mycoplasma spp., Babesia spp.), hepatic lipidosis, intoxications (e.g. zinc, onions and paracetamol), genetic disorders and hypophosphataemia.

Hepatic jaundice: This form of jaundice happens when there is hepatocellular dysfunction (direct injury to the liver itself), causing issues processing bilirubin. Common causes of damage to the hepatocytes include drug reactions (e.g. carprofen, paracetamol, azathioprine or methimazole), toxins (e.g. sago palm, paracetamol, xylitol, copper or aflatoxins), immune mediated or inflammatory disease, infectious diseases (e.g. FIP, Leptospirosis, Toxoplasmosis and Coccidioidomycosis), neoplasia or genetic abnormalities that may affect normal copper metabolism. Hepatic lipidosis can also contribute to hepatic jaundice by damaging hepatocytes.

Post-hepatic jaundice: This is when the outflow of bile is affected, causing accumulation of bilirubin and can be further divided into intraluminal or extraluminal obstruction of bile ducts and be partial or complete in nature. Intraluminal causes include mucoceles, choleliths, inflammation or infection, strictures, neoplasia or parasites. Extraluminal causes include pancreatitis, neoplasia, lymphadenopathy and duodenal foreign body.

Clinical presentations and prioritisation of care

As nurses in the emergency room, our role is not to diagnose, but it is to identify clinical concerns and ensure appropriate escalation to a veterinarian based on order of urgency. To be able to do this appropriately, we need to become comfortable identifying possible causes to help guide history taking and clinical examination of incoming jaundiced patients. As discussed earlier, with many different causes leading to variable jaundiced conditions, each presentation will have variable levels of stability. Accurate history taking is essential to aid escalation and eventual diagnosis due to jaundice being secondary in nature. Owners should not just be questioned about the clinical signs leading to the presentation, but of any pre-existing known diagnosis. Signalment should be considered for potential genetic disorders, using species, breed and the age of patient. History should be inclusive of vaccination status, parasitic control, current medications, travel and potential access to toxins. Timeline is important as acute jaundice presentation is more likely to have a post-hepatic obstruction, an acute toxicity or IMHA than a genetic cause or chronic liver changes for instance. A physical primary survey should be included in the assessment and triage of any incoming patient to the emergency room. Results from this may further narrow the focus of secondary surveys with veterinarians or help signal the immediate need for veterinary intervention. Identification of jaundice often comes from seeing the yellow tinge of mucous membranes, sclera or skin and may range from a subtle undertone to obvious yellow/orange colour. Patients with pre-hepatic jaundice will likely be anaemic and may present in a form of haemolytic shock evidenced by affected perfusion parameters such as dull demeanour, pale mucous membranes, tachycardia or bradycardia, abnormal pulse quality and hypothermia. If hypoperfusion and cardiovascular instability is evidenced, veterinary intervention should be prioritised. Patients with bleeding disorders caused by hepatic dysfunction will present with similar signs of hypoperfusion from hypovolaemic shock but may also have evidence of petechiae or ecchymoses. If shock is identified, patients may require supplemental oxygen or veterinary prescribed fluid therapy intervention. Identification of fever in the face of jaundice, signals a likely infectious or inflammatory process. Active cooling is contraindicated for fever, unless temperatures risk cytotoxicity, but quick action is indicated in the face of critical patients that meet SIRS/SEPSIS criteria. Abdominal palpation and pain scoring should occur in

jaundice patients as hepatic or post hepatic causes are often extremely painful. Fast identification of pain will lead to early provision of analgesia. Neurological status should also be assessed. Severe states of shock may alter demeanour and level of consciousness. Neurological decline in jaundice patients can also signal other clinical diseases. In hepatic jaundice patients, the reduction in ammonia filtration ability can lead to hepatic encephalopathy. If hyperbilirubinaemia is left untreated or reaches extremely high levels, kernicterus can occur where the hyperbilirubinaemia causes damage to the brain itself. Both presentations can be severe and patients may be comatose. Lastly, barrier nursing should be considered with jaundice patients as protection for personnel, due to the risk of a zoonotic diseases or protection for patients, due to immunocompromise.

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NURSING MANAGEMENT OF PARVOVIRUS PUPPIES IN CRITICAL CARE

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Learning objectives:

- Understand the basic pathophysiology of CPV (canine parvovirus)
- Be able to recognise common clinical signs and symptoms
- Have a good understanding of key nursing points for critical care patients
- Be aware of treatments and therapy for CPV
- Feel confident nursing isolation patients within a busy ICU

Proceeding:

Introduction

Canine parvovirus (CPV-2) remains one of the most significant infectious diseases affecting young dogs, particularly unvaccinated or incompletely vaccinated puppies between six and twenty weeks old. Infection commonly results in severe gastrointestinal upset, profound fluid and protein losses, and an increased risk of secondary bacterial sepsis. Survival depends heavily on severity of disease, rapid supportive treatment and intensive nursing care. Due to the virus's persistence in the environment and high contagiousness, strict barrier nursing is essential within the hospital setting.

Pathophysiology

Canine parvovirus is a small, non-enveloped, single-stranded DNA virus that replicates within rapidly dividing cells. Transmission occurs through the faecal–oral route, with viral shedding beginning several days before clinical signs appear. The virus can survive on surfaces for many months, making infection control vital. CPV targets intestinal crypt cells, resulting in gastrointestinal inflammation, malabsorption, vomiting and haemorrhagic diarrhoea. Bone marrow suppression leads to neutropenia and lymphopenia, weakening immune response. Increased gastrointestinal permeability may lead to bacterial translocation, which can result in systemic inflammatory response syndrome, with severe cases progressing to disseminated intravascular coagulation, multi-organ dysfunction and death.

Clinical presentation and diagnosis

Affected puppies often present with acute lethargy, anorexia, pyrexia, vomiting with or without haematemesis, and diarrhoea which may be haemorrhagic. Diagnosis is commonly supported by in-clinic antigen testing, although false negatives may occur early in infection. Faecal polymerase chain reaction testing is considered the diagnostic gold standard. Imaging such as radiography or ultrasound may be required to exclude gastrointestinal obstruction or complications such as intussusception.

Nursing care

Treatment of CPV is largely supportive and should be tailored to each patient's problem list. Intensive nursing care is fundamental, with priorities including fluid therapy, nutritional support, analgesia, antiemetic therapy and close monitoring. Keeping the patient clean can be a difficult task with ongoing diarrhoea but is important to prevent perineal scalding and contamination of intravenous lines. It is important to consider enrichment when nursing puppies, as playing and interaction are a vital part of development.

Infection control and barrier nursing

Parvovirus patients must be managed in strict isolation due to environmental persistence and high transmission risk. Isolation protocols include high level personal protective equipment, patient-specific equipment and consumables, double-bagging of waste and minimising movement between isolation and general ICU areas.

Fluid therapy and monitoring

Aggressive crystalloid therapy is often required due to dehydration and ongoing gastrointestinal losses. Accurate recording of ins and outs is essential. Nurses must monitor patients closely to assess volume status. Colloid or blood product support may be indicated in cases of refractory hypotension, anaemia or severe hypoalbuminaemia.

Nutritional support

Early nutritional intervention supports gastrointestinal integrity and immune function. Enteral feeding is preferred when tolerated, often via nasoesophageal or nasogastric tubes, which can be placed by trained veterinary nurses without sedation when tolerated. Nutrition should be calculated using resting energy requirements (RER) and puppies may require higher calorie intake for growth, depending on age.

Conclusion

Canine parvovirus infection remains a critical disease requiring intensive supportive care. Veterinary nurses play a vital role in improving outcomes through meticulous monitoring, accurate fluid management, early nutritional support, effective analgesia and strict infection control. With robust nursing protocols, survival rates for CPV puppies in ICU can be significantly improved.

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THE SWEETNESS THAT COULD KILL YOU: DKA

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Learning objectives:

- What exactly is DKA, and what do we need to know about it?
- What signs and symptoms are we likely to see?
- What diagnostics are most important?
- What is the treatment plan?
- What is the prognosis, and how do underlying or concurrent diseases play a role in this?

Proceeding:

Introduction

Diabetic ketoacidosis (DKA) is a life-threatening complication of diabetes mellitus that frequently presents in critically ill veterinary patients. These patients require intensive care, advanced monitoring, and diligent nursing support. Successful outcomes depend not only on medical management but also on the veterinary technician's understanding of the underlying pathophysiology, anticipated complications, and specific nursing considerations required throughout hospitalization.

Pathophysiology

DKA represents a state of cellular starvation resulting from absolute or relative insulin deficiency. Without insulin, glucose cannot enter cells, leading to hyperglycemia. Concurrent elevations in counter-regulatory hormones - glucagon, cortisol, and epinephrine - exacerbate hyperglycemia and promote lipolysis.

Hyperglycemia causes osmotic diuresis, resulting in dehydration and electrolyte loss. As intravascular osmolality increases, water shifts from the intracellular space, worsening cellular dehydration. The body compensates by metabolizing fat and protein for energy, producing ketone bodies including acetone, acetoacetate, and beta-hydroxybutyrate. Accumulation of ketones leads to metabolic acidosis.

DKA is commonly precipitated by concurrent diseases, including pancreatitis, urinary tract infection, hyperadrenocorticism, chronic kidney disease, or other systemic inflammatory conditions.

Clinical Presentation

Clinical signs of DKA are often severe and nonspecific. Common findings include anorexia, vomiting, lethargy, dehydration, weakness, polyuria, polydipsia, hypothermia, hypotension, and shock. Tachypnea or Kussmaul respirations may be present as compensation for metabolic acidosis.

Species-specific findings include diabetic neuropathy with a plantigrade stance in approximately 10% of cats and cataract formation in up to 40% of dogs. Hepatomegaly and secondary pancreatitis are frequently observed in both species.

Electrolyte Abnormalities

Electrolyte disturbances are a defining feature of DKA and require frequent monitoring and adjustment.

Hyponatremia is expected due to osmotic dilution caused by hyperglycemia. Normal sodium concentrations in the presence of marked hyperglycemia indicate inappropriate free water loss and hyperosmolality.

Hypokalemia is the most common electrolyte abnormality. Clinical signs include generalized weakness, cervical ventroflexion in cats, arrhythmias, and respiratory muscle weakness. Potassium supplementation is routinely required, often at high levels, and total potassium delivery must not exceed 0.5 mEq/kg/hr.

Hypophosphatemia may be present at admission or develop during treatment. Severe deficits can cause hemolytic anemia and muscle weakness. Supplementation with potassium phosphate may be required, with careful calculation of total potassium administration.

Hypomagnesemia results from chronic losses and inadequate intake. Magnesium deficiency may impair correction of hypokalemia, as magnesium is required for cellular potassium uptake. Concurrent supplementation is often necessary.

Acid–Base and Cardiovascular Effects

Metabolic acidosis is common in DKA patients and contributes to lethargy, vomiting, hyperventilation, decreased myocardial contractility, peripheral vasodilation, and altered mentation. Treatment focuses on correcting dehydration and suppressing ketogenesis through IV fluids and insulin therapy.

In cases of severe acidemia (pH < 7.10), sodium bicarbonate supplementation may be considered. Use remains controversial and should be reserved for refractory cases.

Acidosis adversely affects cardiovascular function by decreasing cardiac output, lowering arrhythmia thresholds, reducing responsiveness to catecholamines, and altering vascular tone.

Diagnostics

Initial diagnostics include blood glucose measurement, urinalysis, ketone assessment, serum electrolytes, and blood gas analysis. Once DKA is confirmed, further diagnostics should be pursued to identify underlying disease.

Pancreatitis is common and should be evaluated using pancreatic lipase immunoreactivity testing and abdominal ultrasound. Urine culture is recommended to assess for urinary tract infection. Abdominal ultrasound may also reveal adrenal enlargement consistent with hyperadrenocorticism in dogs. Thoracic radiographs may be indicated to identify pneumonia or other inflammatory processes.

Treatment

Treatment should begin immediately upon diagnosis. IV fluid therapy is the cornerstone of initial management and addresses dehydration, hypovolemia, and hyperosmolality. Buffered crystalloid solutions such as Normosol-R or Lactated Ringer's solution are preferred. Fluid deficits should be corrected over 24-48 hours.

Insulin therapy should be delayed until adequate hydration is achieved, as fluid therapy alone can significantly reduce blood glucose and ketone concentrations. Early insulin administration may exacerbate hypokalemia.

Short-acting regular insulin (Humulin R) is used to control hyperglycemia and suppress ketogenesis. The target rate of glucose reduction is 50-100 mg/dL/hr (2.7-5.5 mmol/L/hr). Insulin may be administered via intermittent intramuscular injection or continuous rate infusion. Blood glucose should be monitored every 2-4 hours, with insulin and dextrose adjustments made as indicated.

IV Access and Monitoring

Reliable IV access is essential in DKA patients due to frequent blood sampling and electrolyte supplementation. Peripheral catheters are often placed initially; however, central venous or sampling catheters are recommended for ICU patients.

Monitoring should include mentation, perfusion parameters, pulse quality, blood pressure, urine output, catheter sites, and daily renal values. Electrolytes and ketones are typically reassessed 2-4 times daily.

Supportive Care and Nutrition

Supportive care should address concurrent disease processes. Opioid analgesics are appropriate for patients with painful conditions such as pancreatitis. Antiemetics including maropitant, ondansetron, or metoclopramide are commonly required. Routine acid suppression is not recommended unless gastric ulceration is suspected or confirmed. Antibiotics should be administered when infection is identified.

Nutritional support is critical. Many DKA patients present after several days of inadequate intake. Feeding tubes should be considered if patients have not eaten for three days or longer, including time prior to hospitalization. Voluntary intake should be encouraged once nausea is controlled. In-hospital feeding should prioritize adequate caloric intake over strict diabetic diets to prevent food aversion.

Prognosis

Despite the severity of disease, approximately 70% of patients with DKA survive to hospital discharge. Average hospitalization duration is approximately six days for dogs and five days for cats. Recurrence rates are estimated at 7% in dogs and up to 40% in cats. With vigilant monitoring and comprehensive nursing care, successful outcomes are achievable.

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NURSING THE TRAUMATIC BRAIN INJURY PATIENT

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Learning objectives:

- Pathophysiology of traumatic brain injury
- Understanding patient presentation and carrying out appropriate triage
- Tailoring patient care and needs
- Nursing care
- Prognosis

Proceeding:

Traumatic brain injury (TBI) is common in small animals following road traffic accident (RTA), falls and altercation with other (larger) animals. Other causes can also include malicious intent to cause harm by humans.

Primary injury and secondary injury can occur following head trauma. Primary injury occurs immediately following the trauma, secondary injury can occur hours, even days following known trauma. Secondary head trauma occurs due to the release of inflammatory mediators, neurotransmitter excitement and change in the cellular membrane permeability.

Immediate intervention is required for these patients, following primary survey triage exam. In cases of trauma, extracranial injuries need to be addressed, including severe wounds, ventilation, oxygenation and any evidence of airway obstruction. Also addressing hypovolaemia and potential haemorrhage.

A minimum data base (MDB) blood sample and venous or arterial blood gas should be assessed during the triage. Packed cell volume (PCV) and total solids (TS) can indicate haemorrhage, blood glucose (BG) to assess severity of injury and CO2 to identify hypoventilation. Jugular sample is to be avoided due to decreased blood outflow from the brain.

Maintenance of cerebral perfusion pressure (CPP) is required, by ensuring adequate oxygen delivery to the brain, and treatment and management of increased intracranial pressure (ICP). CPP can be maintained by maintaining adequate tissue perfusion, for example, maintaining appropriate systemic blood pressure, heart rate, oxygenation and ventilation. Increase in ICP, indicated by hypertension and bradycardia, should be addressed by administering mannitol.

Neurological assessment in the emergency room (ER) should primarily focus on mentation and level of consciousness, pupil size and pupillary light reflex (PLR). Interpretation of pupil size and PLR are indicative of prognosis, with poor to no PLR and bilateral mydriasis following TBI are associated with a grave prognosis. A Glasgow Coma Score (GCS) has been developed for small animals which can be used to indicate improvement and overall prognosis.

Nursing the TBI patient is continuous, requiring round the clock care. The patient should be positioned on a board at a 30degree angle, thus allowing for venous drainage from the cranial vault, minimising risk of increased ICP. Blankets should be avoided where possible, ensuring no compression of the jugular vein preventing venous drainage. Repeat MDB and blood gas assessment, perfusion checks and pain and anxiety management are also required.

Other presentations similar to TBI can include acute hyponatraemia, intracranial space occupying lesion, neoplasia, hepatic encephalopathy and inflammatory brain disease (MUO). If presenting with signs of increased ICP then will require treatment with hypertonic saline (HTS) or mannitol. Then follow up nursing care as per TBI patient with tailored treatment to underlying disease process.

In cases where seizures are present, levetiracetam and diazepam are recommended, currently there is no evidence to suggest that prophylactic treatment with anticonvulsants decreases delayed onset of seizures following TBI.

Prognosis following TBI is variable. GCS can be indicative of outcome. Younger animals are reported to have better outcomes; however, no research has been done to determine this. Some patients can go on to have long term neurological deficits following TBI and develop seizures, therefore long term seizure management may be required in these patients.

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MEDICAL MATH & CRIS: REMOVING THE FEAR FACTOR

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Learning objectives:

- Why is it so important that we learn how to do medical math and not just use a spreadsheet of some kind that does it for us?
- Review of some of the medications that we use in ECC, just so it's not all numbers!
- Step by step instructions of how to work out CRIs.
- We will also cover dilutions and percentages.

Proceeding:

Introduction

Accurate medical calculations are a fundamental responsibility of veterinary technicians, particularly those involved in anesthesia, emergency, and critical care. While electronic calculators, infusion pumps, and spreadsheet tools are widely available, a foundational understanding of medical math remains essential. Veterinary technicians must understand what medications they are administering, why they are indicated, and the precise dose being delivered to ensure patient safety and optimal outcomes.

Many technicians experience anxiety when faced with drug calculations, particularly constant rate infusions (CRIs). With a systematic approach and consistent practice, medical math can become reliable and reproducible. Proficiency in calculations also allows technicians to serve as an essential secondary safety check, reducing medication errors and supporting a culture of shared accountability.

Role of CRIs in Veterinary Medicine

Constant rate infusions are commonly used in veterinary patients to deliver medications in a controlled and consistent manner. CRIs are utilized intraoperatively for analgesia and total intravenous anesthesia (TIVA), in critical care patients requiring insulin or antiarrhythmic therapy, and postoperatively for pain management. This delivery method minimizes fluctuations in plasma drug concentrations, avoiding peaks and troughs that can compromise efficacy or safety.

When calculating a CRI, it is recommended to write out all known variables, including patient weight, prescribed dose, duration of infusion, and drug concentration. Identifying the final desired unit (typically milliliters of drug and diluent) helps guide the calculation process.

Fentanyl CRI Example

Fentanyl is a potent μ-opioid receptor agonist commonly used for intraoperative and postoperative analgesia. It has a rapid onset and short duration of action, making it well suited for CRI

administration. Fentanyl CRIs should always be preceded by a loading dose to rapidly achieve therapeutic plasma concentrations.

Diluting fentanyl so that 1 mcg/kg/hr equals 1 ml/hr allows for simple dose adjustments.

Example:

Patient weight: 15 kg

Loading dose: 5 mcg/kg IV

CRI dose: 3 mcg/kg/hr

CRI duration: 10 hours

Drug concentration: 50 mcg/ml

Loading dose calculation:

15 kg × 5 mcg/kg = 75 mcg

75 mcg ÷ 50 mcg/ml = **1.5 ml**

CRI calculation:

15 kg × 3 mcg/kg/hr = 45 mcg/hr

45 mcg/hr × 10 hr = 450 mcg

450 mcg ÷ 50 mcg/ml = **9 ml fentanyl**

Total CRI volume:

3 ml/hr × 10 hr = 30 ml

Diluent volume:

30 ml − 9 ml = **21 ml saline**

Metoclopramide as an IV Fluid Additive

Metoclopramide is a prokinetic and antiemetic medication frequently administered as a CRI via IV fluids at 1-2 mg/kg/day.

Example:

Dose: 2 mg/kg/day

Patient weight: 23 kg

IV fluid rate: 120 ml/hr

Bag volume: 1 L

Drug concentration: 5 mg/ml

Bag duration:

1000 ml ÷ 120 ml/hr = 8.33 hr

Daily dose:

23 kg × 2 mg/kg/day = 46 mg/day

Hourly dose:

46 mg ÷ 24 hr = 1.92 mg/hr

Total drug needed:

1.92 mg/hr × 8.33 hr = 16 mg

Volume to add:

16 mg ÷ 5 mg/ml = **3.2 ml**

Potassium Chloride and Maximum Safe Delivery

Potassium chloride (KCl) is commonly added to IV fluids to correct hypokalemia. Because rapid potassium administration can cause life-threatening arrhythmias, the maximum safe delivery rate (K max) must always be calculated.

K max formula:

0.5 mEq/kg/hr

Example:

Patient weight: 26 kg

IV fluid rate: 130 ml/hr

KCl concentration: 95 mEq/L

K max:

26 kg × 0.5 mEq/kg/hr = 13 mEq/hr

K concentration per mL:

95 mEq ÷ 1000 mL = 0.095 mEq/ml

Hourly delivery:

0.095 mEq/ml × 130 ml/hr = 12.35 mEq/hr

Since 12.35 mEq/hr is less than the patient's K max of 13 mEq/hr, this concentration is safe to administer.

Lidocaine CRI Example

Lidocaine is used as an antiarrhythmic, analgesic adjunct, and anti-inflammatory agent. Preparing a CRI that allows for easy dose adjustment improves safety and efficiency.

Example:

Patient weight: 9.3 kg

Dose: 20 mcg/kg/min

CRI rate: 2 ml/hr

Duration: 12 hours

Drug concentration: 2% lidocaine

Convert dose to mg:

20 mcg = 0.02 mg

mg/min:

9.3 kg × 0.02 mg/kg/min = 0.186 mg/min

mg/hr:

0.186 mg/min × 60 = 11.16 mg/hr

Total mg needed:

11.16 mg/hr × 12 hr = 133.92 mg

Convert % to mg/ml:

2% = 20 mg/ml

Volume of lidocaine:

133.92 mg ÷ 20 mg/ml = 6.7 ml

Total CRI volume:

2 mL/hr × 12 hr = 24 ml

Diluent volume:

24 ml − 6.7 ml = **17.3 ml saline**

Conclusion

Medical math and CRI calculations are critical skills for veterinary technicians working in emergency and critical care environments. By approaching calculations methodically and verifying work whenever possible, technicians can reduce errors and improve patient safety. Regular practice and collaboration with colleagues are essential, and calculations should always be double-checked to prevent dosing errors. Mastery of these skills supports excellent patient care and reinforces the technician's role as an integral member of the veterinary healthcare team.

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THE DELICATE BALANCE: NEONATAL ANAESTHESIA DEMYSTIFIED

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Learning objectives:

- Recognise the key physiological differences in neonatal patients that impact anaesthetic management in the ECC setting
- Identify common risks and complications associated with neonatal anaesthesia during emergency procedures
- Apply practical strategies to support the neonate undergoing anaesthesia in the ECC setting
- Anticipate and respond to anaesthetic changes to improve patient safety and outcomes

Proceeding:

The Neonate Patient

Neonates may require anaesthesia for a number of reasons, such as a congenital defect correction, emergency procedures for foreign body obstruction or trauma, laceration repairs and potentially restraint for diagnostic imaging. Each of these reasons requires a risk/benefit assessment due to the neonate's limited physiological reserves and inability to communicate pain or distress.

The neonate refers to patients from birth to around two weeks of age, in cats and dogs. During this period, these patients present their own sets of challenges due to their physiology and metabolic needs. During this period, rapid physiological changes occur as the neonate transitions from intrauterine to extrauterine life. The neonatal patient's organs and body systems are immature and functionally distinct from the adult dog or cat. This particularly applies to the nervous system, respiratory and cardiovascular system, all of which are depressed during anaesthesia. Neonatal cats and dogs have poor reflexes during this time and are often thermoregulation deficient. All of these considerations during the developmental stage will directly influence drug responses, metabolic demands and the risk profile for anaesthesia.

Physiological Differences

Neonate patients have larger tongues, small airways and a high oxygen demand. However, due to their infancy, they have fewer alveoli which are overall less compliant. Therefore, their functional residual capacity (FRC) is lower, increasing rapid desaturation risk. In addition, their chest walls are more compliant, reducing effective ventilation efforts and putting these patients at risk of barotrauma. Due to their infancy, the neonates have not matured physiologically, meaning the cardiac muscle is weaker, so positive inotropy is less effective at changing the stroke volume. Cardiac output is therefore largely heart rate dependant, and there is limited ability to increase stroke volume. Baroreceptor responses are immature and the neonate primary relies on peripheral vasoconstriction as a defence against hypotension.

These patients have a thermoregulation deficiency, due to their high surface area-to-volume ratio predisposing them to rapid heat loss, as well as an inability to shiver. This can create profound hypothermia, which can lead to unwanted bradycardia, prolonged drug metabolism, and coagulopathies. The metabolism in neonates is also impaired due to limited glycogen stores and immature gluconeogenesis, resulting in hypoglycaemia. Drug metabolism in the liver and elimination by the kidneys are also reduced due to immature enzymatic pathways and a low glomerular filtration rate (GFR) leading to more profound anaesthesia. Finally, neonatal patients are more susceptible to infection and sepsis due to their underdeveloped immunity, increasing the risk of post-operative complications and nosocomial infections.

Anaesthesia Strategies

Prior to the anaesthetic, baseline vitals and bloodwork should form part of the pre-assessment and stabilisation; any hypoglycaemia, dehydration and hypothermia should be addressed before anaesthetising. Maintenance of a normal body temperature should be the main goal, with strategies including;

Warm recovery area and operating table.

Use circulating warm water blankets.

Warm IV fluids and surgical scrub (but not overheated).

Minimise clipping and use of spirit.

Monitor temperature every 3-5 minutes intra-operatively.

Hypoglycaemia should be addressed with dextrose supplementation if the blood glucose is less than 3.3 mmol/L (60 mg/dL), though care should be taken to avoid hyperglycaemia. Dehydration and fluid deficits should be monitored and corrected with isotonic crystalloids with careful monitoring for fluid overload.

Choice of anaesthetic drugs is limited due to drug metabolism in the immature liver, and the neonate may be sensitive to some choices due to immature kidney function and altered protein binding. Drugs that cause profound cardiovascular depression. Induction agents such as propofol and alfaxalone may be used if they are titrated slowly. However, Etomidate is an ultra-short-acting imidazole derivative for anaesthetic induction, and is particularly suitable for neonates due to its minimal effects on heart rate and blood pressure. Local blocks will reduce the need for opioids, which should be used sparingly, as well as the need for large concentrations of cardio-respiratory depressive inhalants such as isoflurane or sevoflurane. Supplemental oxygen and flow by rates may need to be kept higher to counteract respiratory depression. Uncuffed or low-pressure high volume cuffed endotracheal tubes should be used, and care should be taken intubating as the neonatal larynx is fragile. Monitoring should include monitoring the heart rate and rhythm (via electrocardiogram), pulse oximetry and capnography, as well as blood pressure, temperature, mucous membrane colour and pulse quality.

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ANAESTHESIA CASE STUDIES

Chloe Fay [1]

[1] IVC Evidensia, Ringwood, United Kingdom

Learning objectives:

- Apply structured anaesthetic decision-making to a range of clinical and emergency case scenarios
- Identify common anaesthetic challenges and complications using case studies
- Interpret patient assessment and monitoring data to guide anaesthetic adjustments
- Develop practical, adaptable anaesthetic plans tailored to individual patient risk
- Reflect on outcomes to improve future anaesthetic management and patient safety

Proceeding:

Structured Anaesthetic Decision-Making

Anaesthesia in the emergency and critical care (ECC) setting is rarely ideal or predictable. Patients often present with limited physiological reserve, incomplete diagnostic information, and time-critical surgical needs. A systematic approach will improve consistency, in addition to reducing cognitive overload in high-stress situations.

Anaesthetic decision-making should be guided by:

Patient stability and ASA status

Life-threatening abnormalities

Procedure urgency

Anticipated anaesthetic risks

Correction of immediately life-threatening abnormalities, such as hypovolaemia, hypoxia, severe electrolyte disturbances, or hypoglycaemia, is the primary priority before induction of ASA IV–V emergency patients, if possible. While full diagnostics may be desirable, delaying anaesthesia for non-essential imaging can worsen outcomes. Often patients will present with hypotension/shock, hypoxia/ respiratory compromise, neurological injury, pain and potentially cardiac disease.

These factors necessitate careful drug selection and dose titration. Analgesia should not be withheld as untreated pain increases sympathetic stimulation, oxygen demand, and stress responses. Instead, drug choices should minimise haemodynamic effects while providing effective analgesia.

For many critically ill small animal patients, fentanyl is commonly preferred due to its rapid onset, short duration, and minimal cardiovascular depression when appropriately titrated. Continuous rate infusions allow for smoother analgesic control and reduced inhalant requirements.

Case-Based Considerations: Neurological, Cardiac and Respiratory Patients

Patients with acute head trauma present unique anaesthetic challenges. The primary goals are to:

Maintain cerebral perfusion pressure

Avoid increases in intracranial pressure (ICP)

Prevent hypoxia and hypercapnia

Protocols incorporating an opioid combined with a benzodiazepine, followed by careful titration of alfaxalone or propofol, are commonly utilised. Etomidate (typically 0.3-1.0 mg/kg IV) is a superior induction agent for head trauma patients due to its ability to decrease ICP while maintaining stable cerebral perfusion and cardiovascular function. Opioids and benzodiazepines will provide sedation and analgesia while minimising abrupt changes in cerebral blood flow. Alpha-2 agonists and acepromazine are generally avoided due to their effects on cerebral perfusion and blood pressure. Ketamine alone is not ideal in this context due to concerns regarding sympathetic stimulation and ICP, though the latter has been dispelled in more recent years.

Patients with cardiac disease undergoing anaesthetic must prioritise cardiovascular stability. Most importantly, tachycardia should be avoided whilst maintaining forward cardiac output. Excessive heart rates reduce diastolic filling time and worsen regurgitation, while excessive increases in systemic vascular resistance can exacerbate cardiac workload. Patients should be closely monitored for their heart rate, blood pressure trends, and perfusion parameters, with anaesthetic depth and analgesia adjusted accordingly. Use of local anaesthetic techniques will reduce the concentration of cardio-depressive inhalants needed.

Respiratory patients have minimal tolerance for hypoventilation or hypoxia. Therefore, anaesthesia carries significant risk due to:

Reduced functional lung capacity

Ventilation–perfusion mismatch

Rapid desaturation during induction or apnoea

Stress-induced worsening of respiratory effort

Even brief periods of inadequate oxygen delivery can result in critical deterioration. Preoxygenation in these patients is essential and should be performed for as long as the patient tolerates, ideally using a flow-by or mask technique without increasing stress. A minimal handling, rapid-onset induction is preferred. An opioid (such as fentanyl) combined with a benzodiazepine allows for anxiolysis and analgesia while preserving respiratory drive. Once intubated, oxygen supplementation is critical. Ventilation should be gentle, with close monitoring of end-tidal CO_2. Positive pressure ventilation may be necessary but must be carefully controlled to avoid worsening pneumothorax or barotrauma.

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Abstract & Literature Review Stream, Thursday 4 June 2026

VETLIT YEAR IN REVIEW – ICU

Simon Cook [1, 2]

[1] Royal Veterinary College, London, United Kingdom
[2] VetLit.org, London, United Kingdom

Learning objectives:

- Review recent developments relating to ER and ICU literature
- Critically appraise emerging research relevant to ECC practice
- Translate impactful research findings into ECC practice

Proceeding:

These sessions will explore the most exciting and impactful literature in the fields of ER and ICU 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 to include evaluation of their impact and implications. The primary focus will be veterinary literature, but human landmark publications may also be included.

VETLIT YEAR IN REVIEW – ER

Simon Cook [1, 2]

[1] Royal Veterinary College, London, United Kingdom
[2] VetLit.org, London, United Kingdom

Learning objectives:

- Review recent developments relating to ER and ICU literature
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VETLIT YEAR IN REVIEW - INTERNAL MEDICINE

Christopher Scudder [1]

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

Learning objectives:

- Evaluate the strength of evidence and clinical applicability of key studies to determine their relevance to daily ECC patient management.
- Analyse the limitations and potential biases of highlighted studies.
- Describe novel diagnostic tools, biomarkers, or therapeutic interventions that have emerged in the past year for internal medicine conditions.
- Identify the most significant internal medicine research findings from the past year that impact emergency and critical care clinical practice.

Proceeding:

This presentation will discuss key developments in internal medicine literature from the past year which have relevance to patients presenting to emergency and critical care practices. Each study will be briefly summarised with key findings, limitations, and implications for daily practice highlighted. The goal is to provide evidence-based updates of internal medicine and also to encourage readers of veterinary literature to consider whether the design of a particular study has relevancy to their practice.

VETLIT YEAR IN REVIEW - NEUROLOGY

Abbe Crawford [1]

[1] Royal Veterinary College, 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:

It is daunting task to keep on top of the veterinary literature, particularly given the ever-growing volume of new publications. In this “Neurology year-in-review” session we will recap and review relevant articles published over the last ~12 months from journals including JVECC, JVIM, JSAP and Frontiers. The emphasis will be on papers that apply to emergency and critical care, with a range of relevant topics to be covered including seizure management, spinal cord disorders, trauma, inflammatory and infectious diseases of the nervous system. We will discuss the main findings of each paper, alongside any pertinent features of the methods and considerations for application to clinical practice. The aim of the session is to provide an overview of new understanding and current research focuses in veterinary neurology.

Main Stream, Friday 5 June 2026

DEXMEDETOMIDINE IN THE ER AND ICU

Angela Briganti [1]

[1] University of Pisa, Pisa, Italy

Learning objectives:

- Select ECC-appropriate indications
- Design titration-based dosing plans
- Predict and manage haemodynamic effects
- Integrate dexmedetomidine into multimodal analgesia/sedation bundles,
- Identify special safety considerations

Proceeding:

Dexmedetomidine (DEX), a highly selective α2-adrenergic agonist, is increasingly used in veterinary emergency and critical care (ECC) to provide titratable sedation with intrinsic analgesic-sparing and sympatholytic properties. A key practical advantage in the ECC environment is preservation of ventilatory drive compared with many alternative sedatives, supporting its use in both mechanically ventilated and spontaneously breathing patients when close monitoring is available. Human ICU literature consistently highlights this “cooperative” sedation profile (patients are more rousable) and minimal respiratory depression, while also emphasizing predictable dose-related haemodynamic adverse effects—especially bradycardia and blood pressure changes.

Clinical indications in the ECC patient (dogs/cats):

Ventilator tolerance and sedation strategies: In human intensive care, dexmedetomidine (DEX) provides light, cooperative sedation with minimal respiratory depression. Randomized trials comparing DEX with propofol during neurosurgical procedures demonstrated preservation of cerebral blood flow velocity and regional brain oxygenation, supporting its physiologic stability in neurologically vulnerable patients. Meta-analyses in ICU populations suggest that DEX does not consistently shorten overall ICU length of stay but may modestly reduce duration of mechanical ventilation in selected subgroups and lower delirium incidence. However, bradycardia remains more frequent compared with other sedatives. From a veterinary ECC perspective, these findings support the use of DEX when: light, titratable sedation is required, neurologic reassessment is desirable, ventilatory drive preservation is advantageous.

Delirium, neuroprotection, and brain injury: Human meta-analyses evaluating ischemic brain injury show that DEX reduces inflammatory mediators (TNF-α, IL-6), neuro-injury biomarkers (S100-β, NSE), and stress hormones, suggesting a neuroprotective profile. Additionally, perioperative data demonstrate increased circulating BDNF levels following DEX administration, a biomarker associated with neuronal survival and synaptic plasticity. In neonatal populations, systematic reviews suggest that DEX provides effective sedation with potential neuroprotective properties and reduced seizure risk during therapeutic hypothermia. Importantly, respiratory compromise appears lower compared with opioids and benzodiazepines. While veterinary ICU outcome trials are lacking, these data

support mechanistic plausibility for: reduced neuroinflammatory injury, improved stress response modulation, potential protection in traumatic brain injury or hypoxic events. However, caution is warranted in hypotensive or bradycardic patients where cerebral perfusion may be compromised.

Microcirculation, endothelial function, and organ protection: Beyond sedation, DEX appears to exert microcirculatory and endothelial effects. In endotoxemic rat models, DEX attenuated intestinal microcirculatory dysfunction, reduced endothelial injury markers, preserved tight junction integrity, and decreased bacterial translocation. Experimental sepsis models also demonstrate improved intestinal epithelial barrier function and reduced mucosal apoptosis.

Metabolomic analyses in healthy human volunteers reveal that DEX induces broad reductions in circulating oxylipins and bile acids, molecules implicated in inflammatory signaling and mitochondrial stress pathways. These changes have been hypothesized to relate to organ-protective mechanisms. Collectively, these findings suggest that DEX may: modulate endothelial dysfunction, improve microvascular perfusion, and attenuate inflammatory cascade activation. However, translational extrapolation to naturally occurring canine and feline disease requires caution.

Pulmonary and mitochondrial effects: Experimental sepsis-induced lung injury models demonstrate that DEX attenuates oxidative stress, reduces inflammatory cytokines, improves oxygenation index, and modulates mitochondrial dynamics. In fentanyl-induced muscle rigidity models, dexmedetomidine reversed decreases in respiratory compliance and restored oxygenation by reducing muscle rigidity and metabolic demand. They support the concept that DEX may reduce ventilatory burden not only through sedation but via metabolic modulation.

Risk considerations and cerebral physiology: While DEX preserves regional brain oxygenation in controlled settings, experimental and volunteer data suggest potential alterations in cerebral blood flow regulation under certain conditions. Therefore: avoid loading doses in haemodynamically unstable patients, monitor MAP and perfusion targets closely, consider individual cerebrovascular autoregulation variability. Bradycardia and vasoconstriction remain predictable pharmacodynamic effects and should be anticipated in advanced ECC patients.

Conclusion

Dexmedetomidine should not be considered merely a sedative but a physiology-modulating agent. In human ICU medicine, it provides light, cooperative sedation associated with reduced delirium and preserved brain oxygenation. Preclinical and translational data support anti-inflammatory, microcirculatory, endothelial, and mitochondrial protective effects.

In veterinary emergency and critical care, these findings justify a goal-directed, titrated use of dexmedetomidine in carefully selected canine and feline patients, particularly when ventilatory preservation, neurologic assessment, or multimodal opioid-sparing strategies are desired.

However, robust outcome data in veterinary ICU populations remain limited, and use should remain individualized, with strict haemodynamic monitoring and structured sedation scoring.

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SIMPLE PROTOCOLS FOR SAFE SEDATIONS AND GA

Angela Briganti [1]

[1] University of Pisa, Pisa, Italy

Learning objectives:

- Perform a rapid pre-sedation and pre-anesthetic assessment to identify risk factors and guide protocol selection.
- Select appropriate sedation and general anesthesia protocols based on patient status, procedure type, and available resources.
- Apply dose-sparing and multimodal strategies to minimize cardiovascular and respiratory complications.
- Implement basic monitoring and troubleshooting steps to recognize and manage common anesthetic complications early.

Proceeding:

"There are no safe anaesthetic agents, only safe anaesthetists."

Safe sedation and general anaesthesia are integral to emergency and critical care practice. However, they are frequently perceived as complex and high-risk, particularly in haemodynamically unstable patients. This lecture focuses on simple, practical, and reproducible approaches that can be safely applied in emergency rooms and intensive care units.

Rather than memorizing complex drug "recipes," clinicians should rely on core physiological principles. Emphasis will be placed on patient assessment, rational drug selection, dose adjustment, monitoring, and early prevention of common complications. A principle-based approach enhances safety, efficiency, and clinician confidence when managing critically ill patients.

Start with the Patient

Go directly to the core of the problem: What is the primary disease process? What are its systemic consequences? How might anesthesia exacerbate these alterations?

Risk stratification is essential in all patients, but it becomes critical in emergency and ICU cases. A comprehensive preoperative evaluation of the patient as a whole is key to anticipating complications.

Minimum database considerations should include: Haematology and biochemistry panels, thoracic imaging (radiography and/or ultrasound), blood gas analysis.

If our goal is to return the same patient from anesthesia, we must clearly understand the patient's baseline physiological status beforehand.

This information allows construction of an individualized risk profile, particularly crucial in cardiac patients.

A Surgery Safety Checklist is a valuable tool to ensure team communication and structured evaluation of critical points before induction.

Drug Selection: Weighing Risk and Benefit

The primary objective of any anesthetic protocol in critical patients is to minimize cardiovascular depression. This requires a thorough understanding of both cardiac pathophysiology and drug pharmacodynamics.

For each drug, the clinician should ask: Is this drug truly necessary? What is the risk–benefit ratio in this specific patient? Preparedness is essential: predictable side effects should be anticipated and recognized early. As Goethe stated, “We only see what we know.”

Maintenance Strategies

Total intravenous anesthesia (TIVA) with propofol has been shown to provide better cardiovascular stability compared with inhalant anesthesia, although it requires pharmacological expertise to prevent drug accumulation.

Alfaxalone has minimal cardiovascular impact and is relatively straightforward for TIVA use, but recovery quality may be suboptimal.

Dexmedetomidine infusions can provide cardiovascular stability and improved recovery quality in both human and veterinary patients. However, clinicians must remain aware of potential delayed hemodynamic responses, particularly in cases of hemorrhage.

In many cases, a “liquid” maintenance technique may be preferable to volatile anesthesia.

Analgesia: The Cornerstone of Anesthetic Stability

Optimal anesthesia relies on an effective analgesic plan.

Opioids are generally well tolerated, with dose-dependent side effects. Dose reduction through multimodal strategies is advisable whenever possible. Butorphanol may be appropriate for premedication due to its limited cardiovascular and respiratory effects, but it is not suitable for significantly painful procedures.

A well-structured intraoperative and postoperative analgesic protocol is fundamental for smooth recovery.

Locoregional Anesthesia: A Game Changer

Locoregional anesthesia (LRA) offers significant advantages: Reduction in systemic opioid and anesthetic requirements, prolonged postoperative analgesia, improved hemodynamic stability

Ultrasound-guided techniques enhance precision, efficacy, and safety by reducing local anesthetic volume requirements.

The placement of perineural, epidural, or wound catheters enables continuous analgesia.

A comfortable postoperative patient breathes more effectively, mobilizes earlier, resumes eating and drinking sooner, and demonstrates improved cardiovascular stability. LRA should be considered a core skill for intensive care clinicians managing both cardiac and non-cardiac patients.

Monitoring: Use What You Have, Aim for What Matters

Ideal monitoring in cardiac patients would include: Cardiac output (CO),assessment of contractility, tissue oxygen delivery (DO_2)

In addition to standard monitoring:ECG, temperature, pulse oximetry, capnography, invasive blood pressure (IBP).

Invasive blood pressure monitoring is strongly recommended, allowing beat-to-beat hemodynamic assessment. Careful evaluation of the arterial waveform or plethysmographic curve can provide valuable information about vascular tone and circulatory status.

While advanced hemodynamic monitoring is ideal, clinicians must optimize the tools available in their specific setting.

Recovery: A Critical Phase

Recovery is often underestimated. Monitoring is frequently discontinued too early, and extubation is mistakenly considered the end of risk.

Cardiac patients should recover with continued monitoring (ECG, blood pressure, pulse oximetry) and supplemental oxygen. Early recognition of complications during recovery is essential to improving outcomes.

A smooth recovery is not only more comfortable — it is safer.

Practical Take-Home Messages

Clearly define your patient's pathophysiological status.

Use a Surgery Safety Checklist.

Anticipate perioperative complications — be "Cassandra" for a moment.

Weigh the positive and negative impact of every drug.

Consider TIVA when appropriate.

Optimize analgesia — locoregional techniques make a significant difference.

Monitor as comprehensively as possible; prioritise hemodynamic assessment.

Aim for a calm, controlled, and well-monitored recovery.

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INFLAMMATORY BRAIN DISEASES

João Miguel De Frias [1]

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Learning objectives:

- Explain why the brain is a site of immune privilege.
- Identify the main inflammatory brain diseases in dogs.
- Identify the main inflammatory brain diseases in cats.
- Recognise the challenges of treating neuroinflammation.

Proceeding:

Why is the brain a site of immune privilege?

The brain is protected not only mechanically by being enclosed in a bony structure, but also by the blood-brain barrier (BBB). The BBB is a semi-permeable membrane formed by capillary endothelial cells linked by tight junctions, supported by surrounding pericytes, a basement membrane, and astrocyte end-feet that are responsible to stop the influx of most blood-borne substances from entering the brain. Furthermore, BBB excludes more than 98% of small molecule drugs and all macromolecular therapeutics from access to the brain. The brain immune surveillance is done predominantly by the resident inflammatory cell called microglia. The BBB protects the central neurons from the presence of systemic inflammation and infection. In many inflammatory brain diseases, the BBB is disrupted allowing systemic inflammation (activated macrophages, lymphocytes, neutrophils) to propagate the inflammatory process.

What are the main inflammatory brain diseases in dogs?

Dogs are susceptible to a number of brain inflammatory conditions, including infectious (such as bacterial, viral, protozoal, rickettsial, fungal, parasitic, and algal) and immune-mediated causes. While in humans infectious causes are considered predominant, in dogs auto-immune diseases are the most common brain inflammatory diseases. However, the prevalence of infectious diseases may differ across the globe. Meningoencephalitis of unknown origin (MUO) is the most common immune-mediated brain disease in dogs. This term includes the histopathological subtypes of granulomatous meningoencephalomyelitis (GME), necrotising meningoencephalitis (NME) and necrotising leucoencephalitis (NLE). While a definite diagnosis can only be made post-mortem, the following criteria has been established for clinical diagnosis of MUO: 1) Dogs should be over 6 months of age; 2) Multifocal, diffuse or focal neurological examination and intra-axial hyperintense lesions on T2-weighted magnetic resonance image (MRI) 3) CSF with pleocytosis with over 50% of monocytes 4) Infectious diseases should be ruled out. The prognosis is considered guarded. Relapses are common with higher risk found in cases with abnormalities in MRI/CSF in repeated investigations. Other brain immune-mediated diseases affecting dogs include eosinophilic meningoencephalitis of unknown origin, idiopathic hypertrophic pachymeningitis and idiopathic generalized tremor syndrome.

What are the main inflammatory diseases in cats?

Cats present in a more similar fashion to human patients, as most of the brain inflammatory brain diseases in cats are infectious. The most common include feline infectious peritonitis (FIP) and Toxoplasmosis. FIP clinical diagnosis relies in the combination signalment (pedigree or rescue cats from multi-cat households that are less than 4 years of age), history (recent stressful event), clinical signs (including ocular signs), albumin/globulin ratio, serology titters and alpha-1 acid glycoprotein level. MRI can reveal meningeal and ependymal contrast enhancement with a resulting obstructive hydrocephalus. Toxoplasmosis clinical diagnosis criteria include serological evidence of an active infection; other causes of clinical signs are excluded and a positive response to treatment. Although less common, auto-immune diseases can also affect cats, such as meningoencephalitis of unknown origin and feline limbic encephalitis.

How to treat neuroinflammation?

The primary aim of the treatment of inflammatory brain disease is to address the underlying cause. However, the brain unique defensive mechanism (BBB) makes the selection of the treatment of pivotal importance. For infectious causes, it is essential to consider the BBB penetrance prior to start antibiosis treatment. For immune-mediated diseases, the treatment focuses on mitigating the neuroinflammatory status. Most auto-immune brain diseases are treated with high doses of corticosteroids alongside a second line of immunosuppressive medications, which, ideally, cross the BBB. Finally, anti-viral treatment is now available, such as the metabolite GS-441524 or Remdesivir for FIP.

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5 STRATEGIES TO IMPROVE OUTCOMES IN VETERINARY TRAUMA PATIENTS

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Learning objectives:

- Apply lung-protective respiratory strategies to reduce ARDS and ventilator-associated complications in thoracic trauma.
- Implement haemostatic resuscitation principles and evidence-based fluid selection to improve survival in haemorrhagic shock.
- Prevent secondary neurological injury through targeted ICP management, appropriate osmotherapy, and evidence-based use of TXA and steroids.
- Initiate early multimodal analgesia to prevent respiratory deterioration and reduce mortality in trauma patients.
- Use point-of-care ultrasound (POCUS) to accelerate triage, guide immediate interventions, and improve diagnostic accuracy over radiography.

Proceeding:

Five evidence-based strategies are presented. Where veterinary-specific data is limited, recommendations are extrapolated from the best available human trauma literature.

Strategy 1: Protect the Lungs Early

Pulmonary contusions occur in 23–38% of dogs following blunt force trauma, with maximum destruction in the first 24 hours. Contusions affecting more than 20% of lung volume significantly increase ARDS and pneumonia risk. Lung ultrasound offers superior diagnostic sensitivity to radiography (90% vs 66.7%); CT remains gold standard. Maintain euvolaemia rather than restricting or aggressively administering fluids; use diuretics only for proven overload. Do not administer steroids. Prioritise non-invasive support and high-flow nasal oxygen; when mechanical ventilation is required, use tidal volumes of 6 mL/kg with plateau pressure not exceeding 30 cmH_2O. Correct pneumothorax before positive pressure ventilation, using low-bore chest tubes.

For rib fractures and flail chest, aggressive pain control is the single most impactful intervention to prevent respiratory failure; epidural analgesia is preferred. When emergency intubation is indicated (apnoea, SpO_2 below 90% despite supplementation, GCS below 9, SBP below 90 mmHg, or severe tachypnoea), use RSI with ketamine combined with midazolam and low-dose propofol; avoid etomidate. Capnography is mandatory; target normocapnia, as abnormal $EtCO_2$ markedly increases mortality (46% vs 29%).

Strategy 2: Resuscitate with Blood, Not Crystalloids

Excessive crystalloid causes haemodilution, acidosis, and hypothermia, negating the benefit of plasma resuscitation. Limit crystalloids to one bolus and prioritise blood products early, using balanced plasma-to-RBC ratios of at least 1:2, ideally 1:1; plasma and platelet deficits independently

predict mortality. Permissive hypotension with lower MAP targets reduces 24-hour mortality in uncontrolled haemorrhage.

Fluid selection matters: in non-TBI patients, balanced crystalloids such as Ringer's lactate prevent dilutional acidosis. In TBI patients, use 0.9% saline — a meta-analysis of 15 trials (35,207 patients) found balanced solutions associated with increased TBI mortality (OR 1.31, 95% CI 1.03–1.65). Monitor ionised calcium and base excess at admission: lower values at admission prior to transfusion are prognostic for severe haemorrhagic shock and should prompt early blood product mobilisation.

Strategy 3: Prevent Secondary Neurological Injury

In TBI, management is focused on preventing secondary damage. Maintain CPP (CPP = MAP - ICP), targeting ICP below 22 mmHg and CPP of 60–70 mmHg (extrapolated from human guidelines). Use a tiered approach: head elevation at 30° (less than 45°), adequate sedation, and hyperosmolar therapy with hypertonic saline preferred over mannitol, given as symptom-based boluses rather than continuous infusions. Reserve brief hyperventilation for acute herniation only. Do not use steroids — the underlying cytotoxic oedema does not respond to corticosteroids, and RCTs demonstrate increased harm. Administer TXA (15–20 mg/kg IV) only within 90 minutes of injury; beyond 2 hours it may cause harm. Seizure prophylaxis with levetiracetam for 7 days or fewer is reasonable, though withholding is equally acceptable.

In TBI, non-contrast CT is first-line imaging modality; however, goal would be for prognostication rather than surgical decision-making (epidural, subdural hematomas) in veterinary medicine. In spinal cord injury, use CT for spine clearance — radiographs cannot exclude cord injury. MRI is indicated when neurological deficits are present: cord oedema carries a better prognosis than haemorrhage or transection. Do not use methylprednisolone. Maintain MAP at 85 mmHg or above. Neurological status at presentation (SCI) and bilateral unresponsive pupils at presentation (TBI) are the strongest outcome predictors.

Strategy 4: Start Multimodal Analgesia Early

Untreated pain in trauma directly causes respiratory failure: it suppresses cough, retains secretions, worsens hypoxaemia and shunting, and increases ARDS risk. Early, aggressive analgesia is not supportive care — it is a primary intervention that changes outcomes.

For haemodynamically stable patients, fentanyl provides effective systemic analgesia but carries risk of respiratory depression. For haemodynamically unstable patients or those with TBI, ketamine at sub-anaesthetic doses is the preferred first-line agent due to NMDA antagonism and sympathomimetic blood pressure support. NSAIDs should only be introduced once the patient is haemodynamically stable with adequate renal perfusion and no coagulopathy. Dexmedetomidine at up to 1µg/kg/hr offers relief from anxiety and pain. Locoregional techniques should be initiated as early as feasible: epidural analgesia is the gold standard for flail chest; intercostal and paravertebral blocks are alternatives when epidural access is unavailable.

Strategy 5: Use POCUS to Drive Faster, Better Decisions

Thoracic POCUS (T-POCUS) should be the first-line imaging tool for unstable trauma patients. Pneumothorax is the highest-priority finding: absent lung sliding, absent B-lines, A-lines only, and the lung point to confirm diagnosis and estimate size. On M-mode, the barcode sign (stratosphere sign)

replaces the normal seashore sign. Haemothorax presents as fluid with swirling echoes; the jellyfish sign indicates lung collapse within effusion. Contusions appear as focal B-lines with patchy distribution, frequently missed on early radiographs. Diaphragmatic rupture may be identified by visualising abdominal organs within the thorax. Serial examinations are essential as lesions evolve and should drive immediate interventions such as thoracocentesis.

Abdominal POCUS using the fluid score (AFS, 0–4) guides transfusion decisions: scores of 3–4 predict anaemia, with approximately 25% requiring transfusion. CT remains first-line for TBI and spine clearance; MRI is reserved for SCI prognostication.

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RECOVER CPR GUIDELINES: BASIC LIFE SUPPORT

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Learning objectives:

- Demonstrate the correct chest compression technique for a dog or cat given a description of the patient's chest conformation and size.
- Demonstrate the correct bag-mask and mouth-to-nose ventilation techniques for a dog or cat in CPA.
- Explain how the approach to ventilation differs when the patient is endotracheally intubated.

Proceeding:

Introduction

The RECOVER 2024 Guidelines present rigorously developed, evidence-based recommendations for veterinary CPR, created through an international collaboration using GRADE methodology and systematic PICO-driven review of the veterinary and human literature; these proceedings summarize key updates to the BLS and associated monitoring recommendations.

Early, High-Quality BLS Improves Outcomes

Immediate initiation of high-quality BLS is strongly associated with improved return of spontaneous circulation (ROSC) and survival to discharge. BLS consists of chest compressions and ventilation, both of which are essential for maintaining perfusion and oxygen delivery to vital organs. Chest compressions should be delivered at a rate of 100–120 compressions per minute, with ventilation at 10 breaths per minute. This balance optimizes systemic blood flow while ensuring adequate oxygenation during resuscitation.

Chest compressions should begin immediately in any animal that is nonresponsive and apneic or agonal. Delays in compressions reduce the likelihood of ROSC and long-term functional survival. Early compressions maintain coronary and cerebral perfusion pressure, limit myocardial ischemia, and prime the heart for successful defibrillation when indicated. Delayed initiation allows accumulation of metabolic waste and worsens myocardial injury, reducing responsiveness to subsequent interventions.

Chest Conformation Determines Body And Hand Positioning

Animal size and thoracic conformation directly influence optimal patient positioning and compression technique. When compressions are delivered in lateral recumbency:

Round-chested dogs benefit from hand placement over the widest part of the thorax, utilizing the thoracic pump mechanism.

Keel-chested dogs (e.g., Greyhounds) benefit from compressions directly over the cardiac silhouette, engaging the cardiac pump mechanism.

Wide-chested dogs (e.g., Bulldogs) may benefit from dorsal recumbency with compressions applied to the sternum. Small dogs (approximately <8 kg) and cats typically require a circumferential two-thumb technique or one-handed compressions to ensure control and safety.

Compression depth must be adjusted to patient position. In lateral recumbency, compress the chest one-third to one-half of its width. In dorsal (sternal) recumbency, compress approximately one-quarter of thoracic depth to effectively compress the heart between the sternum and spine.

Ventilation Remains A Priority

Ventilation should be initiated as soon as possible after chest compressions begin. Adequate ventilation is particularly critical in asphyxial arrests, where hypoxemia is the primary driver of CPA. Without ventilation, circulated blood lacks sufficient oxygen to support myocardial and cerebral function, leading to poor resuscitation outcomes. Early integration of ventilation improves oxygen delivery and survival by limiting the consequences of prolonged hypoxemia.

Visible chest rise should be used as the primary indicator of adequate tidal volume. When airway pressure can be monitored, peak inspiratory pressure should not exceed 40 cm H_2O to avoid barotrauma and impaired venous return. Purpose-built bag-valve systems with pop-off valves help prevent excessive airway pressures.

When CPR is performed in patients already on mechanical ventilation, conversion to manual ventilation is recommended. Manual ventilation allows real-time adjustment of breath timing, volume, and pressure, avoids over-ventilation, and improves synchronization with compressions. Mechanical ventilators may not adapt rapidly enough during CPR, and excessive intrathoracic pressure can reduce cardiac output.

If endotracheal intubation is not immediately possible, a tight-fitting facemask with oxygen supplementation should be used to deliver positive-pressure breaths rather than mouth-to-nose ventilation. Facemasks provide higher and more consistent oxygen delivery, reduce rescuer exposure risk, and create a better seal to minimize oxygen leakage. Evidence supports improved ventilation quality and resuscitation outcomes when facemasks are used in non-intubated patients.

Compression Cycles And Minimizing Interruptions

Chest compressions should be delivered in continuous, uninterrupted 2-minute cycles. Sustained compressions maintain perfusion pressure and blood flow to the heart and brain. Even brief interruptions markedly reduce perfusion pressure, and the first compressions after a pause are less effective due to the time required to re-establish flow.

Pauses between cycles should be limited to less than 10 seconds and reserved for essential assessments such as pulse checks or rhythm evaluation once ECG monitoring is available. Effective team coordination is required to ensure that tasks are performed efficiently within planned pauses. Communication regarding timing and roles is essential to minimize hands-off time and preserve CPR quality.

Chest compressions should only be interrupted when there is strong objective evidence of ROSC, including a palpable femoral pulse independent of compressions, a marked rise in end-tidal CO_2 (during ALS), or spontaneous movement or breathing. Limiting interruptions to these situations maximizes perfusion and improves the likelihood of neurologically favourable outcomes.

Summary Of Major BLS Updates

RECOVER 2024 emphasizes immediate, uninterrupted, high-quality chest compressions; ventilation guided by visible chest rise and safe airway pressures; patient-specific positioning based on thoracic conformation; conversion to manual ventilation during CPR; and the use of facemasks with oxygen when intubation is not feasible. Collectively, these updates reinforce a physiology-driven, evidence-based approach to veterinary CPR aimed at improving ROSC chances and meaningful survival.

RECOVER CPR GUIDELINES: ADVANCED LIFE SUPPORT

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Learning objectives:

- Describe the priority order for initiating advanced life support interventions during CPR in dogs and cats, including monitoring, vascular access, and drug therapy.
- Compare and contrast the updated pharmacologic recommendations for non-shockable versus shockable arrest rhythms, including the roles of epinephrine, atropine, vasopressin, and antiarrhythmic agents.
- Discuss the evidence supporting key changes in the 2024 RECOVER ALS guidelines, such as the recommendation against high-dose epinephrine and the approach to escalating defibrillation energy.
- Identify current knowledge gaps in veterinary CPR research and recognize how the quality of available evidence influences the strength of clinical recommendations.

Proceeding:

The Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative was established to provide evidence-based guidelines for cardiopulmonary resuscitation (CPR) in dogs and cats. This lecture will review the key ALS recommendations from the 2024 RECOVER Guidelines and discuss the evidence supporting them.

Advanced life support (ALS) is defined as the aspect of CPR performed after basic life support (BLS) has been initiated; ALS measures are delivered while BLS is ongoing. ALS encompasses drug therapies such as vasopressors, anticholinergics, and antiarrhythmics; correction of electrolyte disturbances and volume deficits; and electrical defibrillation. Once BLS is underway, ALS interventions should be initiated in a specific priority order: establishing monitoring with electrocardiography (ECG) and capnography, obtaining vascular access (preferably intravenous [IV], with intraosseous [IO] as an acceptable alternative), and administering reversal agents if indicated. End-tidal CO_2 ($ETCO_2$) monitoring serves as a critical tool both for confirming endotracheal tube placement (values greater than or equal to 12 mmHg likely indicate proper placement) and for guiding optimization of cardiac output during CPR, with a target of 18 mmHg or greater.

The guidelines address the management of both non-shockable and shockable arrest rhythms. For non-shockable rhythms (asystole and pulseless electrical activity), vasopressors remain the cornerstone of pharmacologic therapy. Epinephrine at a standard dose of 0.01 mg/kg IV is recommended every 3 to 5 minutes, and notably, high-dose epinephrine is no longer recommended during CPR in dogs and cats. Atropine (0.04 mg/kg IV or IO) may be administered once during CPR for patients with non-shockable rhythms, particularly when the arrest was preceded by bradycardia due to high vagal tone. Repeated doses of atropine are not recommended.

For shockable rhythms (ventricular fibrillation and pulseless ventricular tachycardia), electrical defibrillation is the primary treatment. Biphasic defibrillators are recommended over monophasic defibrillators. The initial shock should be delivered at a standard biphasic dose (2 J/kg), and if unsuccessful, subsequent shocks should be delivered at double the initial dose (4 J/kg). The guidelines recommend against using epinephrine before the first defibrillation attempt in shockable rhythms and suggest using vasopressin (or epinephrine if vasopressin is unavailable) for shockable rhythms that persist beyond the first shock. Lidocaine is suggested as adjunctive therapy for refractory shockable rhythms in dogs but is not recommended in cats. Esmolol is suggested for shockable rhythms that do not convert after initial defibrillation, and amiodarone may be considered in cats. Additional pharmacologic interventions addressed by the guidelines include fluid therapy, calcium for hyperkalemia management, and naloxone administration for suspected opioid-related arrests. Routine fluid boluses are recommended against in euvolemic patients during CPR, while fluid therapy is recommended for patients with known or suspected hypovolemia. Calcium administration is recommended when hyperkalemia is known or suspected to have contributed to the arrest.

Despite these advancements, the overall quality of evidence remains very low due to the near absence of clinical data in dogs and cats, and many recommendations are extrapolated from human and experimental animal studies. Continued research in veterinary species is essential to close these knowledge gaps and further improve CPR outcomes for dogs and cats.

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LOW NA/K RATIOS: IT IS NOT ALWAYS ADDISON'S

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Learning objectives:

- Identify patients with an abnormal sodium-potassium ratio
- Recognize the most common causes of a low sodium-potassium ratio
- Develop a diagnostic approach to identify the common causes of a low sodium-potassium ratio
- Understand the treatment approach to patients with a low sodium-potassium ratio

Proceeding:

A low ratio of blood sodium-to-chloride concentration (Na:K) is a common tool used to help identify patients with hypoadrenocorticism. The normal Na:K in dogs ranges from 27:1 to 40:1. As hypoadrenocorticism is uncommon in cats, an established range for Na:K has not been established.

Pathophysiology

The pathophysiology for the development of a low Na:K can be divided into a few common mechanisms. Low Na:K can be the result of hyponatremia, hyperkalemia or both these abnormalities. As a consequence, pathophysiologic mechanisms are mechanisms of hyponatremia, hyperkalemia or both. One mechanism of a low Na:K is depletion of effective circulating volume (ECV). Decreased ECV will upregulate the renin-angiotensin-aldosterone system and stimulate antidiuretic hormone (ADH) release. This results in reductions in glomerular filtration rate (GFR) and renal reabsorption of sodium and water. If the animal continues to drink water but does not eat, there will be more water reabsorbed than sodium, leading to hyponatremia. Hyperkalemia can also develop if glomerular filtration rate is reduced for a significant period of time as this can impair renal potassium excretion. Another mechanism of low Na:K is urinary tract disease. Kidney disease is commonly associated with hyponatremia due to inability to adequately excrete sufficient free water and hyperkalemia is a result of inadequate GFR due to intrinsic renal disease or post renal obstruction. As azotemia is a common finding in dogs with an Addisonian crisis, it can be a diagnostic dilemma to differentiate dogs with Addisons from dogs with acute kidney injury from blood work changes alone. By evaluation of history, signalment, and in some cases, abdominal imaging, these cases can usually be distinguished. With an adrenocorticotrophic hormone stimulation test as the definitive tool to confirm the diagnosis of hypoadrenocorticism.

Causes

A recent retrospective study of dogs and cats at the author's institution identified a low Na:K in approximately 7% of animals that had blood electrolyte concentrations evaluated. The most common primary disease process in dogs was gastrointestinal disease (35%) while hypoadrenocorticism was an uncommon cause, accounting for 2% of the dogs with a low Na:K. As hypoadrenocorticism

commonly causes gastrointestinal signs, it is important that all dogs with a low Na:K and gastrointestinal signs are not assumed to have hypoadrenocorticism. A reported cause of low Na:K is body cavity effusions such as chylothorax. Body cavity effusions were evident in 15% of dogs and 12% of cats with low Na:K in our study. Effusions can lead to this electrolyte abnormality through depletion of ECV which is often exacerbated by large volume paracentesis. Although some dogs with gastrointestinal disease develop abdominal effusion, it is interesting to note that most of the cases in this study did not have effusion. In contrast to dogs, the most common cause of a low Na:K in cats was kidney disease (52%) and lower urinary tract disease (30%), although gastrointestinal disease was still a relatively common finding in this population (25% of cases). When dogs and cats with a low Na:K were compared, dogs had a lower median sodium concentration, and cats had a higher median potassium concentration. This fits with the common underlying diseases in these populations.

Treatment

Treatment of a low Na:K needs to be tailored based on the underlying mechanism by which it has developed. Hyponatremia secondary to low ECV generally responds to volume replacement alone. If severe hyponatremia is evident, monitoring during volume replacement to avoid overly rapid resolution of hyponatremia is important. If the predominant electrolyte abnormality is hyperkalemia, then emergency interventions may be warranted.

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HEMODIALYSIS: WHEN? WHY? HOW? DO THEY LIVE?

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Learning objectives:

- Recognize evidence-based indications for hemodialysis initiating in animals with severe acute kidney injury and understand the importance of timely, proactive intervention.
- Compare intermittent hemodialysis and continuous renal replacement therapy with respect to mechanisms, clinical applications, advantages, and limitations in the management of acute kidney injury.
- Apply fundamental principles of renal replacement therapy prescription to guide platform selection, treatment modality choice, and safe delivery of extracorporeal therapies in dogs and cats with acute kidney injury.
- Identify the major prognostic determinants of acute kidney injury, including severity, etiology, reversibility, comorbidities, in animals with acute kidney injury undergoing hemodialysis

Proceeding:

Acute kidney injury (AKI) is defined as an injury to renal parenchyma, often associated with a decrease in kidney function. AKI may result in the accumulation of uremic toxins leading to a wide range of clinical signs and clinicopathological disturbances, some of which are life threatening. Prognosis and long-term outcome are multifactorial and depend on the severity (which dictates the window of opportunity for recovery), the underlying etiology (which often dictates reversibility), comorbidities, complications, and treatment availability. The prognosis of AKI patients is guarded; however, when considering the prognosis for the individual patient, one has to take into consideration that the reported mortality rates in veterinary medicine are highly influenced by euthanasia, which may lead to overestimation of true fatality rates of aggressively managed patients. The long-term outcome is largely determined by renal reversibility, with a proportion of survivors developing chronic kidney disease. In many cases, the kidneys are able to recover from the initial insult, providing a sufficient time frame; however, the consequences of uremia might lead to death before recovery occurs. In severe AKI cases (i.e., IRIS AKI Grades 4 and 5), conventional medical management provides only a limited window of opportunity for recovery. In contrast, in the absence of fatal complications or financial constraints, renal replacement therapies can substantially extend this window of opportunity for recovery, potentially allowing for unlimited time for healing and regeneration. Therefore, renal replacement therapies improve outcomes of animals with AKI.

Hemodialysis effectively restores homeostasis in AKI patients by facilitating the removal of uremic toxins, correcting electrolyte acid base abnormalities, and fluid overload. Hemodialysis should be considered when conventional medical management *is expected to be insufficient*. Timely initiation is critical, as uremic toxins exert systemic effects and contribute to progressive dysfunction of body organ systems. Delayed intervention is associated with increased morbidity and mortality,

particularly when organ damage has already developed. *Therefore, hemodialysis should be applied proactively in patients with advanced AKI, before the onset of multiorgan dysfunction*, rather than as a salvage therapy once irreversible systemic complications are established. Hemodialysis is indicated for animals with severe AKI characterized by progressive azotemia (serum creatinine concentration > 5 mg/dL and trending up despite adequate hydration), persistent anuria or severe oliguria, refractory metabolic derangements (e.g., severe metabolic acidosis), electrolyte abnormalities (e.g., hyperkalemia), and clinically significant fluid overload refractory to diuretic therapy.

Renal replacement therapy can be delivered through multiple platforms and treatment modalities, most commonly intermittent hemodialysis (IHD) and continuous renal replacement therapy (CRRT), which together offer complementary approaches for the management of AKI. Intermittent hemodialysis is characterized by short (4-6 hours) treatments with high solute and fluid clearance rates, whereas CRRT provides slower clearance applied over prolonged periods, typically exceeding 24 hours, which confers advantages as the treatment can be delivered bedside in the ICU setting in animals with cardiovascular instability. CRRT is better designed to deliver gradual correction of uremia and overhydration. Importantly, the defining features of renal replacement therapy are determined by the treatment prescription rather than the platform (i.e., dialysis machine) that is used to deliver the treatment. Intermittent therapies may be delivered using platforms designed for continuous treatment, although with reduced efficiency, and continuous treatments can be delivered on intermittent hemodialysis systems, albeit with some potential practical and technical constraints.

The goals of the lecture are to provide guidance on the appropriate timing of dialytic intervention, practical tools for platform selection, and an introduction to fundamental principles of prescription writing for animals with AKI.

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Advanced Stream, Friday 5 June 2026

UNIQUE AND EMERGING INTOXICATIONS: EXPERIENCE FROM A VETERINARY DIAGNOSTIC LABORATORY

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Learning objectives:

- Appreciate several intoxications that might be overlooked in a clinical setting.
- Understand clinical presentations, mechanisms of toxic action, and diagnostic and treatment approaches for these toxicants.

Proceeding:

Mushrooms

Potentially toxic mushrooms are a small percentage of thousands of mushroom species. Unfortunately, there is no simple approach to identify toxic from non-toxic mushrooms without appropriate experience and training. Confirmed mushroom intoxications in pets are relatively uncommon, likely due to lack of a history of exposure and availability of toxicologic testing. The majority of confirmed cases are caused by hepatoxic mushrooms that contain amanitin; exposure to these mushrooms is serious and life-threatening. In the absence of a history of exposure and positive identification of an ingested mushroom, diagnostic tests are available to confirm exposure to amanitin-containing mushrooms. Another group of mushrooms causing serious illness and death contain muscimol and ibotenic acid. These toxins interfere with glutamate and GABA-mediated neurotransmission causing seizures and tremors followed by CNS depression.

Blue-green algae (Cyanobacteria)

Harmful algal blooms (HAB) are a global problem. All HAB are potentially hazardous, although there is no way to determine potential risk from visual clues. Toxic algae are most commonly found in fresh water environments, but they can proliferate in brackish waters as well. There are two major classes of toxins produced by HAB. Anatoxins are neurotoxic and produced mainly by *Anabaena* spp. Clinical signs of intoxication are due to cholinergic stimulation and include rapid onset of rigidity and muscle tremors followed by paralysis, cyanosis and death. The second class of toxins are hepatotoxic microcystins. Clinical signs of intoxication include those related to acute liver failure. Algal toxins affect most species, although dogs are the most likely to be intoxicated due to their frequent exposure to HAB. Given the rapidity of onset of clinical signs and death following toxin exposure, treatment is often not possible. Testing can be helpful in assessing the risk of a bloom and making a diagnosis of exposure/intoxication.

Tremorgenic Mycotoxins

Penitrem A and roquefortine are tremorgenic mycotoxins produced by *Penicillium* spp. Pet exposure to these toxins occurs through ingestion of moldy foods or access to compost piles. Penitrem A alters the spontaneous release of neurotransmitters such as glutamate, aspartic acid, and GABA. Clinical

signs often develop within minutes to a few hours following ingestion, with GI and neurologic signs most commonly exhibited. The objectives of treatment are decontamination when appropriate, controlling tremors and seizures and providing supportive care.

Grapes

Ingestion of grapes, raisins, currants and sultanas has been associated with acute renal failure (AKI) in dogs. Recently, tartaric acid has been strongly implicated as the causative agent. Dogs are uniquely sensitive to intoxication because they lack a renal anion transporter (OAT-4) that normally excretes organic acids into renal tubules. It is thought that the build-up of tartaric acid in proximal in renal tubular cells causes ATP depletion and cell death. Appropriate decontamination is recommended if more than 1 grape or raisin is ingested per 5 kg. Treatment for AKI follows standard protocols.

Diethylene Glycol

Diethylene glycol (DEG) can be found in lotions, skin creams, deodorants, brake fluid, lubricants, wall paper strippers, and heating/cooking fuel. DEG intoxication has similar pathophysiologic and clinical effects to ethylene glycol, with the exception that oxalate crystalluria does not occur following DEG ingestion. Like ethylene glycol, DEG reportedly has a sweet taste which can be attractive to some animals.

Adverse Effects of Decontamination Strategies

In general, common decontamination strategies such as use of emetics, activated charcoal, and lipid emulsions (LE) are not associated with adverse effects (AE). However, AE have occurred from the use of hydrogen peroxide to induce vomiting and LE to alter the distribution and clearance of lipophilic toxicants. Hydrogen peroxide (3%) can cause significant GI mucosal damage. LEs have been associated with a variety of adverse effects including pancreatitis, corneal lipidosis, hemolysis, and fat overload syndrome. In addition, LE can interfere with some clinical laboratory tests.

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EXTRACORPOREAL THERAPIES FOR INTOXICATIONS

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Learning objectives:

- Describe the role of extracorporeal therapies in the management of severe intoxications in companion animals and identify clinical scenarios in which enhanced toxin removal is indicated.
- Understand the key toxicokinetic properties of intoxicants, including molecular weight, protein binding, and volume of distribution, that determine suitability for extracorporeal removal.
- Compare the mechanisms, capabilities, and limitations of extracorporeal modalities used for toxin removal, including intermittent hemodialysis, continuous renal replacement therapy, hemoperfusion, plasma adsorption, and therapeutic plasma exchange.
- Apply toxicologic and clinical principles to select the most appropriate extracorporeal platform and modality for specific intoxications.
- Recognize the phenomenon of toxin rebound following extracorporeal therapy and incorporate appropriate monitoring and treatment strategies to achieve sustained detoxification.

Proceeding:

Intoxications represent a frequent clinical problem in veterinary medicine and remain an important cause of morbidity and mortality in companion animals. In many cases, toxic exposures can be managed successfully with supportive medical therapy alone, particularly when the ingested dose is low, the toxic compound has rapid endogenous clearance, the clinical consequences are mild, or a specific antidote is available. However, a subset of intoxications is associated with severe and potentially life threatening systemic effects, in which spontaneous elimination is insufficient or too slow to prevent irreversible organ injury or death. In these situations, active enhancement of toxin removal becomes a critical component of patient management. Extracorporeal therapies provide an effective means of accelerating the clearance of selected endogenous and exogenous solutes and have an expanding role in the treatment of severe intoxications in veterinary patients.

Several extracorporeal modalities are available for toxin removal, including intermittent hemodialysis, continuous renal replacement therapy, hemoperfusion, plasma adsorption, and therapeutic plasma exchange. Each modality is characterized by distinct mechanisms of solute removal, technical requirements, efficiencies, and risk profiles. Appropriate selection of extracorporeal therapy requires a thorough understanding of both the toxicologic properties of the compound and the operational principles of each technique. Key characteristics of the intoxicant that influence extracorporeal removal include molecular weight, degree of protein binding, volume of distribution, water solubility, and endogenous clearance rate. Among these variables, volume of distribution is a fundamental determinant, as extracorporeal techniques can remove solutes only

from the intravascular compartment. Toxins with a large volume of distribution, typically exceeding 2L/kg, are extensively sequestered within tissues and are therefore poorly amenable to extracorporeal removal despite intervention.

Hemodialysis is one of the most widely used extracorporeal platforms for toxin removal. Intermittent hemodialysis relies primarily on diffusion across a semipermeable membrane, driven by concentration gradients between the blood compartment and the dialysate compartment. Diffusive clearance is highly dependent on solute molecular weight and protein binding, with optimal efficiency achieved for small, water soluble compounds with minimal protein association. In general, intermittent hemodialysis is most effective for solutes with molecular weights below 5000 Daltons and low protein binding. The addition of convective transport, achieved through ultrafiltration and solvent drag, can enhance solute removal and partially mitigate the limitations imposed by molecular size. Convection is less sensitive to molecular weight than diffusion, although solutes must still be sufficiently small to traverse the membrane pores.

When toxins exhibit high protein binding or molecular weights beyond the effective range of dialysis membranes, adsorption based techniques may offer a more suitable alternative. Hemoperfusion employs sorbent materials, such as activated charcoal or synthetic resins, to directly bind solutes from the blood. This modality can effectively remove compounds with molecular weights up to approximately 40 K Daltons and retains efficacy even for highly protein bound substances, including NSAID. Plasma adsorption systems share similar principles but may provide enhanced selectivity depending on the sorbent material used.

For toxins with very large molecular weights or when protein binding severely limits other modalities, therapeutic plasma exchange represents a viable option. This technique removes plasma containing the toxin and replaces it with donor plasma or other replacement fluids, thereby eliminating solutes independent of molecular size. Therapeutic plasma exchange can be performed using either centrifugal or membrane-based technologies, each associated with specific logistical considerations, costs, and complication profiles.

Selection of the optimal extracorporeal modality is rarely based on toxin characteristics alone. Clinical decision making is also influenced by patient stability, anticipated complications, availability of equipment, and operator expertise. Regardless of the platform and modality, clinicians must recognize that even after successful extracorporeal removal and apparent clinical improvement, toxin rebound is common due to redistribution from peripheral tissues back into the intravascular space. Ongoing monitoring and, in some cases, repeated treatments are therefore essential to achieve sustained detoxification.

The goals of this lecture are to introduce the various platforms and modalities available for toxin removal and to provide guidance on identifying which toxins are appropriate candidates for removal using each extracorporeal platform.

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PLASMA EXCHANGE: CORE CONCEPTS AND CURRENT APPLICATIONS IN VETERINARY MEDICINE

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Learning objectives:

- Define total plasma exchange (TPE) and describe its fundamental principles.
- Understand the historical development and established indications of TPE in human medicine.
- Identify current and emerging applications of TPE in veterinary medicine.
- Compare similarities and differences in indications, techniques, and outcomes between human and veterinary TPE.
- Appreciate the limitations, risks, and future directions of TPE in veterinary clinical practice.

Proceeding:

Therapeutic plasma exchange (TPE) is an extracorporeal blood purification technique in which plasma is separated from cellular components to remove circulating high–molecular weight pathogenic substances, including autoantibodies, immune complexes, paraproteins, inflammatory mediators, and protein-bound toxins. First described by John Jacob Abel in 1914, the technique became widely adopted in human medicine during the late twentieth century and has progressively expanded into veterinary emergency and critical care over the past two decades as extracorporeal technologies have become more accessible.

The therapeutic benefit of TPE is multifactorial. The primary mechanism is rapid reduction of circulating pathogenic solutes that are largely confined to the intravascular space, allowing prompt decrease in disease burden. In addition, TPE exerts immunomodulatory effects through complement removal, alteration of Fc-receptor interactions, and cytokine modulation, creating a transient immunologic reset that is particularly valuable during the delayed onset of immunosuppressive therapies. In hyperviscosity syndromes, plasma removal improves microcirculatory rheology, enhances tissue perfusion, and reduces thrombotic risk, which often explains the rapid clinical improvement observed following treatment.

Veterinary indications for TPE continue to expand but remain supported primarily by case series and retrospective studies. The most frequently reported applications include refractory immune-mediated hemolytic anemia and immune-mediated thrombocytopenia, intoxications involving highly protein-bound drugs such as non-steroidal anti-inflammatory agents, hyperviscosity associated with multiple myeloma or severe leishmaniasis, neurologic immune-mediated disorders including myasthenia gravis and acute polyradiculoneuritis, and renal vasculopathic conditions such as

cutaneous and renal glomerular vasculopathy. In these settings, TPE is typically employed as adjunctive or bridging therapy rather than definitive disease control.

Plasma exchange modalities are classified according to the separation technique. Although the terms plasmapheresis and TPE are often used interchangeably, TPE specifically refers to plasma removal followed by replacement. In veterinary patients, separation is achieved either by centrifugal TPE (cTPE) or membrane-based TPE (mTPE). Both techniques require placement of a double-lumen large-bore jugular catheter positioned near the right atrium to ensure adequate extracorporeal blood flow and safe reinfusion.

In centrifugal systems, plasma is separated according to density gradients generated by centrifugal force, resulting in efficient separation with relatively smaller processed blood volumes. However, limitations include lower achievable blood flow rates, prolonged treatment duration, increased anticoagulant requirements, and extracorporeal circuit volumes that may restrict use in smaller patients. Membrane-based TPE separates plasma via filtration across semipermeable hollow-fiber membranes with pore sizes typically ranging from 0.2 to 0.6 µm. Membrane permeability is characterized by the sieving coefficient, which determines solute removal efficiency. For example, immunoglobulin G (150 kDa) demonstrates near-complete membrane passage, whereas the larger immunoglobulin M (approximately 950 kDa) shows slightly reduced but still clinically meaningful clearance. Filtration efficiency may decline due to membrane fouling, reflected by increased transmembrane pressure; maintaining filtration fractions below approximately 20–25% reduces this risk.

Effective anticoagulation is essential for circuit patency. Regional citrate anticoagulation is frequently preferred, particularly in centrifugal systems, as it provides localized anticoagulation while minimizing systemic bleeding risk and may be advantageous in coagulopathic patients. Citrate chelation of ionized calcium necessitates continuous calcium supplementation and monitoring to prevent hypocalcemia, metabolic alkalosis, and electrolyte disturbances. Systemic unfractionated heparin is more commonly used in membrane-based systems and is administered as a pre-treatment bolus followed by continuous infusion targeting clotting times approximately 1.5–2 times baseline. Hybrid approaches may be considered to optimize circuit lifespan while minimizing metabolic complications.

Complications of TPE are generally predictable and manageable with appropriate monitoring. Metabolic abnormalities include hypocalcemia, electrolyte disturbances, metabolic alkalosis, and hypothermia. Hemodynamic instability may occur due to extracorporeal volume shifts and oncotic changes, particularly in critically ill or hypovolemic patients. Hematologic effects include thrombocytopenia and coagulation factor depletion, while technical complications encompass catheter dysfunction, circuit clotting, hemolysis, and membrane fouling. Infectious risks related to vascular access and hypersensitivity reactions to replacement fluids should also be considered.

The kinetics of solute removal during TPE follow an exponential decay model approximating first-order elimination for intravascular solutes. Exchange of one plasma volume removes roughly 60–65% of circulating target substances, with diminishing returns during larger exchanges due to redistribution from extravascular compartments. Consequently, serial treatments are often required in antibody-mediated diseases, and exchange volumes exceeding approximately 1.5 plasma volumes provide limited additional clearance while increasing procedural risk and resource utilization.

Accurate treatment prescription requires estimation of plasma volume, calculated as (0.09 × body weight in kg) × (1 − hematocrit) in dogs and (0.06 × body weight in kg) × (1 − hematocrit) in cats. To reduce hemodynamic complications, extracorporeal circuit volume should generally remain below 15% of total blood volume, although circuit priming may mitigate this limitation. Replacement fluid selection remains clinically important; although no ideal solution exists, fresh frozen plasma is most commonly used in veterinary patients because it restores coagulation factors, immunoglobulins, and oncotic support, often supplemented with isotonic crystalloids.

Overall, TPE represents an increasingly valuable adjunctive modality in veterinary critical care, offering rapid removal of pathogenic plasma constituents and meaningful clinical stabilization when applied with appropriate patient selection, prescription accuracy, and vigilant monitoring.

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NEUROLOGICAL ABNORMALITIES - DIFFERENTIATING THE EFFECTS OF SYSTEMIC DISEASE FROM PRIMARY BRAIN PATHOLOGY

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Learning objectives:

- Recognise how systemic illness can affect the brain
- Make distinctions between primary and secondary brain disease
- Briefly review the clinical signs of forebrain, cerebellar or brainstem pathology
- Identify signs of structural or focal brain pathology

Proceeding:

How can systemic illness affect the brain?

The brain can be affected by systemic illness. In fact, sickness behaviour, such as fever, anorexia, lethargy, altered sleep and social withdrawal are adaptive brain changes that are aimed at increasing survival. Brains are highly active organs metabolically and consume 20-25% of daily whole-body glucose. Therefore, it can be challenging to distinguish the effects of systemic disease from diffuse brain pathology.

What can differentiate between primary and secondary brain disease?

Dogs and cats can be presented with a typical neurological manifestation (such as a generalised tonic-clonic seizure) as a manifestation of illness elsewhere in the body (reactive seizures). Therefore, it is important to not assume that there is a structural brain pathology when presented with neurological clinical signs. A stepwise approach to ascertain if there is a primary or secondary brain disease is necessary. This includes not only ancillary tests, but a thorough history combined with a physical and neurological examination. Moreover, repeat examinations are very important to monitor trends and amend plans when necessary.

What are the clinical signs of forebrain, cerebellar or brainstem pathology?

Knowledge of the clinical signs seen with specific brain localisation is important. Forebrain pathology is characterised by decreased in mental status and abnormal behaviour, with very often a normal gait. Compulsive behaviour, such as circling, is a very characteristic clinical sign for forebrain pathology, but it is not limited to structural pathology. Seizures are of the most common clinical signs of forebrain disease seen in animals. Cerebellar lesions cause clinical signs related to the rate, range, direction, and force of voluntary movements. Therefore, dysfunction of this part of the brain causes a cerebellar ataxia, characterised by dysmetria (mostly perceived as hypermetria clinically), intention tremors and titubation. Vestibular clinical signs can also be seen with cerebellar lesions (paradoxical vestibular syndrome). Finally, the brainstem is essential for life, as it is the command centre for involuntary control of the cardiovascular and respiratory systems. It is also indispensable for the consciousness state through the Reticular Activating System (RAS). The brainstem is composed by the

nuclei of all cranial nerves, except cranial nerves I (olfactory) and II (optic nerve). Assessment of cranial nerve function can aid in recognising if a disease process is affecting the brainstem. This can be of particular significance when presented with a dog or cat with diffuse brain clinical signs, where there are concerns about increased intracranial pressure. Given importance for life, it is essential for the clinician to recognise if there are brainstem clinical signs.

How to identify clinical signs of structural or focal brain pathology?

Differentiating diffuse from unilateral clinical signs could be the key to distinguish primary from secondary brain pathology. Structural brain pathology, such as neoplasia or inflammatory disease, very often cause abnormalities in only one side of the body. There are three main abnormalities that could be present with focal structural forebrain pathology, including contralateral decreased or absent menace response, nostril sensation and postural reactions. In the other hand, with unilateral brainstem clinical signs, particularly cranial nerve dysfunction (such as vestibular signs), ipsilateral primary structural pathology could be considered. However, while the lateralised clinical signs can raise the suspicion for structural brain disease, when presented with cases with neurological abnormalities that are diffuse, bilateral and symmetrical this is more challenging. For those cases, when systemically illness has been ruled out, and no improvements are perceived with supportive treatment, investigations for primary brain pathology are indicated.

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STAYING ON TRAC WITH TRANSFUSION REACTIONS

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Learning objectives:

- Improve understanding of the pathophysiology underlying commonly encountered acute transfusion reactions (TRs).
- Increase confidence in identifying and managing acute TRs using recently published veterinary definitions.
- Be aware of the incidence of acute TRs using current definitions and appreciate possible associated risk factors.

Proceeding:

Introduction: Acute transfusion reactions (TRs) remain important potential complications of blood product administration in dogs and cats. While many transfusions occur uneventfully, early recognition of reactions is essential to reduce morbidity and mortality. This presentation reviews the pathophysiology of acute TRs and summarises current veterinary definitions and management recommendations, with emphasis on the Association of Veterinary Hematology and Transfusion Medicine (AVHTM) Transfusion Reaction small Animal Consensus Statement (TRACS). Recent data regarding incidence and risk factors for TRs in dogs and cats, using TRACS criteria, are also presented.

Classifying Transfusion Reactions: The TRACS guidelines, published in 2021 in the Journal of Veterinary Emergency and Critical Care, provide the first consensus-derived veterinary definitions for individual clinical TRs. Each reaction type is associated with specific diagnostic criteria, including required clinical signs, laboratory abnormalities, and temporal association with blood product administration. TRACS also outlines alternative diagnoses to exclude, and provides consensus recommendations for prevention, monitoring, and treatment. Prior to TRACS, inconsistent terminology and variable definitions across studies hindered comparison of findings and limited interpretation to a wider population. Adoption of standardised TRACS terminology improves communication within the veterinary team and with clients, enhances TR surveillance, research, future benchmarking and the identification of possible risk factors.

Transfusion Reaction Pathophysiology: Relevant acute TRs in small animal practice include febrile non-haemolytic TRs (FNHTRs), acute haemolytic TRs (AHTRs), allergic reactions, transfusion-associated circulatory overload (TACO), and transfusion-related acute lung injury (TRALI).

FNHTRs generally arise from cytokine release by recipient immune cells or donor leukocyte-derived pyrogens. TRACS defines an FNHTR as development of pyrexia exceeding 39.2°C with an increase of at least 1°C, in the absence of another aetiology. Proper classification is essential because FNHTRs may resemble more severe reactions.

AHTRs may be immunologic or non- Immunologic AHTRs result from donor–recipient incompatibility, leading to complement-mediated intravascular haemolysis or extravascular haemolysis via opsonisation and phagocytosis. Non-immunologic haemolysis may result from mechanical, osmotic, or thermal injury and may present with haemoglobinaemia or haemoglobinuria. Clinical signs may vary dependent on the aetiologic cause, but both carry morbidity and mortality risk.

Allergic reactions are usually IgE-mediated responses to donor plasma proteins and may manifest as dermatologic, gastrointestinal, or respiratory signs. Anaphylaxis is rare.

TACO arises when transfusion induces volume overload, leading to pulmonary oedema. Cats may be particularly susceptible due to compensatory blood volume expansion associated with anaemia.

TRALI is an acute, immune-mediated pulmonary reaction. Reports in veterinary medicine are limited, and cases may not meet full TRACS criteria.

Transfusion Reaction Identification and Management: Effective detection of TRs requires monitoring prior to, during, and after transfusion. The AVHTM monitoring sheet and therapeutic and diagnostic algorithms assist clinicians in detecting TRs and how to maage them. When a TR is suspected, transfusion should be paused immediately while clinical assessment and diagnostics are performed.

Incidence of Transfusion Reactions in Dogs and Cats: Two recent large, prospective, multicentre studies applying TRACS definitions provide current estimates of TR incidence. In 858 dogs receiving 1542 units, acute TR incidence was 8.9%. FNHTRs were most common in packed red blood cell transfusions (4%), while allergic reactions were most frequent with plasma (3.2%). Higher pRBC dose and older units increased odds of FNHTR and AHTR. In 444 cats receiving 608 units, overall incidence was 7.8%, with FNHTRs predominating in RBC-containing transfusions (5.7%). Use of infusion pumps and older RBC units were associated with higher FNHTR odds. No cases of TRALI, septic reactions, or anaphylaxis were reported.

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UNCONTROLLED HEMORRHAGIC SHOCK: PRACTICALITIES OF MASSIVE TRANSFUSION

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Learning objectives:

- Understand the pathophysiology of hemorrhagic shock
- Describe how to perform massive transfusion
- Understand complications of massive transfusion
- Understand appropriate diagnostic tests to perform during hemorrhagic shock

Proceeding:

Learning Outcome 1: Understand the pathophysiology of hemorrhagic shock

Hemorrhagic shock is a consequence of both hypovolemia (reduced stroke volume secondary to blood loss resulting in decreased preload) and decreased arterial oxygen content due to decreasing hemoglobin concentration as fluid redistributes into the intravascular space and when 'clear' IV fluids are given as part of treatment. Acute traumatic coagulopathy (ATC) / acute coagulopathy of trauma shock (ACOTS) results and contributes to ongoing blood loss in patients with hemorrhagic shock. Hypothermia and acidosis that result from shock lead to reduced coagulation factor activity, creating the well-recognized 'lethal triad' of trauma. Without early intervention, these processes become self-perpetuating and rapidly fatal.

In veterinary medicine, massive transfusion has been defined as transfusion of a volume of whole blood or blood components exceeding the patient's estimated blood volume within a 24-hour period, approximately 80–90 mL/kg in dogs and 50–60 mL/kg in cats. An alternative and increasingly useful concept is that of critical bleeding or massive hemorrhage, which emphasizes the magnitude and rate of blood loss rather than the volume transfused, such as loss of an entire circulating blood volume within 24 hours or 50% within 3 hours.[1]

Critical bleeding most commonly occurs into the peritoneum, but may also include bleeding into the pleural space, subcutaneous space, gastrointestinal tract, or external bleeding. Some of the most common causes of large volume hemoperitoneum include bleeding from the liver, spleen, great vessels, or adrenals. Hemorrhage may be due to vascular injury from trauma, a bleeding neoplasm, or related to surgery particularly in patients with underlying coagulopathy. Anticoagulant rodenticide induced coagulopathy can also cause critical bleeding.

Learning Outcome 2: Describe how to perform massive transfusion

Early management priorities include rapid hemorrhage control, establishment of vascular access to facilitate rapid fluid administration, active patient warming, and activation of a massive transfusion protocol (MTP). Early communication with the blood bank and coordinated team-based care are critical to minimise delays and errors during resuscitation.

The ideal transfusion product for massive transfusion is fresh whole blood (FWB), as it provides red blood cells, coagulation factors, and platelets in physiologic proportions and is not refrigerated, thereby reducing hypothermia. When FWB is unavailable, stored whole blood is an acceptable alternative and may be suitable for up to 21 days of refrigerated storage.[2] In many veterinary settings, massive transfusion initially relies on component therapy, with packed red blood cells and plasma administered until whole blood donors can be mobilized.

Autotransfusion is an important adjunct in cases of cavitary haemorrhage and represents an efficient use of resources while reducing compatibility concerns.[3] Large volumes can be safely returned in a short period, although decisions regarding anticoagulant use should be individualized to minimise the risk of citrate toxicity and exacerbation of coagulopathy.

In human medicine, component therapy in a 1:1:1 ratio of red blood cells, plasma, and platelets is standard, but platelet availability remains limited in veterinary medicine. Thawed or refrigerated plasma stored for rapid use may facilitate early hemostatic resuscitation.[4] Cryoprecipitate or factor concentrates can be incorporated when specific deficiencies are identified using coagulation testing. Emerging access to lyophilized platelet products may further improve hemostatic support, and xenotransfusion of canine lyophilized platelets has been described in feline patients. In cats, red blood cell xenotransfusion may be used as a salvage therapy when feline blood supplies are exhausted, although delayed hemolytic reactions should be anticipated.

Antifibrinolytic therapy, particularly tranexamic acid, is recommended in patients with critical bleeding. Evidence from human trauma medicine supports early administration within hours of injury, and veterinary case reports demonstrate benefit in patients with documented hyperfibrinolysis.

Learning Outcome 3: Understand complications of massive transfusion

Patients receiving massive transfusion are at increased risk of transfusion-associated complications, many of which are dose-dependent. A full discussion of transfusion reactions is beyond the scope of this lecture but the reader is directed to consensus resources.[5] Citrate toxicity leading to hypocalcemia and hypomagnesaemia is common, particularly when large volumes are administered rapidly. Prophylactic calcium supplementation, typically initiated after the first one to two units of blood products, is recommended during massive transfusion.

Transfusion-associated circulatory overload (TACO) is another important complication, especially in cats, where assessment of volume status can be challenging. Serial physical examination and point-of-care ultrasound can aid early recognition, and judicious use of diuretics may be required.

Learning Outcome 4: Understand appropriate diagnostic tests during haemorrhagic shock

Serial diagnostic testing is essential during haemorrhagic shock to guide resuscitation. Viscoelastic testing, such as thromboelastography or rotational thromboelastometry, provides real-time assessment of clot formation, strength, and fibrinolysis, allowing targeted haemostatic therapy rather than empiric transfusion alone.

Additional monitoring includes serial packed cell volume or haemoglobin concentration, lactate, acid-base status, and electrolyte concentrations—particularly ionized calcium. Point-of-care

ultrasound is valuable for tracking ongoing haemorrhage, volume status, and identifying complications such as TACO.

Conclusions

Critical bleeding and haemorrhagic shock requires rapid recognition, decisive intervention, and coordinated delivery of massive transfusion. While historically associated with poor outcomes, recent veterinary data demonstrate improving survival when structured protocols, haemostatic resuscitation, and vigilant monitoring are employed. Understanding the underlying pathophysiology, practical execution of massive transfusion, anticipated complications, and appropriate diagnostic testing is essential to improving outcomes in dogs and cats with life-threatening haemorrhage.

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CRRT FOR AKI

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Learning objectives:

- Identify clinical scenarios in which extracorporeal therapies should be considered for animals with acute kidney injury and understand how these therapies extend the window of opportunity for renal recovery.
- Compare continuous renal replacement therapy and intermittent hemodialysis with respect to indications, practical applications, advantages, and limitations in veterinary patients.
- Apply principles of solute kinetics to develop safe and effective CRRT prescriptions, including stepwise dose escalation strategies to achieve controlled reduction of azotemia while minimizing the risk of dialysis disequilibrium syndrome.
- Recognize key safety considerations associated with extracorporeal therapies, including hemodynamic stability, treatment efficiency, and anticoagulation strategies, and integrate these considerations into individualized treatment planning.

Proceeding:

Acute kidney injury (AKI) is common in dogs and cats and is associated with high morbidity and mortality. Patient outcome depends on multiple factors, including etiology as an important determinant of reversibility, severity of injury as the main determinant of the window of opportunity for recovery, concurrent comorbidities, secondary complications, and availability of advanced therapeutic options. The goals of medical therapy are to control clinical signs and clinicopathological abnormalities, maintain adequate hydration and volemic status, correct metabolic derangements such as metabolic acidosis and electrolyte abnormalities, manage complications (e.g., hypertension, bleeding), address damage to affected body organs (e.g., gastroenteritis, pancreatitis, CNS abnormalities) and to provide sufficient time for renal recovery. When medical management alone is expected to be inadequate, extracorporeal therapies should be considered.

Extracorporeal therapies can extend the window of opportunity for recovery, potentially for an unlimited duration, provided that complications do not occur and financial constraints are not limiting. Extracorporeal therapies can be delivered using a variety of platforms. Platforms designed to deliver continuous renal replacement therapy (CRRT) are increasingly utilized in veterinary medicine, creating a need for structured guidance regarding its indications, prescription, and potential complications. CRRT offers particular advantages in animals that are hemodynamically unstable or severely uremic, where gradual solute and fluid removal is required and the treatment can be delivered bedside. In human medicine, CRRT is commonly delivered continuously over several days at a constant clearance rate. In veterinary medicine, however, prolonged treatments are often impractical due to intensive staffing requirements and high costs. As a result, a modified approach is frequently adopted, in which CRRT is delivered over a shorter duration (e.g., 12 to 24 hours), with the goal of gradually restoring homeostasis. Patients that do not recover following the initial treatment

can be transitioned to intermittent hemodialysis therapy (IHD), which is ideally delivered using a designated an IHD platform, but can also be performed effectively, particularly in cats and small dogs, using CRRT platforms.

Most extracorporeal platforms used in veterinary medicine were originally designed for human patients and are therefore capable of delivering very high solute clearance. While this allows for effective solute removal, it also introduces important safety considerations. One of the major risks associated with hemodialysis is dialysis disequilibrium syndrome, which results from rapid solute removal from the intravascular compartment. This resultant osmotic gradient created between body compartments promotes fluid shifting from the intravascular space into the interstitial and intracellular compartments. Expansion of the intracellular compartment is particularly critical within the central nervous system. Consequently, cerebral edema, increased intracranial pressure, and a spectrum of neurologic manifestations may develop, collectively referred to as dialysis disequilibrium syndrome. To minimize the risk of this potentially fatal complication, solute removal should be reduced gradually through appropriate prescription strategies. However, prolonged (and low efficiency) treatments increase costs and logistical burden in veterinary practice, and premature termination may compromise efficacy. Therefore, an optimal balance between treatment efficiency and patient safety must be carefully achieved. To achieve safe and effective solute reduction within this timeframe, a stepwise prescription strategy can be applied. This lecture introduces a modified approach to CRRT prescription aimed at achieving a constant urea reduction ratio and normalization of azotemia of any magnitude within 24 hours while maintaining patient safety. This strategy is based on the fact that solute removal follows first order kinetics, namely solute removal rate is proportional to solute concentration. Therefore, during the early phases of treatment, a given concentration of urea clearance results in a higher solute removal rate than during the later phases of treatment. Thus, to maintain a relatively constant urea reduction ratio, as urea concentrations decrease over time, the prescribed treatment dose must be progressively increased throughout the course of therapy in proportion to the declining urea concentration. Gradual escalation of clearance in proportion to decreasing uremic toxin concentrations allows for stable and safe solute reduction and minimizes abrupt physiological shifts.

The goals of this lecture are to provide practical guidance on indications for dialytic intervention in AKI, to offer tools for efficient and safe CRRT prescription tailored to veterinary patients, and to introduce key considerations regarding anticoagulation strategies during extracorporeal therapy.

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DYSNATREMIA

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Learning objectives:

- Recognize the importance of evaluating volume status when assessing patients with dysnatremia
- Know the common causes of severe hyponatremia and severe hypernatremia
- Use history, physical examination and clinical diagnostics to identify the likely cause of dysnatremia
- Understand the key aspects of treatment of dysnatremia

Proceeding:

Treatment of dysnatremia is complex and a complete review of the topic cannot be provided in this short presentation. The aim of this discussion is to highlight the key concepts in clinical diagnosis and development of treatment plans.

Clinical Consequences

Acute severe changes in plasma sodium concentration can have immediate neurologic consequences related to either neuronal cell swelling or cell shrinkage. If changes in plasma sodium concentration occur slowly enough, cell volume will be protected by compensatory changes in intracellular osmolality that will return cell volume to normal in the face of the abnormal plasma sodium concentration. Osmotic adaptation of brain cells takes time and is generally considered to take 48 hours to have made substantial changes and several days to be complete. Osmotic adaptation is the reason that patients can present with severe dysnatremia with little to no evidence of neurological abnormalities. Once osmotic adaptation to severe dysnatremia has occurred, rapid alterations in plasma [Na] can have neurological consequences. Overly rapid reductions [Na] in a patient with chronic hypernatremia could lead to cerebral edema while overly rapid resolution of severe, chronic hyponatremia can lead to myelin degeneration (osmotic demyelination syndrome) and the subsequent neurological abnormalities may take 3 to 5 days to be apparent.

Causes of Dysnatremia

Plasma sodium concentration reflects the relative quantities of sodium and water present. In the clinical setting, hyponatremia almost always is due to a gain of electrolyte-free water. Hypernatremia can be due to a gain in sodium or a loss of water.

Key Concepts in Treatment of Dysnatremia

When developing a treatment plan for dysnatremia cases, four key questions should be considered.

Severe vs not severe: As a rule of thumb, severe dysnatremia is a sodium concentration of ³ 15 mmol/L out of the reference interval. Generally, strict guidelines for treatment of dysnatremia are only necessary for severe abnormalities.

Acute vs chronic: Dysnatremia of < 48 hours duration is considered acute. If the duration of dysnatremia is > 48 hours or unknown, it should be considered as chronic for treatment purposes.

Symptomatic vs asymptomatic: If patients have neurologic abnormalities at the time of presentation with severe dysnatremia, they should be considered symptomatic. Neurologic signs reported include tremors, ataxia, seizures, obtundation and stupor

What is the underlying mechanism of the dysnatremia? This will help direct the treatment approach to resolve the dysnatremia.

Assessment of the volume status of patients with dysnatremia will help determine the underlying disease process responsible and will help guide therapy. Patients are evaluated as hypovolemic, euvolemic or hypervolemic. Several treatment guidelines for severe dysnatremia have been developed in human medicine and they are not consistent in their recommendations. More recent studies have suggested outcome may be better with faster than slower correction. The optimal approach has not been determined but suggestions for an approach to treatment will be provided in this discussion. Human studies suggest it is the rate of change of plasma [Na] over 24 hours, not the rate of change per hour that is associated with neurological complications. This means if the desired 24 hour rate of change is achieved in a shorter period of time, it is acceptable, if no further change of plasma [Na] occurs for the rest of that 24 hour period. Fluid plans for resolution of dysnatremia do not address the maintenance fluid needs and replacement of any deficits. Isotonic fluid (ideally a fluid with a [Na] within 10 mmol/L of the patient) should also be provided as appropriate. Treatment of dysnatremia can include volume administration, hypertonic saline administration, free water administration, loop diuretic therapy, water restriction and glucocorticoid therapy. The treatment of choice depends on the underlying cause and evaluation of urine osmolality and electrolytes can help guide decision making. This case-based presentation will review diagnostic and treatment algorithms to help clinicians determine the most appropriate treatment plan.

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Nurse & Tech Stream, Friday 5 June 2026

HEART DISEASE IN CATS AND DOGS: UNDERSTANDING THE CONDITIONS WE SEE MOST

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Learning objectives:

- Normal cardiac structure and function
- Compensatory mechanisms to cardiac dysfunction
- Overview of common acquired heart diseases in dogs and cats
- Common emergency presentations
- Treatment goals

Proceeding:

Anatomy and Physiology Overview

The heart is a muscle, located within the mediastinum that is responsible for pumping blood around the body, delivering oxygen and nutrients to the cells and tissues.

Arterial blood pressure (ABP) is essential for circulating blood around the body and maintaining adequate perfusion of the body's tissues and organs. ABP is directly determined by cardiac output and systemic vascular resistance. Cardiac output is directly determined by heart rate and stroke volume, while systemic vascular resistance is determined by vascular tone, blood viscosity, and vessel diameter.

Compensatory Mechanisms

A drop in ABP or its determinants activates two major compensatory mechanisms

The sympathetic nervous system

Renin-Angiotensinogen-Aldosterone System (RAAS)

Activation of these compensatory mechanisms will help to maintain ABP, direct blood flow to major organs (heart and brain), and increase effective circulating volume.

Heart Disease

Heart disease occurs when there are structural or functional abnormalities that affect the heart, valves or blood vessels, often leading to reduced blood flow, compromised oxygen delivery, and impaired function. Heart disease may be congenital (present at birth) or acquired (develop after birth). Heart failure occurs when the heart is unable to pump enough blood to meet the body's metabolic demands.

Heart murmurs

Heart murmurs are caused by abnormal, turbulent blood flow within the heart, which can create vibrations of cardiac structures (valves, walls, or septum) that produce a whooshing noise when auscultated.

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is one of the most prevalent cardiomyopathies and is a primary disease of the heart muscle. It results in gradual dilation of the ventricles and thinning and weakening of the ventricular walls, reducing the ventricles' ability to generate enough pressure to pump blood effectively. DCM most commonly affects large and giant breed dogs, including Dobermanns, Boxers, and Great Danes. Causes can include hereditary factors, nutritional deficiencies, infectious agents, and metabolic derangements.

Clinical signs occur secondary to impaired oxygen delivery (lethargy, weakness, weight loss, collapse), and/or pulmonary congestion (coughing, increased respiratory rate or effort). These patients often present as emergencies in respiratory distress when DCM progresses to heart failure and require immediate stabilisation with oxygen therapy, sedation, and medications. Diagnostics may include echocardiography, radiographs, electrocardiography, and blood tests. Emergency treatment is aimed at improving systolic function, dilating peripheral vessels, eliminating pulmonary congestion, and controlling heart rate and rhythm.

Hypertrophic cardiomyopathy

Hypertrophic cardiomyopathy (HCM) is the most common cardiomyopathy in cats and is characterised by thickening of the left ventricular walls. This thickening reduces the ventricles' ability to relax during diastole, impairing ventricular filling, decreasing stroke volume, increasing atrial pressure, and leading to enlargement of the left atrium. HCM can be hereditary, particularly in certain breeds such as Maine Coons and Ragdolls, and also occur secondary to conditions, such as hyperthyroidism or systemic hypertension.

Clinical signs are most commonly related to the development of pulmonary oedema and/or pulmonary effusion, secondary to congestive heart failure and include lethargy, weakness, dyspnea and coughing. A potentially life-threatening sequelae of HCM is the formation of blood clots in the left heart, which can enter systemic circulation and obstruct blood flow in other areas of the body, often causing sudden hind limb paresis. Many HCM patients present as emergencies in respiratory distress or with thromboembolic complications, requiring prompt stabilisation with oxygen, sedation, and medications. Diagnostics typically include echocardiography, radiographs, electrocardiography, and blood tests, including thyroid function. Although there is no current cure for HCM, treatment focuses on managing heart rate, alleviating pulmonary congestion, and preventing the formation of blood clots.

Myxomatous mitral valve disease

Myxomatous mitral valve disease (MMVD) is the most common acquired heart disease in dogs and is characterised by chronic degeneration of the mitral valve, preventing complete closure of the two mitral valve leaflets.

This allows blood to flow backward, and 'regurgitate' into the left atrium during systole. Over time, the left atrium and ventricle start to dilate due to the longstanding leak across the mitral valve into

the left atrium. As the mitral valve continues to degenerate, the leak in the mitral valve worsens, and congestive heart failure can develop.

MMVD most commonly affects older, small breed dogs, including Cavalier King Charles Spaniels, Chihuahuas, and Miniature Poodles, and is influenced by hereditary factors and age-related degenerative changes.

Clinical signs of MMVD include the presence of a new heart murmur, cough, increased resting respiratory rate, laboured breathing, exercise intolerance, lethargy and weakness. Many patients present as emergencies in respiratory distress when MMVD progresses to left-sided heart failure, requiring immediate stabilisation with oxygen therapy, sedation, and medications to relieve the pulmonary congestion.

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ARTERIAL MOTIVES: SAMPLING AND ANALYSIS

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Learning objectives:

- Refresh the venous and arterial circulatory system pathophysiology
- Become confident with arterial sampling indications/contraindications
- Become confident safely obtaining arterial blood samples
- Understand what is analysed from arterial samples
- Be confident calculating oxygen indices and understanding results

Proceeding:

The blood vasculature is a closed circulatory system made up of arteries, veins and capillaries. During contraction of the heart, nutrient and oxygen rich blood is pumped via the arterial system to tissues. The venous system circulates low oxygen and low nutrient blood from the tissues back to the heart. This varies only in the pulmonary vasculature. Here, the pulmonary artery carries the low oxygen blood from the heart to the lungs for gas exchange and the pulmonary vein carries high oxygen blood back to the heart to enter the arterial system. Each vessel plays a crucial and distinct role. They are lined with endothelial cells and surrounded by smooth muscle cells and elastic fibres of varying thickness to allow expansion and recoil. Arterial walls have thicker smooth muscle cell layers and smaller lumens which makes them more difficult to penetrate. This means when trying to enter these vessels with a needle, there is a higher risk of spasming, haemorrhage and pain. Arterial blood is used to assess arterial oxygen content (CaO2) which is relied upon for delivery of oxygen to tissues. CaO2 depends on haemoglobin concentration and binding affinity, the degree of oxygen saturation and a small fraction dissolved in plasma. The following can be measured:

Haemoglobin (Hgb/Hb)

Percentage of oxygen-carrying haemoglobin (SpO2) [normal >95%]

Arterial partial pressure of oxygen (PaO2) [normal = 80-105mmHg]

Think of SpO2 as the oxygen tank (reservoir) and PaO2 as the regulator (driving force). Venous blood will reflect tissue function, whereas arterial blood reflects pulmonary function. Arterial carbon dioxide (PaCO2) can also be interpreted which further strengthens assessment of lung function and gas exchange.

Indications and contraindications

SpO2 is used to assess oxygenation as a non-invasive tool but has limitations and barriers to accuracy. SpO2 and PaO2 are directionally but not linearly related which is represented in the oxygen

dissociation curve. If we need to assess lung function and gas exchange in our patients, PaO2 is therefore the gold standard. Indications for arterial sampling include assessment of pulmonary function, assessment of oxygen carried to tissues, quantifying or replacement for questionable pulse oximetry, indications for or response to oxygen supplementation, prognostic indicators or identification and staging of acute respiratory distress syndrome (ARDS). Arterial sampling is contraindicated with coagulopathy, thrombocytopaenia, if the patient is conscious and intolerable or due to lack of skill or equipment.

Sampling technique and analysis

Common sites for sampling include the dorsal metatarsal, femoral, sublingual, coccygeal, radial, brachial and auricular arteries. Local anaesthetic may be considered. Assistant should help restrain and apply pressure post sample but should not 'raise' like with a vein, this will occlude the artery. If repeat samples are required and no contraindications, an arterial catheter should be considered. If a room air sample is required, ensure oxygen supplementation is withheld for 5-10 minutes prior. Ensure minimal exposure to atmospheric air for accuracy.

Step 1:

Prepare necessary equipment and analyser for immediate use. Have patient in the appropriate position depending on choice of artery

Step 2:

Palpate the artery of choice to assess location, clip an appropriate window and perform a surgical clean of area

Step 3:

Wash hands thoroughly +/- gloves. Palpate the artery with the first two fingers of the non-dominant hand

Step 4:

Drawback plunger on syringe with needle attached to the volume required. Insert needle at 45-60° angle with dominant hand while simultaneously palpating artery with non-dominant hand

Step 5:

Arterial puncture should result in filling of the syringe with arterial blood; further aspiration may be required depending on syringe used

Step 6:

Once the sample is obtained, the assistant is to apply pressure for 5 minutes and a pressure bandage to the area for at least 15 minutes to avoid haematoma formation

Step 7:

Run sample immediately with minimal exposure to atmospheric air for accurate results.

Interpretation

A PaO2 of <80mmHg on room air indicates mild hypoxaemia and a PaO2 of <60mmHg on room air indicates severe hypoxaemia. To interpret results, there are a variety of oxygen indices that can be utilised depending on what is being assessed and what fraction of inspired oxygen (FiO2) the patient is breathing. Each of these come with specific indications and limitations.

5x Rule (FiO2 x 5 = PaO2) [normal on room air = 80-105mmHg]

120 Rule (PaCo2 + PaO2 = 120)

P/F Ratio (PaO2/FiO2 (as a decimal [40% FiO2 = 0.4])) [normal is 350-500]

A-a gradient (room air) ([150-PaCO2/0.8] – PaO2) [normal = 0-15mmHg]

Which oxygen index to use will depend on the primary purpose of sampling. If using for diagnosis by a veterinarian, it is important to consider which one is appropriate on room air or supplemental oxygen. The A-a gradient and 120 rule are suggested for use if on room air due to the inclusion of PaCO2 in their formulas, with the A-a gradient being superior. Performing these on supplemental oxygen therapy can lead to misdiagnosis, as results may not correlate. The P/F ratio is suggested for use on supplemental oxygen due to its exclusion of PaCO2 in the formula, which may lead to misleading diagnosis if PaCO2 is outside normal range. In staging of ARDS, the veterinary consensus guidelines advise the inclusion of P/F ratios into the diagnostic criteria and suggested to be able to use this oxygen index on room air if PaCO2 is within normal limits. The 5-x rule can be used in any scenario as a general overview, quick and easy calculation. Any abnormal results should be quickly reported to the veterinarian in charge of the patient and oxygen therapy provided to patient if indicated by results.

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THE POWER OF DEBRIEFING: BUILDING STRONGER TEAMS & BETTER PRACTICES

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Learning objectives:

- Initiate advanced life support interventions in a dogs or cat in cardiopulmonary arrest, including monitoring, vascular access, and administration of reversal agents
- Diagnose a non-shockable arrest rhythm and devise a plan for vasopressor, parasympatholytic, and buffer therapy, including drug, dose and frequency
- Diagnose a shockable arrest rhythm and devise a plan for defibrillation and drug therapy, including dose and frequency

Proceeding:

Team debriefing in veterinary emergency and critical care

Emergency and critical care environments demand rapid decision-making, technical precision, and coordinated teamwork. These high-pressure situations are inherently complex and prone to communication breakdowns or performance variability. Team debriefing has emerged as a critical tool to support reflection, reinforce effective behaviors, and identify opportunities for improvement following cardiopulmonary resuscitation (CPR) events.

Conducting CPR team debriefs

Debriefing typically occurs shortly after a resuscitation attempt, while details remain fresh. The primary objectives are to review the sequence of events, assess CPR quality, examine team communication, and identify actionable improvements for future cases. Sessions are facilitated by a trained leader or senior team member who establishes a non-judgmental, psychologically safe environment.

Most debriefings follow a structured format:

Event review: The team reconstructs the timeline of the arrest and resuscitation, highlighting key decisions such as CPR initiation, medication administration, and role assignments.

CPR quality assessment: Technical performance is reviewed, including compression depth and rate, ventilation, and adherence to resuscitation guidelines. When available, objective data from CPR feedback devices or defibrillators are incorporated.

Team dynamics: Communication, leadership, and coordination are discussed, with attention to role clarity, information flow, and handoffs.

Reflection and learning: Participants identify what went well and what could be improved. The facilitator ensures balanced participation and focuses discussion on learning rather than blame.

Summary and action plan: Key takeaways are consolidated, and specific next steps are identified, such as targeted training, protocol refinement, or follow-up review.

Variations in debriefing approaches

Debriefing models vary based on time, staffing, and institutional resources. Group debriefings promote shared learning and team cohesion, while individual verbal debriefs provide focused, immediate feedback. Written debriefings, though less interactive, ensure consistent messaging across large or rotating teams. Evidence suggests all formats can be effective, particularly when applied consistently and intentionally.

Impact on CPR quality and patient outcomes

The quality of CPR is a major determinant of resuscitation success. Multiple studies demonstrate that structured debriefing improves CPR performance metrics, including compression depth and rate. Improvements in these parameters are associated with increased return of spontaneous circulation (ROSC) and survival to discharge.

Regular debriefing has been shown to improve adherence to recommended compression depths (50–60 mm) and maintain appropriate rates, even in settings with high baseline performance. Importantly, debriefing continues to yield incremental gains in experienced teams, underscoring its role in performance refinement rather than remediation alone.

Improving communication and teamwork

Effective communication is central to successful resuscitation, particularly in multidisciplinary teams operating under stress. Debriefing creates space for open dialogue, allowing team members to clarify perspectives, address misunderstandings, and strengthen shared mental models.

Teams that routinely debrief demonstrate improved coordination and reduced errors during subsequent emergencies. Even brief, individual verbal debriefings have been shown to positively influence communication and team dynamics, suggesting that meaningful improvement does not require lengthy or formal sessions.

Supporting continuous learning and improvement

Debriefing reinforces a culture of continuous learning by embedding reflection into routine practice. Teams that regularly analyze performance develop stronger clinical reasoning, improved decision-making, and greater adaptability over time. The reflective process encourages teams to evaluate not only outcomes, but also processes, fostering durable improvements in practice.

Evidence indicates that the greatest gains occur when debriefing is integrated into routine workflow rather than treated as an optional add-on. Regularity reinforces learning and normalizes feedback as part of professional growth.

Challenges to implementation

Despite clear benefits, implementation barriers remain. Time constraints, variable team composition, and organizational culture can limit consistency. However, studies demonstrate that even short, focused debriefings can yield meaningful improvements when time is limited.

Tailoring debriefing to team experience and context improves relevance and engagement. Organizational culture also plays a critical role: environments that prioritize psychological safety, provide facilitator training, and explicitly support debriefing are more likely to see sustained benefits.

Recommendations for optimizing debriefing

Evidence supports several strategies to strengthen debriefing programs:

Standardize frameworks to ensure consistency in timing, structure, and content.

Train facilitators to guide discussion effectively and maintain a learning-focused tone.

Integrate debriefing into routine practice to reduce time barriers.

Tailor sessions to team composition and event complexity.

Promote a learning culture that values reflection and non-punitive improvement.

Veterinary-specific evidence

Veterinary literature on debriefing remains limited but growing. One study evaluating a postarrest debriefing tool in a veterinary university hospital reported that debriefing improved perceived team performance in 66% of participants. Equipment issues were frequently identified, and closed-loop communication was underutilized. While some staff reported hesitancy to speak openly, the tool was effective in identifying strengths and training needs, highlighting both the promise and areas for refinement in veterinary settings.

Summary: five key take-home points

Structured reflection improves performance: Post-CPR debriefing enhances CPR quality metrics that directly influence patient outcomes.

Communication and teamwork strengthen: Debriefing improves coordination and reduces errors in future emergencies.

Learning becomes routine: Regular debriefing embeds reflection and professional growth into daily practice.

Barriers exist but are manageable: Even brief debriefs can be effective despite time and staffing constraints.

Support and standardization matter: Clear frameworks, trained facilitators, and organizational commitment are essential for success.

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RECOVER CPR GUIDELINES: NEWBORN RESUSCITATION

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Learning objectives:

- Describe the key physiologic changes that occur during the immediate transition from intrauterine to extrauterine life in newborn puppies and kittens.
- Recognize which newborn animals require resuscitation and apply appropriate initial stabilization steps within the first minutes after birth.
- Implement the RECOVER Newborn Resuscitation algorithm, including airway management, ventilation, chest compressions, and appropriate drug use.

Proceeding:

The RECOVER Initiative's Newborn Resuscitation Guidelines address the management of dogs and cats during the first few hours of life, termed the "newborn" period, when animals transition from intrauterine to extrauterine physiology. This transition involves rapid cardiopulmonary and metabolic changes, making the immediate postnatal period particularly vulnerable. Perinatal mortality remains high in small animals: approximately 6% of naturally born puppies, 11% of puppies born following dystocia, and 8–12% of puppies delivered by Cesarean section are stillborn. Additionally, up to 13% of puppies born by C-section that initially survive die within the first two hours of life. These statistics highlight the importance of prompt recognition and management of compromised newborns. The RECOVER Initiative defines terminology to clarify developmental stages. The newborn period refers to the first few hours after birth when physiological transition occurs. The neonatal period extends from birth until the start of weaning (typically 4–5 weeks), while pediatric animals are those from weaning until sexual maturity. Importantly, the RECOVER Newborn Guidelines apply specifically to the immediate postnatal period rather than the entire neonatal stage.

Unlike adult cardiopulmonary resuscitation (CPR), newborn resuscitation focuses primarily on identifying animals that do not require intervention. Resuscitation may be withheld only if all three of the following criteria are met: the animal was delivered by normal vaginal parturition (eutocia), the dam can provide immediate postpartum care, and the newborn is vigorous, defined as breathing ≥15 breaths per minute, vocalizing, and demonstrating strong reflex irritability and spontaneous movement. Newborns that do not meet all criteria, including all puppies and kittens delivered via Cesarean section, should receive immediate supportive interventions. The first 1–2 minutes after birth are critical for establishing effective oxygenation and ventilation. Initial resuscitation measures should be performed simultaneously and include clearing the airway, drying and warming the newborn, providing tactile stimulation, and initiating ventilation if needed. Fetal membranes should be removed immediately, and visible fluid around the nose and mouth should be gently cleared. Routine suctioning is not recommended, but brief suctioning may be performed if excessive fluid or meconium is present. Historical practices such as "swinging" newborns to remove fluid should be avoided due to the risk of trauma and delayed ventilation.

Maintaining body temperature is essential during resuscitation. Newborns should be dried with warm towels and kept in an environment that maintains a rectal temperature between 35–37°C (95–99°F). Tactile stimulation during drying may also promote respiratory effort. Ventilation is the most important intervention during newborn resuscitation. Positive pressure ventilation (PPV) should be initiated immediately in apneic or gasping newborns using a tight-fitting mask at 20–30 breaths per minute. Chest rise and an increasing heart rate indicate effective ventilation. In contrast to adult CPR, bradycardia in newborns is usually caused by hypoxemia rather than primary cardiac disease; therefore, ventilation should be prioritized over drug therapy. Routine administration of 100% oxygen is not recommended initially. PPV should begin with room air, with supplemental oxygen added if the heart rate fails to improve. If heart rate remains below 120 beats per minute despite effective ventilation, further interventions may be required, including endotracheal intubation. Chest compressions should be initiated if the heart rate remains below 50 beats per minute after at least 30 seconds of effective ventilation with oxygen. Compressions are performed using one or two fingers over the cardiac area, compressing the chest to one-third to one-half its width. A compression-to-ventilation ratio of 4:1 is recommended, targeting approximately 120 compressions and 30 breaths per minute. Pharmacologic interventions are rarely required during early resuscitation. However, epinephrine may be administered if the heart rate does not improve after coordinated ventilation and chest compressions. Reversal agents such as naloxone, atipamezole, or flumazenil may be indicated if the dam received opioids, α2-agonists, or benzodiazepines prior to delivery.

The RECOVER Newborn Resuscitation Guidelines represent the first comprehensive evidence and consensus-based recommendations for neonatal resuscitation in veterinary medicine. Developed through a systematic literature review and expert consensus addressing 28 PICO questions, the guidelines provide practical recommendations summarized in a clinical algorithm designed for both pre-delivery preparation and real-time decision support. Implementation of these guidelines aims to standardize neonatal resuscitation practices and improve survival outcomes in newborn puppies and kittens.

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TAKE A BREATH- UNDERSTANDING COMMON RESPIRATORY DISEASES IN CATS AND DOGS

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Learning objectives:

- Normal respiratory physiology
- Respiratory disease
- Distinguishing abnormal breathing patterns
- Emergency presentations

Proceeding:

The respiratory system consists of the upper airways (nose, mouth, pharynx, larynx) and lower airways (trachea, bronchi, bronchioles, and alveoli), with the distal trachea, bronchi, and lungs housed within the rib-protected thoracic cavity. In addition to facilitating gas exchange, the respiratory system warms, humidifies, and filters inspired air.

Ventilation occurs through negative pressure generated by coordinated contraction of the diaphragm and intercostal muscles, drawing air through the airways into the lungs, where gas exchange occurs. Effective oxygen diffusion across the alveolar–capillary membrane requires adequate ventilation, functional alveoli, appropriate pulmonary perfusion, and normal respiratory control. Disruption of any component of this system can result in respiratory compromise.

Upper Airway Disease

The upper airways facilitate olfaction and thermoregulation and protect the lower airways from aspiration of food and water. Upper airway obstruction is a common cause of respiratory distress and requires rapid recognition and intervention.

A typical presentation is a young brachycephalic dog with acute respiratory distress following mild exertion, characterised by exaggerated inspiratory effort, stertor, and stridor. Upper airway disease increases airway resistance, significantly increasing the work of breathing. Conditions such as brachycephalic obstructive airway syndrome (BOAS), laryngeal paralysis, tracheal collapse, and upper airway masses primarily obstruct airflow during inspiration. Complications include hyperthermia, aspiration pneumonia, and noncardiogenic pulmonary oedema.

Regardless of the underlying cause, these patients are extremely unstable, and handling them can result in rapid decompensation. Prompt management includes oxygen supplementation and anxiolysis and/or sedation, with endotracheal intubation or tracheostomy required in severe cases.

Lower Airway Disease

The most common lower airway disorders in dogs and cats include structural diseases (tracheal or bronchial collapse and bronchiectasis), airway infections, and inflammatory airway diseases (feline asthma and chronic bronchitis).

Airway inflammation causes mucosal oedema, airway smooth muscle hypertrophy and constriction, and excessive production of airway secretions.

Consider a middle-aged cat presenting with episodic dyspnoea and coughing. In severe cases, these cats will have expiratory distress; however, this can be difficult to detect due to tachypnoea causing short inspiratory and expiratory times. These cats may have crackles or wheezes due to fluid or mucus accumulation within the airways.

Airway inflammation is treated with corticosteroids and bronchodilators are indicated when there are signs of bronchoconstriction.

Pulmonary Parenchymal Disease

Pulmonary parenchymal disease affects the terminal and respiratory bronchioles, interstitium, alveoli, and pulmonary vasculature. Conditions within this category include pneumonia, pulmonary oedema, interstitial lung disease, pulmonary neoplasia, and traumatic pulmonary parenchymal injury.

These disorders reduce functional lung capacity and impair gas exchange by decreasing functional alveolar surface area and/or increasing diffusion distance. This commonly results in hypoxaemia due to ventilation–perfusion (V/Q) mismatch, intrapulmonary shunting, and hypoventilation.

Clinical signs may include coughing, increased respiratory rate with variable effort, exercise intolerance, respiratory distress, and pulmonary crackles.

For example, a dog may present with acute onset tachypnoea and hypoxaemia following aspiration during anaesthetic recovery.

The primary aims of therapy are to maintain airway patency and provide oxygen supplementation, treat any underlying infection, and address predisposing conditions.

Pleural Space Disease

Pleural space disease describes abnormalities and accumulations within the pleural space, including pleural effusions, pneumothorax, or space occupying structures, such as diaphragmatic hernias, or masses, that impair lung expansion during inhalation.

Clinical signs include, rapid shallow breathing, paradoxical breathing pattern (chest falls on inspiration and the abdomen expands), open-mouth breathing, orthopnea, cyanosis and decreased lung sounds.

Consider a cat presenting open-mouth breathing and a rapid, shallow breathing pattern following trauma. Accumulation of air or fluid within the pleural space restricts expansion of the lungs, causing decreased tidal volume, total vital capacity, and functional residual capacity, as well as hypoxemia and hypoventilation. Other treatments are dependent on the underlying disease.

Chest Wall Abnormalities

The chest wall anatomy is essential for functional respiration. Diseases that can affect the chest wall include congenital disorders, neoplasia, and trauma (rib fractures, flail chest, penetrating wounds).

Pain and concurrent injuries, such as pulmonary contusions and pneumothorax, can impair effective ventilation, leading to hypoventilation and hypoxaemia.

Spinal cord and neuromuscular Disease

Any disease that impairs the movement and function of the diaphragm or inspiratory muscles has the potential to cause hypoventilation, hypoxaemia and hypercapnia, despite normal lung parenchyma and airways. Respiratory failure may manifest rapidly and require ventilatory support.

Causes include; disease or injury of the cervical neurons that facilitate breathing Toxins (snake envenomation, tick paralysis, botulism) Autoimmune (Myasthenia gravis)

Respiratory Look-alikes

Increases in respiratory rate and effort can be caused by conditions that are unrelated to the respiratory system, such as pain, acidosis, impaired oxygen delivery (anaemia, shock, hypotension, or dyshaemoglobins), opioid administration, or brain dysfunction (elevated intracranial pressure, neoplasia, or vascular events). Treatment depends on the underlying cause.

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YOU'RE MY HERO: SMOKE INHALATION

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Learning objectives:

- What damage can smoke inhalation cause to our patients?
- What symptoms will we see in patients presenting with smoke inhalation +/- thermal burns?
- What are some of the immediate treatments these patients will need?
- What are the chances of smoke inhalation and thermal burn patients recovering?

Proceeding:

Introduction

Smoke inhalation injury is a common and potentially life-threatening consequence of residential and enclosed-space fires in veterinary patients. While cutaneous burns and singed fur may be immediately apparent, inhalation injury is frequently less obvious on initial examination and may progress over time. Veterinary technicians play a critical role in early recognition, monitoring, and supportive care of these patients to reduce morbidity and mortality.

Pathophysiology

Smoke inhalation injury may occur with or without concurrent thermal burns and is associated with exposure to heat, particulate matter, and toxic gases. Patients exposed to fires in enclosed spaces are at increased risk due to higher concentrations of combustion byproducts. Burning synthetic materials such as plastics, nylon, rubber, wool, and acrylic increases the likelihood of inhaling toxic chemicals.

Common clinical consequences of smoke inhalation include upper airway obstruction, bronchospasm, small airway occlusion, pulmonary infection, and respiratory failure. Direct thermal injury to the upper airway, deposition of soot, and inhalation of toxic gases contribute to mucosal damage and inflammation. This inflammatory response may progress to acute respiratory distress syndrome (ARDS).

Inhalation of toxic combustion products may cause chemical tracheobronchitis and severe pulmonary injury. Soot adheres to airway surfaces, prolonging local inflammation and tissue damage. Secondary mucosal sloughing may occur 12-72 hours after exposure, resulting in airway obstruction.

Carbon Monoxide and Cyanide Toxicity

Carbon monoxide (CO) exposure is common in fire victims and results in the formation of carboxyhemoglobin (HbCO), which shifts the oxyhemoglobin dissociation curve to the left and impairs oxygen delivery to tissues. This leads to cellular hypoxia, reduced myocardial function, central nervous system injury, and increased risk of reperfusion injury.

Clinical signs of CO or cyanide toxicity include altered mentation, ataxia, collapse, syncope, hypotension, and coma. Cyanide toxicity causes severe cellular hypoxia by inhibiting oxidative phosphorylation. Delayed neurologic dysfunction may occur up to 4-5 days following exposure. Methemoglobinemia may also develop following inhalation of certain combustion products.

Untreated CO or cyanide toxicity can rapidly result in death due to hypoxia.

Clinical Presentation and Progression

Clinical signs of smoke inhalation may be immediate or delayed, with severity often peaking within 24 hours of exposure. Progression of injury can continue over 24-36 hours, necessitating close monitoring.

Patients may present in shock due to hypoxia, hypotension, or cardiovascular collapse. Common respiratory signs include tachypnea, dyspnea, wheezing, stridor, cyanosis, open-mouth breathing, orthopnea, and abnormal lung sounds. Cherry-red mucous membranes, while classically described with CO poisoning, are rarely observed.

Patients presenting without respiratory distress may be observed for 6-8 hours and discharged if stable, with re-evaluation recommended at 72 hours. Patients with respiratory compromise should be monitored for at least 24-48 hours.

Prognosis

Prognosis is influenced by the severity of inhalation injury, presence of thermal burns, age, and development of complications such as pneumonia. Mortality rates are significantly higher in patients with concurrent burns. Dogs and cats with smoke inhalation alone have reported survival rates approaching 90%. Secondary bacterial pneumonia is a common late complication and carries a mortality rate of up to 50%.

Diagnostics

Initial assessment should include a thorough physical examination, with particular attention to singed whiskers, facial burns, soot in the nares, oral cavity, and pharynx, and evidence of airway edema.

Diagnostic testing may include pulse oximetry, arterial blood gas analysis, thoracic radiographs, and lactate measurement. Hypoxemia is indicated by $PaO_2 < 60$ mmHg, while impaired ventilation is reflected by $PaCO_2 > 60$ mmHg. Metabolic acidosis due to hyperlactatemia or respiratory acidosis from hypoventilation may be present.

Pulse oximetry may appear falsely normal in patients with CO poisoning, as standard devices cannot differentiate oxyhemoglobin from carboxyhemoglobin. Co-oximetry provides more accurate assessment when available. Thoracic radiographs may initially appear normal but can worsen within 24-72 hours.

Initial Stabilization and Treatment

Immediate administration of high-flow, humidified 100% oxygen is the most critical intervention. Oxygen therapy reduces the half-life of HbCO and improves tissue oxygen delivery. In facilities

without the ability to measure HbCO, CO exposure should be assumed, and oxygen therapy provided for a minimum of four hours, with continuation as indicated.

IV access should be established promptly, allowing for blood sampling and medication administration. If full laboratory evaluation is not feasible, priority should be given to blood gas analysis, electrolytes, and lactate.

Analgesia should be initiated early in patients with painful injuries. Opioids such as methadone, hydromorphone, fentanyl, or morphine are preferred and may be administered intermittently or as a constant rate infusion. Cough suppressants should be avoided, as airway clearance is essential. NSAIDs should be withheld until the patient is stable and renal function is confirmed.

IV fluids are typically administered at maintenance rates. In patients with hypovolemic shock, low-volume resuscitation with hypertonic saline may be considered to minimize pulmonary edema and airway secretions.

Supportive and Ongoing Care

Ocular injuries should be addressed promptly. Eyes should be flushed with sterile saline, and fluorescein staining performed to evaluate for corneal ulceration.

Respiratory rate and effort should be monitored hourly. Oxygen supplementation via nasal cannula or oxygen cage may be sufficient for mild to moderate injury. Mechanical ventilation is indicated if patients cannot maintain adequate oxygenation or ventilation or become fatigued. Ventilatory support may be required for 2-4 days.

Bronchoscopy may be indicated to assess for airway necrosis and mucosal sloughing 12-72 hours post-injury. Removal of debris via suction may significantly improve respiratory function and may be required over multiple days.

Electrolytes should be monitored closely. Hyperkalemia may occur initially due to cellular injury, followed by urinary potassium losses after 48 hours. Hyponatremia may develop due to antidiuretic hormone release and may require adjustment of fluid composition.

Additional monitoring parameters include PCV/TS, ECG, arterial blood pressure, and repeat thoracic imaging as indicated. Antibiotic therapy should be reserved for patients with confirmed or suspected secondary bacterial pneumonia.

Conclusion

Smoke inhalation injury is a complex and dynamic condition requiring vigilant monitoring and supportive care. Early oxygen therapy, thorough assessment, and proactive nursing management are essential to improving outcomes. With timely intervention and appropriate monitoring, many dogs and cats recover successfully, particularly when exposure duration is limited and complications are minimized.

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CRITICAL CARE NURSING OF THE VENTILATED PATIENT

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Learning objectives:

- Recognise the decompensating respiratory patient
- Learn how to set up the ventilator and all supplies
- Understand the importance of infection prevention and control in ventilator patients
- Be able to critically nurse anaesthetised patients and provide recumbency and oral care
- Confidently assess oxygenation and oxygen dependence using blood gas analysis

Proceeding:

Introduction

Patients requiring ventilation within the intensive care unit require rapid stabilisation and intervention. It is important to recognise the patient with respiratory deterioration and be prepared to intervene quickly in these cases. Getting a patient on to the ventilator is a team effort which requires team co-ordination, clear communication and preparedness for any complications that could arise. This lecture will discuss how to set up and initiate ventilator care and delve into the ongoing nursing care of the patient with particular emphasis on preventing ventilator associated pneumonia (VAP) and assessing the patient's oxygenation and ventilation status. VAP is the most common and severe complication of ventilation; nursing care should focus on mitigating the risk of this occurrence.

Oxygenation and ventilation

Oxygenation refers to the movement of oxygen from the alveoli into the pulmonary capillaries and is assessed using SpO_2 and PaO_2. Ventilation describes the elimination of carbon dioxide from the pulmonary capillaries into the alveoli and is evaluated using end tidal CO_2 monitoring and blood gas analysis. A clear understanding of these processes is essential when managing mechanically ventilated patients. Different calculations such as the Alveolar-Arterial PO_2 Gradient (A-a gradient) can aid us in assessing oxygenation status and severity of pulmonary disease.

$PAO_2 = 150 - PaCO_2$

The measured PAO_2 from the arterial blood gas is then subtracted from the calculated PAO_2 from the calculation above. Results should be below 10mm Hg, values above 20 mm Hg indicate decreased ability to oxygenate, although it is important to note this calculation is only reliable for room air blood gas analysis. For patients receiving oxygen supplementation at the time of blood gas draw, the PaO2/FiO2 ratio (P/F ratio) is considered more beneficial.

$PaO_2 / FiO_2 = P/F\ ratio$

The normal P/F ratio is around 500, values much lower than this indicate severe pulmonary disease. These equations assess oxygenation without taking CO2 into account, and therefore do not assess ventilation. This is best assessed by measuring the 'CO2 gap'

$PaCO_2$-$EtCO_2$

This should be 2-5 mmHg, and a bigger gap can indicate alveolar dead space where ventilation is not efficient. A large gap would indicate a need to initiate or increase ventilation.

When to ventilate

Mechanical ventilation is indicated in patients with hypoventilation resulting in hypercapnia and hypoxaemia. Causes include sedation overdose, neuromuscular disease, upper airway obstruction such as brachycephalic obstructive airway syndrome, pleural space disease, respiratory fatigue, cervical spinal cord disease, pneumonia, and acute respiratory distress syndrome (ARDS). Blood gas analysis plays a critical role in identifying respiratory acidosis and guiding clinical decision-making.

Setting up the Ventilator

Rapid and thorough preparation is essential when initiating ventilation. The use of checklists, pre-calculated emergency drugs, and clear allocation of team roles improves patient safety during this high-risk period. Intubation, monitoring connection, oxygen supplementation, and induction of unstable patients require close coordination, with emergency drugs and reversal agents readily available.

Nursing care

Nursing care of the ventilated patient is multifaceted and includes strict hand hygiene and barrier nursing, continuous monitoring, maintenance of venous access, recumbency care, airway management, oral and ocular care, nutritional support, urinary management, and accurate record keeping. Ventilator bundles may be used as a means of standardising care and reducing ventilator-associated complications. This intense nursing requires vigilance and attention to detail and patients should be nurses 1:1 to allow this. Scrupulous record keeping is vital in monitoring trends and aiding continuity of care between shifts, as well as clear communication with colleagues.

Conclusion

Ventilation of the veterinary patient requires intense nursing and an organised team. Reducing VAP is one of the biggest challenges in these patients and so strict barrier nursing and vigilant monitoring is essential. The veterinary nurse should be able to recognise the patient in respiratory distress or fatigue and act quickly to ensure rapid stabilisation and initiation of ventilation. Assessing oxygenation and ventilation is a vital skill for the ICU nurse to ensure quick interventions and improved outcomes.

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ACID BASE

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Learning objectives:

- Understand what pH represents and why it is clinically relevant in emergency and critical care patients
- Describe the body's buffer systems and the role of the Henderson Hasselbalch equation in acid–base balance
- Review the principles of strong ion theory and its relevance to blood gas interpretation

Proceeding:

The Basics

In emergency and critical care, patients often present with an acid-base imbalance causing clinical symptoms such as cardiovascular dysfunction, neurological depression, abnormal respiration, as well as vomiting and even seizures depending on the underlying causes of imbalance. When beginning to look at acid base as a concept, it is first important to understand what pH is and how this affects the patient. pH is the conventional way of describing the concentration of hydrogen ions, based on a logarithmic scale. As it is a logarithmic scale, a small change in pH represents a significant change in hydrogen ion concentration. The pH considered compatible with life is from 6.8 to 8, with normal for most mammalian species being around 7.4 and kept within a very tight range. When a patient has a blood pH lower than normal (an increase in blood H+ concentration) the patient is considered to have an acidemia, 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, which is often what is being assessed when looking at blood gases. An acidosis can be respiratory or metabolic in origin, i.e. primary respiratory or metabolic acidosis.

In a similar vein, alkalaemia is a blood pH higher than normal. 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. As with acidosis, an alkalosis can be respiratory or metabolic in origin, i.e. primary respiratory or metabolic alkalosis. 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.

Buffer Systems and Henderson-Hasselbalch Equation

As a physiologic protective mechanism, the body will attempt to rectify the altered pH so that the body isn't exposed to long periods of acidosis or alkalosis. Sustained pH changes will also alter the response to drug therapies, as they are assessed for their effectiveness under normal pH levels. The Henderson-Hasselbalch equation can account for some of the compensatory mechanisms that happen within the body in 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. The equation below is the Henderson-Hasselbalch equation:

pH= 6.1 + log ([HCO3-}/[0.03 x PCO2])

Simplified, an acid is a hydrogen ion (or proton) donor, a base is a hydrogen ion acceptor. From this equation it is evident that pH is a consequence of the ratio of pCO2 and bicarbonate; the pH is directly related to bicarbonate, a base, (HCO3) (that is, as bicarbonate increases, pH increases) and inversely related to pCO2, an acid, (so as pCO2 goes up, pH goes down). 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. Buffer systems within the body maintain acid-base balance. The two main buffer systems are the lungs and kidneys and they are directly related; regulation of pCO2 by ventilation (blowing off acid, or retaining acid), 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. Other buffer systems within the body including in the gastrointestinal system and non-carbonate buffers such as plasma proteins and phosphates will also have a small effect on the changes seen to the pH.

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. A patient will never have respiratory acidosis and respiratory alkalosis simultaneously.

Further Theories

Strong Ion Theory, proposed in 1981, works on the basis that strong ions and proteins affect acid–base status. This theory treats body fluids as a system and accounts for other buffering systems besides the lungs and kidneys, which are directly and inversely proportional to each other and responsible for the body correcting its pH — therefore, not only HCO_3– and PCO_2 affect the pH. Weak anions such as H+, OH–, HCO_3–, and HA are reliant on the independent parameters and attempt to create equilibrium.

The independent variables — pCO_2, ATOT (total weak non-volatile acids such as albumin), and SID (Strong Ion Difference) — control arterial plasma acidity. The body maintains electroneutrality through dissociation equilibria involving weak electrolytes. Since Na+ and Cl– are the most abundant strong ions, it is relatively simple to calculate SID from Na+ and Cl–. Strong anions also include L-lactate and urate, which can have significant influence over pH.

A high SID equates to alkalosis due to a loss of Cl– or a gain of Na+. A low SID equates to acidosis due to a gain of Cl– or loss of Na+. ATOT comprises acids that are not fully dissociated at a pH of 7.4 and will not cause changes to respiratory or renal mechanisms; these include proteins such as albumin and phosphate.

Unmeasured strong ions include lactate and ketones (β-hydroxybutyrate, acetoacetate), which are often seen in critical care patients. These ions contribute to base excess (BE); since unmeasured strong ions are anions, they cause an acidosis and decrease BE.

BE 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 pCO_2 of 40 mmHg 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 component because it is not influenced by pCO_2.

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Leadership Stream, Friday 5 June 2026

INFECTION PREVENTION AND CONTROL IN THE ICU/ER

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Learning objectives:

- Understand the importance of infection prevention and control within an ICU and ER setting.
- Be able to identify common risks within an ICU and ER setting.
- Understand standard and transmission-based precautions and how to implement them to mitigate infectious risks.
- Be able to identify specific risks in high-dependency patients and understand how to mitigate them.
- Understand the ongoing educational requirements for IPC within an ICU and ER setting.

Proceeding:

Infection control is a pivotal component of patient care in the veterinary intensive care unit (ICU) and emergency room (ER), where animals are often critically ill, immunocompromised, or exposed to invasive procedures. The high patient turnover, frequent intensive handling by staff, and use of medical devices significantly increase the risk of hospital-associated infections (HAIs). Effective infection control protocols are therefore essential to protect patients, veterinary staff, students, and clients.

Hand hygiene

One of the most important elements of infection prevention and control is hand hygiene. Hands are a key pathway for transmission of infectious agents, including multidrug-resistant organisms. All personnel should perform hand hygiene according to the 'Five Moments of Hand Hygiene'. Alcohol-based hand rubs are effective in most situations, while handwashing with soap and water is preferred when hands are visibly soiled or when dealing with certain pathogens, such as spore-forming bacteria, due to the reduced efficacy of alcohol-based hand rub against these specific pathogens.

Personal protective equipment

Personal protective equipment (PPE) plays a critical role in preventing the spread of infection. PPE should be selected based on the staff and patient risk levels and the procedures being performed. Reverse barrier nursing should be implemented for immunocompromised patients.

Isolation

Patients suspected or confirmed to have contagious diseases should be promptly and clear identified and managed using standardised isolation protocols. Physical separation, dedicated equipment, clear

signage, and good communication between clinical staff can help minimize potential cross-contamination between patients.

Environmental decontamination

Environmental cleaning and disinfection are equally important in high-risk areas such as the ICU and ER. All surfaces, kennels, and equipment must be cleaned and disinfected regularly using products effective against veterinary pathogens. High-touch areas, including door and drawer handles, infusion pumps, and monitoring equipment, require more frequent disinfection. Each area should have a clear cleaning protocol and should specify the appropriate disinfectant, dilution, contact time, and cleaning frequency. Compliance should be routinely monitored to highlight areas for improvement.

Invasive devices

Invasive medical devices such as intravascular catheters, urinary catheters, and endotracheal tubes are common sources of infection in critical care patients. Strict aseptic technique during placement, regular maintenance, and prompt removal when no longer needed are essential to reduce device-associated infections. Patients should be assessed daily for their requirements and ongoing necessity of devices.

Antimicrobial stewardship

Antimicrobial stewardship is an increasingly important component of infection control within the veterinary industry. Judicious use of antibiotics helps limit the development of antimicrobial resistance, which poses a serious global threat to both animal and public health. Antimicrobial therapy should be guided by clinical judgment, and treatment should be based on culture and sensitivity results whenever possible.

Education and compliance

Staff education and compliance monitoring are fundamental to the success of infection control programs. Regular training ensures that all staff understand current protocols and the rationale behind them. Protocol updates, audits, feedback, and a professional culture of accountability can help reinforce good practices and encourage continuous improvement.

Infection control in the veterinary ICU and ER requires a multifaceted approach that includes hand hygiene, PPE use, environmental decontamination, device management, antimicrobial stewardship, ongoing staff education, and audit and surveillance for compliance. Consistent implementation of these measures is essential to reducing healthcare associated infections and improving patient outcomes in critical care settings.

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MANAGING AN OUTBREAK SITUATION IN THE ICU

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Learning objectives:

- Understand what an outbreak is.
- Understand the common causes and pathogens seen in an outbreak in a veterinary ICU.
- Identify how to detect an outbreak and the immediate responses required.
- Understand the steps required to control an outbreak.
- Identify the protocols recommended to prevent future outbreaks.

Proceeding:

Outbreaks of infectious disease in small animal veterinary intensive care units (ICU) pose significant risks to patient outcomes, staff safety, hospital function, and reputation. Patients admitted to the ICU are often immunocompromised, require invasive devices, and are hospitalised for longer periods, making them more susceptible to hospital-associated infection (HAI). Therefore, early recognition and structured investigation of infectious outbreaks are essential parts of infection control in small animal critical care units.

Recognising an outbreak

Suspicion of an outbreak in a small animal ICU should be raised when there is an increase in hospital-associated infections, identification of unusual or multidrug-resistant organisms, or clustering of similar clinical disease among patients over a defined period. Timely detection relies on routine surveillance, awareness of baseline infection rates, and effective communication between all hospital staff and diagnostic laboratories.

First response

Once an outbreak is suspected, immediate containment measures must be implemented. These include reinforcing hand hygiene compliance, escalating the use of personal protective equipment, isolating affected patients, and minimizing patient movement within and outside of the ICU. Dedicated equipment for infected patients and enhanced environmental cleaning should be initiated while the investigation is ongoing and only de-escalated once the outbreak is declared cleared.

Investigation

The outbreak investigation begins with the development of a clear case definition, considering species, clinical signs, and diagnostic findings such as culture and sensitivity reports. Affected and exposed patients should be identified, and detailed case mapping is performed to evaluate potential contamination pathways within the ICU. Review of patient records can help identify shared risk factors such as procedures, devices, personnel, or clinical areas.

Diagnostics

Diagnostic testing is central to confirming the outbreak. Samples from affected patients should be collected for culture and susceptibility testing, and, where appropriate, molecular typing. Environmental sampling of kennels, clinic surfaces, monitoring equipment, and shared devices may be useful in identifying environmental reservoirs or points of transmission. Collaboration with diagnostic laboratories, such as designated times to submit samples, can help to improve timely interpretation of results.

Review of practices

Assessment and evaluation of routine hospital infection control protocols is essential. Hand hygiene adherence, cleaning and disinfection protocols, equipment handling and aseptic technique during invasive procedures should be reviewed for compliance. Any gaps identified should be addressed immediately, and staff education revisited.

Communication

Clear and consistent communication throughout the outbreak is vital. ICU staff should have regular updates on case status, control measures, and workflow adjustments. Hospital management and infection control teams should be engaged early to support decision-making and supply resources. Documentation of the investigation process encourages transparency within the hospital and provides useful evidence for future use.

Outbreaks can be particularly concerning in a small animal veterinary ICU and require a considerable multifaceted response. A proactive approach with clear structure not only limits outbreak impact but also reinforces long-term infection control practices and improves patient safety in small animal critical care settings.

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THRIVE OR SURVIVE: TOOLS TO SUPPORT THRIVING

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Learning objectives:

- Understand what can happen to us physiologically, emotionally, and cognitively when we become overwhelmed.
- Recognise when our team is becoming overwhelmed
- Consider how to apply the tools discussed to help yourself and your team start to thrive again.

Proceeding:

Emergency and critical care (ECC) environments expose clinicians to sustained high-pressure situations. Multiple unstable patients, time-critical decision-making, and unpredictable caseloads can create conditions where stress levels exceed an individual's cognitive and emotional capacity. While moderate stress can enhance focus and performance, overwhelming stress reduces cognitive bandwidth. This shift moves clinicians from a state of thriving to one of surviving. Understanding how to recognise and manage this transition is essential for maintaining patient safety, effective communication, and team cohesion.

Physiological, Emotional and Cognitive Responses to Overwhelm

When we become overwhelmed, the sympathetic nervous system is activated, preparing the body for threat: increasing heart rate, respiration, and muscle tension. While adaptive in short bursts, prolonged activation leads to physical fatigue and reduced fine motor control.

Emotionally, overwhelm can look like irritability, withdrawal, anxiety or a sense of loss of control. These emotional shifts can influence interactions with colleagues, patients, and clients, often reducing empathy and slowing emotional processing.

Cognitively, overwhelm narrows attentional capacity. Executive functions, such as planning, prioritisation, working memory, logical reasoning and problem-solving can become impaired. Individuals may struggle to sequence tasks, maintain situational awareness, or adapt to changing patient needs which, in turn, can increase the risk of errors, communication breakdowns, and delayed decision-making.

Recognising overwhelm in teams

Key indicators include:

Reduced clarity in communication

Increased frequency of small errors

Decreased ability to share mental models

Emotional escalation or withdrawal

Loss of adaptability or creative problem-solving

Leaders and team members must be able to identify these signs early to allow timely intervention for supportive action rather than reactive behaviour.

Without recognition cognitive bandwidth decreases, clinical decision-making becomes less accurate and more susceptible to bias and patient safety can be compromised through missed cues, incomplete assessments or ineffective prioritisation. In parallel staff wellbeing deteriorates contributing to moral distress, compassion fatigue and burnout; without action this risks becoming a repeating circle of events.

Tools

Without tools to widen cognitive bandwidth, individuals and teams risk functioning below their usual clinical capability, and organisational culture can shift toward survival-mode behaviours.

Helpful tools include:

Controlled Breathing

Techniques such as box breathing provide a rapid, evidence-based method for activating the parasympathetic nervous system. Slow, controlled respiration reduces physiological arousal and supports re-engagement of executive function.

Micro-pauses and Reset Moments

Short, purposeful pauses between tasks create cognitive separation and reduce the load associated with continuous task switching. These pauses can be as brief as one structured breath cycle or a 10-second grounding technique.

Cognitive Offloading

Using checklists, whiteboards or structured communication systems reduces the demand on working memory. By externalising cognitive tasks, teams maintain clarity even during peak workload.

Relational Awareness

Overwhelm can be mitigated through situational awareness not only of the clinical environment but also of team emotional states. Leaders who monitor team dynamics, anticipate pressure points and intervene early can prevent escalation. Supportive interactions, for example clear task allocation, verbal reassurance or brief huddles can help stabilise team function.

Leadership Considerations

Effective leadership in high-pressure settings relies on awareness, presence and adaptability. Rather than relying on directive communication under stress, leaders benefit from maintaining an overview of the environment, identifying emerging risks and supporting team members who show signs of narrowing bandwidth. Leadership actions such as redistribution of workload, brief resets, or clarifying priorities helps to widen bandwidth, strengthen team resilience and support overall performance.

COMPASSION FATIGUE IN THE EMERGENCY CRITICAL CARE VETERINARY PROFESSIONAL

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Learning objectives:

By the end of this presentation, participants will be able to understand the following:

- Define compassion fatigue and distinguish it from related conditions such as stress and burnout with specific reference to the unique emotional demands of veterinary practice.
- Identify risk factors and contributing conditions for compassion fatigue in veterinary professionals including chronic exposure to grief, euthanasia, difficult client interactions, and financial stressors.
- Recognize common signs and symptoms of compassion fatigue at the emotional, behavioral, interpersonal, and occupational levels including factors/indicators associated with elevated suicide risk.
- Describe evidence-informed prevention and intervention strategies for compassion fatigue (e.g., self-care practices, emotional processing, workplace-level supports, and when to seek professional help).

Proceeding:

The field of veterinary medicine is deeply rewarding and provides meaningful opportunities to heal animals. At the same time, the work environment can be intensely pressured and emotionally demanding especially among emergency critical care professionals (ECC). ECC veterinary professionals (i.e., veterinarians and veterinary technicians/nurses) often work long hours under stressful conditions, where the priority of saving animals frequently supersedes the acknowledgment and processing of intense emotions or the promotion of self-care. Unfortunately, the combination of chronic stress, limited self-care, and insufficient emotional processing can contribute to compassion fatigue. In the United States, these pressures may be compounded by significant educational debt, with more than one quarter of veterinary graduates accruing over $200,000 in student loans during training (American Veterinary Medical Association [AVMA], 2019).

What Is Compassion Fatigue?

Individuals who enter veterinary medicine are typically highly dedicated, compassionate professionals motivated by improving the lives of animals. Over time, however, some may become disenchanted or disillusioned experiencing a gradual decline in enthusiasm and sense of purpose. This disillusionment can extend beyond work, and can negatively affect relationships and overall life functioning. Chronic exposure to death, dying, grief, and emotionally intense environments has been linked to interpersonal difficulties, anger, frustration, loneliness, cynicism, and even suicide (Bartram

& Baldwin, 2010). When veterinary professionals experience deterioration across emotional, interpersonal, and occupational domains, they may be experiencing compassion fatigue (Ayl, 2013).

Definition

Compassion fatigue refers to the emotional, physical, mental, social, and spiritual changes that occur as a result of sustained empathic engagement with individuals experiencing trauma, such as death and dying (Ayl, 2013). It is more severe than general work-related stress and differs from burnout. Compassion fatigue is associated with a deep awareness of others' suffering coupled with a strong desire to relieve that suffering (Penson et al., 2000). It has also been conceptualized as secondary traumatic stress or vicarious trauma (Keidel, 2002). Stress typically involves excessive workload, long hours, and high expectations, whereas burnout is characterized by emotional depletion, apathy, and loss of motivation. Importantly, stress and burnout are often alleviated by time away from work such as vacations. Compassion fatigue, however, is more pervasive and damaging. It may involve emotional numbing or restricted affect and is particularly prevalent in professions with prolonged exposure to suffering, loss, and difficult clients. Recent studies suggest that over 50% of veterinary professionals experience compassion fatigue (Grindlay et al., 2020; Reijula et al., 2023). Continuous exposure to client grief, trauma, and death can leave practitioners feeling overwhelmed, angry, exhausted, and emotionally depleted. When unaddressed, compassion fatigue may result in career dissatisfaction, high turnover, substance misuse, personality changes, and suicide (Bartram & Baldwin, 2010; Collins & Long, 2003). Family members and colleagues are often the first to notice these changes, as affected individuals may become irritable, withdrawn, or disillusioned with a profession they once loved.

Contributors to Compassion Fatigue

Multiple factors contribute to compassion fatigue in veterinary medicine. These include frequent interactions with distressed or angry clients who project grief onto veterinary staff, repeated exposure to death and euthanasia, and the emotional burden of navigating end-of-life decisions for beloved pets. Clients may struggle to understand euthanasia or reject medical recommendations leading to prolonged animal suffering and increased moral distress for the professional. Financial constraints that prevent clients from affording life-saving treatments may further evoke guilt, frustration, or anger in veterinary professionals. Nearly daily exposure to grief, loss, and ethically complex decisions in the critical care professional significantly elevates the risk for compassion fatigue.

The Most Severe Outcome: Suicide

Suicide represents a tragic and extreme outcome potentially associated with compassion fatigue. While suicidality is multifactorial, compassion fatigue may interact with biological and environmental vulnerabilities to increase risk. The normalization of euthanasia within veterinary practice—where ending life is framed as a humane solution to suffering—may inadvertently reduce inhibitions toward suicide among professionals (Kirwan, 2005). Bartram and Baldwin (2008) found that veterinarians in the UK had a suicide rate four times higher than the general population and twice that of other health professionals. Recent Austrian studies similarly reported elevated suicide risk among male and female veterinarians compared to the general population and other medical professions (Zimmermann et al., 2023; Zrinka & Steiger, 2025). These alarming findings underscore the urgent need for enhanced self-care and systemic support within the profession.

Symptoms of Compassion Fatigue

ECC veterinary professionals experiencing compassion fatigue may find their work emotionally exhausting and increasingly devoid of meaning. Common symptoms include guilt, sadness, anger, emotional volatility, irritability, and a sense of detachment. Tasks once experienced as rewarding may feel burdensome or thankless, contributing to emotional withdrawal and interpersonal conflict.

Treating Compassion Fatigue

It is essential that veterinary professionals do not ignore or suppress difficult emotions associated with their work. These emotions must be acknowledged, processed, and supported rather than buried. Without intervention, compassion fatigue can affect both professional performance and personal well-being. Although compassion fatigue can erode passion for veterinary medicine, there is hope. Effective interventions include individual self-care practices, organizational support, and professional mental health services. Evidence-based well-being programs have demonstrated positive effects on compassion satisfaction and reductions in burnout and secondary traumatic stress (Rohlf et al., 2024).

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Nurse Advanced Stream, Friday 5 June 2026

PATHOPHYSIOLOGY OF NAUSEA AND VOMITING

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Learning objectives:

- Define nausea, vomiting, regurgitation and ileus
- Describe the pathophysiology of each process
- Discuss pharmacological treatment options for each process and the rational for drug choice
- Describe nursing considerations for the critical patient experiencing nausea, vomiting, regurgitation and ileus

Proceeding:

Many ECC patients display nausea, vomiting, regurgitation, and ileus. These symptoms may be associated with primary disease processes like pancreatitis or secondary to treatments such as chemotherapy. While veterinary teams excel at identifying pain, gastrointestinal symptoms often go unrecognized due to subtle presentation. Understanding the underlying physiology enables nurses and technicians to identify which pharmacological agents will benefit their patients.

Gastrointestinal Physiology

Gastrointestinal function involves complex coordination between the central and enteric nervous systems. Critical illness commonly alters GI motility, resulting in esophageal dysmotility, delayed gastric emptying, functional obstruction, and colonic dysmotility. After swallowing, peristalsis moves food to the stomach, where mixing with acid, pepsin, gastrin, and protective mucus occurs. Partially digested food passes into the small intestine, where most digestion and absorption happens in the duodenum with bile and pancreatic enzymes. The large intestine absorbs water and electrolytes to form feces.

Nausea and Vomiting

Nausea represents a subjective feeling of needing to vomit—a protective evolutionary response. In humans, nausea proves more disabling and longer-lasting than vomiting itself. Defining nausea in veterinary patients challenges clinicians due to its subjective nature. Studies score nausea based on observed behaviors: salivation, lip licking, lethargy, restlessness, and circling behavior.

Nausea and vomiting can exist independently; patients may experience severe nausea without vomiting, making behavioral observation paramount. Research has explored biomarkers like arginine vasopressin and cortisol, which correlate with nausea-like behavioral responses, though further evaluation is needed.

Vomiting (emesis) involves active contractions of gut and thoracoabdominal musculature, expelling digested food with or without bile. Pathophysiology involves stimulation of the vomiting center in the medulla oblongata through the CNS, autonomic nervous system, and endocrine system. Principal

receptors include histamine (H1), acetylcholine (M1), serotonin (5-HT3), dopamine (DA2), and neurokinin (NK1).

Before vomiting, reverse peristalsis moves small intestinal contents into the stomach. As the upper GI tract distends, nerve impulses reach the vomiting center, triggering contractions that expel vomitus. Numerous causes include medications, toxins, GI disorders, CNS disorders, and metabolic causes. Common sequelae include aspiration pneumonia, reflux esophagitis, abdominal pain, and inadequate nutrition.

Regurgitation

Regurgitation involves passive passage of esophageal contents into the mouth. The esophagus contains smooth and striated muscle regulated by the vagal nerve and autonomic nervous system. Regurgitation occurs when the lower esophageal sphincter relaxes and esophageal muscular activity alters. Causes include hypomotility, inflammation, or obstruction. Regurgitation typically occurs after eating, contains undigested food without bile, and may manifest as burping and gulping. Aspiration pneumonia represents a common critical complication.

Ileus

Ileus involves temporary lack or abnormal GI muscle contraction patterns, causing fluid or gas accumulation, abdominal distention, and reduced ingesta movement. Gastric emptying requires coordinated muscle contraction and relaxation regulated by vagal nerve function. Five small intestinal contractile patterns aid movement: peristalsis, stationary segmenting contractions, giant contractions, migrating contraction clusters, and migrating motor complexes.

Pathophysiology involves "pump failure" from motor dysfunction or "excessive feedback" with inhibitory pathway activation. Inflammatory mediators damage muscularis, releasing cytokines and nitric oxide, causing muscle paralysis and intestinal dilation. Concerns include predisposition to regurgitation or vomiting, increasing aspiration pneumonia risk and decreasing calorie absorption.

Pharmacological Support

Understanding physiology guides rational drug selection. Antiemetic and antinausea drugs inhibit emetic pathways, targeting serotonin (5-HT3), dopamine (DA2), and neurokinin (NK1) receptor antagonists. Prokinetic drugs treat GI dysmotility. Technicians should understand medication pharmacology: mechanism of action, effects, side effects, drug interactions, and administration techniques. In critically ill patients, route selection matters—oral preparations may not absorb if vomiting occurs, necessitating alternative routes or medications.

Non-Pharmacological Support

Non-pharmacological strategies include treating metabolic abnormalities to maintain electrolyte homeostasis and normal acid-base balance. Early mobilization encourages GI movement and blood flow. Appropriate pain management using multimodal approaches minimizes opioid-related GI motility effects. Balanced fluid resuscitation avoids both inadequate perfusion and GI edema. Early enteral nutrition improves GI blood flow, minimizes bacterial translocation, and stimulates normal GI physiology.

Conclusion

While the true incidence of nausea, vomiting, ileus, and regurgitation in veterinary ICU populations remains unknown, these symptoms are commonly observed. Untreated, they impact patient welfare and increase complication risks. Nurses and technicians play crucial roles in identifying subtle signs, ensuring appropriate pharmacological agents are prescribed, administering medications safely, and incorporating non-pharmacological interventions. Understanding pathophysiology enables comprehensive, evidence-based care that optimizes patient outcomes in critically ill patients.

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THERMOREGULATION IN THE CRITICAL PATIENT

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Learning objectives:

- Understand normal thermoregulatory pathophysiology
- Understand adaptive thermoregulatory mechanisms
- Classify hypothermia and hyperthermia
- Analyse physiological effects of abnormal temperatures
- Analyse responses to interventions

Proceeding:

Temperature is controlled by the thermoregulation centre in the anterior chamber of the hypothalamus, acting as the 'thermostat' of the body, setting the temperature range to maintain normal metabolic function. The body communicates temperature through nerve cells from both peripheral and central thermoreceptors. They receive the temperature and transmit signals to the hypothalamus which then triggers behavioural or physiological compensatory mechanisms. Behavioural adaptive mechanisms are voluntary decisions to actively seek out warmer or cooler areas and utilise conduction (direct) and radiation (indirect) transfer of heat to or from another object. Physiological adaptive mechanisms are involuntary autonomic responses to correct temperature such as: water loss via skin, piloerection/pilorelaxation, cutaneous vasomotor tone changes, salivation and panting, shiver reflex, hormone release, increased glycogenesis, heat shock protein synthesis, vasodilation (diverting warm blood peripherally for dissipation of heat via the skin) or vasoconstriction (diverting warm blood to central circulation to sustain vital organ function).

Hypothermia

Hypothermia in canines and felines is defined as a temperature <37.2°C and can be primary (exposure to a cold environment) or secondary (triggered by another primary cause such as disease, clinical syndromes, toxicity, drug administration, or ongoing recumbency). Hypothermia initially triggers peripheral vasoconstriction and an increase in heart rate to prevent heat loss and ensure adequate cardiac output. If sustained, the following can occur:

Cardiovascular: bradycardia and eventual decreased systemic vascular resistance, alpha-1 adrenergic receptors lose affinity for noradrenaline and blood pressure decreases. Cardiac pacemaker cells and myocardial conduction become compromised, can lead to arrythmias such as ventricular fibrillation and asystole

Respiratory: a decrease in cellular metabolism will decrease carbon dioxide levels and may affect respiratory drive

Acid base: respiratory and metabolic changes affect acid base

Coagulation: decreased platelet production and factor function

Kidneys: suppressed antidiuretic hormone secretion and marked diuresis risking hypovolaemia

Immune response impairment

Hyperthermia

Hyperthermia in canines and felines can be defined as a temperature above 39.2°C and divided into the below classifications.

Non-pyrogenic: there is a high core body temperature, but the hypothalamus thermoregulatory range is normal. Primary causes include exposure to high temperatures or humidity. Secondary causes: increased metabolic demand or inefficient heat loss mechanisms. Malignant hyperthermia can be triggered by the administration of drugs, with high mortality rates.

Pyrogenic: there is a high core body temperature, and the hypothalamus thermoregulatory range is increased to above the normal range. Pyrexia is a secondary occurrence induced when exposed to a pyrogenic trigger. Cytokines then travel to the hypothalamus, bind to the endothelial cells and causes production of prostaglandin E2 (PGE2). PGE2 causes the thermoregulatory cells to increase the set point, tricking the hypothalamus into thinking higher is normal.

During hyperthermia, peripheral vasodilation initially occurs to increase heat loss and heat shock proteins are utilised to protect cells. If sustained, the following issues can occur:

Cardiovascular: hypovolaemia can lead to hypoperfusion and ischemic damage. Arrythmias follow cardiac myocyte cytotoxicoses, ischaemic damage and release of excess free radicals.

Hypoglycaemia: due to overwhelming metabolic demand/failure of glycogenesis

Hypoxia: due to overwhelming oxygen demand

Gastrointestinal: direct mucosal cytotoxicity and ischemia from hypoperfusion leads to sloughing and risks bacterial translocation

Coagulation: platelets, coagulation and fibrinolysis are activated, resulting in microthrombi which leads to disseminated intravascular coagulation

SIRS/Sepsis: heat shock proteins and other cytokines can cause systemic overwhelm and hypoperfusion/hypoxic states can also lead to multiple organ disfunction

CNS: nerve cytotoxicity, cerebral hypoperfusion and microthrombi can cause cerebral oedema

Exertional heat stroke/malignant hyperthermia may cause rhabdomyolysis and subsequent renal damage

Interventions

Warming and cooling techniques are passive or active. Passive techniques concentrate on maintaining body temperature. Active techniques utilise an external source to provide heat or cooling. In critical patients with comorbidities, intervention becomes more intricate as professionals

must consider primary causes and compensatory mechanisms that may be influenced. As always, consider risk vs benefit and assess the patient as an individual. Controlled warming of the core and treatment of the primary cause is essential for hypothermia to avoid drastic surface warming leading to peripheral vasodilation and rewarming shock. Patients should also be closely monitored for an 'after drop' as cool blood from extremities re-enters central circulation. Rapid active cooling of patients with non-pyrogenic hyperthermia should be actioned quickly and ceased when rectal temperature reaches 39.5°C to avoid rebound hypothermia. Active cooling of pyrexic patients is contraindicated unless patients are at risk of cytotoxicity due to extreme hyperthermia. Responses to interventions should be monitored closely to avoid risks and ensure patient safety.

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MASTERING SIRS, SEPSIS, SEPTIC SHOCK AND MODS

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Learning objectives:

- Purpose of inflammation
- Systemic Inflammatory Response Syndrome (SIRS)
- Sepsis
- Treatments
- Multi Organ Dysfunction Syndrome (MODS)

Proceeding:

Systemic inflammatory response syndrome (SIRS) and sepsis are complex diseases that are often related to one another, and result in high mortality. Sequelae of SIRS and sepsis include cardiovascular collapse, disseminated intravascular coagulation (DIC), Multiple Organ Dysfunction Syndrome (MODS), septic shock, and death.

Inflammation

Inflammation is a complex response to harmful stimuli, such as pathogens, trauma or toxins designed to protect the host from disease, by;

Eliminating the source of the inflammationRemoving damaged tissuePromoting healing

Normal pro-inflammatory response

The pro-inflammatory response stimulates inflammation, via the release of chemicals, which increases blood flow and recruitment of cells to the affected area, causing signs such as redness, swelling and heat.

Normal anti-inflammatory response

The anti-inflammatory response, known as the compensatory anti-inflammatory response syndrome (CARS) is intended to regulate inflammation and prevent an excessive immune response which could damage the body's tissues.

SIRS

SIRS occurs when there is dysregulation of pro-inflammatory and anti-inflammatory responses. Pro-inflammatory mediators gain entry into systemic circulation and instigate global activation of the immune system. Excessive inflammation disrupts homeostasis and results in vasodilation, increased vascular permeability and dysregulates coagulation.

Sepsis

Sepsis is SIRS with a suspected source of infection.

Clinical signs of SIRS and sepsis

Signs of early SIRS/sepsis include fever, depressed mentation, increases in heart rate (HR) and respiratory rate (RR), injected or muddy mucous membranes (MM), rapid capillary refill time (CRT) (<1 second), and bounding pulses.

It is important to note that there is a fundamental difference between cats and dogs in the haemodynamic response to systemic inflammation, with cats more likely to experience bradycardia and hypotension.

Diagnosis of SIRS

Dogs exhibiting 2/4 of the diagnostic criteria, or cats exhibiting 3/4 are considered to have SIRS. The diagnostic criteria include abnormalities to temperature, heart rate, respiratory rate or white blood cell count.

Diagnosis of Sepsis

Sepsis requires rapid diagnosis and treatment, so it is essential for veterinary nurses to know the clinical signs associated with it. Early recognition and treatment have been shown to improve mortality in human patients by up to 50%.

Obtaining blood for cultures allows for specific identification of the infectious pathogen and the antimicrobial therapy required to treat it.

Treatment of SIRS and Sepsis

Fluid Therapy A mainstay of treatment is fluid resuscitation which aims to resolve hypovolaemia, maintain daily requirements, and replace ongoing losses.

Vasopressor Therapy If fluid therapy fails to restore cardiovascular stability, vasopressor therapy may be used to increase systemic vascular resistance and mean arterial pressure, helping to maintain adequate perfusion of vital organs

Nutrition Nutritional support is essential for the recovery of any patient experiencing a critical illness. Early enteral nutrition is recommended to help maintain gastrointestinal mucosa integrity, prevent bacterial translocation, maintain glycemic control, and promote wound healing and normal immune responses.

Pain Control Controlling pain reduces morbidity and mortality. The use of a validated pain scoring system (e.g., Colorado, Glasgow, and Feline Grimace Pain Scale) will help to evaluate the pain and guide analgesia administration.

Antimicrobial therapy Prior to receiving blood culture results, broad spectrum antibiotics should be prescribed and administered to any patients with known or suspected sepsis. Broad spectrum antibiotics cover gram-positive, gram-negative, anaerobic organisms.

Infectious source control for patients with known or suspected sepsis Involves removing the source of the infection (either medically or surgically) as early as possible, and is critical to the outcome.

Monitoring Monitoring and evaluation of volume and perfusion status with the use of blood pressure monitoring, serum lactate measurements, central venous pressure (CVP), central venous oxygen

saturation, serial body weights, and comparison of fluid intake and output should be carried out regularly.

Severe sepsis

Severe sepsis is a term used to describe sepsis with one or more end-organ failures.

Septic shock

Septic shock is defined as hemodynamic instability that persists despite adequate intravascular volume resuscitation. Such sequelae of sepsis increase mortality and require immediate intervention to support the circulatory system.

Multiple Organ Dysfunction Syndrome (MODS)

MODS is the potentially reversible dysfunction of two or more organ systems caused by a life-threatening systemic inflammatory response. Once present, treatment is supportive and aimed at aggressively managing the SIRS/sepsis while providing organ-specific support.

Sequential organ failure assessment (SOFA) scoring

Sequential organ failure assessment (SOFA) scoring is one of the most common organ dysfunction scoring systems in human medicine, and it is predictive of outcome in dogs. Higher SOFA scores indicate increased severity of the organ failure. The six organ systems evaluated are cardiovascular, respiratory, neurological, renal, hepatic, and haematological.

To ensure effectiveness and assist with early recognition, a patient's SOFA score should be documented at admission and repeated throughout the hospital stay.

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STATE OF THE UNDEAD: POST-ARREST CARE

Karen Roach [1]

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Learning objectives:

- What exactly happens in a patient's body when it has been dead for a period of time?
- What complications can arise from what happened while the patient was for all intents and purposes, dead?
- What monitoring and treatments are most important during the post arrest period?

Proceeding:

Anyone who has worked in veterinary medicine for any length of time, especially if it has been emergency or critical care, has likely been involved in CPR many times. The hard truth is that we rarely get these patients back; this stems from the fact that so many of our patients are so badly injured, or have very serious underlying diseases. Successful CPR depends on the cause of the arrest. If the patient was healthy prior to the arrest – this usually encompasses arrests that are caused by anesthetic medications, or easily rectified problems (kinked endotracheal tube, improper anesthetic depth, allergic reaction). Patients that arrest due to serious disease or trauma are less likely to be successfully resuscitated, and if they are, will be considered extremely critical and unstable in the post resuscitation stage. Sometimes when we do get a patient back, it always seems a bit surreal, "did that really happen?" "we actually got a patient back?!" and then, you think, "NOW what?!" We are so focused on doing life-saving CPR with the goal of having the patient exhibit ROSC (Return of Spontaneous Circulation), but since it happens so infrequently, when it does happen, there is always this moment of pause where we try to think of what comes next.

The best place to start is to realize that this patient WAS truly dead for a period of time, and to think of all the systems in the body that have been affected by this, what changes have possibly happened in that period. After an animal has been through a cardiac arrest, they go into what is called post cardiac arrest syndrome. There are 4 major components to this: brain injury, myocardial dysfunction, systemic ischemia/reperfusion injury, and also persistence of the underlying disease. All these things do need to be addressed with an overall approach of focusing on optimizing respiratory and cardiac function, and neuroprotection.

The first priority following successful resuscitation is respiratory support. Ventilation is not always spontaneous immediately following arrest, and other supportive treatments will not be useful if the patient is not receiving enough oxygen. Most patients may require mechanical ventilation with 100% oxygen for a period of time for optimal respiratory support. The respiratory system does need to be closely assessed during this time. Respiratory system assessment includes auscultation, respiratory rate and depth, Hb, EtCO2, and arterial blood gases. If the patient is ventilating properly, mechanical ventilation of course will not be necessary, but oxygen supplementation, preferably via nasal cannulas, does need to be administered. If the patient is not being mechanically ventilated, then

respiratory rate and effort does need to be closely watched. Hypoventilation puts patients at risk for cerebral vasodilation, increased ICP, peripheral vasodilation and this all leads to compromised perfusion. Hyperventilation with decreased PCO2 causes cerebral vasoconstriction and has the potential to worsen cerebral ischemia.

If possible, collect an arterial blood gas immediately following successful ROSC. It is also recommended to place an arterial line for ease of repeat blood work and direct blood pressure monitoring. Placing nasal cannulas for oxygen supplementation is a must if a patient does not require mechanical ventilation. We want to see the PaO2 >60mmHg to assure adequate Hb saturation for effective oxygen delivery to tissues. Nasal oxygen cannulas provide an FiO2 of approximately 40%, which is safe to continue for a prolonged time. Oxygen toxicity can occur with oxygen of over 50% for >12-24 hours, and results in severe pulmonary damage.

It may become necessary to take thoracic radiographs to check for rib fractures, pulmonary contusions, or pneumothorax from CPR. But the patient should not be moved and manipulated for radiographs until they are breathing spontaneously, and their cardiovascular system stabilized.

Myocardial depression and cardiac arrhythmias (frequently VPCs and ventricular tachycardia) are commonly seen in the post-arrest patient. Monitoring should include continuous ECG and arterial blood pressure in order to help optimize organ perfusion. Sustained v-tach >160/min is most commonly treated with lidocaine at 1-4mg/kg IV, then 30-80mcg/kg/min CRI. Refractory ventricular arrhythmias can be treated with magnesium at 0.15mEq/kg IV, then 0.75-1.0mEq/kg/day CRI. Bradycardia should be treated with atropine or glycopyrrolate. Sinus tachycardia can be due to fear, anxiety, pain, hypoxia, or hypovolemia. Analgesia should be provided for post-arrest patients.

Since post-arrest patients can have poor perfusion, IV fluids should be administered to help maintain this. And because of the lack of proper perfusion to the heart during the arrest, many patients may also require a positive inotrope to support the cardiovascular system for 24-48 hours. (Dobutamine 5-10mcg/kg/min or Dopamine 5mcg/kg/hr – adjust dose based on HR, BP). Patients on IV fluids should have the following parameters monitored: arterial BP, urinary output, lactate – and if these do not improve, MODS (multiple organ dysfunction syndrome) will likely follow.

During cardiac arrest, the brain can be damaged through blood glucose and ATP being depleted. Cerebral perfusion can be improved by maintaining blood pressure at 80-120mmHg. Increased intracranial pressure can be reduced by furosemide and/or mannitol. Hypothermia is actually neuroprotective, so avoiding hyperthermia is key, and no active rewarming should be performed. Maintaining blood glucose in the normal range will decrease further harm to the brain. Calcium channel blockers (such as nimodipine & lidoflazine) can prevent cerebral vessels from vasoconstricting and prevent neuronal damage as well. Serial neuro exams should be performed, looking at presence of palpebral or corneal reflex, response to external stimuli, PLR, as well as size and symmetry of pupils. It should be expected that blindness is very common post-arrest, and it can be temporary or permanent.

There are other body systems that need to be monitored in this post-arrest period, including the renal and gastrointestinal systems, as well as keeping an eye out for coagulation disorders.

Keeping a patient from re-arresting can be challenging and will require close monitoring and excellent nursing care. There are some good prognostic indicators that can be watched for. These

include blood gas values and pulmonary function starting to improve, lactate, acid base, and electrolyte values returning to normal, cardiac arrhythmias resolving, coagulation parameters normalizing, normal urinary output, and return of normal neurologic function.

In summary, we should not be afraid of the “undead”, but be vigilant, use all your knowledge, powers of observation, and nursing skills to keep our post-arrest patients alive, and do our best to prevent them from arresting again.

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Main Stream, Saturday 6 June 2026

EXPOSURE OF DOGS AND CATS TO DRUGS OF ABUSE

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Learning objectives:

- Gain an appreciation for the types of drugs of abuse (DA) intoxications, other than THC, likely to be presented to small animal emergency clinics
- Appreciate the different exposure scenarios as determined from a retrospective review of confirmed DA intoxications
- Understand the clinical presentations and diagnostic approaches for each class of drugs of abuse
- Understand the difference between screening vs. confirmatory testing in drugs of abuse cases

Proceeding:

While known animal exposures to human "drugs of abuse" (DA) have been considered relatively uncommon in veterinary medicine, the trends are changing, and marijuana and amphetamines are among the 20 toxicants most frequently consulted about with the Pet Poison Helpline in the United States (US). Veterinarians need to be well-versed in the most frequently encountered DA, the potential clinical courses these exposures can take, and appropriate therapeutic approaches. Additionally, many DA "street" drugs are not pure and may consist of combinations of substances, making the clinical assessment more complex. Confirmed cases of DA exposure over a 10-year period submitted to the California veterinary diagnostic laboratory were reviewed. Fifty-seven samples tested positive for DA: 53 were from dogs and 4 were from cats. While this would seem to be a relatively small number of positive cases, it is important to keep in mind that confirmatory testing is not widely available and testing is often not performed on suspect cases due to cost considerations or patient recover. The most common drugs in confirmed exposure cases were methamphetamine and amphetamine followed by cocaine, fentanyl, morphine, THC, heroin, 3,4-methlylendioxy-methamphetamine (MDMA), and LSD. In about half of the cases, multiple drugs were found in tested samples.

Amphetamines

Methamphetamine, amphetamine, MDMA, and other designer amphetamines collectively belong to the group of drugs known as amphetamine-type stimulants (ATS). Amphetamines are part of a class of psychotropic drugs initially developed for human use in the treatment of conditions such as attention deficit hyperactivity disorder (ADHD) and narcolepsy. Methamphetamine currently ranks as the second most widely abused drug worldwide and ATS have become the most popular illegal psychostimulants in the world. The most common clinical signs reported for amphetamine

intoxications include seizures, agitation, hyper-reactivity, tremors, ataxia, circling, mydriasis, tachypnea, tachycardia, and hyperthermia, all consistent with sympathomimetic effects.

Cocaine

Cocaine is the natural alkaloid found in the leaves of the coca plant (*Erythoxylum coca* and *E. mongynum*). Although cocaine has legitimate medical uses, it is one of the most commonly abused drugs globally and is reported to be in the top five illicit substance exposures reported to animal poison control centers in the US. Similar to amphetamines, cocaine has a strong sympathomimetic effect with clinical signs consistent with nervous system stimulation: hyperactivity, hyperesthesia, tremors, seizures, tachycardia, hypertension, and hyperthermia.

Opioids

Interestingly, given the human opioid (e.g., fentanyl and heroin) abuse epidemic in the US, relatively few cases were confirmed in pets. The opioid drug class includes more than 25 different compounds. Opioids can be opioid receptor full agonists, partial agonists, full antagonists, or agonists/ antagonists. Cats and dogs can present with different clinical signs when exposed to the same compound due to different opioid receptor locations within the CNS. In dogs, clinical signs include early CNS excitation followed by lethargy, ataxia, stupor, seizures, coma and death. Death is often attributed to respiratory depression and hypoxia. Aggression, excitation, seizures or depression, and variable respiratory effects are reported in cats.

Case Management

In general, case management should focus on prevention and/or controlling life-threatening central nervous system and cardiovascular signs. This might include early decontamination such as emesis and activated charcoal, hospitalization for intravenous fluid treatment, and addressing symptomatic signs. There are no antidotes for many DA. However, for opioids, naloxone, an opioid antagonist, can be used to reverse CNS and respiratory depression.

Confirmatory Testing

Point-of-care (POC) on-site urine multidrug tests or UDST are often used in clinical settings to diagnose DA exposures/intoxications. POC exhibit a reasonable correlation with confirmatory mass spectrometry methods. However, the limited number of reported cases is insufficient for making a precise assessment of false positive or negative rates. In cases where POC UDST is used, the diagnosis should be supported by a history, when available, and clinical examination findings consistent with drug exposure.

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EFFECTIVE USE OF VETERINARY TOXICOLOGY LABORATORIES TO DIAGNOSE SUSPECTED BUT UNKNOWN INTOXICATIONS

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Learning objectives:

- Be able to formulate a diagnostic plan for cases of suspected intoxication from an unknown toxicant
- Understand what non-targeted toxicology testing is and its strengths and limitations
- Appreciate the need for obtaining correct biological samples in the proper amounts for toxicologic testing
- Appreciate reasons why toxicologic testing is useful in situations in which case outcomes are unaffected

Proceeding:

Animals are often presented to the veterinarian with a variety of clinical signs and a complaint by the owner that the animal was “poisoned.” In reality, the incidence of true poisoning in animals is low. Diagnosis of intoxication generally depends on the consistency of several diagnostic criteria including the history, clinical signs, clinical laboratory evaluation, postmortem lesions in the case of deaths and toxicologic testing of appropriate samples. Unfortunately, in many cases, a history of exposure to a particular toxicant is not available and in the case of finding an animal dead, no clinical signs are observed.

Testing for Unknowns

There is a general impression that if there is no history of exposure to a specific toxicant toxicology testing is not possible. However, advances in analytical testing platforms permit broad screening of samples for a variety of toxicant classes; this screening approach is termed non-targeted testing. The use of powerful screening tools for both organic and inorganic toxicants is a viable option in many cases of suspected exposure to an unknown chemical. Non-targeted testing for organic toxicants most often involves the use of mass spectrometry (MS). Mass spectrometry coupled with gas or liquid chromatography is a highly effective qualitative and quantitative analytical technique used to identify a wide range of toxicologically relevant compounds. Non-targeted testing using MS involves three broad steps: isolation of compounds from biological samples, separation of chemicals using gas or liquid chromatography and ionization of each separated chemical into fragments (mass spectra or “fingerprints”) which are unique to given chemical. Mass spectra are then compared to libraries of spectra with the goal of identifying what the unknown chemical is. For metals, there are several techniques that allow for the identification of dozens of metals in a sample with a single analysis. The limitations to non-targeted testing include the inability of some chemicals to be detected by the methods used and the unknown sensitivity of the testing approach for a given chemical.

Sample Selection

Depending on the specifics of a case, antemortem testing either gastric contents (vomit), urine or serum/plasma can be useful. Stomach contents are often the sample of choice in cases of acute onset of clinical signs, while urine can be a useful sample if clinical signs have been present from several hours up to one or two days postexposure. Appropriate postmortem samples for analysis include stomach contents, urine, liver, kidney, brain and fat. To be as thorough as possible, it is often recommended to test more than one sample, although costs associated with testing might restrict analysis to only one. Thus, the testing of the most appropriate sample is a critical choice to be made. In such a situation, a toxicologist can assist with the decision. Sample size can also be a factor in obtaining useful results; test sensitivity generally declines as the size of samples decreases and small samples can preclude additional testing if initial testing is negative. In many cases, toxicological testing takes some time and therefore should not be counted on to help direct early case management. Despite this limitation, test results can sometimes be available within 24 hours.

Is There a Need for Toxicology Testing?

In cases of suspected intoxication, toxicology testing is often not pursued, particularly when an animal is treated symptomatically and supportively and makes a full recovery. Additionally, the cost of testing might be prohibitive. However, in cases of suspected intoxication, toxicologic testing (negative or positive) can help relieve client anxiety, identify sources of exposure that might put other animals at risk, and be crucial in cases where subsequent litigation is a possibility.

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KEYNOTE LECTURE: CRITICAL TRANSFORMATION FOR CRITICAL CARE – HOW WE CAN USE ROUTINELY COLLECTED DATA TO IMPROVE PATIENT OUTCOMES

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Learning objectives:

- Uses of routinely collected healthcare data in human research
- Uses of routinely collected healthcare data in quality improvement
- How can we use the data around us effectively

Proceeding:

In this lecture, I will explore the expanding opportunities for discovery science that arise from the systematic use of clinical data. I will begin by framing clinical datasets, particularly electronic health records, biobank linkages, imaging repositories, and longitudinal monitoring streams, as a fundamentally new empirical substrate for hypothesis generation and mechanistic investigation. Rather than viewing these data as merely operational or administrative by-products of care delivery, I will argue that they will increasingly function as core scientific instruments.

I will first discuss how phenotyping will enable the discovery of previously unrecognised disease subtypes. By integrating laboratory measurements, diagnostic imaging, prescribing patterns, and narrative clinical documentation, I will demonstrate how latent structure in routine care data will reveal heterogeneous disease trajectories. I will emphasise that temporal modelling approaches will allow us to identify prodromal signals and transition states that precede overt diagnosis, thereby opening new windows for early intervention research.

A further section of the lecture will examine how clinical data will transform drug discovery and repurposing science. I will describe how naturally occurring treatment variation across health systems will create quasi-experimental conditions that I will be able to analyse using modern causal inference techniques. These approaches will allow me to detect off-target therapeutic effects, characterise heterogeneity in treatment response, and identify safety signals that may suggest new biological mechanisms.

I will also consider opportunities in biomarker discovery. By applying machine learning methods to multimodal clinical inputs such as electrocardiograms, radiological imaging, and wearable sensor streams, I will illustrate how latent physiological signatures will be extracted at scale. These signals will support the development of dynamic risk prediction tools and surrogate endpoints suitable for both observational and interventional research.

Importantly, I will devote attention to population-level discovery. Clinical datasets will enable me to quantify health disparities, investigate structural determinants of disease, and examine how environmental and socioeconomic exposures will interact with biological risk. I will suggest that linking clinical records to geospatial and policy datasets will generate new forms of systems-level epidemiology.

Finally, I will conclude by outlining the concept of the learning health system. I will argue that future healthcare environments will operate as continuous discovery platforms in which clinical practice will generate data, data will generate hypotheses, and those hypotheses will feed back into improved care delivery. Through advances in data harmonisation, privacy-preserving analytics, and federated research infrastructures, I will propose that clinical data will become one of the most powerful engines of translational discovery science in the coming decades.

RESEARCH GRANT WINNER 2024: POPULATION PHARMACOKINETICS OF INTRAVENOUS AMPICILLIN IN DOGS IN THE POLYURIC PHASE OF ACUTE KIDNEY INJURY

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Background:

Antimicrobial dosing in critically ill patients with acute kidney injury (AKI) is challenging. During the oligoanuric phase, reduced renal clearance leads to drug accumulation and potential toxicity. Conversely, the polyuric phase is commonly assumed to restore or augment renal clearance, raising concerns about subtherapeutic antibiotic exposure — a concept known as augmented renal clearance (ARC). Ampicillin, a hydrophilic beta-lactam excreted primarily by the kidneys, is a first-line treatment for leptospirosis in dogs. Pharmacokinetic data during the polyuric phase of AKI remain scarce in veterinary critical care.

Hypothesis/Objectives:

We aimed to characterize ampicillin pharmacokinetics during the polyuric phase of AKI and hypothesized that polyuria increases clearance, leading to reduced drug exposure and potential underdosing.

Methods:

A prospective population pharmacokinetic study was conducted in client-owned dogs (n = 12) with leptospirosis-associated AKI in the polyuric phase (urine output >4 mL/kg/h for ≥24 h, IRIS AKI stage ≥4). All dogs received IV ampicillin/sulbactam 22 mg/kg q8h via infusion pump. Serial plasma ampicillin concentrations were measured by HPLC-MS/MS. Data were analyzed using nonlinear mixed-effects modeling. Monte Carlo simulations were performed to evaluate target attainment for several dosing regimens.

Results:

Median creatinine at inclusion was 962.5 µmol/L; median urine output was 12 mL/kg/h. Ampicillin pharmacokinetics were best described by a two-compartment model with first-order elimination. Clearance was markedly reduced (median 0.060 L/kg/h, range 0.013–0.260) compared to previously reported values in healthy dogs (~0.655 L/kg/h) and critically ill non-azotemic dogs (~0.420 L/kg/h). Elimination half-life was substantially prolonged (median 273 min, range 75–1243 min), with substantial interindividual variability. Monte Carlo simulations demonstrated sustained exposure above MIC targets for both Leptospira spp. (MIC = 0.5 mg/L) and E. coli (MIC = 2–8 mg/L) throughout the dosing interval. A reduced regimen of 10 mg/kg q24h was sufficient to attain target therapeutic exposure in this population.

Conclusion:

These preliminary results show ampicillin clearance remains markedly reduced in dogs with AKI even during the polyuric phase, indicating that polyuria does not reflect restoration of glomerular filtration nor tubular secretion. Standard dosing regimens (22 mg/kg q8h) can result in significant drug overexposure in this population. These findings have direct implications for antimicrobial stewardship in veterinary critical care.

RESEARCH GRANT WINNER 2026: THE EFFECT OF ERYTHROMYCIN AS A GASTROINTESTINAL PROKINETIC ON THE FAECAL MICROBIOME AND METABOLOME IN HOSPITALISED DOGS

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Introduction:
Erythromycin is a macrolide antibiotic that also acts as a motilin receptor agonist to stimulate gastrointestinal motility. No studies have investigated the potential adverse effects of using an antibiotic as a prokinetic drug, including the development of dysbiosis in the patient, or by promoting antimicrobial resistance more broadly.

Objectives:
The objective of the proposed study is investigate the effects of prokinetic erythromycin on the faecal microbiome in hospitalised dogs. We hypothesise that while dysbiosis index (DI) and the proportions of primary and secondary bile acids will change over time in hospitalised dogs, there will be no difference between dogs receiving erythromycin and those not receiving erythromycin as a prokinetic.

Methods:
The proposed study is a pragmatic, prospective, observational, cohort study. All dogs hospitalised with a primary veterinarian in the Emergency and Critical Care (ECC) departments of two referral hospitals that are expected to be hospitalised for >24 hours will be eligible for enrolment with informed owner consent. Dogs prescribed macrolides for non-prokinetic (i.e. antimicrobial) indications and dogs prescribed lincosamide antimicrobials (eg. clindamycin) for any indication will be excluded. Treatment with erythromycin will not be directed by the study, but rather at the discretion of the treating veterinarian. Thus, enrolled dogs may be treated with erythromycin during hospitalisation (experimental group) or not (control group), but individual group allocation will not be known until the time of discharge sample collection. Based on a previous study by our group, we expect that approximately 50% of dogs hospitalised by the ECC service are treated with erythromycin during their hospital stay, so we expect relatively even group allocation. Data will be collected on the use of erythromycin as a prokinetic (yes/no) and dose, frequency, and duration of therapy.

Faecal samples will be collected hospital admission, and again at discharge from the ECC service, and frozen at -80C. Batched faecal samples from 25 dogs treated with erythromycin and 25 dogs in the control group will be sent to Texas A&M Gastrointestinal Laboratory on dry ice. Quantitative polymerase chain reaction (qPCR) assays will be used to measure the abundance of 16 core bacterial taxa, from which the DI derived. A gas chromatography-mass spectrometry (GC-MS) quantitative assay will be performed to assess concentrations of 29 targeted faecal metabolites including long-chain fatty acids, zoosterols, phytosterols, and unconjugated bile acids. Results of faecal metabolite quantification will be reported as nanograms per milligram of dry faecal matter, and the percentages of primary and secondary bile acids relative to the total unconjugated bile acids. pPCR results for individual bacterial taxa, the DI index, bile acid concentrations, and bile acid proportions will be

compared at admission and discharge within and between erythromycin versus no-erythromycin groups.

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CHALLENGING SHOCK CASES

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Learning objectives:

- Understand interacting mechanisms of shock in clinical cases
- Describe a rational approach to the clinical recognition of shock
- Prescribe appropriate shock treatments

Proceeding:

Shock refers to a systemic failure of cellular energy generation. Circulatory shock is caused by inadequate oxygen delivery to meet oxygen consumption needs. Shock causes substantial cellular dysfunction and can be rapidly fatal if not identified and appropriately treated. Major factors determining oxygen delivery into the arterial circulation are the oxygen content of the blood and the cardiac output. Additionally, for blood to perfuse tissue capillaries, adequate arterial blood pressure is required. Mean arterial pressure is affected by cardiac output and peripheral vascular resistance. Thus, shock develops when there is inadequate oxygenation of the blood, reduced cardiac output, or pathologic vasodilation. Cardiac output relies upon an adequate heart rate, sufficient venous return (preload), lack of an impediment to outflow (afterload), and sufficient contractility. Clinically, the major phenotypic categories of shock are vasoconstrictive and vasodilatory. These refer to patterns of change in six key perfusion parameters: mentation, heart rate, pulse quality, mucous membrane colour, capillary refill time (CRT), and peripheral temperature. In both categories of shock, mentation will become progressively decreased (obtundation through to coma). Tachycardia is common with both categories of shock in dogs. Severe shock in dogs, and shock of varying severity in cats, may be associated with bradycardia. Vasoconstrictive shock is typically associated with decreased pulse quality, pale mucous membranes, a prolonged CRT, and cool peripheries. Vasodilatory shock is typically associated with bounding pulse quality, hyperemic mucous membranes, a rapid CRT, and warm peripheries. Four categories of shock are considered based on underlying pathophysiology. Three are vasoconstrictive: hypovolemic, obstructive, and cardiogenic shock. Distributive shock is vasodilatory. Hypovolemic shock is caused by a reduced circulating blood volume, reducing preload and therefore cardiac output. Obstructive shock is due to an obstruction to venous return (or less commonly arterial outflow) that reduces cardiac output. Cardiogenic shock involves decreased cardiac output due to intrinsic cardiac failure, resulting in reduced contractility and/or abnormal heart rate. Distributive shock is caused by pathologic vasodilation, as in sepsis. Complex cases of shock may involve multiple mechanisms. Careful assessment of perfusion parameters and an understanding of pathophysiology can aid in a rational approach to these cases.

Anaphylactic shock

Anaphylaxis is a life-threatening acute systemic type 1 hypersensitivity reaction that results in multiple organ dysfunction. Anaphylactic shock is typically described as vasodilatory in the human literature. However, vasoconstrictive shock, shock with features of both vasoconstriction and vasodilation, or hypotension alone are reported in dogs with anaphylaxis. This is due to the combination of arterial vasodilation (maldistributive shock), decreased hepatic venous drainage (obstructive shock), and fluid losses into the gastrointestinal tract or secondary to spontaneous hemoperitoneum (hypovolemic shock). Treatment considerations include intravascular volume expansion with IV fluids, vasopressor agents (notably epinephrine), as well as the role of antihistamines.

Shock in gastric dilatation and volvulus (GDV)

Dogs with GDV often also present with vasoconstrictive shock due to multiple mechanisms. Classically GDV is thought to result in obstructive shock due to the markedly dilated gas filled stomach resulting in increased abdominal pressure +/- direct compression of the caudal vena cava that impedes venous return to the heart. It is also likely that dogs with GDV have some degree of hypovolemia associated with blood loss from rupture of the short gastric arteries, fluid loss of into the gastrointestinal tract, sequestration of blood in the spleen, and inability to drink to replace lost fluid. Cardiogenic shock also occurs in a subset due to malignant tachyarrhythmias, and potentially myocardial dysfunction. Compounding assessment of these cases is that lactate production and hyperlactatemia will result not only from global hypoperfusion due to shock, but also the gastric necrosis that occurs in GDV. Treatment includes intravascular volume expansion with IV fluids, resolving the obstructive component by gastric decompression, and addressing any other specific abnormalities, such as the use of anti-arrhythmics.

Septic shock

Sepsis is life-threatening organ dysfunction caused by a dysregulated host response to illness. Septic shock is a subset of sepsis where profound circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality. Pathologic vasodilation results in hypotension due to inadequate peripheral vascular resistance. Typically, this is identified when shock persists despite initial fluid resuscitation. The appropriate volume and type of intravenous fluid to administer is challenging to determine, due to variability in fluid responsiveness and the degree of true hypovolemia in this patient cohort. Whilst catecholamine vasopressors are the next-line therapy, they may be ineffective due to critical illness-related corticosteroid insufficiency. Some septic patients also develop cardiogenic shock due to myocardial dysfunction, which requires inotropic therapy to maintain cardiac output.

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EUGLYCEMIC DKA - CASE BASED MANAGEMENT STRATEGIES

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Learning objectives:

- Define euglycemic diabetic ketoacidosis and explain how it differs from traditional DKA
- Recognize key diagnostic features of eDKA, including the limitations of blood glucose testing
- Explain the pathophysiologic role of SGLT2 inhibitors in promoting ketosis despite euglycemia
- Describe the rationale for fixed-dose regular insulin CRI paired with early dextrose supplementation in eDKA
- Apply a safe, practical ER management strategy for eDKA while acknowledging current evidence limitations

Proceeding:

Euglycemic diabetic ketoacidosis (eDKA) is an increasingly recognized, life-threatening complication in cats treated with feline SGLT2 inhibitors such as bexagliflozin or velagliflozin, among others. In contrast to "classic" DKA, eDKA can present with normal or only mildly increased blood glucose despite clinically significant ketosis and metabolic acidosis, which creates a major ER pitfall: clinicians may falsely down-rank the severity of illness because they anchor on normoglycemia. The core premise of this session is that ketosis + acidosis = a medical emergency regardless of blood glucose levels, and that the treatment priorities are to (1) stop ketogenesis with insulin, (2) do so safely by pairing insulin with early dextrose support, and (3) aggressively prevent iatrogenic complications—especially electrolyte shifts—while acknowledging that veterinary evidence is still evolving and many recommendations are extrapolated.

1) **Defining eDKA and how it differs from "traditional" DKA**

In general, a diagnosis of eDKA can be relatively straightforward when combining the following factors: SGLT2 inhibitor exposure plus (a) ketosis and (b) high anion gap metabolic acidosis, with a blood glucose that is normal to mildly elevated (often <250 mg/dL in human-based definitions). Euglycemic DKA may be considered "nonhyperglycemic" DKA in cats with SGLT2 inhibitor history, increased anion gap metabolic acidosis, and ketonemia/ketonuria in a sick patient. Since the blood glucose concentrations may be normal (or even occasionally low) despite substantial ketonemia, delayed diagnosis is a real risk.

2) **Why SGLT2 inhibitors create this phenotype: glucose off-loading + relative insulin deficiency**

SGLT2 inhibitors reduce blood glucose by blocking renal glucose reabsorption, increasing urinary glucose loss. Approximately ~90% of glucose uptake occurs in the early proximal tubule via the sodium glucose cotransporters, and inhibition decreases renal glucose reabsorption and increases glucose excretion—so a cat can be clinically ill and ketotic while still appearing "euglycemic" on spot glucose checks.

It is important to remember that cats require residual β-cell function (endogenous insulin) to prevent ketosis; when insulin becomes inadequate (or the insulin:glucagon ratio shifts), unchecked lipolysis and ketone generation can occur even while glucose is being "handled" by renal dumping. Interestingly, eDKA in SGLT2-treated cats is often detected within 2 weeks of starting therapy, and frequent monitoring for betahydroxybutyrate levels early in therapy can aid in timely detection.

3) **Diagnosis: don't let glucose "falsely reassure" you**

A safe, practical approach to these patients in the ER hinges on early ketosis recognition, avoidance of anchoring on normal glucose, and centering diagnostics around: betahydroxybutyrate measurement + the use of a venous blood gas to confirm metabolic acidosis.

What to measure in the ER:

- Blood β-hydroxybutyrate (BHB) (preferred ketone metric): it is strongly advocated to preferentially test blood BHB because it is more sensitive than urine dipsticks (dipsticks detect acetoacetate and can miss clinically significant BHB-dominant ketosis), and because BHB can be reliably trended. While ketone meters measure β-hydroxybutyrate, values must still be interpreted in context of the patient's clinical status and blood gas values. Urine glucose/ketones may confuse the picture in SGLT2 cats—glucosuria is expected from the drug, and urine ketones can be falsely negative or underrepresent severity. This is also why blood BHB is a better diagnostic anchor than urine dipsticks in suspected eDKA
- Venous blood gas (pH, HCO3−/base excess) to confirm metabolic acidosis
- Electrolytes (especially potassium and phosphorus)
- Blood glucose (useful, but not decisive), why BHB is the key ketosis test

To summarize: clinical criteria for diagnosis of eDKA include: glucose <250 mg/dL + acidosis (pH <7.3 or HCO3− <18) + ketosis (preferably BHB >2.4 mmol/L).

4) **Case-based application:**

The case discussed during this talk is used to demonstrate the exact diagnostic trap eDKA creates: the cat was started on an SGLT2 inhibitor, became progressively lethargic with reduced appetite, and had normal-range glucose on chemistry while showing marked ketonemia on a ketone meter and required escalation of care.The ER-level "lesson" is not that every SGLT2 cat will do this—it is that history + BHB + blood gas is how you prevent a miss.

5) **Treatment priorities: what you do first, and why**

Core principle: insulin is required to stop ketogenesis. In patients with DKA, insulin is crucial to stop ketone production—this is the first priority; glucose control is secondary once ketogenesis is reversed. Treatment of eDKA is similar to hyperglycemic DKA, except for the crucial difference: immediate dextrose with insulin to allow insulin therapy despite lower glucose levels.

Fixed-dose regular insulin CRI + proactive dextrose: when the traditional sliding scale may not work! A specific emphasis of this session is on the use of fixed-dose regular insulin CRI paired with early dextrose. A fixed-rate CRI delivers insulin at a set dose while dextrose supplementation is adjusted to maintain safe BG; this approach is the ideal method for eDKA because these cats are normoglycemic/mildly hyperglycemic and still need substantial insulin, so aggressive dextrose supplementation is necessary to allow these patients to tolerate high insulin doses without risking

life-threatening hypoglycemia. While ideal evidence-based insulin recommendations are lacking in veterinary patients with eDKA, current anecdotal and expert recommendations include: dextrose bolus prior to starting insulin, then regular insulin ~0.1 U/kg/hr (CRI preferred), with a dextrose infusion started around 0.25–0.3 g/kg/hr and BG checks q2h with adjustments.

- Why early dextrose matters: It allows you to administer enough insulin to halt ketogenesis without causing hypoglycemia—especially because the drug may continue to drive glycosuria for days after discontinuation.

Fluids + electrolyte resuscitation

Aggressive electrolyte supplementation may be necessary in these patients, similar to traditional DKA—especially potassium and phosphorus— and is driven by the physiology of total body depletion with extracellular shifts during acidemia, then rapid intracellular shifts once insulin starts.

A key operational safety point: consider starting insulin CRI once the patient is euvolemic and potassium is roughly ≥3.0 mEq/L and be prepared for high dextrose needs—often requiring central line access for safe administration.

Stop the inciting drug

A key emphasis here is the immediate discontinuation of SGLT2 inhibitors in any cat with acute lethargy/hyporexia/dehydration/weight loss and assessment for DKA regardless of blood glucose levels. Typically, once a cat develops eDKA, they will require ongoing insulin therapy.

6) Evidence gaps: what we know, and what we're extrapolating

It is important to acknowledge that the veterinary evidence base is limited and protocols are extrapolated from expert recommendations, case series, and human literature. More data are needed to clarify optimal treatment protocols for cats with euglycemic DKA, to supplement this practical guidance (insulin + immediate dextrose, monitoring frequency, electrolyte vigilance).

Ultimately when treating these cats, any protocol should be structured and reproducible (to reduce variation and improve safety), but teams must remain adaptive—especially around dextrose needs, potassium/phosphorus supplementation needs, comorbid drivers (pancreatitis, infection, hyporexia), and owner constraints.

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Advanced Stream, Saturday 6 June 2026

MACROCIRCULATION AND MICROCIRCULATION - PHYSIOLOGY, ASSESSMENT, AND TREATMENTS

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Learning objectives:

- Explain the physiology of the macro- and microcirculation.
- Analyze the regulatory mechanisms that control blood flow.
- Use clinical tools to assess macro- and microcirculatory blood flow.
- Identify common microcirculatory derangements.
- Integrate the information for application to clinical patients.

Proceeding:

Introduction

When cellular energy production fails to meet energy demands, a state of shock ensues. Hypoperfusion is one of the most common causes of insufficient oxygen delivery to the tissues; therefore, the veterinarian's goal when treating patients with evidence of poor perfusion is to normalize global oxygen delivery. The clinical parameters frequently used to monitor patients with evidence of shock include heart rate, pulse quality, blood pressure, mucous membrane color, capillary refill time, extremity temperature, mentation, blood lactate, +/- central venous oxygen saturation, and point-of-care ultrasound findings. However, the heterogenous distribution of capillary blood flow and subsequent regional hypoxia that frequently occurs in shock states is difficult to assess with these more global macrocirculatory parameters. Subsequently, some critically ill patients develop multiple organ dysfunction syndrome (MODS), multiple organ failure (MOF), and subsequent death, despite rapid recognition and aggressive treatment for shock.

The microcirculation is most likely the first organ to fail in patients with MODS and MOF due to both infectious and non-infectious causes. The multitude of pathogenic factors that occur in patients with critical illness affect virtually every cellular component of the microcirculation. In humans, microcirculatory distress that persists for at least 24 hours was found to be a single independent predictor of patient outcome. Novel technologies are now enabling clinicians to visualize and assess the microcirculation, subsequently diagnosing microcirculatory dysfunction, formulating more accurate prognoses, and treating patients more effectively to improve outcomes in severely ill patients.

The Microcirculation

The microcirculation consists of vessels less than 100µm in diameter and includes arterioles, capillaries, and venules. It contains the biggest endothelial surface within the body (>0.5 km^2), and 10 billion capillaries (diameter 5-9 µm) are responsible for transporting oxygen and nutrients to and removing waste products from all cells of the body, ensuring adequate immunological function, and delivering therapeutic drugs to target cells in disease states. The microcirculation is comprised of

endothelial cells lining the inner walls of microvessels, smooth muscle cells (mostly in arterioles), red blood cells, leukocytes, and plasma components. The main determinants of flow within the capillaries include the driving pressure, arteriolar tone, hemorheology, and capillary lumen patency.

The vascular endothelial cells are covered and protected by the endothelial glycocalyx, a surface layer that plays a key role in regulating blood flow and preserving microcirculatory integrity (a permeability barrier) and function. Estimates of glycocalyx integrity are currently calculated using videomicroscopic software that measures the available width of the glycocalyx-free space in the vessel by tracking the movement of red blood cells. The calculated value is known as the perfused boundary region (PBR), and an increase in PBR indicates damage to the endothelial glycocalyx layer, which allows red blood cells to move within a wider vessel lumen.

Monitoring the Microcirculation

Several different invasive and noninvasive techniques can be used to assess microcirculatory flow and the adequacy of tissue oxygenation in critically ill patients. Microcirculatory dysfunction occurs secondary to many different disease processes. Damage to the microcirculation has been documented in patients with infectious or non-infectious causes of severe inflammatory diseases, various shock states (e.g., cardiogenic shock), diabetes, hypertension, and cardiac bypass. Evaluating the effects of different therapeutic strategies on both macro- and microcirculatory parameters is a current area of research.

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WHAT'S THE RELEVENCE OF ANAEMIA IN CRITICAL ILLNESS?

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Learning objectives:

- Understand the physiology of hematopoiesis
- Understand the frequency, and physiology, of anemia in critical illness
- What can we do to manage/address anemia in critical illness?

Proceeding:

Overview erythropoiesis and iron metabolism

The committed erythrocyte producing stem cells (CFU-E) in the bone marrow respond to growth inducers (e.g interleukin-3) and differentiation inducers to give rise to red blood cells (RBC). The cells in the erythrocyte series are as follows in chronological order:

- *Proerythroblast:* condensation of chromatin starts here via modification of histone tails
- *Basophil erythroblasts*
- *Polychromatophil erythroblast*- synthesis of hemoglobin begins at this stage
- *Orthochromatic erythroblast*
- *Reticulocyte*: Nucleus has been condensed and extruded and endoplasmic reticulum reabsorbed. Contains a small amount of basophilic organelles (golgi apparatus, mitochondria, ribosomes for hemoglobin synthesis) that disappear after 1-2 days, with the cell becoming mature erythrocytes. Usually <1% of RBC population as they have a short life span
- *Erythrocytes*

Tissue hypoxia from anemia, due to decreased oxygen delivery, stimulates release of erythropoietin (EPO) from the kidneys and to a lesser degree from the liver. Renal hypoxia increases the transcription factor hypoxia-inducible factor 1 (HIF-1), which binds to the hypoxia response element in the erythropoietin gene, and induces transcription of mRNA for EPO. Other stimulants of EPO production include norepinephrine, epinephrine and prostaglandin. EPO production peaks by 4 days and binds to EPO receptors on erythroid cell precursors of the bone marrow.

Actions of EPO include:

- Direct stimulation of hematopoietic stem cells to produce proerythroblasts
- Hastens maturation of proerythroblasts to the next stages
- Prevents apoptosis of erythroid progenitors
- Inhibits expression of hepcidin in the liver

Vitamin B12 and folic acid are important in the final maturation of RBCs, and play a role in DNA synthesis, nuclear maturation and cell division in the erythroblast phase. Chronic deficiency leads to a maturation failure anemia.

Heme is formed when protoporphyrin IX (made up of 4 pyrroles) and iron combine. Each heme molecule then combines with globin, to form a single hemoglobin subunit, and 4 of these subunits bind to form the hemoglobin molecule that we know of. Each hemoglobin molecule contains 4 iron atoms, and each atom binds loosely to one molecule of oxygen.

65% of total body iron is in the form of hemoglobin, and 15-30% is stored in the reticuloendothelial system and liver.

- Non-heme/dietary iron absorbed from the duodenum combines with apotransferrin (a type of globulin) to form transferrin (iron transport protein) and is transported to tissues
- In cells, iron combines with apoferritin (protein), to form ferritin. Ferritin is the storage form of iron
- Transferrin binds strongly to cell membranes of erythroblasts, and is endocytosed and delivered to the mitochondria where heme is synthesized
- Hemoglobin released from senescent RBCs are ingested by macrophages of the reticuloendothelial system (Liver Kupffer cells, spleen, and bone marrow). This is the main source of iron for continued RBC production.
  (1) Hemoglobin is first split into globin and heme
  (2) The heme ring is opened to liberate iron and iron is transported as transferrin to the bone marrow for RBC production or stored in the liver as ferritin
  (3) The porphyrin ring is converted by macrophages to bilirubin (biliverdin --> free/unconjugated bilirubin--> conjugated with glucuronic acid or sulfate)
- Free heme in plasma is scavenged by albumin and haptoglobin and brought to the reticuloendothelial system

Iron homeostasis:

- Hepcidin is the key regulator and is produced by hepatocytes
- Ferroportin is a cellular exporter of iron in duodenal epithelial cells, macrophages, hepatocytes
- Hepcidin binding to ferroportin results in degradation of ferroportin, which then decreases iron availability due to inhibited intestinal iron uptake and inhibited release of cellular iron from macrophages and hepatocytes
- Erythroferrone (ERFE) is a protein secreted by RBCs in the marrow and is induced by EPO. As a negative feedback mechanism, ERFE directly decreases hepcidin expression in the liver

Clinical pathological evidence of erythropoiesis

- *Polychromatophils* - Blue staining with Diff-Quik due to ribosome presence, for hemoglobin production. Ribosomes (producing hemoglobin) stain as dark blue granules when stained with new methylene blue.
- *Reticulocytes (>60-65,000/uL)*

- *Nucleated red blood cells (erythroblasts):* includes basophillic, polychromatophilic and orthochromic metarubricytes. Sometimes these cells are misidentified, and counted, as lymphocytes on automated cell counts
- *Anisocytosis* - *V*ariation in RBC size due to the presence of the larger reticulocytes
- *Higher red blood cell distribution width* - Describes variation in RBC cell size (anisocytosis)
- *Macrocytic hypochromic anemia* - Due to larger reticulocytes which are also hypochromic as hemoglobin synthesis is not fully complete

Anemia of critical illness

Anemia of critical illness is frequent in humans, with almost 95% of patients becoming anemic on day 3 of hospitalization. In veterinary medicine, 43-56% of dogs and ~70% of cats become anemic during hospitalization. In humans, anemia of critical illness has been shown to persist in 45% of patients 12 months following discharge from the ICU; the veterinary percentage is not known.

While inflammation is a known culprit in the pathogenesis of anemia of critical illness in the ICU, there are many contributing causes:

1. Inflammatory response in critical illness leading to dysregulation of iron homeostasis (iron-restricted erythropoiesis)
2. Inhibited EPO production, and activity, due to the inflammatory response and reduced renal function in critical illness
3. In ICU patients, ERFE rapidly decreases due to low EPO and results in high levels of hepcidin. IL-6 also upregulates hepcidin. This leads to decreased iron absorption and mobilization.
4. Iatrogenic anemia from frequent phlebotomy, leading to hospital acquired anemia
5. Increased fragility of RBC due to inflammation and subsequent erythrophagocytosis by the reticuloendothelial system

Overall, ICU patients have low serum iron, low total iron binding capacity and low transferrin saturation, but increased serum ferritin.

Treating anemia of critical illness based on physiology

1. Focusing on anemia prevention and treating the cause of anemia, rather than just reducing the number of transfusions
2. Reducing phlebotomy volumes
3. Transfusions – Generally there is a restrictive transfusion trigger of hemoglobin at <7g/dL for most ICU patients, but considering a lower threshold of 9-10 g/dL for cardiac patients and traumatic brain injury
4. Intravenous iron therapy - Shown to increase hemoglobin concentration at discharge but not reduce transfusion requirements. The author currently uses Iron Dextran 10-20mg/kg intramuscularly in trauma patients with persistent anemias.
5. Oral iron - May be ineffective due to hindered absorption by other medications and increased hepcidin levels in ICU patients
6. Vitamin B_{12} supplementation - No literature in acute anemia
7. Erythropoietic stimulating agents (e.g. EPO) - There are conflicting opinions on this, with some use in combination with iron supplementation. Patients have improved hemoglobin concentrations at discharge, but no reduction in transfusion requirement has been noted. Use of

stimulating agents has been associated with increased thrombotic events when there is no prophylactic anticoagulation.

8. Modulating hepcidin with antagonists and stabilizers of ferroportin
9. Increasing HIF by inhibiting its degradation by HIF-prolyl hydroxylase

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SEPTIC SHOCK

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Learning objectives:

By the end of this session, participants will be able to:

- Define sepsis and septic shock in small animals using contemporary concepts adapted from Sepsis-3.
- Recognize the limitations of SIRS criteria and explain why organ dysfunction and perfusion assessment are central to diagnosis.
- Identify key diagnostic indicators of septic shock, including vasopressor-dependent hypotension and evidence of organ dysfunction.
- Describe core treatment strategies for septic shock, including antimicrobials, fluids, vasopressors, and source control.
- Discuss areas of controversy and limited evidence, including corticosteroid use and optimal resuscitation targets in veterinary patients.

Proceeding:

Sepsis and septic shock remain among the most common and lethal syndromes managed in small animal emergency and critical care, yet veterinary-specific definitions and high-quality treatment evidence are still limited. This session is designed to give participants a review of key literature and help provide a framework to move beyond the diagnostic pitfalls of SIRS-only thinking and recognize shock states earlier using organ dysfunction and perfusion assessment, as well as implement core, time-sensitive therapies (antimicrobials, fluids, vasopressors, and source control) while navigating persistent controversies and evidence gaps in veterinary ECC.

1) **Definitions**: sepsis vs septic shock

Our current understanding of veterinary sepsis aligns with the contemporary Sepsis-3 definitions in people that describe sepsis as not just “infection + inflammation,” but rather infection driving life-threatening organ dysfunction through a dysregulated host response. The human Sepsis-3 definition was developed to solve a longstanding problem: older definitions leaned heavily on physiologic inflammatory criteria (SIRS), which are common in many non-septic illnesses and sometimes absent in truly septic patients—making them an unreliable gatekeeper for life-threatening infection. Veterinary-specific sepsis definitions are forthcoming and will greatly shape our ever-evolving understanding of this complex and dynamic disease process.

Sepsis (core definition): Life-threatening organ dysfunction caused by a dysregulated host response to infection.

Septic shock (subset of sepsis): A subset where circulatory and cellular/metabolic abnormalities are profound enough to substantially increase mortality, typically manifesting clinically as persistent

hypotension requiring vasopressors despite adequate volume resuscitation, with evidence of metabolic/perfusion derangement (lactate is a commonly referenced marker in Sepsis-3 constructs).

As a by-product of the same conceptual shift: the term “severe sepsis” is increasingly considered redundant once sepsis is defined by organ dysfunction, not by SIRS severity.

Practical clinical implication: when you suspect infection, you should be asking, “Is there organ dysfunction and/or perfusion failure?” rather than “do they meet the SIRS criteria?” because that question may better predict what patients are in danger of decompensation.

2) Diagnosing sepsis: SIRS is a smoke alarm, not a diagnosis

Why SIRS alone is insufficient: temperature, heart rate, respiratory rate, and WBC changes can occur with infection or many non-infectious insults; likewise, septic patients may fail to mount classic inflammatory patterns due to immunosuppression, comorbidities, or medications. The SIRS criteria are neither sensitive nor specific, and while severity scores can help with illness assessment they should not be used as sole diagnostic criteria or prognostication tools.

What replaces SIRS-centered thinking: organ dysfunction + perfusion assessment

To operationalize “organ dysfunction,” veterinarians are urged to consider what they can see and measure at cage-side with their patients:

Mentation changes, hypoglycemia, hypotension, weak pulses, prolonged CRT, hypothermia—especially late-stage sepsis signs. Laboratory evidence of organ dysfunction (e.g., renal indices, hepatobiliary changes, coagulation abnormalities, hypoglycemia, hypoalbuminemia) and metabolic derangement such as lactate elevation. Recognition that microcirculatory failure can precede macro-hemodynamic collapse (“cryptic shock”), so a normal blood pressure does not equal adequate perfusion. Sepsis is organ dysfunction from infection; shock is ongoing perfusion failure—and early identification of these is vital to effective therapeutic management of these patients.

3) What septic shock looks like: Septic shock is defined as vasopressor-dependent hypotension and evidence of organ dysfunction, after adequate fluid resuscitation. This fits with the clinical construct that septic shock is sepsis plus persisting hypotension requiring vasopressors despite adequate volume resuscitation, and associated cellular/metabolic dysfunction.

Species nuances that are clinically relevant here include the key difference between canine and feline manifestations of sepsis. Dogs (and humans) more commonly show a hyperdynamic “warm shock” pattern early (hyperemic mucous membranes, rapid CRT, tachycardia), while cats often present differently with evidence of “cold shock” (bradycardia, hypotension, hypothermia), making pattern recognition and “shock suspicion” more challenging. This reinforces why relying on one pattern (e.g., “warm shock”) can be dangerous.

The “shock is not one thing” concept:

A key emphasis for clinical practice is the reality that while septic shock is a form of distributive shock, patient clinical presentations are frequently multifactorial (e.g., a dog with septic peritonitis may present with vasodilatory shock but may also have a component of hypovolemic shock from GI losses and third spacing; some patients may also have cardiogenic components depending on cause and myocardial dysfunction).

This concept is increasingly relevant as septic patients are dynamic in their clinical course, and may require varying degrees of support with IV fluids, vasopressors and inotropes depending on where they are in the continuum of sepsis.

4) Treatment strategies: "time-sensitive bundle" logic translated to veterinary reality. Key treatment objectives focus on four pillars: early antimicrobials, fluid therapy, vasopressors, and source control. Early recognition of sepsis facilitates targeted management starting immediately at presentation with rapid targeted resuscitation, early antimicrobials, and controlling the source.

Early antimicrobials: control the driver of the dysregulated host response. Early parenteral antimicrobials and source control are central; delays in appropriate therapy are strongly associated with worse outcomes in human septic shock, and veterinary studies suggest timely/appropriate empiric therapy can be feasible and may improve outcomes (with caveats and mixed results).

Fluid therapy: resuscitate, but don't confuse blood pressure with perfusion. Early volume resuscitation is critical, but assessing volume status and fluid responsiveness is challenging in veterinary patients, and static measures are not reliably predictive, which is where dynamic assessment (including POCUS) can help. The use of clear resuscitation endpoints focusing on organ function is preferred over the approach of "give fluids until the blood pressure is okay." A "normalizing" blood pressure can coexist with ongoing microcirculatory failure and cellular oxygen debt (cryptic shock), so a good septic shock resuscitation plan must include perfusion endpoints, not BP alone.

Vasopressors: when hypotension persists after adequate volume resuscitation. Vasopressors are typically indicated when hypotension persists after restoration of intravascular volume. A MAP target of ~65 mmHg is commonly recommended during/after initial resuscitation (human guideline basis), while acknowledging uncertainty and patient-specific adjustments (e.g., chronic hypertension). Norepinephrine is typically deployed as a first-line vasopressor in veterinary medicine (although clear evidence pointing to its superiority over other vasopressors is lacking in veterinary patients). Pragmatically, if a patient is euvolemic and remains hypotensive at high norepinephrine dosing, referral/advanced care escalation should be strongly considered.

Source control: the highest-leverage intervention when feasible. Source control as a central management priority alongside resuscitation and antimicrobials. This is variably feasible in veterinary patients in the context of "surgical sepsis" (septic peritonitis from GI perforation, urogenital sepsis, pyometra, or sepsis from contaminated wounds/abscesses). Decision making in veterinary patients is often multifactorial and complex and subject to many constraints (stability, cost, surgeon/staffing, referral availability).

Practical takeaway: In veterinary medicine, "source control" is often the point where ideal physiology meets reality. The session aims to help clinicians make disciplined choices that prioritize (1) stabilizing enough to survive intervention, while (2) not delaying definitive control until the patient's physiology collapses.

5) Controversies and evidence gaps

As our understanding of sepsis in small animals evolves, so do areas of ongoing controversies and limited evidence—especially corticosteroids and optimal resuscitation targets.

Corticosteroids: classically considered a means to support patients with critical illness related corticosteroid insufficiency (CIRCI), corticosteroids may be considered in vasopressor-refractory shock, but evidence is limited in veterinary patients—so they are not routine for "all sepsis." Physiologic corticosteroid supplementation may be considered as an "as indicated" supportive strategy within broader sepsis management.

Resuscitation endpoints and targets: Lactate measurement and lactate trends are commonly used as resuscitation elements, but also underscores ongoing debate around which endpoints best represent adequate perfusion and oxygen delivery. Ideally, resuscitation endpoints should be considered holistically as a multi-parameter set (mentation, pulse quality, mucous membranes, blood pressure, lactate, etc.), reinforcing that sepsis resuscitation is not "one number medicine."

6) Putting it together: a "cage-side algorithm (conceptual)

Suspect infection (confirmed or presumptive) using the whole clinical picture—not SIRS alone.
Identify organ dysfunction early (mentation, glucose, renal/hepatic/coagulation changes, lactate, urine output).
Assess perfusion and shock phenotype (macro + micro), remembering cryptic shock exists.
Treat immediately with bundled logic: early antimicrobials, fluids with reassessment of responsiveness, vasopressors if persistent hypotension after adequate volume, and source control when appropriate.
Name uncertainties explicitly (evidence gaps) and use structured decision-making when data are limited—especially around steroid use and targets.

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NON-CARDIOGENIC PULMONARY OEDEMA: PATHOPHYSIOLOGY AND TREATMENT

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Learning objectives:

- Describe normal endothelial fluid physiology.
- Compare the genesis of non-cardiogenic *versus* cardiogenic pulmonary oedema.
- Review specific scenarios leading to non-cardiogenic pulmonary oedema.
- Consider treatment options for non-cardiogenic pulmonary oedema.

Proceeding:

Oedema is the abnormal accumulation of fluid in tissues.[1] Non-cardiogenic pulmonary oedema (NCPE) is the accumulation of interstitial and/or alveolar fluid not primarily due to elevated capillary hydrostatic pressure secondary to elevated left atrial pressure, as is found in congestive heart failure or fluid overload.[1,2,3] The principle mechanism is increased endothelial permeability[2,3], though other factors may contribute, such as decreased lymphatic drainage of the interstitial space.[1,2] The important difference between NCPE and cardiogenic pulmonary oedema (CPE) is the status of the left atrial pressure and thus the pulmonary venous pressure.

Heart failure, which is the natural sequela of most cardiac diseases, is defined as the inability of the heart to meet the metabolic needs of the tissues without elevated filling pressures.[4] As the left heart pressure increases, this pressure is reflected backward into the pulmonary veins. As the pulmonary veins are downstream of the pulmonary capillaries, the pulmonary capillary hydrostatic pressure necessarily increases to maintain forward flow.[5] The elevated capillary hydrostatic pressure increases fluid efflux into the interstitial space leading to oedema formation. Interestingly, elevated left atrial pressure, via this same mechanism, is the cause of Group 2 pulmonary hypertension.[5]

In cases of NCPE, the left atrial pressure *is not elevated*.[1,2] The primary cause of increased fluid efflux into the interstitial space is *increased vascular permeability.*[2,3] Said differently, the *resistance to fluid extravasation decreases*, allowing increased flow from the pulmonary capillary into the interstitial space. Alternatively, fluid efflux can be driven by an increased transcapillary pressure gradient when the pressure within the capillary (capillary hydrostatic pressure) increases relative to the pressure in the pulmonary interstitium.[1] This occurs in some situations *without an increase in left atrial pressure;* thus, it is non-cardiogenic despite elevated capillary hydrostatic pressure.
We therefore see three subcategories of NCPE based on their pathophysiological mechanism:
1) increased permeability pulmonary oedema
2) increased transcapillary pressure gradient pulmonary edema (without left atrial hypertension)
3) combined increased permeability & pressure gradient-driven oedema.

Naturally, patients often do not fit neatly into one category. For example, it is not uncommon to encounter a patient with underlying cardiac disease *and* acute NCPE. This patient may already have elevated left atrial pressure or have decreased left atrial tolerance to fluid administration. Similarly,

iatrogenic fluid overload can increase both left atrial pressure and capillary hydrostatic pressure: fluid overloading a patient with increased permeability pulmonary oedema will worsen oedemagenesis. Fluid unloading (e.g. diuresis) a patient with NCPE and without fluid overload, however, may be detrimental as it can dangerously decrease cardiovascular filling pressures and volumes. It is therefore important to recognize the category of pulmonary oedema (NCPE vs CPE), consider the pathophysiological mechanism of the NCPE (increased permeability vs pressure-gradient driven vs combined), recognize comorbidities (i.e. cardiac disease, volume status and other causes of fluid intolerance) and to critically consider our goals in manipulating fluid status (i.e. fluid loading vs unloading).

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GI PROKINETICS IN THE ICU: DOES ERYTHROMYCIN BEAT METOCLOPRAMIDE?

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Learning objectives:

- Understand the mechanisms of action of metoclopramide and erythromycin
- Understand adverse effects of prokinetics
- Describe clinical evidence for the use of prokinetics in human and veterinary critical care
- Prescribe rational treatment plans for ICU cases with GI dysmotility

Proceeding:

Learning outcomes: Understand the mechanisms of action of metoclopramide and erythromycin, and understand adverse effects of prokinetics

Metoclopramide exerts dual antiemetic–prokinetic effects. Centrally, it antagonizes dopamine D_2 receptors (and weakly 5-HT_3 at higher concentrations) in the chemoreceptor trigger zone to reduce nausea and vomiting. Peripherally, it agonizes 5-HT_4 receptors, enhancing acetylcholine release from myenteric neurons, thereby increasing lower esophageal sphincter tone, antral contractility, and gastric emptying. These receptor actions explain both therapeutic benefit and dopaminergic adverse effects. Regarding adverse effects, tachyphylaxis is reported in humans and likely occurs in animals also. Metoclopramide undergoes renal excretion and so should be used cautiously in patients with reduced GFR. Extrapyramidal signs such as hyperactivity, hallucinations, and behavior change, which are reported in humans, have not been well characterized in dogs. Standard doses vary from 0.1 - 0.5 mg/kg IV, IM or PO q 6 - 12h. In a veterinary ICU setting, it is often used as a CRI at 1 - 3mg/kg/day.

Erythromycin acts as a motilin-receptor agonist, reproducing phase-III migrating motor complexes at low doses and generating strong antral contractions at higher doses—effects that accelerate gastric emptying. As with metoclopramide, tachyphylaxis develops over days, which can limit duration of effect. Early canine and human physiology work demonstrated erythromycin's motilin-mimetic activity and its cholinergic pathway dependence, forming the mechanistic basis for clinical use as a prokinetic. Prokinetic doses of erythromycin are much lower than antimicrobial doses and hence may not affect the patient's microbiome, but further investigation is warranted. Both drugs can also prolong the QT interval in humans.

Learning outcome: Describe clinical evidence for prokinetics in human and veterinary critical care

Prokinetic agents reduce enteral feeding intolerance and gastric residual volumes in human ICU patients, but do not improve mortality and have uncertain effects on ICU length of stay. A 2016 meta-analysis concluded that prokinetics improve tolerance to gastric feeding compared to placebo or no intervention but effects on patient centered outcomes such as development of pneumonia, ICU length of stay, and mortality were unclear.[1]

Recent consensus guidelines for the provision of enteral nutrition recommend that "In critically ill patients with gastric feeding intolerance, intravenous erythromycin should be used as a first line prokinetic therapy… Alternatively, intravenous metoclopramide or a combination of metoclopramide and erythromycin can be used as a prokinetic therapy."[2] Other important components of the management of ileus in humans include weaning narcotic analgesics when possible, daily monitoring of electrolytes and supplementation as needed, ensuring euvolemia and euhydration, early enteral nutrition, and regular ambulation.[2,3]

Evidence for prokinetic use in veterinary medicine is sparser but growing. In healthy cats, a crossover study using ultrasonographic endpoints showed both metoclopramide and erythromycin shortened gastric emptying times and increased antral motility.[4] Another study in healthy cats also supported the role of oral erythromycin or azithromycin in stimulating gastric emptying.[5] In anesthetized dogs, high-dose metoclopramide given as a bolus plus CRI reduced gastro-esophageal reflux risk, suggesting a role in peri-operative regurgitation prophylaxis.[6] Interestingly in a study investigating the total passage time through the gastrointestinal tract in dogs undergoing capsule endoscopy, the longest transit time occurred in the group receiving metoclopramide (691.33 min) and the shortest in the group receiving cisapride (584.17 min).[7] A recent multi-center veterinary cohort published by our group describes ICU prokinetic usage patterns in dogs—metoclopramide predominates, with rising erythromycin use and increased dual-agent therapy—highlighting practice variability and the need for prospective trials.[8]

Learning outcome: Prescribe rational treatment plans for ICU cases with GI dysmotility

1) Confirm dysmotility phenotype and ensure multimodal management. Rule out mechanical obstruction and correct precipitating factors (opioids, electrolyte abnormalities, overhydration etc.), reduce unnecessary sedatives, and initiate enteral nutrition.[9]

2) Commence first-line prokinetics. Metoclopramide (0.2 – 0.5 mg/kg IV/PO/SC q6-8h or CRI 0.01 – 0.02 mg/kg/h) and/or Erythromycin (dogs ~0.5 – 1-3 mg/kg PO q8–12h)

3) If feeding intolerance or other adverse effects of GI motility persist – escalate to dual prokinetics (if not already). In humans this is when post-pyloric feeding would be considered. Add additional prokinetics (eg. cisapride)

4) Reassess at least daily and de-escalate when able. Discontinue prokinetics once feeding tolerance is achieved or if adverse effects arise; continue non-pharmacologic measures.

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Nurse & Tech Stream, Saturday 6 June 2026

COMMON ECG ABNORMALITIES IN THE ER AND GP SETTING

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Learning objectives:

- Understanding electrical conductance through the heart
- Confidence in identifying abnormal ECG rhythms
- Determining which rhythms require treatment and why
- Getting the most from your ECG

Proceeding:

ECG abnormalities are a common finding in both the emergency room (ER) and general practice (GP) setting. Different rhythm abnormalities warrant varying treatment protocols, with some ECG abnormalities not requiring treatment.

Some of the most common arrhythmias witnessed in the emergency setting are ventricular in origin, for example; premature ventricular complex (VPC), accelerated idioventricular rhythm (AIVR) and ventricular tachycardia. Of these arrhythmias, the ventricular tachycardia (V-tach) is the one that warrants immediate emergency treatment and intervention. VPCs and AIVR generally do not require treatment unless accompanied by the R-on-T phenomenon.

Ventricular arrhythmia can be due to multiple factors, including; primary heart disease, metabolic and electrolyte disorders, myocarditis caused by systemic inflammation/shock, and traumatic injury to the thorax.

Ventricular fibrillation (V-fib) is an immediate life-threatening rhythm that requires CPR and treatment with biphasic defibrillation in order to restore a perfusing rhythm. As well as V-fib requiring immediate treatment, addressing V-tach and the R-on-T phenomenon rapidly is required by using a sodium channel blocker, such as lidocaine.

Arrhythmias that are atrial in origin can also be seen in ER and GP, although perhaps less frequently than ventricular, due to ventricular arrhythmia being common in the emergency patient from an underlying disease process.

The most common atrial arrhythmias include atrioventricular (AV) blocks: AV first degree, AV second degree Mobitz type I, AV second degree Mobitz type II , AV third degree, atrial fibrillation, and flutter (rare) and finally sinus tachycardia and bradycardia.

For second-degree AV block Mobitz II and third-degree, the patient requires immediate stabilisation and immediate referral to a cardiologist, where pacing will likely be required. Second-degree type II heart blocks can rapidly progress into third-degree, risking complete block and thus evolving into asystole when the escape rhythm fails. The escape rhythm is a protective rhythm which becomes evident when the sinoatrial (SA) node fails, thus allowing the atrioventricular (AV) node and ventricles to take over. This can be identified on ECG as wide QRS complexes (ventricular) with

lacking preceding P waves; this rhythm is slow and typically regular, generally less than 40 bpm with some rates dropping below 20 bpm.

Atrial fibrillation can commonly be seen in larger breeds and is identified on ECG as an irregular tachycardia with irregular R – R intervals and an 'erratic' baseline. Although not generally immediately life-threatening, this arrhythmia does require rapid treatment with antiarrhythmics and rate control drugs. Underlying causes can include dilated cardiomyopathy (DCM) in larger breed dogs and myxomatous mitral valve disease (MMVD) in smaller breeds, where structural heart damage has occurred.

Sinus tachycardia is common in most emergency presentations, a regular rhythm with rates exceeding 140 bpm in dogs and 220 bpm in cats, which are non cardiac in origin. Reasons for this can include; hypovolaemia, pain and anxiety. Sinus bradycardia, although not always common, can also be seen. A regular sinus bradycardia in a patient with abnormal mentation should be taken seriously, as this could indicate an increase in intracranial pressure (ICP), so it is paramount to always check a blood pressure on all patients that present in an emergency setting. Other reasons for sinus bradycardia could include a high vagal tone, also presenting as a sinus arrhythmia. These patients generally require no treatment, however need to be treated if the patient has experienced syncopal episodes.

Finally, a normal rhythm on ECG that presents as changes in amplitude of QRS complexes could indicate a pericardial effusion; known as electrical alternans. Physical movement of the heart 'swinging' in the pericardial sac causes the spikes in complexes to change in size from this movement. Immediate intervention is required to prevent further effusion and tamponade.

Obtaining an ECG on admission or at the time of presentation can give a large amount of information and should always, where possible, be included during any triage or admission. If on auscultation there are any concerns regarding arrhythmia, or concerns in pulse quality, this could be indicative of arrhythmias requiring treatment.

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FROM HYDRATION TO RESUSCITATION: THE FUNDAMENTALS OF FLUID THERAPY

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Learning objectives:

- Fluid compartments in the body
- Indications for fluid therapy
- Types of fluids available for use
- Patient monitoring
- Prevention of fluid overload

Proceeding:

Fluid therapy is administered almost daily to veterinary patients, which can make it easy to forget that fluids are a drug and must be treated as such. Like any drug, fluid therapy can cause harm, and inappropriate selection, dosing, or rate of administration may result in adverse outcomes. Decisions regarding fluid type, volume, and rate should always be based on the patient's physiological needs, underlying disease processes, and estimated tolerance.

Fluid Balance

Maintaining appropriate fluid balance is essential to support normal physiological function and to prevent dehydration or fluid overload. Dogs and cats are composed of approximately 60% water. Around two-thirds of this water is contained within cells as intracellular fluid (ICF). The remaining third is extracellular fluid (ECF), which is further divided into interstitial fluid (approximately 15%) and intravascular fluid (approximately 5%). To deliver appropriate fluid therapy, clinicians must first determine which fluid compartment(s) need replenishing or what derangement needs to be corrected.

Indications for Fluid Therapy

Indications for fluid therapy include:

Correction of dehydration

Expansion of intravascular volume

Restoration of adequate tissue perfusion

Maintenance of hydration

Clinical signs of fluid deficits vary depending on the fluid compartment affected. Dehydration is the result of fluid loss within the interstitial and intracellular spaces. As dehydration progresses, deficits may extend into the intravascular, resulting in concurrent hypovolaemia. Estimation of dehydration

is based on physical examination findings, including body weight, skin turgor and mucous membrane moisture.

Multiple formulas exist to calculate fluid requirements, and there is no evidence that one is superior to another. Regardless of the method used, fluid plans must be tailored to the patient and should take into consideration physical examination findings, laboratory results, and ongoing losses.

Correction of dehydration is typically achieved using buffered isotonic crystalloids, such as Lactated Ringer's Solution (LRS), administered intravenously over 12–24 hours. Patients receiving fluid therapy must be monitored regularly. End points include improved skin turgor and mucous membranes, normalisation of urine specific gravity, increased urine output and body weight.

Hypovolaemia and Shock

Reduced intravascular volume, impaired perfusion, and shock require rapid intravascular volume expansion to restore tissue perfusion. This is achieved through the administration of repeated fluid boluses, with continuous reassessment between doses.

Buffered isotonic crystalloids are commonly administered at:

5–10 mL/kg in cats15–20 mL/kg in dogs

Boluses are delivered over 15–30 minutes, with therapy guided by clinical end points including improvement in mentation, heart rate, capillary refill time and colour, arterial blood pressure, and pulse quality.

When patients present with both dehydration and hypovolaemia, hypovolaemia must be corrected first to restore perfusion, followed by slower correction of dehydration over 12–24 hours.

Types of Fluids

A variety of fluids are available, and the most appropriate fluid is dependent on multiple factors. Fluids are most simply categorised as replacement or maintenance solutions.

Crystalloids

Crystalloid fluids are the most commonly used and include both replacement and maintenance formulations. Replacement crystalloids, such as LRS, Hartmann's, and Plasma-Lyte 148, contain water and electrolytes in concentrations similar to plasma. 0.9% sodium chloride is also a replacement crystalloid, although it is not considered balanced.

Replacement vs Maintenance Fluids

Replacement fluids are indicated for resuscitation and correction of fluid deficits and electrolyte abnormalities. Maintenance fluids are designed to meet daily water and electrolyte requirements once intravascular volume and electrolyte derangements have been corrected. They are formulated for long-term administration and more closely reflect physiological sodium losses.

Hypertonic fluids contain a higher solute concentration than intracellular fluid and rapidly expand intravascular volume by drawing fluid from the interstitial and intracellular compartments.

Colloids

Colloids, both synthetic and natural, contain large molecules that remain within the intravascular space. The use of synthetic colloids is controversial in both human and veterinary medicine, with evidence linking them to acute kidney injury and coagulopathies.

Monitoring Fluid Therapy

All patients receiving fluid therapy require vigilant monitoring to detect and prevent overhydration, also known as fluid overload. Fluid overload can cause further complications, including but not limited to pulmonary oedema, hypoxaemia, impaired organ perfusion, delayed wound healing, and increased infection risk.

Clinical signs of fluid overload include:

Weight gain, particularly ≥10% above admission weight

Tachypnoea or increased respiratory effort

Serous nasal discharge

Abnormal lung sounds

Peripheral oedema

Early recognition relies on regular monitoring, and prompt adjustment of the fluid plan following detection significantly improves patient outcomes.

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NURSE RETENTION

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Learning objectives:

- Understand why nurse retention matters
- Explore strategies to reduce attrition
- Identify retention strategies
- Identify concrete actions leaders can implement to improve nurse retention within their own teams.

Proceeding:

Introduction

Veterinary nursing teams are the backbone of clinical practice, particularly in high-intensity environments such as emergency and critical care. Despite this, veterinary nursing continues to experience high levels of burnout, dissatisfaction, and attrition. Retention is often discussed reactively, once staff have already disengaged or left. However, retention should instead be addressed proactively, as a core component of patient safety, workforce wellbeing, and economic stability.

This lecture focuses on why nurse retention matters, what factors drive nurses to stay or leave, and—critically—what leaders can realistically influence within their own practices.

Why Nurse Retention Matters

Impact on Patient Care

Retention could impact patient outcomes. High turnover results in fewer experienced nurses, reduced continuity of care, and increased reliance on less experienced staff. These factors are associated with higher rates of medical errors, inefficiencies in workflow, and poorer overall patient outcomes. In critical care settings, where teamwork and familiarity with protocols are essential, frequent staff turnover poses significant risks to quality and safety.

Human Factors and Staff Wellbeing

The loss of colleagues is not only a logistical issue but also an emotional one. Nurses frequently report grief, frustration, and disengagement when valued team members leave. Remaining staff often absorb additional workload, leading to chronic fatigue, moral stress, and declining mental and physical health. Over time, this can create a self-perpetuating cycle of burnout and further attrition.

Economic Consequences

From a financial perspective, retention is not optional. Replacement costs for veterinary staff are estimated to be approximately 50% of an individual's annual salary when recruitment, onboarding,

training, and lost productivity are considered. Additional expenses include overtime pay to cover staffing gaps and the administrative burden of repeated recruitment cycles. Retention, therefore, is a core economic strategy rather than a management concept.

Retention Versus Attrition:

Evidence suggests that nurses do not leave solely because of workload or pay. Instead, attrition is usually multifactorial. Factors commonly associated with retention include feeling valued, supported by leadership, fairly compensated, and able to see a future within the organization. Conversely, toxic workplace cultures, lack of recognition, limited career progression, and inflexible scheduling consistently drive nurses away.

The concept of a "veterinary hierarchy of needs" highlights that basic requirements—such as fair pay and psychological safety—must be met before higher-level motivators like professional fulfillment and development can be effective. Importantly, leaders must recognize that decisions supporting retention may not always feel immediately logical or efficient, particularly in the short term.

Fair Pay and Recognition

Fair compensation remains a foundational retention factor. Pay structures should reflect qualifications, experience, and additional responsibilities, including specialist skills and leadership roles. Transparency is essential; unclear or inconsistent pay progression breeds dissatisfaction and mistrust.

Recognition is equally important. Verbal appreciation, acknowledgement of achievements, and genuine interest in individual staff members all contribute to nurses feeling valued. Simple, consistent recognition practices can have a positive impact on morale and engagement.

Career Development and Progression

A lack of career progression is one of the most frequently cited reasons for nurses leaving the profession. Retention improves when nurses can envision a future within their current organization.

Strategies include:

Establishing clear nursing levels or frameworks

Creating senior clinical, specialist, or mentorship roles

Supporting further education and certification

Offering structured pathways into education, training, or leadership positions

Importantly, career progression does not need to be limited to management or administrative roles. Clinical expertise, education, and specialization should be equally valued and rewarded.

Leadership and Workplace Culture

Leadership quality is a decisive factor in retention. Job satisfaction is reported higher when leaders prioritise wellbeing, communicate openly, and actively support professional growth. Caring leadership does not mean avoiding difficult conversations; rather, it involves fairness, consistency, and psychological safety.

A supportive workplace culture is built intentionally through:

Regular team meetings

Workshops and shared learning opportunities

Social events and informal connections

Transparent communication and feedback

Strong teams demonstrate what has been described as “team stickiness,” a sense of belonging that makes staff want to stay, even during challenging periods.

Meaningful Work and Purpose

Nurses are more likely to remain in roles where they feel their work matters. Meaningful work is defined by a sense of purpose, connection to a greater cause, and alignment between personal and organizational values.

Practices should articulate a clear mission and actively connect daily tasks to that mission. When nurses understand how their work contributes to patient outcomes, team success, and organizational goals, engagement and retention improve.

Flexibility and Work–Life Balance

Shift patterns, scheduling transparency, and reasonable accommodation of holiday and roster requests can influence retention. While operational constraints are real, even small degrees of flexibility can demonstrate respect for staff as individuals with lives beyond work.

What Leaders Can Affect Directly

While some systemic issues sit outside individual control, leaders can directly influence:

Team culture and psychological safety

Recognition and appreciation practices

Communication and transparency

Support for development and education

Fairness and consistency in decision-making

Retention is not achieved through a single initiative but through cumulative, everyday leadership behaviours.

Conclusion

Nurse retention is fundamental to sustainable veterinary practice. It affects patient care, staff well-being, and financial viability. Evidence and experience both demonstrate that nurses stay where they feel valued, supported, fairly compensated, and able to grow. Leaders play a central role in shaping these conditions. By focusing on what can be influenced directly—culture, leadership, recognition, development, and flexibility—practices can reduce attrition and build resilient, engaged nursing teams for the future.

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Resident Stream, Saturday 6 June 2026

PHYSIOLOGY OF MECHANICAL VENTILATION

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Learning objectives:

- Define compliance and resistance
- Understand how the equation of motion relates to ventilator operation
- Understand the difference between static and dynamic compliance
- Identify resistance and compliance abnormalities from the pressure-time scalar

Proceeding:

Mechanical ventilation describes the use of a machine, 'the ventilator' to move gas in and out of the lungs. The ventilator uses changes in pressure to generate gas flow into the lungs. The pressure needed to deliver a given tidal volume will need to overcome the resistance of the airways as well as the elasticity (also known as compliance or elastance) of the lungs. Compliance can be defined as the change in volume for a given change in pressure. Airway resistance is another major contribution to the pressure needed to generate an inspiratory breath from the ventilator. Resistance is equal to the pressure in the system divided by the gas flow rate.

Airway pressure

The equation of motion describes the factors that determine the total pressure needed to generate a ventilator breath. It is the basis of computer analysis for pulmonary mechanics, built into modern ventilators. From the equation of motion it can be seen that the pressure needed for a ventilator breath can be reduced by decreasing tidal volume, increasing compliance, decreasing airway resistance or increasing inspiratory time.

Compliance

Compliance can be calculated as dynamic (gas is still moving) versus static (no gas moving). Dynamic compliance is determined using peak inspiratory pressure (PIP) while static compliance uses plateau pressure (Pplat), measured when all gas flow has stopped. To measure plateau pressure, an inspiratory hold maneuver must be performed. The airway pressure will drop from PIP to Pplat as gas redistributes in the lung. The magnitude of the difference between PIP and Pplat reflects resistance to gas flow. While the magnitude of Pplat reflects the distending pressure of the respiratory system. In health, it is expected that healthy dogs and cats will have minimal resistance in the system and the difference between dynamic and static compliance will be small.

Transpulmonary pressure

Although Pplat is commonly used as a surrogate for lung distension, it includes the pressure needed to distend both the lung and the chest wall. In order to evaluate the compliance of the lung alone, it is necessary to evaluate transpulmonary pressure (TPP). Transpulmonary pressure represents the

distending pressure of the lung and is calculated as the difference between alveolar pressure and pleural pressure. If the lung is air filled, the TPP is a positive value. There are ventilator strategies described that target TPP to set optimal PEEP and tidal volume, with the aim to minimize lung injury. To determine TPP, alveolar pressure is considered equal to Pplat as with no gas moving, the alveolar pressure has equilibrated with the breathing circuit. In the clinical setting, pleural pressure is commonly estimated by measurement of esophageal pressure, although it is recognized that this approach has limitations.

I:E ratio & Respiratory Rate

Respiratory rate can be set on all ventilators. A normal respiratory rate of 15 – 20 breaths is usually selected when the patient is initially established on the machine. This can then be changed as appropriate for the patient. The ratio of the duration of inspiration to exhalation (I:E ratio) may be preset by the operator or maybe a default setting within the machine. Commonly an I:E ratio of 1:2 is utilized to ensure the patient has fully exhaled prior to the onset of the next breath. As respiratory rates are increased the expiratory time will be sacrificed in order to 'squeeze' in the necessary number of breaths. High respiratory rates can lead to a situation known as breath stacking or intrinsic positive end expiratory pressure (intrinsic PEEP) as the animal is not able to fully exhale before the start of the next inspiration.

Positive End Expiratory Pressure

Extrinsic PEEP is a baseline phase variable that can be set on most modern ventilators and maintains positive pressure (a pressure greater than atmospheric pressure) in the airway during exhalation. PEEP will increase the functional residual capacity and may lead to recruitment of collapsed alveoli which will improve gas exchange. PEEP can also reduce cyclic collapse and reopening of lung units (atelectrauma), so may have lung protective effects. PEEP can also have detrimental effects, these include overdistension of more compliant lung regions which can create alveolar dead space as well as cause lung injury. PEEP can also compromise venous return and lead to cardiovascular instability.

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EXTRA-PULMONARY EFFECTS OF MECHANICAL VENTILATION

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Learning objectives:

- Describe the effects of positive pressure ventilation on the cardiovascular, renal, and neurologic systems.
- Consider ways to mitigate adverse extra-pulmonary effects during positive pressure ventilation.

Proceeding:

Positive pressure ventilation (PPV) can be life-saving. Effective use requires detailed understanding of pulmonary physiology. However, a lung-centric approach overlooks the extra-pulmonary consequences of PPV. In particular, the cardiovascular, renal and neurologic systems are fundamentally – and often adversely – affected by PPV. Appreciating these effects is essential to managing the whole patient, not just the lungs.

Cardiovascular effects

The heart and the lungs are intimately related. Changes in intrathoracic pressure are transmitted to the heart.[1,2] Additionally, the pulmonary vascular is in series with the heart: downstream of the right heart and upstream of the left heart. Therefore, changes in a) intrathoracic pressure (ITP) and b) pulmonary blood flow affect the cardiovascular system.[1,2,3]

The ITP is necessarily increased with PPV. By adding positive end expiratory pressure (PEEP), ITP is increased further. Oxygenation is determined mostly by fraction of inspired oxygen (FiO_2) and mean airway pressure, so we intentionally increase the pressure thus the ITP in our mechanically ventilated patients.

Consequent to increasing ITP, the venous return to the right atrium is decreased. This is because the downstream pressure to venous return – the right atrial pressure – is increased by increasing ITP. [1,2,3] Initially, the left ventricular stroke volume may increase due to ventricular interdependence, amongst other things; however, quickly the stroke volume decreases due to decreased venous return. Therefore, PPV can decrease cardiac output. This effect is particularly relevant in volume underloaded patients, though is also relevant in normovolaemic patients.

PPV increases right ventricular afterload by increasing pulmonary vascular resistance (PVR). The lungs normally rest at an equilibrium point called *functional residual capacity (FRC).* At FRC, the PVR, which is determined by the ITP and the recoil pressures of thoracic structures, is at its lowest.[4] Increasing ITP progressively collapses the alveolar pulmonary vasculature, leading to decreased usable vascular volume and increased PVR. Increasing PVR increases right ventricular afterload and the work the right heart must do to eject blood into the pulmonary circulation. Importantly, PVR exists as a U-shaped curve with FRC at the PVR nadir and both increases *and* decreases in lung

volumes from FRC leading to increased PVR. When alveolar units are collapsed (low lung volume), PVR is increased: the application of PPV and PEEP can open alveolar units and open collapsed vessels that are tethered to these units, thereby *decreasing* PVR.[4] Some of the increase in right ventricular afterload can also be offset by the reduction in hypoxic pulmonary vasoconstriction with the improved oxygenation achieved by PPV.

Renal effects

PPV can reduce cardiac output, which in turn decreases renal perfusion. This predisposes patients to pre-renal azotemia and acute kidney injury.[1,2] Additionally, decreased venous return caused by increased ITP has a backward, congestive effect on renal blood flow that can result in renal oedema and reduce kidney function.[1] Adding to this, reduced renal perfusion and reduce glomerular filtration result in sodium and water retention, contributing to volume overload. Inconsistent evidence in human medicine supports low-volume ventilation strategies to reduce the impact of PPV on the kidneys, though further evidence is needed.[2]

Neurological effects

Hypercapnia results in cerebral arterial vasodilation and increased cerebral blood flow (CBF). Conversely, hypocapnia reduces CBF.[1,2] High-volume ventilation may be associated with cerebral neuronal apoptosis or injury supporting so-called lung-protective ventilation strategies.[2,5] However, high levels of PEEP have been associated with decreased CBF secondary to decreased mean arterial pressure and there is additional concerns that high PEEP – thus high ITP – can impair venous draining from the brain, resulting in elevated intracranial pressure. [2,5] Importantly, PEEP improves oxygenation and the injured brain does not like hypoxia: PEEP may be necessary. Additionally, PEEP seems to have minimal negative brain effects in mild or moderate traumatic brain injury, so limiting PEEP may only be necessary with severely injured brains.

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PHYSIOLOGY OF COAGULATION

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Learning objectives:

- Know the key aspects of the *in-vivo* coagulation pathway
- Be familiar with changes in the coagulation pathway in response to inflammation
- Describe the pathophysiology of disseminated intravascular coagulation

Proceeding:

Introduction

Hemostasis is the process of forming a blood clot to seal an injured vessel, in contrast thrombosis refers to the formation of a pathologic blood clot. Changes in coagulation are common in patients with severe systemic disease, an understanding of normal hemostasis and the influence of inflammation on coagulation is important for the critical care clinician.

Normal Hemostasis

In health the balance between pro and antithrombotic processes is such that vessel damage is repaired effectively without excessive bleeding or excessive coagulation. The major prothrombotic factors in coagulation include cell membranes, platelets and the soluble coagulation factors. Prothrombotic processes have a procoagulant influence on the system. Cell membranes of platelets, microparticles and endothelial cells maintain an anticoagulant surface in health, inhibiting coagulation. In response to cell trauma or inflammation, the distribution of phospholipids on the cell surface changes to ones that promote coagulation and provide a scaffold for fibrin formation.

Platelets

Platelets are very dynamic cells that are covered in membrane bound receptors. Upon activation, platelets undergo several changes including a shape change, degranulation, activation of receptors, adhesion to the vessel wall and aggregation with other platelets. Numerous stimuli can activate platelets including exposed collagen, von Willebrand Factor (vWF), adenosine diphosphate, thrombin and thromboxane A2. Activation of platelets leads to formation of the platelet plug (primary hemostasis) and provides a surface for the activation of coagulation factors.

Secondary coagulation

Secondary coagulation refers to the activation of the coagulation cascade that ultimately results in the formation of thrombin. Tissue factor (TF), previously known as factor III of the extrinsic pathway is the primary initiator of coagulation in both normal and disease states. Tissue factor is the only membrane bound member of the coagulation cascade; it is an extremely pro-coagulant molecule that also has cell signaling actions and interacts with numerous pathways other than coagulation. In health, active TF is found outside of the bloodstream, when it is exposed to blood following vessel

injury, it binds and activates factor VII and initiates coagulation. Thrombin is central to the coordination of coagulation, it cleaves fibrinogen to fibrin, activates factor XIII to crosslink fibrin, activates fibrinolysis, initiates cell repair and activates inflammation.

Fibrinolysis

Fibrinolysis is a vital aspect of normal hemostasis. Plasmin is responsible for the degradation of fibrin. Plasmin is cleaved from plasminogen (which is bound to fibrin within the clot) by tissue plasminogen activator (tPA) and/or urokinase. These are produced and released by endothelial cells in response to injury or thrombin. In addition to fibrin, plasmin also degrades FVa and FVIIIa.

Anticoagulant processes

There are numerous endogenous anticoagulant processes that in health prevent excessive clot formation in response to vessel injury. These include activated Protein C, tissue factor pathway inhibitor (TFPI) and antithrombin (AT). They are activated simultaneously with coagulation and tend to have an anti-inflammatory influence on the system.

Changes in coagulation with disease

Proinflammatory disease processes will have procoagulant effects. This is a complex process including activation of cell membranes, activation of platelets, expression of TF on mononuclear cells and endothelial cells, reduced anticoagulant processes and inhibition of fibrinolysis. In its severe form, this can lead to intravascular fibrin formation, a syndrome known as disseminated intravascular coagulation (DIC). This is a procoagulant disease process but can be challenging to identify in the clinical setting. In a subset of patients, a consumptive coagulopathy can develop leading to bleeding tendencies. Clinically, this late coagulopathic phase of DIC is most readily identified. The diagnosis of DIC is not specific and is based on a constellation of laboratory abnormalities suggestive of a consumptive coagulopathy existing in a patient with a primary disease process considered likely to cause DIC.

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AN ION OFT FORGOTTON - HOW TO CARE ABOUT CHLORIDE

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Learning objectives:

- Understand the physiology of chloride
- Understand the effect of hyperchloremia on the kidney
- Identify iatrogenic causes of dyschloremia
- Treat cases of dyschloremia

Proceeding:

The plasma chloride concentration is often abnormal in critically ill patients. However, this abnormality is not always given the attention that it deserves. Both hypochloremia and hyperchloremia are associated with mortality in both dogs and cats.[1-4] Hospital-acquired dyschloremia has been associated with a worse prognosis than community-acquired dyschloremia, raising the possibility that iatrogenic contributions to dyschloremia may have prognostic significance.[3,4]

Physiology of dyschloremia

The plasma chloride concentration is affected by free water balance. Thus, assessment of dyschloremia first involves calculation of the corrected chloride concentration. This assesses the impact of free water balance using the plasma sodium concentration. The equation is $[Cl^-]_{corrected} = [Cl^-]_{measured} * [Na^+]_{mid\text{-}normal} / [Na^+]_{measured}$. Corrected dyschloremia is associated with abnormalities in the metabolic acid-base status. Due to the need to maintain electroneutrality, free water-independent changes in the concentration of one plasma anion must be associated with a concurrent opposing change in another anion. As chloride and bicarbonate represent the two major plasma anions, changes in chloride concentration are generally associated with opposing changes in bicarbonate concentration. Thus, hyperchloremia is usually associated with a metabolic acidosis, and hypochloremia is usually associated with a metabolic alkalosis. Common causes of hyperchloremic metabolic acidosis include gastrointestinal bicarbonate loss, renal bicarbonate loss (renal tubular acidosis), and iatrogenic administration of high-chloride, low-bicarbonate fluids. Common causes of hypochloremic metabolic alkalosis include loss or sequestration of gastric fluid and loop diuretic administration. Separate to the acid-base effects, hyperchloremia is a potential contributor to acute kidney injury (AKI), due to disruption of tubuloglomerular feedback.[5] Delivery of chloride to the macula densa is the mechanism of measurement of tubular flow in this important renal autoregulatory response. Increased tubular flow results in afferent arteriolar constriction and efferent arteriolar dilation, reducing glomerular filtration rate (GFR). However, hyperchloremia results in a higher tubular fluid chloride concentration, interfering with the calibration of this feedback mechanism. Excessive afferent arteriolar constriction occurs, resulting in pathologically decreased GFR and AKI. Whilst there is no direct evidence for hyperchloremia-induced AKI in

veterinary medicine, one study documents an association between plasma chloride concentration and AKI in dogs.[6] Whilst the hyperchloremia may have been a causative factor in the AKI, it is also important to note that the AKI may have also contributed to hyperchloremia. Renal tubular acidosis, which is a hyperchloremic metabolic acidosis, may occur in critically ill animals with AKI.[7] Thus, hyperchloremia and AKI may represent a vicious cycle where each abnormality may predispose to the other.

Iatrogenic contributions to hyperchloremia

Intravenous fluid therapy (IVFT) can substantially contribute to an excessive chloride load. Isotonic crystalloid fluids form the mainstay of intravenous fluid therapy in veterinary medicine. Whilst use of balanced isotonic crystalloid fluids is common, some veterinarians opt for 0.9% sodium chloride (NaCl) for routine IVFT.[8] There is evidence for a higher incidence of AKI and mortality when 0.9% NaCl is used for routine fluid resuscitation in human medicine, compared to balanced isotonic crystalloids.[9-11] Whilst use of balanced crystalloids for resuscitation and maintenance is an important consideration, there are several other potential sources of chloride. Drug dilution and flush solutions are often based on 0.9% NaCl. The volume of fluid administered in this manner is often poorly documented and a major contributor to 'fluid creep', a situation where fluid ins are substantially higher than patient requirements or prescribed IVFT. Hypertonic crystalloids, used for rapid volume expansion and treatment of cerebral oedema, are usually concentrated NaCl (e.g., 7%). There is some research in human medicine into the use of balanced hypertonic crystalloids.[12,13] Potassium chloride supplementation of IVFT is common.[8] The excess chloride may be a contributor to excessive chloride load, and research into other formulations such as potassium acetate is ongoing.

Iatrogenic contributions to hypochloremia

The major iatrogenic contributor to hypochloremia is loop diuretic administration for congestive heart failure. Hypochloremia is associated with a poorer prognosis in dogs with stable congestive heart failure.[14] Research into chloride concentration in decompensated heart failure and chloride supplementation as an aid to management of heart failure is ongoing. Excessive gastric suctioning and excessive bicarbonate administration are other potential iatrogenic contributors to hypochloremia.

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SEMI-QUANTITATIVE ACID-BASE ANALYSIS

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Learning objectives:

- To revise the traditional approach to acid-base analysis, recognising its deficiencies in assessing the metabolic component;
- Understand the principles behind the Stewart approach, and apply them to interpretation of acid-base disorders;
- Understand the influence of anionic and cationic charge on hydrogen ion flux;
- Be aware of the quantitative effect of certain ions on Metabolic acid-base disorders;
- Apply all 3 techniques to clinical case examples, using a holistic approach to gain maximal insight into acid-base analysis

Proceeding:

Acid-base analysis can be a vital tool in the diagnosis and monitoring of ECC patients. Whilst analysis of the Respiratory/Ventilatory aspects of acid-base balance are relatively straightforward and aligned between different techniques, interpretation of Metabolic contributions is more complex. This is because the Respiratory 'side' is concerned with a simple process – transport of CO_2 from the tissues where it is generated, to the pulmonary capillaries where it diffuses into the alveolar space and hence lost from the body with alveolar ventilation. Except in periods of extreme exertion (e.g., strenuous exercise, seizures, etc.), cellular CO_2 production is also relatively constant, meaning that levels in mixed venous blood are fairly stable, with therefore levels in arterial blood being almost entirely determined by the minute volume. Whilst transport of CO_2 does have some biochemical limitations, functionally, this process also means that the Respiratory system has an almost infinite and rapidly adjustable ability to remove H^+ from the body via the equation:

$CO_2 + H_2O$ «-» H_2CO_3 «-» $H^+ + HCO_3^-$

This is commonly referred to as *Respiratory Compensation*, with **hyperventilation** (and hypocapnia) **generating a Respiratory Alkalosis to 'compensate' for a Metabolic Acidosis**; likewise, **hypoventilation leads to** hypercapnia and **a Respiratory Acidosis that could 'compensate" for a Metabolic Alkalosis**, with the caveat that eventually hypoxaemia will eventually trigger ventilation.

Whilst oftentimes in our critical patients, Respiratory acid-base changes will be compensatory, clinicians should not forget to consider *primary* pathological drivers of both hypo- and hyper-ventilation.

Assessing the Metabolic 'side' of acid-base balance is much more complicated, reflecting as it does biochemical and metabolic processes at the level of the cell, as well as physiological and pathological processes occurring in tissues, with particular emphasis on the gastro-intestinal, renal, and circulatory systems. As a result of this, as clinicians we have to 'bundle' many of these processes

together, with different techniques for acid-base interpretation allowing greater insight at the expense of analytical complexity.

Considering the 'traditional'/Henderson-Hasselbalch technique, the use of **Base Excess** allows us to assess the overall Metabolic acid-base balance, but tells us little of the *processes* causing it: assessment of the 'appropriateness' of the [HCO_3^-] (compared to the PCO_2), as well as examination of the Anion Gap can add some detail, but unfortunately, both of these parameters can obscure significant pathologies. To gain further detail, therefore, we will examine and combine 2 other techniques – Stewart's Strong Ion Theory and (Fencl's) Semi-Quantitative analysis of Base Excess.

The 'Stewart approach' (or rather, Peter Constable's simplification!) revolves around the concept of electrochemical balance and the principle that pH (that is, the [H^+]) is determined by water dissociation:

$2\ H_2O$ «-» $H_2O + H^+ + OH^-$ «-» $H_3O^+ + OH^-$

which in turn is determined by the levels of other charged particles in solution. Stewart grouped these electrochemically active particles into 3 groups, each *independent* of external influence (other than by [H^+]):

PCO_2 – partial pressure of CO_2

↑ **CO_2** indicates an acidotic effect ↓ **CO_2** indicates an alkalotic effect

Strong Ion Difference (SID) – concerned with those substances *always* present in an ionised form at physiological temperature & pressure; often abbreviated to [Na^+] – [Cl^-]

↓ **SID** indicates an acidotic effect ↑ **SID** indicates an alkalotic effect

A_{tot} – the sum of weak acids (and bases, although these are usually ignored)

↑ **A_{tot}** indicates an acidotic effect ↓ **A_{tot}** indicates an alkalotic effect

However, whilst the concept of water dissociation underlies the Stewart approach, considering electrochemical balance instead allows (for most people) an easier explanation, and also enables understanding of the semi-quantitative approach.

Consider a model consisting of a small unit of a tissue. Within the extracellular fluid of that unit, there has to exist electrochemical balance in order to allow the cellular use of charged particle gradients across membranes. **Electrochemical balance means that the net cationic (positive) charge equals the net anionic (negative) charge**.

If additional anionic (negative) charge enters that tissue unit, for example, by an increase in [Cl^-], then in order to re-balance the electrical charge, additional cations (positive ions) will be required: these could be *any* cations, however, most of the readily available cations (Na^+, K^+, Ca^{2+}, Mg^{2+}, etc.) have significant metabolic roles so instead, **H^+ are liberated from buffer molecules** (some will leave cells) **to increase cationic charge and restore electrochemical balance**. **This clearly results in an increase in [H^+] and therefore creates a metabolic acidosis**. This *could equally be driven by a reduction in extra-cellular cationic charge,* causing the same H^+ flux.

The converse is therefore also true – **additional extra-cellular cationic charge (or a reduction in anionic charge) would result in a movement of** free cations – i.e., **H^+ - out of solution and onto buffers** (or into cells); **this would therefore result in a decrease in [H^+] and a metabolic alkalosis**.

The final step is to combine what we know of the overall metabolic acid-base balance – the Base Excess, together with the electrochemical principle (above) and some quantitative data determined experimentally. This creates the following set of equations, which (usually via an Excel worksheet!) can be used to analyse the individual contributions of these routinely measured components.

Dogs

ΔBE = 0.25*(Patient [Na^+] – Normal [Na^+]) *normal [Na+] = 146mEq/L*

ΔBE = Normal [Cl^-] – Corrected Patient [Cl^-] *normal [Cl^-] = 110mEq/L*

ΔBE = 0.37*(Normal [Albumin] – Patient [Albumin]) *normal [Alb] = 33g/L*

OR

ΔBE = 0.3*(Normal [TP] – Patient [TP]) *normal [TP] = 65g/L*

Cats

ΔBE = 0.22*(Patient [Na^+] – Normal [Na+]) *normal [Na^+] = 156mEq/L*

ΔBE = Normal [Cl^-] – Corrected Patient [Cl^-] *normal [Cl^-] = 120mEq/L*

ΔBE = 0.37*(Normal [Albumin] – Patient [Albumin]) *normal [Alb] = 30g/L*

OR

ΔBE = 0.3*(Normal [TP] – Patient [TP]) *normal [TP] = 72g/L*

Dogs & Cats

ΔBE = 1.8*(Normal [Phos] – Patient [Phos]) *normal [Phos] = 1.6mmol/L*

ΔBE = -1*Patient [Lactate] *normal [Lact] = 0mmol/L*

Unmeasured Cations consist of K^+, Ca^{2+}, Mg^{2+}, etc. Unmeasured Anions consist of SO_4^{2-}, ketones, uraemic acids, exogenous acids, etc.

Oral Abstracts - Original Study, Thursday 4 June 2026

CAPILLARY REFILL TIME IN HEALTHY DOGS: REFERENCE VALUES AND INFLUENCE OF INDIVIDUAL CHARACTERISTICS – AN INTERIM ANALYSIS

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Introduction

Capillary refill time (CRT) is widely used in canine clinical practice, yet reference values remain empirical and poorly documented. This study aimed to establish reference values for CRT in dogs and to assess the influence of individual characteristics.

Methods

This prospective study included apparently healthy dogs. Dogs were excluded when CRT measurement was not feasible due to handling difficulties or aggressiveness. CRT was measured after an 8-s compression of the labial mucosa, performed by the handler while a second observer recorded the time. In the first 20 dogs, three consecutive measurements performed by three different observers were used to assess repeatability and inter-observer reproducibility using variance component analysis and intraclass correlation coefficients (ICC); in the remaining dogs, three consecutive measurements were obtained by a single observer. Analyses were conducted on the mean CRT per dog. Reference values were calculated using parametric and robust methods, and associations with individual characteristics were assessed using linear models.

Results

Fifty-nine dogs were included, and two were excluded (n=57). Inter-individual differences accounted for the largest proportion of total CRT variance (~47%), followed by intra-observer residual variability (~31%) and a dog × observer interaction (~22%), with no systematic observer effect. When the mean of three repeated measurements performed by the same observer was considered, intra-observer variability was reduced. Inter-observer agreement was moderate (ICC = 0.60; 95% CI: 0.35–0.80). The 95% reference interval for mean CRT was 0.48–1.63 s using parametric methods and 0.47–1.64 s using robust methods. For a single CRT measurement, the corresponding reference intervals were 0.33–1.81 s and 0.35–1.74 s, respectively, with overlapping confidence intervals for all limits. Mean CRT increased slightly with age (0.037 s/year; 95%CI: 0.003–0.072). No significant associations were identified with body weight, size, physical activity level, behavioral scores, systolic arterial pressure, sex, or neuter status. Heart rate and neuter status showed non-significant trends toward shorter and longer CRT, respectively.

Conclusions

This study provides data-driven reference values for CRT in dogs, highlights moderate measurement variability, supports averaging repeated measurements, and identifies age as the only individual factor modestly associated with CRT.

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HOW DOES PIMOBENDAN INCREASE STROKE VOLUME IN AWAKE DOGS? INSIGHTS FROM ADVANCED ECHOCARDIOGRAPHY.

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Introduction:

Intravenous pimobendan is commonly used in veterinary emergency and critical care, particularly in the management of cardiogenic shock, for its rapid inodilatory effects. Myocardial contraction involves distinct components (radial, circumferential, and longitudinal), which can be investigated using speckle tracking echocardiography. Our study aimed to characterize the echocardiographic components of left heart contraction after intravenous pimobendan administration in dogs, and to determine the determinants of the associated increase in stroke volume.

Methods:

Five healthy conscious male Beagles received a single intravenous bolus of pimobendan (0.15 mg/kg). Echocardiographic examinations were performed at baseline and over the 24 hours post-administration (T30min, T2h, T4h, T6h, T8h). Left ventricular systolic function were assessed using speckle-tracking–derived longitudinal, circumferential, and radial strain. Stroke volume and cardiac output were calculated. Plasma concentrations of pimobendan and its active metabolite, O-desmethylpimobendan (ODMP), were measured using liquid chromatography - tandem mass spectrometry. Longitudinal data were analyzed using linear mixed models to describe temporal changes in myocardial strain and to assess the associations between myocardial contraction components and stroke volume, as well as between plasma concentrations of pimobendan and its active metabolite and stroke volume.

Results:

Pimobendan induced a significant increase in left ventricular systolic function. Radial strain increased significantly from T30min to T6h, with a peak at T2h. Circumferential strain increased between T30min and T4h, predominantly at the endocardial and mid-myocardial levels. Longitudinal strain showed significant enhancement from T30min to T8h. Apical segment strain increased more than basal and mid-ventricular segments, resulting in an accentuated base-to-apex strain gradient. Stroke volume increased significantly from T30min to T8h and was most strongly associated with longitudinal strain and particularly with apical contraction. Between plasma pimobendan and ODMP concentrations, only ODMP concentration was associated with an increase in stroke volume.

Conclusions:

Pimobendan produced a sustained enhancement of left heart function lasting up to 8 hours. Although radial strain showed the largest absolute increase, the increase of stroke volume was more closely related to improvements in longitudinal myocardial deformation.

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CLINICAL OUTCOME AND PROGNOSTIC FACTORS IN 232 DOGS WITH ACUTE KIDNEY INJURY TREATED WITH INTERMITTENT HEMODIALYSIS: A RETROSPECTIVE STUDY COMPARING LEPTOSPIROSIS VERSUS NON-LEPTOSPIROSIS CASES (2013–2025)

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Background: Comparison of outcomes and prognostic factors of dogs with acute kidney injury (AKI) secondary to leptospirosis (L-AKI) or other causes (NL-AKI) undergoing intermittent hemodialysis (IHD) have not been evaluated within a single cohort.

Objective: To describe in-hospital outcomes and identify prognostic factors in dogs with AKI treated with IHD, and to compare these between L-AKI and NL-AKI dogs.

Design: Retrospective study

Setting: University teaching hospital

Methods: Medical records of dogs with AKI treated with IHD (2013–2025) were reviewed. Leptospirosis status was defined according to the ACVIM 2023 consensus statement. Number of body systems affected, biochemical variables (at admission and peak during hospitalization), urine output at admission, survival status and azotemia at discharge were recorded.

Results: In univariate analyses, survivors were younger than non-survivors (median 3 [1–5] vs. 5 [2–8] years, $p<0.001$), and L-AKI dogs had a higher survival rate than NL-AKI dogs (60% vs. 38%, $p=0.002$). In the overall population, mortality was associated with anuria at admission (49% in non-survivors vs. 17% in survivors, $p<0.001$) and a greater number of affected body systems ($p=0.0041$), whereas body system involvement did not differ between L-AKI and NL-AKI dogs. In multivariate analysis of the overall population, NL-AKI dogs experienced higher mortality than L-AKI dogs (OR 2.78; 95% CI, 1.07–7.24). Mortality was independently associated with a higher number of affected body systems (OR 4.63 per additional system; 95% CI, 1.81–12.79), anuria at admission (OR 3.13; 95% CI, 1.08–9.05), increased age (OR 1.17 per year; 95% CI, 1.02–1.36), and higher creatinine concentration at admission (OR 1.001 per µmol/L; 95% CI, 1.000–1.002). Within the L-AKI population,

mortality was independently associated with increased potassium concentration at admission (OR 1.74 per mmol/L; 95% CI, 1.03–3.15) and increased age (OR 1.27 per year; 95% CI, 1.04–1.63).

Clinical Relevance: In this large cohort of dogs with AKI treated with IHD, L-AKI was associated with better short-term prognosis than NL-AKI. In L-AKI, prognosis worsened with increasing age and admission potassium levels. These results highlight the prognostic relevance of AKI etiology, age and admission potassium concentration in dogs treated with IHD.

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IMPACT OF INTERMITTENT RENAL REPLACEMENT THERAPY ON AMPICILLIN/SULBACTAM PLASMA CONCENTRATIONS IN DOGS WITH OLIGURIC ACUTE KIDNEY INJURY

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Introduction:

Intermittent renal replacement therapy (IRRT) efficiently removes small, hydrophilic solutes but can alter the clearance of renally eliminated antibiotics such as β-lactams, potentially leading to insufficient time above minimum inhibitory concentration (MIC) and subtherapeutic antimicrobial exposure.

Objectives:

To evaluate the impact of IRRT on plasma concentrations of ampicillin and sulbactam in dogs with oliguric AKI.

Methods:

This prospective, single-center observational study enrolled client-owned dogs with oliguric (<0.5 mL/kg/h) IRIS stage 4 AKI caused by suspected or confirmed infectious origin undergoing intermittent low-efficiency hemodiafiltration. Ampicillin/sulbactam (22 mg/kg IV q8 h) was administered via syringe pump over three minutes, 30 minutes pre-IRRT session. EDTA blood samples were collected immediately before and after IRRT session and immediately centrifuged, separated, frozen, and then analyzed within 3 months. Plasma ampicillin and sulbactam concentrations were quantified using validated high-performance liquid chromatography–tandem mass spectrometry. The change in plasma concentrations were evaluated in relation to published MICs for *Leptospira* spp. and *E.coli* (<0.5 µg/mL and 8 µg/mL, respectively). Drug concentrations were analyzed using descriptive statistics and presented as median (min-max). Ethical approval (no. 2396) and written informed owner consent were obtained.

Results:

Eight dogs were included (median age 3.5 years [0.5–10.5]; median body weight 21.6 kg [10.5–48.7]. Diagnoses included leptospirosis (n = 4), pyelonephritis (n = 1), suspected toxic injury (n = 1), and unknown etiology (n = 2). Admission serum creatinine and urine output during the sampling period were 1,154 µmol/L (883–1,645) and 0.02 mL/kg/h (0–0.69), respectively. Ampicillin and sulbactam concentrations decreased from 75.6 µg/mL (53.2-355.2) and 64.6 µg/mL (40.8-199.4) before dialysis to 21.7 µg/mL (10.8-57.4) and 18.7 µg/mL (8.6-44.3) after dialysis, respectively.

Conclusion

IRRT resulted in a marked decrease in ampicillin and sulbactam plasma concentrations but consistently remained above the MICs of both Leptospira *spp.,* and *E.coli*. Dose optimization studies are needed to define dosing regimens in dogs undergoing IRRT.

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CHARACTERIZATION OF FELINE UROABDOMEN AND ASSESSMENT OF THE ABDOMINAL FLUID-BLOOD CREATININE AND POTASSIUM RATIOS FOR ITS DIAGNOSIS IN CATS

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Introduction:

Uroabdomen is caused from extravasation of urine into abdominal cavity, resulting in peritonitis. Abdominal fluid creatinine (AFCr) or potassium (AFK+) to blood creatinine (BCr) or potassium (BK+) ratios can be used to diagnose uroabdomen. However, the literature describing feline uroabdomen, or testing the historical cut-offs for these tests is limited. This study aimed to describe a referral center feline uroabdomen population and evaluate the cut-off values and diagnostic performance of AFCr:BCr and AFK+:BK+ to diagnose uroabdomen.

Methods:

Medical records of feline patients diagnosed with uroabdomen admitted to a referral teaching hospital between 01/01/10 – 05/01/26 were analyzed. Cats were included if the diagnosis of uroabdomen was confirmed using diagnostic imaging or exploratory surgery. If the abdominal fluid and blood biochemistry were performed within 24h of each other, they were used for ratio analyses. A control group without uroabdomen in which AFCr:BCr and AFK+:BK+ analyses were performed for clinical purposes was included. Standard inferential statistics were performed to assess association with survival and receiver operating curves and Youden's Index (YI) were used to assess the Sensitivity/Specificity (Sens/Spec) of different cut-offs.

Results:

Seventy cats were included. Twenty-six (37%) cats and thirteen controls were used for ratio analysis. Uroabdomen was most commonly caused by trauma (40%) and iatrogenic damage (36%). Mean time between abdominal fluid/blood testing was 45 minutes (IQR 18-60min). Surgically managed cases had higher survival compared to medically managed cases (survival rates 87% and 50%, p=0.0278). Area under the curve (AUC) equaled 0.94 (C.I. 0.87-1; p<0.0001) for creatinine and 0.82 (C.I. 0.66-0.98; p=0.0016) for potassium. Historical cut-offs for AFCr:BCr (2:1) and AFK+:BK+ (1.9:1) resulted in Sens/Spec values of 77%/100% (YI=0.77) and 27%/92% (YI=0.19), respectively. New potassium cut-off (1.2:1) increased Sens/Spec to 77%/83% (YI = 0.60). AFCr:BCr (2:1) had lower YI that AFCr:BCr (1.7:1) (YI=0.81; Sens/Spec 88%/92%).

Conclusions:

New potassium cut-offs improved the performance of AFK+:BK+, which remained less precise than creatinine based on its lower AUC. New suggested ratios retain the limitations of potentially over- and underdiagnosis. Consequently, these ratios should be used in conjunction with diagnostic

imaging findings, compatible history and clinical interpretation for accurate diagnosis of feline uroabdomen.

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COMPARISON OF OBLIQUE VERSUS OUT-OF-PLANE ULTRASOUND-GUIDED CEPHALIC CATHETER PLACEMENT IN CARDIOVASCULARLY COMPROMISED DOGS

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Introduction: Vascular access can be challenging in cardiovascularly compromised dogs. Ultrasound-guided peripheral catheter placement in emergency patients has been described as a less invasive alternative to venous cutdown or intraosseous access. The success rate of oblique (OBL) and out-of-plane (OOP) ultrasound-guided catheter placement in hemodynamically unstable dogs remains unclear.

Objective: To compare success and procedure-related adverse event rates, risk factors, and the effect of operator experience for OBL and OOP catheter placement in dogs with cardiovascular compromise.

Design: Prospective randomized clinical study

Setting: University teaching hospital

Methods: Dogs requiring peripheral catheter placement with a shock index > 1 or tachycardia and ≥ 2 altered perfusion parameters were prospectively enrolled. Dogs were randomized to OOP or OBL placement by trained clinicians. Catheter placement was considered successful if achieved within three attempts and five minutes. Data collected included success rate, attempts, time, procedure-related adverse events, and patient- and operator-related factors.

Results: Ninety dogs (mean age 7,4±4.0 years; mean weight 24.2±15.1 kg), were enrolled by 10 clinicians, 40 dogs underwent OBL, 50 OOP placement. Overall success rate (78.8%) did not differ significantly between techniques (OBL 73.0%; OOP 83.3%; p = 0,25). Mean number of attempts (OBL 1.5±0.76; OOP 1.4±0.70) and median time to placement (OBL 1.2min (0.62–4.5); OOP 1.1min (0.42–2.8)) in successful placements were not significantly different (p=0,79 and p=0,36, respectively). Failure was mostly due to time (15.4%), reaching maximal attempts (7.7%), or clinician decision (3.3%). The overall procedure-related adverse event rate was 37.8% and did not differ between techniques (OBL 40%; OOP 36%; p=0,7). Most common procedure-related adverse events were improper catheter positioning (24.4%) and hematoma formation (18.9%). Reduced patient

cooperation, increased perceived difficulty, higher number of attempts, and longer procedure time were associated with lower success and higher placement failure rates ($p \leq 0.014$). Success rates seemed to increase with clinician experience, from 54.2% for the first 3 cases to ≥84.6% after the fourth case.

Conclusion: In dogs with cardiovascular compromise, OBL and OOP techniques yielded comparable success and procedure-related adverse event rates. Success rates seemed to be influenced by operator experience.

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THE CLINICAL UTILITY OF FLOW-CYTOMETRY FOR EVALUATING IMMUNE-MEDIATED ETIOLOGY IN CATS WITH ANEMIA OR PANCYTOPENIA: A PILOT STUDY

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Introduction:

Bone marrow (BM) diseases resulting in anemia and pancytopenia in cats are numerous; however, discriminating between different causes presents a diagnostic challenge, often requiring invasive approaches including BM biopsy. Additionally, these cases may present with unstable disease, potentially requiring stabilization with blood transfusions prior to further diagnostic evaluation. Non-invasive analysis is, therefore, desirable to guide potential treatment. Flow-cytometric analysis of circulating red blood cell (RBC) populations is supportive for the diagnosis of immune-mediated hemolytic anemia in dogs, but its diagnostic utility has not been evaluated in anemic feline patients. This project aimed to investigate flow-cytometry for the identification of immune-mediated causes of anemia and pancytopenia in cats.

Methods:

A prospective pilot study was performed using residual blood samples from cats with non-regenerative anemia. Surface expression of anti-cat-IgG on peripheral RBC was determined using flow-cytometry, with threshold for positive staining confirmed using appropriate isotype controls. The proportion of cats demonstrating positive IgG expression were compared between groups with non-associative (primary) immune-mediated anemia and anemia of other etiology using Fisher's exact test; level of expression (percentage positive) was compared using Mann-Whitney U test.

Results:

Fifteen cats with non-regenerative anemia (5 male, 10 female) were included. Results of BM biopsy were available for 11/15 cats; if not performed or if results were non-diagnostic, final diagnosis at discharge was used. A non-associative immune-mediated cause was identified in 6 cats, with other reported pathologies including neoplasia (n=4), infection (n=2), aplastic anemia (n=1), BM hypoplasia (n=1), and pre-regenerative anemia (n=1). Anti-cat-IgG staining was more frequently detected in cats with non-associative immune-mediated anemia (6/6) compared with cats with anemia of other etiology (3/9; p =0.028). The percentage of RBC considered "IgG positive" was not significantly difference between groups.

Conclusions:

This study demonstrates the feasibility of flow-cytometry for the detection of circulating IgG-bound RBC in cats with non-regenerative anemia. In this cohort, anti-cat-IgG staining was sensitive for the identification of non-associative immune-mediated anemia (6/6 detected). Anti-cat-IgG staining was also detected in cats with other pathologies not expected to respond to immunosuppression, and

highlights the importance of appropriate screening for underlying infectious or neoplastic disease in such cases.

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SERIAL MEASURED AND CORRECTED PLASMA CHLORIDE IN HOSPITALIZED DOGS WITH DECOMPENSATED MYXOMATOUS MITRAL VALVE DISEASE

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Introduction:

Hypochloremia is associated with mortality in stable congestive heart failure (CHF) in dogs. Changes in plasma chloride concentration ($[Cl^-]$) in decompensated CHF are poorly described. Our primary objective was to describe serial measured and corrected $[Cl^-]$ in dogs hospitalized with decompensated CHF from myxomatous mitral valve disease (MMVD). Secondary objectives were to determine whether initial and serial $[Cl^-]$ are associated with survival to discharge, duration of hospitalization, and furosemide dose. We hypothesized $[Cl^-]$ would decrease during hospitalization, and lower initial and lowest $[Cl^-]$ would be associated with poorer outcomes.

Methods

This was a single-centre retrospective study at a tertiary referral centre. Cases were identified by furosemide fee code search and included if there were serial $[Cl^-]$ measurements and findings consistent with MMVD.

Baseline characteristics, patient outcome, and total duration of hospitalization were recorded. Cumulative furosemide dosage was calculated for every 24-hour period. All $[Cl^-]$ measurements were recorded, with the time relative to the first measurement. Corrected $[Cl^-]$ was calculated.

Change in $[Cl^-]$ over time and effect of furosemide dose were assessed with linear mixed effects models. Associations with mortality were assessed with logistic regression. Associations with duration of hospitalization were assessed with linear regression.

Results:

Overall, 298 $[Cl^-]$ measurements from 69 dogs were collected from 2020-2025. Both measured (-0.155mmol/L/h, $p < 0.001$, $R^2 = 0.284$) and corrected (-0.102mmol/L/h, $p < 0.001$, $R^2 = 0.182$) $[Cl^-]$ significantly decreased over time. Initial 24-hour cumulative furosemide dose significantly influenced this relationship for measured ($p < 0.001$), but not for corrected ($p = 0.347$) $[Cl^-]$.

Mortality was significantly associated with lower initial measured ($p = 0.030$) and corrected ($p = 0.027$) $[Cl^-]$. Mortality was not significantly associated with maximum decrease in measured ($p = 0.498$) or corrected ($p = 0.594$) $[Cl^-]$. Duration of hospitalization was significantly associated with maximum decrease in measured ($p < 0.001$) and corrected ($p < 0.001$) $[Cl^-]$, but not initial measured ($p=0.65$) or corrected ($p=0.20$) $[Cl^-]$.

Conclusion:

In decompensated MMVD, [Cl^-] decreases during hospitalization. Like stable CHF, initial [Cl^-] is associated with mortality. Ongoing case enrolment and investigation into [Cl^-] as a modifiable factor and effects of furosemide is warranted.

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PROSPECTIVE EVALUATION OF THE PREVALENCE AND CHARACTERISTICS OF HEART MURMURS AND GALLOP SOUNDS IN ANEMIC CATS

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Introduction:

Heart murmurs and gallop sounds are often found in anemic cats; however, their association with underlying cardiac disease is unclear. The objectives of this study were to determine the prevalence of heart murmurs and gallop sounds in anemic cats and to investigate the association of auscultatory abnormalities with anemia severity and the presence of heart disease.

Methods:

Single-center prospective observational study. Anemic cats (packed cell volume (PCV) <30%) were eligible for enrolment. On presentation cats were examined and had PCV measured. Each cat underwent echocardiography to assess for cardiac disease. Echocardiographic changes associated with anemia (left atrial and left ventricular dilation with normal wall thickness and systolic function) were not classed as cardiac disease. Cats that remained hospitalized had daily PCV checks and cardiac auscultation.

Results:

Sixty-four client-owned cats were included. 57.8% (37/64) cats had a heart murmur and 23.4% (15/64) had a gallop sound. Mean PCV at enrolment was 17.6% (± 6.37). Cardiac disease was present in 34.4% (22/64) cats. PCV was lower in cats with a murmur compared to those without a murmur (*P* = 0.026) and in cats with a gallop compared to those without a gallop (*P* = 0.006). After adjusting for PCV, the presence of cardiac disease was not associated with increased odds of having a murmur (*P* = 0.636 (OR: 1.31 [95% CI 0.43-3.95])) but was associated with increased odds of a gallop sound (*P* = 0.037 (OR: 3.97 [95% CI 1.09-14.5]). Murmur intensity (*P* = 0.001) and likelihood of a gallop (*P* = 0.022) decreased in individual cats when their PCVs were higher.

Conclusions:

Heart murmurs and gallop sounds are common in anemic cats with and without cardiac disease. Anemic cats with gallop sounds are more likely to have underlying cardiac disease than anemic cats without gallop sounds. This may warrant more cautious fluid administration in anemic cats with gallop sounds. An increase in PCV in anemic cats is associated with a decrease in both murmur intensity and likelihood of a gallop sound.

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EVALUATION OF A NOVEL SELF-FIXATING THORACIC ACCESS PORT FOR RAPID CHEST TUBE PLACEMENT: A PILOT CANINE CADAVERIC STUDY

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Introduction: Thoracic trauma, resulting from blunt force or penetrating mechanisms, is a common emergency in small animal medicine. Pleural air (pneumothorax) or blood (hemothorax) accumulation rapidly leads to severe respiratory and cardiovascular compromise. Conventional management includes needle thoracentesis and placement of thoracostomy tubes, however, these procedures have potential complications and accurate prognosis remains difficult. This preliminary canine cadaveric study evaluates the feasibility of the "C-Lant," a novel, disposable, self-fixating port designed for rapid insertion and replacement of standard chest tubes. This device aims to streamline the stabilization of patients with life-threatening pleural space disease while minimizing iatrogenic injury.

Method: Six fresh canine cadavers were included following owner consent. None had a history of thoracic trauma or chest compression (cardiopulmonary resuscitation). The cadavers were maintained under positive pressure ventilation to mimic physiological conditions. Using a randomized crossover design, a standard over-the-wire chest tube (OWCT) was placed in one hemithorax and the C-Lant device on the other. Deployment time was recorded from the start of insertion to complete fixation. Post-procedure thoracic radiographs were performed to verify tube positioning. Finally, gross necropsy was conducted to evaluate the anatomical placement and screen for any iatrogenic injury to intrathoracic organs.

Results: Six procedures were initiated; five procedures were completed; one C-Lant placement failed due to a technical malfunction. In all cadavers, insertion and securement time was shorter for the C-Lant device compared with the OWCT technique: median 63.5 seconds (range, 27-80), compared to 339 seconds (range, 193-435), respectively. With OWCT placement, diaphragmatic penetration occurred in 3/5 cases, lung lobe laceration in 2/5 cases, and liver lobe laceration in 1/5 cases. Only 1/5 OWCT placements were performed without detectable pathology. With the C-Lant device, correct intrathoracic positioning without gross injury to internal organs was achieved in 4/5 cases. In one case, although the device was positioned within the thoracic cavity, diaphragmatic penetration by the tube occurred, resulting in liver laceration.

Conclusion:C-lant is a novel device designed to facilitate rapid, safe, and straightforward insertion and fixation of chest tubes, potentially by inexperienced veterinary medical providers, while reducing the likelihood of procedure-related complications. Clinical studies are planned.

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NEW OPTIMISM IN CANINE HEMANGIOSARCOMA THROUGH IMPROVED DIAGNOSTIC AND THERAPEUTIC APPROACHES

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Introduction:
Canine hemangiosarcoma (HSA) has historically been viewed as almost uniformly fatal in dogs presenting with spontaneous hemoperitoneum, a perception largely driven by retrospective data subject to substantial bias. This has contributed to frequent preoperative euthanasia decisions and limited pursuit of definitive treatment. The objectives of this study were to reassess the true prevalence of HSA in dogs with hemoperitoneum using prospective data, evaluate the diagnostic utility of commonly employed preoperative staging modalities, and explore how emerging genomic insights may inform novel diagnostic and therapeutic strategies.

Methods:
Prospective data were collected from a nationwide, multicenter, randomized clinical trial (Ethos Precision Medicine Umbrella Study for Hemangiosarcoma: Ethos-PUSH). Dogs presenting with hemoperitoneum secondary to suspected splenic mass rupture underwent standardized staging diagnostics, splenectomy, and histopathologic confirmation. Diagnostic performance of preoperative staging tests was assessed, including thoracic radiography, echocardiography, and abdominal ultrasonography. Tumor and blood samples were collected for genomic analyses, including molecular subtyping and exploratory assessment of circulating tumor DNA. Dogs with confirmed HSA were randomized to one of four genomically matched treatment arms and followed for one year.

Results:
A total of 600 dogs were enrolled across 40 centers. HSA was confirmed histopathologically in 55% of cases, with 37% diagnosed as benign disease (222/600, 95% CI: 33% - 41%). Preoperative ultrasonographic liver lesions demonstrated poor positive predictive value for metastatic disease, with no significant association between liver lesions and liver metastasis ($p > 0.99$, PPV = 25%, NPV = 75%). Survival to hospital discharge following splenectomy was 95%, with a median hospitalization time of 39.5 hours. Genomic profiling identified at least four distinct molecular subtypes of HSA, supporting the rationale for precision-guided therapy. Early survival data from dogs receiving genomically-matched treatment suggests improved longer-term outcomes, with a greater proportion of dogs surviving beyond historically reported median survival.

Conclusions:

These findings challenge longstanding assumptions regarding prognosis in dogs presenting with hemoperitoneum and splenic masses, demonstrating a lower prevalence of HSA than historically reported and high perioperative survival following splenectomy. Integration of improved diagnostics and molecularly informed therapies may shift clinical decision-making away from premature euthanasia and toward personalized, life-extending treatment strategies.

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COMPARISON OF CADAVER-BASED TEACHING AND LIVE ANIMAL TEACHING ON SKILL RETENTION IN UNDERGRADUATE VETERINARY STUDENTS WHEN UNDERTAKING ABDOMINAL POINT OF CARE ULTRASOUND (POCUS)

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Introduction:

Point of Care Ultrasound (POCUS) is commonly used in primary care practice and POCUS is becoming an integral part of undergraduate education. Live animal usage for POCUS teaching raises ethical concerns. The 3R's model advocates for reduction or replacement of live animal teaching where possible. This study aims to assess equivalence of embalmed cadavers as a replacement for live animal teaching in abdominal POCUS.

Methods:

50 students recruited from first and second years of a veterinary undergraduate degree program were randomly assigned to receive live animal or embalmed cadaver training on abdominal POCUS for 1 hour with an instructor and written instructional material. Students undertook an Objective Structure Practical Exam (OSPE) 48 hours later. All students were 'POCUS Naïve' prior to the study and invited to complete a survey on teaching and ethical preferences.

OSPEs had 10 scoring points, correct probe position and diagnostic image acquisition for 4 sites – Diaphragmatico-Hepatic (DH), Spleno-renal (SR), Cysto-Colic (CC) and Hepato-Renal-Umbilical (HRU). Additionally, students identified the gall bladder and caudal vena cava.

Two blinded assessors observed OSPEs. Equivalence of OSPE scores between groups was evaluated using a non-parametric variant of the "two one-sided tests" (TOST) method, with a smallest effect size of interest of ±0.25 mark points. Chi-squared tests were used to assess differences between groups on individual scoring points.

Results:

44 Students successfully completed training on cadavers (n=23) or live animals (n=21) and OSPE.

Cadaver group mean score: 7.32/10 (SD: 1.93, range 3-10). Live animal group mean score: 7.40/10 (SD: 2.48, range 2-10). Results supported statistical equivalence between the two groups.

There were no significant differences between groups in any individual testing points ($p>0.05$).

43 Students completed the survey. 6/43 students agreed that live animal teaching should be minimised. 42/43 students agreed they were comfortable using cadavers for teaching.

Conclusion:

Cadaver teaching of abdominal POCUS produces equivalent skill retention as live animal. No sites were significantly advantaged by a method. Whilst few students expressed concerns about live animal usage, cadaver usage for POCUS training offers an alternative to live animal that aligns with educational best practices without compromising skill development.

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Oral Abstracts – Nurse & Tech Case Presentations, Saturday 6 June 2026

METHIOCARB INTOXICATION: A CASE REPORT

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Introduction:

Methiocarb intoxication is uncommon in small animal emergency practice. This case highlights the use of multimodal decontamination, advanced nursing care and intravenous lipid emulsion in the management of life-threatening carbamate exposure.

Synopsis:

Elsa, a 4-year-old spayed female Golden Retriever weighing 28kg, initially presented after vomiting blue material and was discharged following symptomatic treatment. She represented collapsed, obtunded and hypersalivating, with blue staining of her mouth, coat and perineum. Elsa was tachycardic and tachypnoeic, with pale mucous membranes, prolonged capillary refill time and weak pulses. Muscle fasciculations, vertical nystagmus and miotic pupils were present. Bilateral pulmonary crackles were auscultated, with concurrent hypoxemia. Elsa was positioned with her head and thorax elevated and provided oxygen via flow by, improving oxygenation. An intravenous catheter was placed, and minimum database bloods obtained. Continuous monitoring included electrocardiography, blood pressure and perfusion parameters. Metabolic acidosis with hyperlactatemia and hypotension were identified and a crystalloid bolus was administered via pressure bag. Due to obtundation, emesis was contraindicated. A rapid sequence induction enabled endotracheal intubation to protect the airway, and a dual lumen stomach tube was passed. Gastric lavage was performed using repeated warm water cycles, followed by activated charcoal administration. A stomach contents sample was obtained for toxicological analysis. Dermal decontamination, rectal lavage and an activated charcoal retention enema were undertaken to reduce toxin absorption. Intravenous lipid emulsion was administered as a bolus followed by a constant rate infusion. Post anesthesia, Elsa required high flow nasal oxygen therapy, antimicrobial cover and a metoclopramide constant rate infusion to promote gastrointestinal motility. Intensive care nursing included frequent repositioning, ocular and oral care, nebulization, assisted mobilization and monitoring of electrolytes, renal parameters and cardiovascular status. Elsa progressively improved and was discharged after five days.

Conclusion:

Laboratory analysis confirmed methiocarb exposure, a carbamate that reversibly inhibits cholinesterase activity through carbamylation. Atropine, a competitive muscarinic antagonist used to control cholinergic signs, was not required as the patient stabilized prior to confirmation of exposure. This case emphasizes the importance of airway protection, multimodal toxin elimination, vigilant monitoring, high-quality supportive care and the role of intravenous lipid emulsion in moderately lipophilic toxin exposure.

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A TALE OF TWO TESTICLES: IATROGENIC MISHAPS IN UNINTENTIONAL PROSTATECTOMY OF THE CRYPTORCHID PATIENT

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Introduction:

This case describes a complex postoperative ICU patient requiring advanced critical care nursing following severe iatrogenic urogenital complications. The case is notable for repeated surgical revisions, bladder necrosis, acute kidney injury and septic peritonitis and advanced critical care nursing.

Synopsis:

On 12/5/25 a 1 year 10 month old entire male husky presented through emergency via referral for clinical signs of vomiting, inappetence, and owner noted no urination observed following cryptorchid desexing on 9/5/25. Bloods at regular vet revealed severe renal azotaemia (Crea of 1700, SDMA of 71, Phos of 3, and USG 1.016). Was admitted to ICU for further investigation and supportive care. 13/5/25 patient diagnosed with absent prostate and ligation of prostatic urethra on imaging. The patient underwent speciality surgery for placement of a temporary cystostomy tube, JP drain and wound management of an inguinal incision. The patient commenced with peritoneal dialysis for anuria.

Intensive nursing monitoring included continuous assessment of heart rate, blood pressure, temperature, and perfusion parameters. Urine output was measured hourly via urinary catheter. Fluid therapy was adjusted to match ins and outs to maintain euvolaemia and support renal recovery. Electrolytes and biochemistry were assessed daily, with additional blood gas analysis during periods of instability.

Advanced nursing interventions included sterile management of central venous and urinary catheters, peritoneal dialysis exchanges, and measurement of JP drain output every 1 to 2 hours to detect ongoing leakage. Strict isolation protocols were implemented due to MRSP, including barrier nursing and dedicated equipment. Multimodal analgesia via constant rate infusions with adjunctive medications such as antiemetics, gastroprotectants, and enteral nutrition via oesophageal tube was provided, progressing from partial to full resting energy requirement.

Nurses closely monitored for systemic inflammatory response, hypotension and wound dehiscence. Gradual improvement was observed with decreasing drain output, normalising creatinine and return of voluntary appetite. The patient remains static IRIS AKI 1.

Conclusions:

This case highlights the critical role of vigilant fluid balance monitoring, early identification of postoperative complications, and strict infection control. Advanced nursing skills in dialysis

management, invasive line care and intensive monitoring were central to stabilisation and recovery in a complex ICU patient.

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POST–RETURN OF SPONTANEOUS CIRCULATION (ROSC) NURSING CARE OF A CRITICAL PATIENT

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Introduction:

Post–return of spontaneous circulation (ROSC) care is a critical phase influencing survival and neurological outcome following cardiac arrest. This case describes structured post-ROSC nursing management guided by the RECOVER algorithm, highlighting nursing priorities in haemodynamic stabilisation, respiratory optimisation and neuroprotection.

Synopsis:

A 6-year-11-month-old male entire French Bulldog was admitted for surgical removal of a liver tumour. Pre-anaesthetic assessment identified normal vital parameters and no significant comorbidities. Intraoperatively, approximately 50% circulating blood volume loss resulted in cardiac arrest. Open-chest cardiopulmonary resuscitation achieved ROSC and the patient was transferred to ICU for intensive nursing management.

Primary nursing goals focused on respiratory, cardiovascular and neurological body systems. The patient was comatose with an endotracheal tube in place. Flow-by oxygen supplementation was titrated to maintain SpO_2 between 94–98% to avoid hypo- or hyperoxia, while end-tidal CO_2 remained 30–33 mmHg without requiring intervention. Respiratory effort was monitored closely and intermittent airway suctioning was required. Additional nursing care included oral care, ocular lubrication and recumbency management to minimise secondary complications.

Haemodynamic optimisation proved challenging due to hypovolaemia and suspected myocardial dysfunction. Following arterial line displacement, non-invasive blood pressure monitoring and assessment of physical perfusion parameters were prioritised. Despite fluid resuscitation with stored frozen plasma totalling 20 ml/kg and isotonic crystalloid boluses, hypotension persisted. Post-ROSC lactate measured 16.04 mmol/L, decreasing to 12.57 mmol/L following fluid resuscitation. Vasoactive support included noradrenaline, with adrenaline introduced for additional cardiovascular support. Severe anaemia (PCV 10%) was treated with packed red blood cell transfusion and close monitoring for transfusion reactions. Central venous catheter management and frequent blood sampling were essential nursing interventions.

The patient remained comatose and was monitored closely for seizures. A single seizure was treated with intravenous midazolam. One-hour post-ROSC, Animal Functional Capacity score was 9, Neurological Deficit Score 260, and Modified Glasgow Coma Scale 4/18, indicating severe neurological impairment. Body temperature was 35°C and rewarming was not attempted. Given the guarded prognosis, humane euthanasia was elected

Conclusion:

This case highlights the complexity of post-ROSC nursing management in ECC patients. Key priorities included vigilant perfusion, respiratory and neurological monitoring with RECOVER-guided interventions.

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Poster Abstracts – Original Study

QUALITY CONTROL OF CANINE PLATELET CONCENTRATE PREPARED USING THE BUFFY COAT METHOD

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Introduction:

The buffy coat (BC) method to produce canine platelet concentrate (PC) units is widely used in human medicine but less reported in veterinary medicine. It involves initial high-speed centrifugation to isolate the buffy coat layer (containing leukocytes and platelets), followed by low-speed centrifugation to precipitate contaminated red blood cells (RBC), separating them from platelets (PLT) in suspension. Only one small canine study has been reported on PC preparation by BC, and no guidelines are yet available. Human AABB guidelines require PLT > 5.5 × 10^{10}/unit. This study aimed to describe the BC method for producing canine PC units, and report the obtained quality control results, comparing with the current bibliography.

Methods:

Data were obtained retrospectively from a blood bank program between February 2025 and January 2026. Donors were healthy mixed breed dogs, between 1 and 8 years old, weighing > 20 kg, vaccinated, dewormed, never transfused and PCR/ELISA negative according to the ACVIM donor screening consensus. Mean (SD) pre-donation PLT count was 221x10^3/µL (±71x10^3/µL). A total of 646 canine units of non-leukodepleted whole blood were processed, using the BC method: 2485g, 17 minutes, acceleration 210s and deceleration 720s, followed by 100g, 10 minutes, acceleration 30s and deceleration 300s. Quality control parameters included swirling evaluation, volume, bacteriological culture (Bact/Alert® 3D240), PLT count, residual WBC and RBC levels (Sysmex XN-1000V).

Results:

Of the 646 PC units, four were discarded due to positive bacterial cultures (0,59%). The identified microorganisms were *Staphylococcus pseudintermedius*, *Streptococcus canis*, *Streptococcus dysgalactiae*, *Pasteurella canis*, and *Streptococcus halichoeri*. All units had positive swirling and presented the following mean (SD) results: volume 48 (±5) mL; PLT 3.6×10^{10}/unit (±1×10^{10}/unit); WBC 0.08x10^3/µL (±0.16x10^3/µL); and RBC 0.06×10^6/µL (±0.11×10^6/µL).

Conclusion:

The BC method produces safe canine PC with low bacterial contamination and acceptable residual WBC and RBC levels. Platelet counts were higher than those reported by Hoareau et al. (2014), but below human standards. Lower residual WBC and RBC levels indicate improved leukoreduction and RBC removal, which is in line with AABB guidelines. Guidelines for canine PC are warranted and should reflect the difficulty of achieving human standards.

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CARDIOVASCULAR POINT-OF-CARE ULTRASOUND TO DIFFERENTIATE HEMODYNAMIC SHOCK STATES ASSOCIATED WITH GALLBLADDER WALL EDEMA IN COMPANION ANIMALS PRESENTED TO AN EMERGENCY SERVICE.

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Introduction:

Gallbladder wall edema (GBWE) is an ultrasonographic finding resulting from altered Starling forces in disorders such as anaphylaxis, cardiac tamponade and right-sided congestive heart disease, each of which can result in different hemodynamic shock states. Cardiovascular (CV) point-of-care ultrasound (POCUS) allows for the assessment of cardiac chamber and caudal vena cava (CVC) dimension and collapsibility, which vary depending on hemodynamic mechanisms. The aim was to report the incidence of GBWE on triage POCUS in a cohort of companion animals presented to an emergency service in Belgium and to determine whether CV-POCUS differentiates hemodynamic shock states associated with GBWE**.**

Methods:

Over a ten-month period, emergency clinicians of the University Veterinary Hospital of Liège (Belgium) performed thoracic, abdominal and CV-POCUS (contractility, wall chamber and lumen dimensions, CVC size and collapsibility) in companion animals at triage, guided by clinical questions related to the patients' condition, and recorded their findings. Cases with identified GBWE were retrospectively included. Collected data included clinical parameters, POCUS findings, final diagnosis and outcome. CV-POCUS findings and medical records were independently and blindly reviewed by two ACVECC/ECVECC specialists to determine the suspected hemodynamic shock state associated with GBWE. Disagreements were resolved by a third blinded specialist.

Results:

Sixteen patients (fifteen dogs, one cat) with GBWE were identified in 1376 assessed cases. Based on complete medical record review, specialists did not attribute 8/16 to a single mechanism, 4/16 as obstructive, 2/16 as distributive, and lacked enough data to make any call in 2/16 cases. All patients with obstructive disease had an enlarged, non-collapsible CVC and both patients with distributive disease had a collapsed, compliant CVC. Specificity of CV-POCUS to identify the hemodynamic mechanism was 100% for obstructive and distributive disease (95% CI: 73.5–100% and 76.8–100%, respectively). Sensitivity was 75.0% (95% CI: 19.4–99.4%) and 50.0% (95% CI: 1.3–98.7%) for obstructive and distributive disease. No significant association was detected between hemodynamic mechanisms and CV findings.

Conclusion:

GBWE seems rare in Belgium. CV-POCUS showed high specificity but poor sensitivity to identify hemodynamic mechanisms associated with GBWE. Larger studies are required to understand the clinical relevance of these findings.

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PREVALENCE OF INFECTIOUS DISEASES IN IRISH CLIENT-OWNED HEALTHY DOGS ELIGIBLE TO BECOME BLOOD DONORS

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Introduction

Vector-borne pathogens (VBPs) such as *Babesia* spp., *Ehrlichia* spp., *Anaplasma* spp., *Leishmania spp., Brucella* spp. *and Dirofilaria immitis* pose a recognized risk for transfusion-transmitted infections in dogs, underscoring the need for robust screening of donor candidates. Although Ireland has traditionally been regarded as a low-prevalence setting for many VBPs, evolving factors (e.g., pet travel, climate-driven vector expansion) may shift local epidemiology.

Objective

This retrospective study aimed to determine the prevalence of major VBPs and other transfusion-relevant infectious agents in Irish canine blood donor candidates using PCR and serological test.

Materials and Methods

A total of 2895 Irish client-owned healthy dogs were screened by real time PCR for*Babesia* spp., *Ehrlichia* spp., *Anaplasma* spp., *Leishmania spp. and Brucella* spp. (LightCycler 480II, Roche). Serology was performed for *Ehrlichia* spp.,*Leishmania* spp., and *D. immitis* (ELISA, Gemini Stratec, Novatec). Animals tested positive for RT-PCR and/or serology were excluded from the donor program.

Results:

All PCR assays were negative (0/2895), indicating an estimated molecular prevalence of 0% across all targets. Serologically, one dog was antibody-positive for *Ehrlichia* spp. (0.035%) and one dog was antigen-positive for D. immitis (0.035%). The dog that tested positive for *D. immitis* was tested again 2, 4, and 7 months after the positive result, with consistently negative results, suggesting the presence of an initial false positive.

Discussion

These findings demonstrate an exceedingly low burden of VBPs and other tested infectious agents in the Irish client-owned healthy dogs that are candidates for blood donation. The positive result for *D. immitis* observed in a dog, likely reflect cross-reactivity, assay variability or false positives, rather than established infection. Collectively, the data support the effectiveness of current donor selection and screening measures in Ireland and suggest a low transfusion-transmitted infection risk within this cohort. Continued surveillance remains prudent to detect potential epidemiologic changes and to sustain evidence-based transfusion safety.

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EVALUATION OF THE DELTA NEUTROPHIL INDEX (DNI) IN DOGS WITH SEPTIC SHOCK HOSPITALIZED IN AN INTENSIVE CARE UNIT (ICU)

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Introduction:

The DNI, automatically calculated by the ADVIA 2120i hematology analyzer, reflects the fraction of circulating immature granulocytes. Higher DNI values were reported in dogs with sepsis and septic shock compared to healthy dogs. The aim of this retrospective study in an ICU population was to corroborate the diagnostic performance of the DNI in canine septic shock.

Methods:

Dogs with at least a CBC including DNI measurement during ICU stay (January-December 2025) were reviewed. Septic shock was defined as a state of refractory hypotension requiring vasopressors support. DNI results (%) evaluated upon ICU admission and maximum DNI value measured during ICU stay were recorded and compared between dogs with septic shock and the rest of the ICU population. Data were reported as median and 95% Confidence Interval.

Results:

A total of 198 dogs were hospitalized in ICU: 13 out of 198 (6.6%) were diagnosed with septic shock upon ICU admission. Overall, 18 out of 198 (9.1%) developed septic shock during ICU stay. DNI results were significantly higher in dogs with septic shock (15.5%, 4.2-59.1; n=13) compared to the rest of ICU dogs (-1%, -1.9 to 0.2; n=185), upon admission. During ICU stay maximum DNI values were significantly higher in dogs with septic shock (11.1%, 7.3-45.9; n=18) compared to the rest of ICU dogs (0.5%, -0.9 to 1.5; n=180). A DNI value >2.7% upon ICU admission had a good diagnostic performance for septic shock (sensitivity 92.3% - specificity 76.2%, AUC=0.88; P<0.001). A maximum DNI value >6.6% during ICU stay confirm to have a good diagnostic performance for septic shock (sensitivity 77.8% - specificity 84.4%, AUC=0.87; P<0.001).

Conclusions:

The DNI can be considered for identifying dogs with septic shock in ICU. Further prospective studies are warranted to evaluate the applicability of DNI to predict outcome and response to treatment in septic dogs.

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CORRELATION AND AGREEMENT OF EXTERNAL JUGULAR AND CENTRAL VENOUS OXYGEN SATURATION MEASURED BY BLOOD GAS ANALYSER IN HOSPITALIZED DOGS

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Introduction:

Measurement of central venous oxygen saturation ($ScvO_2$) can aid in shock assessment in dogs. However, a central venous catheter is not always in place at the time that a patient is cardiovascularly unstable, and placement is not always be feasible during patient stabilisation. External jugular venous oxygen saturation ($SejvO_2$) may provide a less invasive and expedient surrogate measure. An experimental model in anesthetised dogs found strong correlation, but insufficient agreement between $SejvO_2$ and $ScvO_2$. The objective of this study is to assess the correlation and agreement of SejvO2 and $ScvO_2$ in hospitalized diseased dogs. We hypothesized that $SejvO_2$ and $ScvO_2$ have good correlation and agreement in hospitalized dogs.

Methods:

This prospective, observational, clinical study was performed at a tertiary referral emergency and critical care service. Paired measurement of $ScvO_2$ and contralateral $SejvO_2$ (ABL90 FLEX, Radiometer Copenhagen) collected within 5 minutes of each other was performed. Once daily repeated sampling was performed in dogs who remained hospitalized with a central venous catheter. Samples with discordant hemoglobin concentrations (>Δ5g/L) or clinically significant oximetry errors were excluded. Data on signalment and disease characteristics were collected.

A linear mixed effects model was created with fixed effect of $SejvO_2$ and random effect of individual dog. Correlation was assessed using Nakagawa's marginal r^2 for mixed models. Agreement was assessed using Bland-Altman method for repeated measures.

Results:

A total of 21 paired samples from 8 dogs have been collected thus far, of which 2 samples were excluded due to excessive hemoglobin difference. Dogs were hospitalized for a range of disease processes, with sepsis present in 3/8 patients. The linear model showed a significant fixed effect of $SejvO_2$ ($p < 0.001$). A moderate correlation between $SejVO_2$ and $ScvO_2$ was found ($r^2 = 0.61$). $SejVO_2$ underestimated $ScvO_2$ with a bias of -2.81% (95% CI, -8.29% to 2.67%), with limits of agreement from -18.89% (95% CI, -25.53% to -12.25%) to 13.27% (95% CI, 6.63% to 19.91%).

Conclusion:

$SejvO_2$ cannot be substituted for $ScvO_2$. Given moderate correlation, ongoing case enrolment with a pragmatic target sample size of 50 paired samples, and further investigation is warranted.

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EVALUATION OF APOLIPOPROTEIN A1 IN DOGS WITH SEPTIC PERITONITIS

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Introduction:

Circulating Apolipoprotein A-1 (ApoA-1) behaves as a negative acute phase protein (APP) during sepsis. Lower ApoA-1 has been documented in dogs with septic shock compared to uncomplicated sepsis, however, it was not able to predict survival in the same population. Dogs with septic peritonitis were reported to have reduced ApoA-1 and a high frequency of death in population of septic dogs of different origin. The aim of the study was to evaluate the diagnostic and prognostic significance of ApoA-1 in dogs with septic peritonitis. Our hypothesis was that a lower ApoA-1 is associated with the diagnosis of septic peritonitis and a worse prognosis.

Methods:

Dogs underwent surgical treatment for peritonitis, classified as septic or sterile based on chemistry analysis, cytology and/or culture of the abdominal fluid were retrospectively included. The development of septic shock and the final outcome were also recorded. ApoA-1 was measured upon admission by a validated immunoturbidimetric method. Data were reported as median and 95% confidence interval or range.

Results:

Forty-seven dogs were included in this study: 39/47 (82%) had a diagnosis of septic peritonitis, while 8/47 (18%) had a diagnosis of sterile peritonitis. Median ApoA-1 was significantly lower in dogs with septic peritonitis, compared to those with non-septic peritonitis (123, 92-137 vs 172, 118-256 mg/dL; p=0.03). According to the AUROC curve analysis, ApoA-1 ≤ 139 mg/dL had a fair accuracy (AUC 0.75) to correctly predict septic peritonitis (p=0.007). Moreover, ApoA-1 was significantly lower in dogs with septic peritonitis due to gastrointestinal perforation, compared to those with septic peritonitis from genito-urinary and unkown origins (110, 58-287 vs 173, 97-306 vs 148, 113-196 mg/dL, respectively; p=0.002). ApoA-1 in dogs who developed septic shock (116, 78-214 vs 132, 108-166 mg/dL; p=0.616), and in non survivors (124, 86-141 vs 136, 113-172 mg/dL; p=0.205) showed lower median values without reaching a statistical significance.

Conclusions:

ApoA-1 confirmed its behavior as a negative APP during canine peritonitis and might help in differentiating the septic nature of the disease. However, ApoA-1 was not able to predict development of septic shock or outcome in this population.

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PREVALENCE AND CHARACTERISTICS OF HOSPITAL-ACQUIRED (HAAKI) IN THE VETERINARY ICU POPULATION

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Intro: In human ICUs, acute kidney injury (AKI) is associated with increased morbidity and mortality. HAAKI reportedly occurs in 12% of dogs with sepsis. The wider prevalence of AKI across the veterinary ICU population is unknown. The aims of the study are to describe the prevalence of HAAKI in a canine and feline veterinary ICU population, determine its effect on outcome and explore associated risk factors.

Methods: This was a prospective observational cohort study with dogs and cats presented as an emergency and hospitalized in ICU for > 48h at a university teaching hospital. Patients without azotemia or with stable blood creatinine measurement within 6 hours of presentation and a minimum of 3 serial creatinine measurements were included. HAAKI was defined as an increase in blood creatinine ≥26.4µmol/l after 48h of hospitalization. AKI Grade was assigned as per the International Renal Interest Society guidelines. Survival to discharge and length of hospitalization were compared between HAAKI and non-HAAAKI patients. Reason for presentation (medical/surgical), procedures (sedation/general anesthesia, mechanical ventilation) and exposure to nephrotoxic drugs were recorded to explore potential risk factors for HAAKI. Data was reported as median and interquartile range. Fisher's exact and Mann Whitney were performed to compare the patient groups and relative risk calculation. P value < 0.05 was considered significant.

Results: Of 1154 ICU patients, 164 met the inclusion criteria (117 dog and 45 cats). Prevalence of HAAKI was 19.1% (31/162) where 83.9% (26/31) were Grade I, 9.68% (3/31) Grade II and 6.45% (2/31) Grade III. HAAKI was associated with non-survival (HAAKI 61% survived (19/31) whilst 83.9% (110/131) non-HAKKI survived, p=0.005). HAAKI was associated with longer ICU stay (HAAKI 98 hours [86-163], non-HAAKI 82 hours [65-117.5], p=0.023). The relative risk of mechanical ventilation for HAAKI was 3.27 (95% CI 1.65–6.46, p=0.004). The relative risk of sedation or general anesthesia was 3.43 (95% CI 0.86–13.6, p=0.046). The reason for presentation and nephrotoxic medication were not associated with HAAKI.

Conclusion: HAAKI occurs commonly in the canine and feline veterinary ICU population and has an effect on survival outcome. Further studies should explore potential risks factors, especially mechanical ventilation.

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MICROBIOLOGICAL ANALYSIS USING URISPONGE® TO PRESERVE URINE SAMPLES FROM SMALL ANIMALS IN THE EMERGENCY ROOM

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Introduction

Clinical signs of lower urinary tract disease (LUTD) frequently prompt emergency visits, being urine cultures essential for diagnosing UTI. Guidelines recommend immediate processing or refrigeration with culture within 24 hours, which is often impractical. This study evaluates UriSponge® for preserving urine samples for 48 hours at room temperature.

Methods

Samples were collected by ultrasound-guided cystocentesis. Each sample was split for the Clinical Biopathology (one aliquot) and Microbiology and Parasitology labs (three aliquots). All samples were streaked in blood agar plates within a laminar flow hood, first one (control) right after harvesting and incubated at 37°C; second was moved to Axygen® microbiology tubes, where the sponge in the UriSponge® device's applicator was inserted and left at room temperature for forty-eight hours; the thrid fraction was stored refrigerated in the collection syringe for the same period. All plates were examined every 24 hours. When sample volume was limited, test strip results were compared before and after sponge contact to ensure preservatives did not affect parameters. Isolated microorganisms were identified using MALDI-TOF technique. To examine associations between categorical variables a chi-square test procedure or Fisher´s exact test were used. Significance was set at $p < 0.05$.

Results

A total of 20 samples, sixteen dog and four cats with LUTD, were harvested. There was perfect agreement (positive or negative culture results) between control and refrigerated samples (p<0,001), control and UriSponge samples (p<0,001), and refrigerated and UriSponge samples (p<0,001). Most commonly recovered microorganism were, *Escherichia coli* (n =4), *Klebsiella spp*. (n=2), *Streptococcus galollyticus* (n=1) and *Staphylococcus pseudointermedius* (n=1). Pathogen could not be identified in one case. No changes were observed in the test strip parameters after contact with the preservatives in the UriSponge® sponge (n=5).

Conclusions

Results show that bacterial cultures using urine samples collected by cystocentesis and stored in the UriSponge ® device at room temperature or refrigerated for 48 hours are reliable. Urine samples can be preserved using the UriSponge® system if a cooling device is unavailable or must be transferred. Urine can be biochemically examined in volume samples after coming into contact with the preservatives in the UriSponge®.

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ANALYSIS OF TARTARIC ACID CONTENT IN VITIS VINIFERA FRUIT (VVF) VARIETIES AS A PREDICTOR OF TOXICITY IN DOGS.

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Introduction: Fresh grapes as well as dried grapes (raisins), cumulatively recorded as *Vitis vinifera* fruit (VVF), are a known toxin for canines. VVF ingestion and subsequent toxicity can lead to acute kidney injury. Decision making in such cases is complicated by individual variations in response to VVF ingestion, plus financial and ethical concerns around recommended treatments. Recent research suggests tartaric acid (TTA) may be the causal toxin, and hypotheses suggest cooking VVF at high temperatures may reduce TTA concentration in foodstuffs. Objectives of this pilot study were a) determine tartaric acid (TTA) content of a range of VVF, and b) assess the impact of cooking (as ingredients within baked goods) on TTA content reduction.

Methods: Representative samples of grapes, raisins, and cooked raisins (e.g. in scones and tea loaf) were homogenized in orthophosphoric acid and TTA measured by high performance liquid chromatography. Grape varieties were compared based on their size, color, seeded vs unseeded, and sourcing (commercial e.g. supermarket vs non-commercial e.g. private residence).

Results: TTA content of fresh VVF ranged from 4.54 - 8.08 grams per kg of VVF fresh weight (5.95 +/- [0.89] g/kg, mean [SD]). Dried VVF had approximately five times greater TTA content (28.84 +/- [3.05] g/kg mean [SD]). Non-commercial and seeded grapes appeared to have different TTA content relative to commercial and unseeded grapes. Dried VVF within baked goods cooked at high temperatures (200°C) recorded similar TTA content as un-cooked fruit, suggesting no temperature-sensitive breakdown of TTA occurred.

Conclusion: TTA content in VVF appeared to vary across some of the factors investigated. Knowledge of these factors at diagnosis could facilitate better decision making. The lack of TTA breakdown in baked goods suggests thermal decomposition of TTA is unlikely to be the cause of reduced toxicity seen in cases ingesting these products. Expanding the findings from this pilot study to include assessment of VVF from confirmed cases of VVF toxicity may provide stronger evidence for potential risk factors.

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OPTIMIZING SEDATION IN FELINE BLOOD DONATION: THE ROLE OF ORAL GABAPENTIN PREMEDICATION

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Introduction:

Sedation is often necessary to ensure the well-being of the animals and the safety of the blood donation procedure.

Oral gabapentin is increasingly used as premedication due to its anxiolytic and mild sedative effects, and also low incidence of adverse effects. However, evidence regarding its impact on IV sedation requirements in feline blood donor programs is limited. This study aimed to evaluate the relationship between gabapentin premedication and the required dose of IV sedation.

Methodology:

A retrospective analysis was performed on 2,100 blood donations from 1,357 indoor, privately owned cats between February 2025 and January 2026. The study population consisted predominantly of female (52%) and domestic shorthair (64%) cats, with a mean age of 4 years (range between 1 and 8 years) and a mean body weight of 4.9 kg (range between 3 and 9 kg)..Donation events were divided into two groups: cats receiving gabapentin premedication (100 mg/cat, administered 90 minutes before donation; n = 267) and cats not receiving gabapentin (n = 1,824). The recorded IV sedative dose in each donation event corresponded to the total administered dose, including any additional boluses when required. Intravenous doses (mg/kg) of ketamine, butorphanol, and diazepam were compared between the two groups using independent-samples t-tests with Welch's correction. All tests were two-tailed.

Results:

Oral gabapentin premedication was associated with a significant reduction in the required IV doses of all evaluated sedative agents. The mean (±SD) ketamine dose was significantly lower in the gabapentin group (0.55 ± 0.19 mg/kg) than in the non-gabapentin group (0.70 ± 0.19 mg/kg; $p < 0.001$). Similarly, the mean butorphanol dose decreased (from 0.029 ± 0.010 to 0.037 ± 0.010 mg/kg; $p < 0.001$), as did the mean diazepam dose (from 0.11 ± 0.04 to 0.14 ± 0.04 mg/kg; $p < 0.001$). Effect sizes were large for all comparisons (Hedges' g = -0.76 ; 95% CI, -0 .89 to -0.63).

Conclusion:

Oral gabapentin premedication was associated with lower IV doses of sedatives during feline blood donation. Overall sedation need depends on donor reactivity during the donation, meaning the bias of potential clinician-driven IV sedatives dose adjustments in gabapentin group is unlikely.

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PREVALENCE OF DIROFILARIA IMMITIS IN A POPULATION OF FELINE BLOOD DONORS

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Introduction:

Feline heartworm infection is characterized by the presence of migrating larvae or adult *Dirofilaria* spp. in host tissues or the pulmonary vasculature. *Dirofilaria immitis*, the primary causative agent of feline heartworm disease, is a zoonotic, mosquito-borne filarioid with a worldwide distribution. While felines contribute to a lesser extent to the transmission of parasites compared to canines, infections do occur and are frequently underdiagnosed due to the low burden of parasites, the transient or absent nature of microfilaremia, and the nonspecific or subclinical nature of clinical signs. The diagnosis is further complicated by delayed or inconsistent antigenemia, particularly in cases of immature or unisex infections. Given the potential for *D. immitis*–infected donors to pose diagnostic, vector, and health risks, screening and prophylaxis are recommended in endemic areas. The objective of this study was to ascertain the prevalence of heartworm antigen in clinically healthy cats enrolled in a feline blood donor program.

Methods:

A retrospective observational analysis was conducted using data from a feline blood donor program. The study population comprised clinically healthy cats, who underwent screening using the SNAP® Feline Triple Test (sensitivity 90.2%, specificity 100%). The primary focus of this test was the detection of *Dirofilaria immitis* antigen. A total of 1,210 samples were collected from 978 cats between February and July of 2025 for the purpose of analysis.

Results:

Of the 1,210 samples that were examined, none exhibited a positive result for the presence of *Dirofilaria immitis* antigen.

Conclusions:

During the study period, no evidence of circulating *Dirofilaria immitis* antigen was detected among clinically healthy cats enrolled in this feline blood donor program. Notwithstanding the 90.2% test sensitivity, the present study indicates an exceedingly low prevalence of detectable heartworm infection within the specified population. However, given the known limitations of antigen testing in cats, particularly in cases of low parasite burden or immature infections, the absence of positive results should be interpreted with caution.

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POST-TRANSFUSION GALLBLADDER WALL THICKENING IN DOGS: A PROSPECTIVE ULTRASONOGRAPHIC STUDY

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Introduction:

Gallbladder wall thickening has been suspected to occur following blood transfusion in dogs. However, this association has never been previously described. The aim of this study was to assess ultrasonographic changes in the gallbladder wall of dogs before and after packed red blood cell (pRBC) transfusion.

Methods:

Client-owned dogs with no ultrasonographic signs of biliary disease receiving pRBC transfusions were prospectively enrolled over a one-year period. A focused ultrasonographic examination using standardised gallbladder wall measurements was performed immediately before, 1h, 12h and 24h post-transfusion, if still hospitalised. Gallbladder wall thickening was defined as >1.3mm.

Results:

Twenty pRBC transfusions administered to sixteen dogs (10 females, 6 males; age range 1-14 years) were included. The underlying cause of anaemia included immune-mediated haemolytic anaemia (n=4), precursor-targeted immune-mediated anaemia (n=4), gastrointestinal haemorrhage (n=4), leukaemia (n=1), immune-mediated thrombocytopenia (n=1), hemophagocytic histiocytic sarcoma (n=1), and acute haemorrhage of unknown cause (n=1). Eleven dogs were DEA1-positive and five DEA1-negative. Follow-up ultrasonographic assessments were available at 1h (20/20), 12h (19/20) and 24h (9/20) post-transfusion.

Gallbladder wall thickening (GBWT) (0.31cm; range 0.21-0.48cm) was documented following 4/20 (20%) transfusions and was detectable 1h post transfusion in all four of them. A median decrease of 32% was documented at 12h (0.21cm; range 0.13-0.25cm), with normalisation documented at 24 hours in the single case with available follow-up (0.1cm). Two GBWT events were documented for the same dog, occurring during separate transfusions from different donors five months apart. Transfusion volumes in GBWT cases ranged from 13-23mL/kg, compared with a median of 15,8mL/kg (range 8.6-30.3mL/kg) in the overall group. One GBWT case received two half-units from two different donors and developed signs of an early transfusion reaction including pigmenturia, hyperthermia, and aural erythema. Another GBWT case developed diarrhoea shortly after transfusion. Two GBWT cases had received prior transfusions, compared with 10/20 in the overall population.

Conclusions:

Gallbladder wall thickening was identified ultrasonographically following 20% of pRBC transfusions, detected within one hour of transfusion completion and showing progressive resolution over 24h. To the authors' knowledge, this is the first description of gallbladder wall thickening following pRBC transfusion in dogs.

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READY TO RESPOND? EUROPEAN VETERINARIANS' PERCEIVED PREPAREDNESS FOR EMERGENCY CASE MANAGEMENT

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Introduction:

Out-of-hours periods account for more than 50% of the regular workday, rendering veterinary emergency services a core component of small animal practice. This survey aimed to evaluate veterinarians' perceived preparedness to manage emergency cases in daily practice.

Methods:

A Europe-wide, anonymous online-survey was conducted among small animal veterinarians from November 2025 until March 2026. The questionnaire addressed the perceived integration of emergency medicine in undergraduate curricula, self-assessed preparedness to manage emergency cases, opportunities for continuing education, and the organization of emergency services. Preliminary data are presented based on 734 responses available at the time of analysis (data cut-off: January 12, 2026).

Results:

Overall, 79% of respondents rated knowledge in emergency medicine as relevant or very relevant for their daily clinical practice. 83% reported feeling not at all or only minimally prepared to manage emergency cases upon graduation, and 72% perceived undergraduate hands-on training in emergency medicine as absent or limited.

In their current professional roles, 28% of respondents reported confidence in their theoretical knowledge but identified insufficient practical experience as a major limitation.

Among veterinarians aged ≤28 years, 22% reported frequently to very frequently reaching their professional limits when managing emergency cases, despite having full case responsibility. Additionally, 34% of veterinarians in this age group reported feeling predominantly to consistently highly burdened during emergency shifts.

The most frequently reported barrier to pursuing further training in emergency medicine was lack of time (44%), followed by insufficient financial support from employers (17%).

Telephone-based professional consultation services were considered a meaningful support option by 42% of respondents.

Conclusion:

Despite the high perceived relevance of emergency medicine in small animal practice, veterinarians report substantial gaps in preparedness at the time of graduation, largely attributable to insufficient practical training and limited acquisition of day-one clinical skills. These deficits contribute to

professional insecurity and elevated stress levels, particularly among younger veterinarians, who often assume primary responsibility for emergency cases. These findings highlight the need for improved practical training and supportive structures to strengthen emergency care competence in veterinary practice.

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KARAKA BERRY TOXICOSIS IN DOGS: A CROSS-SECTIONAL SURVEY AMONG VETERINARY PRACTITIONERS IN AOTEAROA-NEW ZEALAND

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Introduction:

Karaka berry toxicosis (KBT) occurs after ingestion of *Corynocarpus laevigatus* (Karaka) fruit that contain karakin. Karakin is metabolized to 3-nitropropionic acid, an inhibitor of succinate dehydrogenase. 3-Nitropropionic acid disrupts mitochondrial function which can lead to severe neurological signs. It is well recognized in Aotearoa-New Zealand, where the tree is endemic. However, systematic empirical data on epidemiology and clinical management are lacking. We aimed to report New Zealand veterinarians' experiences with KBT in dogs, through a nationwide survey.

Methods:

An internet-based cross-sectional survey, approved by the Massey University Human Ethics Committee (ID: 4000028509) was performed among New Zealand veterinarians between January and May 2025. Questions addressed participant demographics, incidence, seasonality, diagnosis, clinical signs, management approaches, and outcomes.

Results:

Ninety-five veterinarians responded, 54.4% had managed KBT cases in the past, most commonly in the Auckland region and in emergency and critical care settings. Most cases were observed in summer, the season the tree fruits (27/40; 67.5%; *p<0.001*). Diagnoses relied primarily on ingestion history (37/40; 92.5%) and clinical signs (36/40; 92.5%). Spontaneous vomiting was reported as the initial clinical sign of KBT (15/15; 100.0%; *p<0.001*) after which neurological signs developed in most cases (33/34; 97.1%). Particularly, weakness (23/30; 76.7%), altered mentation (22/30; 73.3%) and non-ambulatory status (19/30; 63.3%) were reported as prevalent neurological signs. Nearly half of the respondents (14/30; 46.7%), who observed neurological signs reported episodic seizures. Gastrointestinal decontamination consisted mainly of induction of emesis (26/28; 92.9%) and activated charcoal administration (20/28; 71.4%). Intravenous fluids (35/36; 97.2%) and seizure control (29/36; 80.6%) were the main supportive measures. The reported survival rate was 85.2%.

Conclusion:

This study provides a comprehensive insight into different key aspects of KBT in dogs. Karaka berry toxicosis seems to present with initial gastrointestinal signs progressing to neurological dysfunction. While survival is relatively high, the risk of severe neurological signs highlights the need for early recognition and intervention. These results could serve in educating New Zealand veterinarians on KBT in dogs and provide ground for further research.

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RESILIENCE AND PSYCHOLOGICAL DISTRESS: A LONGITUDINAL STUDY OF WELL-BEING IN HEALTH PROFESSIONS STUDENTS

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Introduction:

Health Professions Students (HPS) in academically challenging graduate programs are at high risk of burnout, depression, and other mental health concerns. Struggles with mental health may have short-term impact on academic performance and student well-being but can also have long-lasting influences on career trajectory and patient care. These challenges affecting our students are multifactorial

and require consideration of individual, social, structural, and institutional factors. While the body of research on HPS is expanding, their wellbeing remains a relatively understudied area. The goal is to identify when students experience psychological distress during their education and which variables, if any, are associated. The findings from this study can identify both risk and protective factors for student success and well-being that may be further explored for future interventions.

Methods:

An anonymous, voluntary survey of 18 psychosocial measures was distributed to UC Davis medical, veterinary, physician associate and nurse practitioner students annually from matriculation (2019) to graduation (2023). The survey included validated tools to measure wellbeing, resilience, and adverse childhood experiences (ACEs). Participants received a nominal gift card to incentivize their particiopation.

Results:

Students across all 3 programs (N = 240) entered with a mean psychological distress score of 2.13/7 with the largest increase in scores from matriculation to year 2. Most students (93%) met the threshold of severe distress at least once during the study. Multiple regression models identified a significant, linear increase in psychological distress over time ($p < .0001$). Multiple linear regression models did not find any significant association between social disadvantage variables and psychological distress. Significant differences were observed across all five timepoints among the three ACEs groups (ACES=0, 1-3 and ≥4) (p-values ranging < 0.0001 to 0.0146). Linear regression

analysis of resilience and psychological distress at matriculation showed a significant negative relationship ($p < 0.0001$).

Conclusions:

Psychological distress is prevalent and worsened over the course of study for health profession students. The largest increased occurred from baseline to year 2. High psychological distress was associated with high childhood trauma (ACEs) and low resilience, but not gender, race/ethnicity, socioeconomic, or first-generation status.

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POINT-OF-CARE ULTRASOUND FOR IDENTIFYING GASTROINTESTINAL OBSTRUCTIONS IN CATS

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Introduction

Identifying gastrointestinal (GI) obstruction in vomiting cats remains challenging for less experienced veterinarians. Using standardized Point-of-Care Ultrasound (POCUS) may improve diagnostic accuracy. Aim of the study was to evaluate a GI-focused POCUS protocol to identify GI-obstructions in vomiting cats.

Material and Methods

This prospective observative study enrolled cats with acute vomiting presented to the emergency service. Cats with known extra-intestinal causes of vomiting were excluded. A modified A-POCUS protocol with five views was applied to assess gastrointestinal dilatation, motility, diameter, wall thickening, peritoneal echogenicity and free fluid. Obstruction was confirmed surgically, non-obstruction by follow-up. Obstructive cases were subclassified into regular obstructions (RO) and linear-foreign-bodies (LFB). Data were analyzed using Mann-Whitney U-test and Fisher exact test. P-values ≤0.05 were considered significant.

Results

One hundred and ten cats (39 obstructed; 71 non-obstructed) were included. The subgroup RO (27/39) consisted of foreign bodies (24), intussusception (2) and neoplasia (1). The subgroup LFB included 12 cats. All patients were correctly evaluated for obstruction with the applied protocol. Obstructed cats were younger (2.0, 0.3–12.0 years), more often painful (51.3%) and had rarely diarrhea (2.6%) compared to non-obstructed cats (years 4.0, 0.1–18.0, p=0.016; painful 28.2%, p=0.022; diarrhea 20%, p=0.031). Obstructive cases had a higher amount of sonographic abnormal views (3, 1–5), more often increased gastric filling (17/39), increased small-intestinal filling at the hepato-renal and umbilical view, intestinal hypermotility at minimum one probe position (28/39), pendular movement (19/39) and peritoneal hyperechogenicity (33/39) compared to non-obstructed cases (abnormal views 1, 0-5, p<0.001; gastric filling 11/60, p=0.003; small-intestinal filling, p<0.001; hypermotility 6/71, p<0.001; pendular movement 0/71, p<0.001; peritoneal hyperechogenicity 14/39, p<0.001). Gastric filling (3/12), occurrence of hypermotility (4/12) or pendular movement (1/12) was not different, but peritoneal hyperechogenicity was more common (12/12) in LFB compared to non-obstructed cases. The occurrence of peritoneal hyperechogenicity or pendular movement was 92% sensitive and 90% specific differentiating any obstruction from non-obstruction, and moderate sensitive (81%) and specific (80%) to differentiate RO from non-obstruction. Peritoneal hyperechogenicity was 100% specific but 80% sensitive differentiating LFB from non-obstruction.

Conclusion

The evaluated protocol offers a reliable tool for diagnosing GI-obstructions in vomiting cats.

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INCIDENCE, TIMING, RISK FACTORS AND OUTCOMES OF VOLUME OVERLOAD IN ANEMIC CATS RECEIVING PACKED RED BLOOD CELL TRANSFUSIONS

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Introduction:

Volume overload (VO) is a poorly characterized complication of fluid therapy in cats. Transfusion associated circulatory overload (TACO) is defined as VO occurring within 6 hours of transfusion. We hypothesized that TACO is common in anemic cats and can occur beyond 6 hours of transfusion. The aim of the study was to determine the incidence, timing, risk factors and outcomes for VO in cats receiving packed red blood cell transfusions (pRBC).

Methods:

Retrospective data was collected from a university teaching hospital from May 2022 to July 2025. Cats were categorized by cause of anemia, and VO was determined based on clinical signs, point-of-care ultrasound findings, and response to diuretics. The incidence and timing of VO (before, within 6 hours, and beyond 6 hours of transfusion) were calculated. Risk factors and survival were analyzed using univariate logistic regression and hospitalization duration was analyzed by student t-test.

Results:

In total 198 cats received 294 pRBC transfusions. The incidence of TACO within 6 hours of transfusion was 5.1% (15/294 transfusions). VO beyond 6 hours of finishing a transfusion was observed in 46/294 transfusions (15.6%). Cats with overload at presentation (n=18) were 4.34 times more likely to develop VO in hospital (95% confidence interval [CI] 1.4-14.04, p= 0.003), as were cats with a gallop soundon auscultation (OR 4.67 [CI] 2.07-10.87, p=<.0001). Body condition score, presence of a heart murmur, rate and number of transfusions, fluids prior to transfusion, severity and cause of anemia were not associated with development of VO. VO was associated with longer hospitalization times (6.5 ± 3.2 vs 4.9 ± 2.7 days; p=0.0003) but not survival (29% vs 32%, p= 0.58).

Conclusions:

VO was commonly encountered beyond 6 hours of transfusion. As such TACO can occur later than previously reported and its timing may need to be expanded. Many anemic cats showed evidence of VO prior to pRBC transfusion indicating they may be a volume intolerant patient group.

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RETROSPECTIVE EVALUATION OF BLOOD-PATCH PLEURODESIS FOR THE TREATMENT OF PERSISTENT PNEUMOTHORAX IN 35 DOGS

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Introduction:

Blood-patch pleurodesis (BPP, injection of fresh blood into the chest cavity), has been anecdotally reported as a treatment modality for pneumothorax in dogs. The objectives of this study were to evaluate the efficacy and clinical outcomes of BPP in a large cohort of dogs and compare them to dogs with pneumothorax managed without BPP.

Methods:

Medical records of 98 dogs diagnosed with pneumothorax were retrospectively reviewed. 35 dogs underwent BPP, while 63 served as controls.

Results:

Dogs treated with BPP were significantly older and more likely to present with dyspnea or cyanosis, undergo CT, require thoracostomy-tube placement and/or continuous suction, have pulmonary bullae, and experience a longer hospitalization period compared with controls ($P<0.05$ for all). The median time from admission to BPP was 3 days (range, 1-6). BPP was performed bilaterally in 13 dogs (37%) and unilaterally in 18 (63%); one, two, or three procedures were performed in 23, 11, and one dog(s), respectively. Blood source was documented in 29 dogs and was autologous in 21 (72%), and allogeneic in eight (28%). The mean administered blood volume was 9mL/kg, divided equally for bilateral procedures. Exploratory-thoracotomy and lung lobectomy were performed in 20/35 BPP dogs (57%), including seven dogs that received BPP postoperatively. Overall survival-to-discharge was 87% (84/98), with no difference between groups. Following BPP, 25 dogs (71.5%) exhibited partial or complete cessation of air leakage, whereas the procedure was unsuccessful in nine dogs (25.7%); efficacy was undocumented in one dog. Long-term success (>20 days without recurrence) was observed in 20 dogs (57%), and short-term success (1-20 days) in five dogs (14.7%). Among dogs with long-term success, 16 (80%) had no recurrence.

Conclusions:

Blood-patch pleurodesis represents a viable and cost-effective option for managing persistent pneumothorax when surgery is delayed or unfeasible, or as a complementary treatment to surgical intervention; However, short- and long-term recurrence rates are substantial and surgery remains the gold-standard for definitive management. Careful case selection is essential to optimize BPP

efficacy. Delaying BPP for at least three days, particularly in traumatic cases, may allow spontaneous resolution. Overall, long term prognosis was favorable.

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COMPARATIVE EVALUATION OF A NEW POINT-OF-CARE BLOOD ELECTROLYTE ANALYZER IN THE VETERINARY SECTOR

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Background:

Electrolyte monitoring is important in companion animal medicine for characterizing and managing metabolic, gastrointestinal, renal, and endocrine disorders. Access to a point-of-care (POC) electrolyte analyzer that is simple to use and generates rapid results is vital in the veterinary emergency and critical care setting. This study aims to assess the performance of the EXIAS e|1^{vet}, a new direct ion-selective electrode POC electrolyte analyzer and compare it with other market-leading veterinary electrolyte analyzers in order to verify its accuracy, reliability and consistency.

Methods:

Residual whole blood samples anticoagulated with lithium heparin from a total of 50 cats and dogs from a primary care veterinary practice were evaluated following established method comparison guidelines (CLSI EP09c). The reference method for analyzing sodium (Na^+), potassium (K^+), and chloride (Cl^-) was the Catalyst One Chemistry Analyser (IDEXX). Ionized calcium (Ca^{2+}) and pH could not be measured using the Catalyst One, therefore the comparator used for assessing these parameters was the epoc Blood Analysis System (Siemens Healthineers). Correlation between results was statistically analyzed using Deming regression, Bland-Altman plots, and Pearson's R^2.

Results:

The EXIAS e|1^{vet} generated results within 25 seconds, used 20 microliters of whole blood and required one 30-second calibration every 12 hours. The Catalyst One generated results within 10 minutes using 700 microliters of blood and no ongoing calibrations. The epoc required a 165 second calibration before every sample, generating results in 40 seconds using 92 microliters of blood.

Preliminary results from the EXIAS e|1^{vet} POC analyzer demonstrated a strong correlation with those from the IDEXX Catalyst One Chemistry Analyser for Na^+, K^+, and Cl^-. The EXIAS e|1^{vet} Ca^{2+} and pH results were also well correlated with the epoc Blood Analysis System, with some discrepancies at extreme values of the available reference range. Analysis of the n=50 samples for further validation is due to be completed by early spring 2026.

Conclusion:

The EXIAS e|1^{vet} achieves accurate and consistent results when compared with other market-leading electrolyte analyzers and requires a much smaller sample volume and decreased analytical time. The

EXIAS e|1^{vet} therefore represents an excellent solution for electrolyte monitoring in small animal veterinary practice.

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PREVALENCE AND DETERMINANTS OF SYSTEMIC HYPERTENSION IN DOGS WITH ANEMIA

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Introduction

Systemic hypertension is often observed in anemic dogs, but this association remains poorly documented. This study aimed to determine its prevalence and identify associated factors. We hypothesized that hypertension may result from mechanisms related to the underlying cause of anemia or from compensatory responses to reduced arterial oxygen content.

Methods

Retrospective study (2010 to 2025). Dogs were eligible if they had anemia (hemoglobin <12 g/dL) and at least one recorded systolic arterial pressure; cases were excluded when anemia was multifactorial. Associations between hypertension and categorical variables were assessed using Fisher's exact test, and residual analysis was used to identify over-represented diseases. Univariable linear regressions were used to assess the association between systolic arterial pressure and individual predictors. A multivariable linear regression model was then built, including both categorical and continuous variables. Homoscedasticity and normality of residuals were evaluated.

Results

Among 151 eligible dogs, 147 were analyzed after exclusions. Hypertension was identified in 44 dogs (30%), including 18 (12%) with moderate hypertension and 26 (18%) with severe hypertension. Hypertension was overrepresented in dogs with leptospirosis, immune-mediated hemolytic anemia, and chronic kidney disease. Analyses were therefore conducted in the overall population and in the subgroup composed of dogs with these diseases. In univariable analyses, systolic arterial pressure was significantly associated with sterilization status (β = –17.7 mmHg, 95% CI –35.1 to –0.37; p = 0.045) and with hematocrit (β = +1.54 mmHg per %, 95% CI 0.23 to 2.86; p = 0.022) in the subgroup. No significant associations were identified with sex, body weight, age, duration of anemia, temperature, or leukocyte count. In the multivariable model, no variable remained independently associated with systolic pressure in the overall population, whereas hematocrit was the only independent predictor in the subgroup (β = +1.49 mmHg per %, 95% CI 0.23 to 2.93; p = 0.034).

Conclusion

Hypertension was most frequent in dogs with leptospirosis, immune-mediated hemolytic anemia, and chronic kidney disease. In this subgroup, systolic pressure was associated with both hematocrit and sterilization status. These findings suggest that hypertension may relate more to disease-specific mechanisms than to anemia itself.

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IMPACT OF VASOPRESSOR ADMINISTRATION ON PERIPHERAL PERFUSION ASSESSED BY TEMPERATURE GRADIENT DURING SEVERE HEMORRHAGIC SHOCK

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Introduction

Vasopressor use during hemorrhagic shock remains controversial. Although these agents can restore macrocirculation and reduce fluid requirements, they may impair perfusion. Temperature gradients measured by infrared thermography provide a surrogate marker of peripheral perfusion. This study evaluated, in a porcine model of severe hemorrhagic shock, the effects of vasopressin and norepinephrine on peripheral perfusion during resuscitation. We hypothesized that vasopressor administration would not be associated with significant hypoperfusion.

Methods

The study included 15 pigs and followed ethical guidelines for animal experimentation. The experimental protocol was approved by the ethics committee (APAFIS#48007-2024021917014478v5). Hemorrhagic shock was induced in 60kg pigs by withdrawing 35 mL/kg of blood over 1 hour, followed by 1.5 hours of sustained hypotension. Animals were assigned to one of three resuscitation strategies. The control group (n=5) received whole blood and Ringer lactate. In addition to fluids, the vasopressin group (n=5) received a continuous infusion of vasopressin at 0.6 mU/kg/min, while the norepinephrine group (n=5) received norepinephrine at 0.2 µg/kg/min. Resuscitation targeted a mean arterial pressure of 65 mmHg, with fluid type adjusted according to hemoglobin concentrations. Peripheral perfusion was assessed by measuring the tarsus–abdomen temperature gradient (GTmean) every 30 minutes using infrared thermography. Changes in GTmean were expressed as ΔGTmean, defined as the difference between each measurement and baseline value. Sample size was calculated to detect significant hypoperfusion, defined as a difference in lactate of 2 mmol/L between groups (α level of 0.05 and a statistical power of 80%). Data were analyzed using linear mixed-effects models.

Results

One animal in the control group died before resuscitation. Before resuscitation, GTmean increased significantly across groups, with a mean rise of +3.8°C (95%CI [2.9; 4.7]). Marked inter-individual variability was observed. During resuscitation, ΔGTmean decreased over time in both the control and vasopressin groups, with slopes of –0.0093 °C/min (95%CI [–0.0127; –0.0057]) and –0.0078 °C/min (95%CI [–0.0109; –0.0046]), respectively. In contrast, no significant change over time was detected in the norepinephrine group (0.0000 °C/min, 95%CI [–0.0033; 0.0032]).

Conclusion

Peripheral perfusion improved during resuscitation in control and vasopressin groups, whereas norepinephrine administration was not associated with a reduction in temperature gradients.

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LONGITUDINAL COMPARISON OF CLINICAL AND POINT-OF-CARE ULTRASOUND SCORES FOR MONITORING PERIPHERAL INTRAVENOUS CATHETER-RELATED VASCULAR CHANGES IN HOSPITALIZED DOGS

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Introduction:

Peripheral intravenous catheters (PIVC) complications (e.g. phlebitis and thrombosis) are traditionally identified through clinical examination in dogs. In people, point-of-care ultrasound (POCUS) has emerged as a sensitive, non-invasive adjunct for detecting early catheter-associated vascular injury, yet its clinical utility in veterinary patients remains poorly defined. This study prospectively evaluated longitudinal trends and interrelationships between standardized clinical and POCUS-based vascular scores in hospitalized dogs with PIVCs. We hypothesized that both scores would increase over time and demonstrate significant longitudinal association.

Methods:

In this prospective observational study, dogs presenting to an emergency service and requiring PIVC placement were consecutively enrolled. Daily paired clinical and POCUS assessments were performed from catheter placement until removal. A previously reported clinical score assessed catheter patency, limb use, erythema, edema, heat, and pain, with scores ≥2 considered suspicious for phlebitis. The POCUS score evaluated vessel wall morphology, venous flow, catheter patency, intraluminal characteristics, and perivascular tissue changes. Baseline relationships were assessed using Spearman correlation, and longitudinal associations were evaluated using linear mixed-effects modeling. Contingency analyses and exact McNemar testing examined relationships between scores and catheter removal.

Results:

From July 2024 to January 2026, 48 dogs [43 (89.6%) cephalic and 5 (10.4%) saphenous catheters] were included. Median catheter duration was 2 (0-4) days. Reasons for catheter removal were animal discharge (27/45, 60%), suspected phlebitis, animal death, soiled bandaging, limb swelling (each 5/45), clinical score ≥2 occlusion (each 3/45), extravasation and self-removal (both 1/45), with multiple reasons possible per dog. Clinical and POCUS scores increased significantly over time (p-value <0.0001). Clinical scores were significantly higher on Day 2 compared with Days 0 and 1 (mean± SE: D0=0.125±0.11, D1=0.333±0.11, D2=1.168±0.18) but remained <2. POCUS scores were significantly increased at Days 2 and 3 compared with Days 0 and 1 (mean± SE: D0=1.542±0.29,

D1=2.471±0.30, D2=4.674±0.47, D3=6.068±0.99). Scores were not correlated at baseline (Spearman r = -0.042, p=0.77), but demonstrated a significant longitudinal association over time.

Conclusion:

Clinical and POCUS catheter scores increased significantly over time, but remained below phlebitis thresholds. The significant association between both scores over time suggests POCUS may be helpful to monitor patients for peripheral catheter-related complications.

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EVALUATION OF THE UTILITY OF A POINT-OF-CARE ULTRASOUND PROTOCOL FOR DETECTING GASTROINTESTINAL OBSTRUCTION IN DOGS

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Introduction:

Differentiating gastrointestinal obstruction (GIO) from other causes of vomiting can be challenging, especially for less experienced veterinarians. Point-of-care ultrasound (POCUS) using simple protocols can improve clinical decision-making. This study evaluated the diagnostic accuracy of different POCUS parameters for detecting GIO in vomiting dogs.

Methods:

This prospective observational study assessed a standardized abdominal POCUS (A-POCUS) protocol containing 5 predefined views performed by emergency veterinarians with standardized training and equivalent POCUS experience in dogs with acute vomiting. Dogs with known extra-gastrointestinal causes of vomiting were excluded. Absence of obstruction was confirmed by clinical follow-up. Assessed parameters included quantitative and subjective gastrointestinal filling, contents, motility, segmental small intestinal dilatation and ascites. Sample size calculation indicated that 30 dogs with GIO and 70 dogs without GIO were required to achieve a statistical power of 0.97. Fisher's exact test was used for statistical analysis; *p*-values ≤0.05 were considered significant. Sensitivity and specificity were calculated.

Results:

A total of 101 cases were included. GIO was confirmed in 33/101 cases (33.3 %) by surgery (n = 31) or endoscopy (n = 2). Obstructions were caused by foreign bodies (n = 30), neoplasia (n = 2), or intussusception (n = 1). The mean body weight of dogs with GIO was 23.1 kg (3.7–67.0 kg) compared to 11.8 kg (2.3–45.0 kg) for dogs without GIO ($p = 0.002$). Overall POCUS sensitivity for detecting GIO was 0.94, specificity 1.00, positive predictive value 1.00, and negative predictive value 0.97. POCUS raised suspicion for obstruction in all cases of intestinal obstruction (n = 30) but only in one case of gastric obstruction (n = 3). The most sensitive parameters were intraluminal small intestinal diameter (ISID) ≥8 mm (0.88) and subjective gastric dilation (0.91). The most specific findings were an ISID ≥8 mm (0.99), subjective segmental small intestinal dilatation in one view (1.00) and pendulum motion (1.00).

Conclusion:

The evaluated gastrointestinal POCUS protocol is an accurate and reliable method for diagnosing intestinal obstruction in dogs with acute vomiting. Its ability to detect gastric obstruction appears limited but the evaluation is restricted due to small case numbers.

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SERUM CHOLESTEROL CONCENTRATION IN DOGS WITH ACUTE HEMORRHAGE: A PRELIMINARY RETROSPECTIVE STUDY

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Introduction:

Hypocholesterolemia (HC) is a documented sequela of trauma, sepsis, gastrointestinal bleeding, surgery, and specific malignancies in people. Studies suggest that HC may serve as a prognostic marker in trauma victims and critically ill populations. While HC in canine patients has been associated with conditions such as sepsis and snake envenomation, its role in hemorrhage remains poorly characterized. This study aims to evaluate the correlation between HC and blood loss in canine patients across various etiologies, including trauma, splenic rupture, coagulopathies, and gastrointestinal bleeding. A secondary goal is to assess the utility of HC as a prognostic indicator within this clinical population.

Method:

Medical records of 110 dogs (2022–2024) transfused for acute hemorrhage were retrospectively analyzed. Baseline serum cholesterol concentrations were evaluated relative to reference intervals and their association with fatality, physiological parameters (vital signs), and clinicopathologic variables (including PCV, total plasma protein [TPP], albumin) was analyzed using Mann-Whitney, Kruskal-Wallis or Chi-square test, as appropriate. Exclusion criteria included prior transfusion before cholesterol sampling, incomplete documentation, or comorbidities known to influence cholesterol metabolism (e.g., chronic hepatic or gastrointestinal disease, endocrinopathies, round cell neoplasia, and snake envenomation).

Results:

The most common causes of blood loss were trauma and neoplasia. The median age was 10 years (range, 2 months-16 years; interquartile range [IQR], 5), the median weight was 22 kg (range, 1-48 kg, IQR, 23). The median cholesterol concentration amongst dogs suffering from blood loss was 152 mg/dL (range, 18-329 mg/dL, IQR, 78), compared with a reference interval of 135-361 mg/dL. Hypocholesterolemia was identified in 34 dogs (31%). Ninety- five dogs (86%) had cholesterol concentrations within the lower third of the reference interval or below. Sixty-eight dogs (62%) survived to discharge. Serum cholesterol concentrations did not differ significantly between survivors and non-survivors. Cholesterol levels were positively correlated with TPP ($r=0.493$, $P<0.001$) and albumin ($r=0.586$, $P<0.001$) concentrations.

Conclusions:

Serum cholesterol concentration may serve as a marker of acute hemorrhage in dogs. The positive correlation between cholesterol, TPP, and albumin concentrations supports vascular loss as a likely mechanism underlying hypocholesterolemia during hemorrhage. Decreased serum cholesterol concentrations were not associated with survival to discharge.

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EMERGENCY ENDOTRACHEAL INTUBATION IN 100 IN DOGS AND CATS: A PROSPECTIVE COHORT STUDY

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Introduction:

Emergency endotracheal intubation is commonly performed in unstable veterinary patients, yet data on common practices and complications are limited. This study aimed to describe patients undergoing emergency intubation and identify factors associated with cardiopulmonary arrest (CPA).

Materials:

A prospective cohort study was performed to include dogs and cats undergoing emergency endotracheal intubation at a university veterinary teaching hospital. Demographic data, indications for intubation, airway management variables, peri-intubation complications, and CPA occurrence were recorded. Continuous variables were analyzed using non-parametric tests and reported as medians (range), while categorical variables were summarized as percentages. Associations with CPA were assessed using the chi-square or Fisher's exact tests and the Mann Whitney U test. Mortality analyses were stratified according to whether intubation occurred during cardiopulmonary resuscitation or not. Significance was set at $p < 0.05$.

Results:

One hundred patients were included (67% dogs, 33% cats). Median body condition score was 4 (range 1–9). The most common indications for intubation were severe non-obstructive hypoxemia (36%) and cardiopulmonary resuscitation (34%), followed by airway obstruction (13%) and apnea or hypoventilation during sedation (8%). Preoxygenation was performed in 88% of cases, most commonly via face mask or flow by (50%), nasal cannulae (28%), or oxygen cage (14%). High-flow oxygen therapy was used for preoxygenation in 12%.

Complications were common, with 78% of patients developing at least 1 complication. Hypotension occurred in 22% of patients, arrhythmias in 14%, hypertension in 6%, and regurgitation or aspiration in 12%. Esophageal intubation was documented in 11%, and endobronchial intubation risk in 20%. Multiple intubation attempts were required in 35% of cases (median 1; range 0–4). Device obstruction occurred in 18%, and coughing in 4%. CPA occurred in 8% of patients. No significant associations were identified on univariable analysis between CPA and body condition score, number of attempts, indications for intubation, airway strategies, or peri-intubation complications (all $p > 0.05$).

Conclusions:

Complications were common following emergency endotracheal intubation and case fatality rate approached 10% in this cohort of dogs and cats. No airway management variables or peri-intubation complications were associated with CPA.

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EFFECT OF CITRATE-PHOSPHATE-DEXTROSE (CPD) ANTICOAGULANT ON POLYMERASE CHAIN REACTION (PCR) RESULTS FOR INFECTIOUS DISEASES; IMPORTANCE IN VETERINARY BLOOD BANKS

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Introduction:

In veterinary medicine, screening blood donors for infectious diseases is a critical safety requirement for hemotherapy. While Ethylenediaminetetraacetic Acid (EDTA) is the reference anticoagulant for PCR, blood units are typically collected in Citrate-Phosphate-Dextrose (CPD). CPD can inhibit PCR by chelating magnesium ions (Mg^{2+}), an essential cofactor for DNA polymerase, potentially leading to amplification failure. Although EDTA is the standard for DNA preservation, preanalytical constraints often result in the unavailability of EDTA samples in veterinary blood banking settings. Consequently, we assessed whether PCR performed on CPD-anticoagulated blood sampled directly from blood bags can sustain testing continuity without compromising amplification performance.

Objective:

This study aimed to compare the PCR amplification success rate of CPD-anticoagulated whole blood versus EDTA (reference standard) in paired samples, and to estimate the relative sensitivity of CPD.

Methods:

A cohort of 113 PCR-positive dogs enrolled in a veterinary blood bank program was included. Paired samples were collected from each subject: whole blood in EDTA tubes (control /reference standard) and whole blood sampled obtained directly from the blood bag containing CPD (test group). Both groups underwent identical DNA extraction and PCR amplification protocols. Paired binary comparisons between CPD and EDTA were assessed using McNemar's test (two-sided $\alpha = 0.05$), and the relative sensitivity of CPD versus EDTA was estimated with 95% confidence intervals (CI).

Results:

Amplification success rates differed significantly between the groups (McNemar's test, $p < 0.001$). PCR assays using DNA from CPD-anticoagulated blood failed in 13 of 113 paired EDTA-positive samples (11.5%), yielding an amplification success rate of 88.5% (100/113). The relative sensitivity of CPD-derived DNA versus the EDTA reference standard was 88.5% (95% CI: 81.1%–93.7%).

Conclusion:

Compared with EDTA (reference standard), CPD-anticoagulated blood showed significantly reduced PCR amplification success and relative sensitivity (88.5%; 95% CI: 81.1%–93.7%) under identical extraction protocols, leading to potential false-negative results in blood donor screening. For sensitive diagnostic applications in veterinary blood banking, EDTA-anticoagulated blood and

rigorous purification protocols are recommended to minimize inhibitor effects and ensure reliable PCR performance.

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EVALUATION OF TISSUE OXYGEN SATURATION VIA NEAR-INFRARED SPECTROSCOPY: A COMPARATIVE STUDY BETWEEN BRACHYCEPHALIC AND NON-BRACHYCEPHALIC DOGS

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Introduction:

Tissue oxygen saturation reflects the balance between oxygen delivery and cellular oxygen consumption and may be altered in conditions associated with chronic airflow limitation. Brachycephalic dogs present anatomical abnormalities consistent with Brachycephalic Obstructive Airway Syndrome (BOAS), traditionally regarded as a localized upper airway disorder but increasingly suspected to have systemic physiological consequences. The objectives of this study were to evaluate peripheral tissue oxygen saturation (StO_2) in awake dogs, compare brachycephalic and non-brachycephalic conformations, and explore the potential influence of airway obstruction severity and body condition score (BCS).

Methods:

Thirty client-owned dogs presented for pre-anaesthetic evaluation were prospectively enrolled. Thirteen dogs were brachycephalic and seventeen were non-brachycephalic (mesocephalic and dolichocephalic). Peripheral StO_2 was measured at rest in the sartorius muscle using near-infrared spectroscopy (NIRS) in awake, unsedated animals under standardized environmental and handling conditions. Brachycephalic dogs were clinically graded for BOAS severity using predefined criteria, and BCS was recorded in all dogs. Descriptive statistics with 95% confidence intervals were calculated, and comparisons between morphological groups were performed using a parametric test for independent samples ($p < 0.05$). Analyses according to BOAS severity grade and BCS were considered exploratory due to limited subgroup sizes.

Results:

Peripheral StO_2 values ranged from 67% to 92%, with an overall mean of 78.0 ± 5.56%. Brachycephalic dogs showed significantly lower StO_2 compared with non-brachycephalic dogs (mean ± SD: 74.23 ± 4.02% vs. 80.88 ± 4.85%; respectively; $p < 0.001$). Variation in StO_2 values was observed across BOAS severity grades and body condition score categories; however, due to limited subgroup sizes, no formal statistical comparisons were performed and no definitive relationship could be established.

Conclusions:

Awake brachycephalic dogs exhibit reduced peripheral tissue oxygen saturation compared with non-brachycephalic dogs, supporting the presence of systemic physiological alterations associated with BOAS. In this study population, the influence of airway obstruction severity and body condition score on peripheral tissue oxygenation could not be determined. Near-infrared spectroscopy represents a clinically relevant, non-invasive tool for assessing peripheral oxygenation, and further studies with larger sample sizes are warranted to clarify potential associations.

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DEVELOPMENT OF TWO-DIMENSIONAL TRAJECTORY PATTERNS FOR FIVE FEEDING TUBE TIP LOCATIONS USING THE CORTRAK2 SYSTEM IN CANINE CADAVERS: A FEASIBILITY STUDY

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Background:

Airway misplacement of nasoenteral feeding tubes is a recognized complication with potentially serious consequences. CORTRAK2 uses electromagnetic tracking to visualize real-time tube position during feeding tube placement, generating a two-dimensional (2D) trajectory graph (x-y coordinates) in three orthogonal planes. As a bedside tool, this system may reduce routine radiographic confirmation in patients where radiography is challenging. CORTRAK2 tracing patterns across different anatomic locations in dogs have not been characterized. The objective of this study was to demonstrate the feasibility of the CORTRAK2 device in canine cadavers and establish typical CORTRAK2 tracing patterns associated with five predefined tube tip locations.

Material and Methods:

Eight canine cadavers (body weight: 30-40.3kg) were used. A 10Fr nasoenteral tube was advanced under endoscopic guidance into five different locations in each cadaver: the right and left main bronchi, esophagus, stomach, and duodenum. When endoscopic duodenal placement was not technically feasible, manual tube placement was performed via laparotomy to achieve duodenal positioning. For each placement, CORTRAK2 tracings displayed in three orthogonal planes (anterior view, lateral view, depth cross section) were recorded. Tube tip positions were confirmed via computed tomography (CT) and three-view radiography. Median x- and y-coordinates were calculated across cadavers and a representative graph for each location was generated.

Results:

The CORTRAK2 system successfully tracked the tube throughout all anatomical locations in two out of three orthogonal planes (anterior and lateral view). However, reliable real-time visualization in the depth-cross section view was only achieved in the duodenum. Distinctive 2D trajectory patterns were observed in the anterior and lateral view and representative tracings for each of the five CT-confirmed locations were created to support pattern recognition in future studies.

Conclusion:

The CORTRAK2 system is reliable for real-time tube tracking in the anterior and lateral viewing planes and produces electromagnetic tracings in canine cadavers that differ by tube tip location, supporting its feasibility as a bedside tool in dogs. The developed trajectory graphs provide a visual reference for future prospective studies aimed to assess the system's diagnostic accuracy in live patients.

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IMPACT OF ROOM TEMPERATURE EXPOSURE ON THE QUALITY OF CANINE AND FELINE PACKED RED BLOOD CELL UNITS

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Introduction:

Ensuring the safety of blood components is of great importance for transfusion medicine. Hemolysis is caused by alterations in erythrocyte morphology and an increase in their fragility, changes that may compromise the safety of blood units. Storage conditions, particularly storage temperature, may play a key role in contributing to increased hemolysis. Exposure to room temperature can occur accidentally in refrigeration equipments (e.g., power outages or improper equipment closure) and affect the quality of stored units. Therefore, periodic quality control (QC) assessments of the units after accidental exposure to room temperature are essential to ensure the safety of transfusions. The objective was to assess the impact of room temperature exposure on the quality of packed red blood cell (pRBC) units.

Methods:

Thirty canine and thirty feline pRBC units, discarded due to positive infectious disease screening, were used. Units were processed in a temperature-controlled room and baseline QC analysis was performed (T0). Units were stored at 2–6°C for 5 days (T1), then exposed to controlled room temperature (18–22°C) for 2 days (T2) and returned to refrigeration for 5 days (T3). The percentage of hemolysis was evaluated at each of the different time points.

Results:

In canine pRBC units the average percentage of hemolysis was 0,10% (0,02–0,33%) at T0, 0,26% (0,07–0,64%) at T1, 0,48% (0,17–0,98%) at T2 and 0,62% (0,23–1,21%) at T3. Four units (≈13%) exceeded the acceptable hemolysis threshold of 0.8% at T2, following exposure to room temperature, while five units (≈17%) surpassed this limit at T3. In feline pRBC units the average percentage of hemolysis was 0,12% (0,00–0,35%) at T0, 0,35% (0,11–0,93%) at T1, 0,55% (0,23–1,12%) at T2 and 1,11% (0,30–3,30%) at T3. None of the 30 feline units exceeded the acceptable hemolysis limit (1.5%) at T2, however, 6 units (20%) exceeded this limit at T3.

Discussion:

Hemolysis increased progressively in both canine and feline pRBC units, with a relevant proportion exceeding acceptable thresholds following exposure to room temperature. Canine units demonstrated earlier susceptibility. The observed variability emphasizes the importance of performing QC on units accidentally exposed to ambient temperature.

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POTENTIAL OF POTASSIUM-ABSORBING FILTERS TO REDUCE TRANSFUSION-ASSOCIATED HYPERKALAEMIA IN DOG

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Introduction

Transfusion-associated hyperkalaemia (TAH) is a recognized complication in humans receiving rapidly administered packed red blood cells (PRBCs) and may precipitate life-threatening arrhythmias. Potassium-absorbing filters (PAFs) have been shown to reduce potassium load during transfusion in human but their application in veterinary practice remains limited. Recently reports of TAH in a dog receiving canine PRBCs highlighted the potential relevance of potassium removal strategies in veterinary medicine. This study evaluated the performance of PAF in a high-potassium solution model and in expired canine PRBC units.

Methods

The experiment was conducted in two phases. Phase 1: two 500 mL of 0.9% NaCl bags, supplemented with potassium chloride to a final concentration of 20 mmol/L were filtered through a PAF at 5 mL/kg/h, with samples collected at five time points from pre-filtration to end-of-bag and subsequently analysed. Phase 2: Five expired canine PRBC units (250–280 mL) were filtered similarly, and sampling obtained at same five time points. At each time point, ten 1 mL samples were collected, and each sample analyzed in ten technical replicates, yielding 5 × 10 × 10 = 250 measurements per phase. All procedures were approved by the Ethical Review Group (AVA 2025-007).

Statistical Analysis

A paired observational design was used. Based on a detectable difference of 2.0 mmol/L, an assumed standard deviation of 0.5 mmol/L, $\alpha = 0.05$, and $\beta = 0.2$ (80% power), five units per phase were sufficient to detect significant changes. Potassium concentrations were analyzed using linear mixed-effects models with filtration volume as a fixed effect and bag as a random effect, with post-hoc Holm correction. Data are reported as mean ± SD and percent change from baseline.

Results

Filtration significantly reduced potassium in both phases. Phase 1: K^+ decreased from 18.64 ± 0.9 to 3.62 ± 2.0 mmol/L (Δ 15.0 ± 4.0 mmol/L, 80.2 ± 17.2%). Phase 2: K^+ decreased from 4.71 ± 0.33 to 1.63 ± 0.66 mmol/L (Δ 3.08 ± 0.65 mmol/L, 65.3 ± 11.5%).

Conclusion

Potassium-absorbing filters effectively reduce potassium concentrations in both high-potassium solutions and canine PRBC units, supporting their potential utility to mitigate TAH in veterinary practice.

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EVALUATION OF THE ASSOCIATION OF THE ABDOMINAL FLUID TO BLOOD BILIRUBIN RATIO AND EFFUSION CYTOLOGICAL FINDINGS WITH THE DIAGNOSIS OF BILIARY PERITONITIS IN DOGS

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Introduction:

To assess the diagnostic value of abdominal effusion to blood total bilirubin ratio (e:b-tBIL), absolute abdominal effusion total bilirubin concentration (e-tBIL), and effusion cytological findings in distinguishing dogs with surgically confirmed biliary peritonitis (BP) from dogs with other causes of abdominal effusions.

Methods:

Retrospective analysis of canine BP with a prospectively enrolled control group at a University teaching hospital. Twenty-seven dogs with surgically confirmed BP and 15 prospectively enrolled dogs with non-BP. The control group included dogs with heterogeneous non-biliary effusions caused by urogenital (5/15), gastrointestinal (4/15), lymphatic (2/15), and cardiovascular (1/15) disorders, hepatobiliary neoplasia (2/15), and soft tissue infection (1/15). Biliary tract rupture was excluded by surgery, comprehensive diagnostic evaluation, or post-mortem examination.

Results:

Bilirubin concentrations in blood and abdominal effusion samples and effusion cytology findings at presentation were evaluated. Median e-tBIL was higher in dogs with BP (51.0 µmol/L [2.98 mg/dL]; interquartile range [IQR] 247.6 µmol/L [14.48 mg/dL]) than in controls (6.4 µmol/L [0.37 mg/dL]; IQR 12.3 µmol/L [0.72 mg/dL]; $p < 0.001$). Receiver operating characteristic curve analysis demonstrated excellent discriminatory ability of e-tBIL for the diagnosis of BP (area under the curve 0.979, 95% confidence interval 0.934–1.000; $p < 0.001$). The optimal cut-off value was 21.15 µmol/L (1.24 mg/dL), determined by Youden's index (0.923), yielding 92.3% sensitivity and 100% specificity.

The median e:b-tBIL did not differ significantly between dogs with BP (1.48; IQR 6.75; range 0.23-27.4) and controls (0.9; IQR 1.16; range 0.17-6.34; $p = 0.586$) and demonstrated poor diagnostic performance (area under the curve 0.564; 95% confidence interval 0.336-0.792).

White bile was identified cytologically in 57.7% (15/26) of BP cases and in no controls ($p < 0.001$), whereas bilirubin crystals were detected in 23.1% (6/26) and 6.7% (1/15) of cases, respectively ($p = 0.232$).

Conclusion:

Absolute abdominal fluid bilirubin concentration showed strong discriminatory ability for identifying BP in this study population, whereas the e:b-tBIL ratio had limited diagnostic value. Cytological identification of white bile may provide a useful adjunctive diagnostic finding. Combined biochemical and cytological evaluation may assist in the clinical assessment of dogs with suspected BP.

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EFFECT OF COGNITIVE AIDS ON CARDIAC ARREST RHYTHM RECOGNITION AMONG SECOND YEAR VETERINARY STUDENTS

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Introduction:

Correct cardiac arrest rhythm identification is important for successful cardiopulmonary resuscitation (CPR) outcomes; however, rhythm recognition during CPR remains challenging for resuscitation teams. The objective of this study was to evaluate the effect of two cognitive aids on cardiac arrest rhythm recognition in veterinary students after minimal curricular CPR training.

Methods:

154 second-year veterinary students attended a 1h curricular CPR lecture and afterwards were invited to complete an online quiz that required diagnosing 12 cardiac arrest electrocardiographic rhythm strips. Students were randomly assigned to only use their lecture knowledge, the Reassessment Campaign on Veterinary Resuscitation (RECOVER) 2024 electrocardiogram algorithm, or a screen shot image consisting of rhythm examples from the RECOVER CPR coach application. Information on additional CPR training was collected. Interactions using a generalized linear mixed model between correct rhythm diagnoses, cognitive aids, and prior CPR training were analyzed.

Results:

A total of 150 students completed the arrest rhythm quiz, of which 50 were assigned no cognitive aid, 44 were assigned the RECOVER electrocardiogram algorithm, and 56 were assigned the RECOVER application screenshot. Overall accuracy of cardiac arrest rhythm recognition was high and ranged from 93-99% (95%CI 86-100%) with no cognitive aid, 89-99% (95%CI 82-100%) with the RECOVER algorithm, and 96-98% (95%CI 91-99%) with the application screenshot. Correct rhythm identification varied by rhythm and was most common for asystole (91%, 95%CI 85–94%) and pulseless ventricular tachycardia (92%, 95%CI 87–95%), followed by ventricular fibrillation (78%, 95%CI 71–84%) and pulseless electrical activity (73%, 95%CI 65–79%). The generalized linear mixed model revealed a significant main effect of rhythm type on correct rhythm identification ($p < 0.001$) and a significant interaction between the provided cognitive aid and the correct arrest rhythm identification ($p = 0.022$). No significant main effects of type of cognitive aid alone ($p = 0.95$) or prior CPR training ($p = 0.30$) were observed.

Conclusions:

Following a CPR lecture, second-year veterinary students demonstrated high overall accuracy in arrest rhythm recognition. Differences in recognition accuracy were rhythm-specific and varied by the type of cognitive aid used, indicating that certain cognitive aids may preferentially support identification of specific arrest rhythms.

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ACCURACY OF TWO SMARTPHONE-BASED ELECTROCARDIOGRAM DEVICES FOR RHYTHM AND HEART RATE MONITORING IN CATS: A PILOT STUDY

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Introduction:

Assessing heart rate and rhythm in cats is often challenging, and rapid evaluation is critical in emergency settings. Smartphone-based electrocardiogram (ECG) devices offer a portable alternative, but their accuracy in cats is not well established. This pilot study aimed to evaluate two such devices, Kardia and Ekuore, for trace readability, rhythm identification, and heart rate (HR) measurement.

Methods:

Ten adult cats were enrolled. ECG recordings were collected for 30 seconds in cats during recovery from anesthesia, with three ECGs acquired simultaneously while the cats were positioned in right lateral recumbency, including two smartphone-based ECG devices (Kardia and Ekuore) and a standard ECG used as the gold standard (GS). Inter-operator agreement among three observers for readability was assessed using Fleiss' kappa. Concordance of HR measurements between smartphone-based devices and the GS ECG was evaluated using the Pearson correlation coefficient (r).

Results:

The median age of the cats was 8.6 years (range 0.5–13.1 years); one cat had a cardiac murmur detected on auscultation. Inter-operator agreement for ECG readability was moderate for Kardia (Fleiss' $\kappa = 0.53$) and almost perfect for Ekuore (Fleiss' $\kappa = 0.81$). All cats showed sinus rhythm; one cat exhibited a right bundle branch block. HR measurements showed a strong correlation between the GS ECG and Kardia ($r = 0.98$), while a moderate correlation was observed between the GS ECG and Ekuore ($r = 0.75$).

Conclusions:

This pilot study provides preliminary data on the accuracy and feasibility of Kardia and Ekuore smartphone-based ECG devices in cats. As recordings were performed in animals not fully awake, further studies are needed to evaluate performance in conscious cats and the devices' ability to detect arrhythmias, which is challenging in this species.

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IDENTIFICATION OF A BIOMARKER TO DETERMINE PRESENCE OF NEOPLASIA OR A BENIGN PROCESS IN CANINE HAEMOABDOMEN, PLEURAL OR PERICARDIAL EFFUSIONS

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Introduction:

Canine hemoabdomen is a common emergency presentation. Cases are often critical at presentation and can require extensive treatment including multiple transfusions, pericardial effusion drainage, and surgery, all before the nature of the lesion is known. It is not possible to definitively identify neoplastic versus benign causes without histopathology.

Development of a biomarker to identify neoplasia from effusions prior to surgery would allow owners to make an informed decision and could improve animal welfare. Cell free DNA (cfDNA) and tumor free DNA (tfDNA) is circulating genetic material that may be used to identify neoplasia. To the author's knowledge this is the first veterinary study to extract cfDNA and carry out shallow whole gene sequencing on effusions and splenic samples to diagnose neoplasia.

Methods:

Single center, prospective, pilot study at a UK university small animal referral hospital. Excess cavity effusions (pleural, peritoneal and pericardial) taken at the time of diagnosis or therapeutic management were placed in sterile lobind cfDNA tubes. Canine splenic ($1cm^3$) samples were collected at the time of surgery and stored at -80°C until batch processing. Exclusion criteria were animals with septic effusions or insufficient samples. Ethical approval was granted by the institute. Thirty-two fluid samples underwent cfDNA extraction and shallow whole genome sequencing. Thirteen splenic samples underwent shallow whole genomic sequencing and analysed using ichorCNA, to identify the genome-scale alterations in patients. Gold standard for neoplastic diagnosis was a histopathology or cytology report by a board-certified pathologist. There was no blinding of samples.

Results:

Of a total of 32 effusions, 25/32 were confirmed or suspected neoplasia and 7/32 benign. On histopathology 7/13 splenic samples were neoplastic and 6/13 were benign. Non-neoplastic effusions and splenic tissue showed no alterations in whole genome sequencing. Results from effusions showed good levels of cfDNA extraction. There were chromosomal abnormalities in ctDNA in effusions and spleens after whole gene sequencing of copy number profiles.

Conclusions:

This study demonstrates ctDNA is an exciting novel concept that can identify neoplasia in effusions and spleens. Further work would be required to ascertain whether a bedside test could identify neoplastic diseases earlier.

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PREVALENCE OF DIABETIC KETOALKALOSIS AND UTILITY OF DELTA RATIO ANALYSIS IN DOGS AND CATS WITH DIABETIC KETOACIDOSIS

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Introduction: Diabetic ketoacidosis (DKA) in dogs and cats is often complicated by mixed acid–base disorders. Traditional analysis may miss these, particularly when metabolic or respiratory alkalosis coexists with metabolic acidosis. Consequently, some DKA patients may present with normal or elevated blood pH, outside conventional criteria. We aimed to determine the prevalence of DKA with neutral or elevated pH ("diabetic ketoalkalosis") in dogs and cats when β-hydroxybutyrate (β-OHB) ≥3.0 mmol/L is used as an independent diagnostic criterion. A secondary aim was to assess whether albumin-adjusted anion gap (AGalb) combined with delta ratio improves detection of mixed disorders.

Methods: Dogs and cats admitted to a single institution between 2024 and 2025 with systemic illness, diabetes mellitus, and β-OHB ≥3 mmol/L were included. A standard Henderson–Hasselbalch (HH) analysis was compared with an advanced HH approach (A-HH) incorporating AGalb and delta ratio. Delta ratio was calculated as [AGalb − mid-normal AG] ÷ [mid-normal HCO_3^- − patient HCO_3^-]; values >2 indicated concurrent metabolic alkalosis and <0.8 suggested concurrent normal anion gap metabolic acidosis (NAGMA).

Results: Forty-three patients met inclusion criteria. Acidemic DKA (pH <7.27 in cats, <7.35 in dogs) occurred in 28/43 (65.1%), neutral pH DKA in 13/43 (30.2%), and diabetic ketoalkalosis (pH >7.42 in cats, >7.43 in dogs) in 2/43 (4.7%). Of the two ketoalkalotic cases, one had high anion gap metabolic acidosis (HAGMA) with concurrent metabolic and respiratory alkalosis, and one had simple respiratory alkalosis. Compared with HH, the A-HH approach detected additional mixed disorders, including HAGMA combined with metabolic alkalosis (3/43, 7%) and HAGMA with NAGMA (6/43, 14%) not captured by HH. NAGMA as the sole abnormality was identified in 6 cases by HH and 8 by A-HH.

Conclusions: Defining DKA as uncontrolled diabetes mellitus with β-OHB ≥3 mmol/L, regardless of pH or bicarbonate, captures the full spectrum from acidemic DKA to diabetic ketoalkalosis. NAGMA may occur as the only metabolic disorder in approximately 15–20% of cases. Incorporating the delta ratio into the HH approach improves recognition of complex mixed disorders in patients with HAGMA.

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Poster Abstracts – Case Report

REDUCING ASPIRATION PNEUMONIA IN CANINE MEGAESOPHAGUS BY ESOPHAGEAL SUCTIONING: A CASE REPORT.

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Background:

Megaesophagus (ME) is characterized by esophageal dilation and impaired motility, often predisposing affected dogs to chronic regurgitation and a heightened risk of aspiration pneumonia (AP), which represents the leading cause of mortality. ME may be idiopathic or secondary to underlying disorders such as myasthenia gravis (MG), hypothyroidism, or esophageal obstruction. In dogs with acquired MG, ME is the most common manifestation, resulting from autoantibody-mediated dysfunction of acetylcholine receptors in striated muscle. Standard management including upright feeding, percutaneous endoscopic gastrostomy tube (PEGt) placement, prokinetics, and treating the primary disease often proves insufficient in refractory cases where persistent esophageal pooling leads to chronic AP.

Case presentation:

A 10-year-old spayed female Border Collie initially presented for inspiratory stridor and intermittent vomiting and was diagnosed with laryngeal paralysis and AP. Following stabilization, unilateral arytenoid lateralization was performed. Two weeks later, the dog was re-presented with pyrexia and frequent regurgitation. Thoracic radiographs revealed generalized ME and recurrent AP. Hypoadrenocorticism and hypothyroidism were excluded, and acetylcholine receptor antibody titers confirmed a diagnosis of MG. Despite multimodal therapy consisting of antibiotics, oxygen supplementation, neostigmine, exclusive PEGt feeding, prokinetics, and gastroprotectants, the dog continued to suffer from persistent esophageal fluid accumulation, regurgitation, and recurrent episodes of AP. To mitigate the risk of further aspiration, an esophageal tube was placed to enable routine manual suctioning of esophageal contents. Nutrition was maintained solely via PEGt. Over a six-month follow-up period, the dog remained clinically stable, and free of recurrent AP despite persistent ME. Serial monitoring showed normalization of C-reactive protein (CRP) levels, and the owner reported a marked reduction in regurgitation frequency.

New/Unique Information:

This case demonstrates esophageal suctioning as a useful adjunctive therapy for refractory ME complicated by recurrent AP. Notably, there are only two previously published reports utilizing this specific intervention in veterinary medicine. Although the technique requires high owner compliance, at-home suctioning can effectively reduce the frequency of AP and improve clinical stability. This case supports the inclusion of esophageal suctioning as part of an individualized, multimodal management strategy to improve clinical stability, quality of life and survival in dogs with severe esophageal dysmotility.

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ACQUIRED DIAPHRAGMATIC HERNIA SECONDARY TO URETHRAL OBSTRUCTION IN A CAT

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Background:

Diaphragmatic hernia (DH) in cats is most often associated with blunt trauma including vehicular trauma, and falls from height, though penetrating trauma and congenital defects are also reported. Clinical signs can range from mild, nonspecific illnesses to severe respiratory compromise.

Case presentation:

A 13-year-old castrated male domestic shorthair cat was presented with a 48-hour history of stranguria. The cat was strictly indoor and had a prior episode of urethral obstruction secondary to urethritis several years earlier. Physical examination revealed a markedly distended, firm urinary bladder, consistent with urethral obstruction. The obstruction was readily relieved via urinary catheterization under general anesthesia. Shortly after bladder decompression, the cat developed acute respiratory distress. Thoracic radiographs demonstrated the presence of DH. There was no known history of trauma, and thoracic radiographs obtained six months earlier confirmed an intact diaphragm. Exploratory surgery identified a right-sided diaphragmatic defect with herniation of the stomach, small intestine, spleen, and right lateral liver lobe. The defect was surgically repaired, and a thoracostomy tube was placed. Although the cat initially recovered uneventfully, recurrent urethral obstruction developed 7 days following discharge. The cat was re-presented for stranguria and bladder distention, and subsequently exhibited progressive dyspnea. Repeat diagnostic imaging confirmed recurrence of the DH, and euthanasia was elected. Necropsy revealed a 4×3 cm circumferential tear within the pars costalis of the diaphragm, accompanied by herniation of abdominal viscera. A separate, intact surgical repair line, secured with sutures, was identified approximately 0.5 cm medial to the new defect. Adjacent to this region, the central tendon appeared focally thinned and atrophic, suggesting pre-existing structural weakness of the diaphragmatic tissue.

New/Unique Information:

The absence of trauma, together with the temporal association between severe urethral obstruction and hernia development, supports a diagnosis of an acquired, non-traumatic DH secondary to an acute increase in intra-abdominal pressure. Focal atrophy of the central tendon likely predisposed the diaphragm to tearing, as recurrence adjacent to an intact repair suggests failure of structurally weakened diaphragmatic tissue rather than suture dehiscence. To the authors' knowledge, this represents the first reported case of DH secondary to urethral obstruction in a cat.

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TRAUMATIC MYOCARDIAL INJURY AND ACUTE CARDIAC FAILURE IN TWO FELINE HIGH-RISE SYNDROME PATIENTS

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Background :

Myocardial injury might be underdiagnosed after trauma as it can occur without obvious thoracic injury. In humans, blunt cardiac injury affects up to 76% of thoracic trauma cases, but its incidence in veterinary medicine is unknown. While blunt trauma is a frequent condition in cats, little is known on acute cardiac failure linked to traumatic myocardial injury in cats. We describe two feline trauma patients who developed blunt-trauma related acute cardiac-failure with elevated cardiac troponin I (cTnI).

Case Presentation :

Two neutered male Domestic Shorthair cats (12 and 7.5 years-old) were presented after high-rise trauma with no prior history of cardiac disease. The first cat, presented 48 hours post-trauma with acute respiratory distress showed more than 3 B-lines per lung ultrasound window, pleural effusion, and left atrial enlargement (LA/Ao >2) on point-of-care ultrasound (POCUS). The second cat, presented immediately after trauma, initially had no respiratory signs or atrial enlargement despite numerous B-lines on POCUS, suspected to be related to pulmonary contusions. Respiratory distress occurred 48 hours later, with persistent B-lines, newly identified left atrial dilation (LA/Ao >1.7), and mild pleural effusion. In both cats, pleural effusion was a modified transudate, consistent with cardiac congestive failure.

Echocardiography revealed myocardial thickening and left atrial dilation. Cardiac troponin I was markedly elevated in both cats (31 and 19.8 ng/ml (<0,09)).

The first cat developed combined congestive and low-output cardiac failure, requiring vasopressors, positive inotrope support, oxygen therapy, and diuretics, but suffered sudden cardiac arrest within 24 hours. The second cat developed congestive heart failure without low-output signs, followed by acute pelvic limb paresis due to arterial thromboembolism. Thrombolysis gave transient improvement, but recurrent congestive failure occurred, and euthanasia was elected.

New/Unique Information :

Both cats developed acute cardiac failure within 48 hours of blunt trauma related to high-rise syndrom. Both cats developped congestive heart failure associated with marked elevated troponine I, exceeding levels reported in phenotype of hypertrophic cardiomyopathy or thromboembolism. This support the putative diagnosis of traumatic myocardial injury. Clinicians should remain alerte to myocardial injury in feline trauma patients as a life-threatening myocardial damage can occur rapidly after trauma.

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MISSILE FRAGMENT-INDUCED GASTROINTESTINAL PERFORATION IN DOGS: REPORT OF TWO CASES

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Introduction:

Modern armed conflicts often result in catastrophic polytrauma caused by high-energy blast effects and ballistic fragmentation. Achieving successful outcomes in a veterinary setting necessitates specialized intensive care to address both life-threatening systemic shock and multifaceted surgical injuries. This case series describes the clinical findings, surgical management, and long-term recovery of two dogs that sustained missile-related shrapnel trauma leading to gastrointestinal (GI) perforation and septic peritonitis.

Case presentation:

Case 1: A 1.5-year-old intact female mixed-breed dog was presented in hemorrhagic shock with penetrating abdominal and over the humeral wounds. Initial point-of-care ultrasound (POCUS) identified a peritoneal effusion. Biochemical analysis confirmed uroperitoneum (effusion creatinine: 8 mg/dL vs. blood 1.95 mg/dL) and cytology suggested no evidence of septic peritonitis. Exploratory laparotomy revealed massive hemoperitoneum, two transmural jejunal perforations, a urinary bladder rupture, and multiple abdominal wall defects. Following surgical stabilization and repair, the patient recovered uneventfully.

Case 2: A 9-year-old intact male was presented in shock with catastrophic injuries to all four limbs, including open comminuted fractures and vascular trauma. While initial POCUS was largely unremarkable, the patient developed a delayed-onset peritoneal effusion by day three. A significant glucose gradient (effusion 47 mg/dL vs. blood 120 mg/dL) and cytological evidence of intracellular bacteria confirmed septic peritonitis. Laparotomy identified a jejunal perforation associated with a metallic fragment, alongside a diaphragmatic perforation and secondary hepatic trauma. Following left forelimb amputation and GI repair, the patient adapted to three-legged ambulation and returned to a high quality of life.

New/Unique Information:

This report describes previously undocumented cases in veterinary medicine, for which few clinical reference points exist. The cases demonstrate the severe effects of high-order explosions, producing an initial shock wave followed by a blast wind of superheated air. Although fragment injuries often predominate, high-velocity shrapnel may cause occult internal organ rupture without obvious external abdominal trauma. Such injuries may be missed during initial stabilization, as shown by delayed septic peritonitis in Case 2. Therefore, blast-exposed patients require thorough, repeated clinical and diagnostic evaluation. Serial point-of-care ultrasonography, radiography, and biochemical

analysis of effusions are essential for early detection of life-threatening internal injury and complications development.

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LIFE-THREATENING MESENTERIC HEMORRHAGE IN A DOG CAUSED BY SUSPECTED ANGIOSTRONGYLUS VASORUM LARVAL MIGRATION

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Background:

Angiostrongylus vasorum infection in dogs is associated with a wide spectrum of clinical manifestations, most commonly respiratory disease and hemostatic disorders. Hemorrhagic diathesis may present as petechiae, ecchymoses, mucosal bleeding, hematomas, or, less commonly, pulmonary or intracranial hemorrhage, and is typically managed medically. Direct arterial rupture secondary to larval migration is rare. To the authors' knowledge, only two cases of arterial rupture have been reported, involving the femoral and the thoracic aorta. Mesenteric vessel rupture resulting in hemoperitoneum has not been previously described.

Case presentation:

A 21-month-old intact male Poodle presented with acute lethargy and anorexia. Physical examination revealed tachycardia with a heart murmur 3/6, tachypnea, pale mucous membranes, and prolonged capillary refill time. Point-of-care ultrasound identified free abdominal fluid, and abdominocentesis confirmed hemorrhagic effusion (fluid PCV 29% versus peripheral PCV 15%). In-house prothrombin time and activated partial thromboplastin time were within reference intervals, and a point-of-care *Angiostrongylus vasorum* antigen test was positive.

Stabilization included intravenous fluids, blood product administration, and analgesia. Due to ongoing hemorrhage, emergency exploratory laparotomy was performed, revealing active mesenteric bleeding associated with focal vascular rupture. Hemostasis was achieved by vessel ligation, and enterectomy was performed due to questionable intestinal perfusion. Mesenteric hematoma and intestinal samples were submitted for histopathology. Postoperative echocardiography identified pulmonary hypertension, and antiparasitic treatment together with sildenafil was initiated. The dog recovered favorably and was discharged two days postoperatively. At four-week follow-up, pulmonary hypertension had resolved and sildenafil was discontinued.

Histopathology revealed intestinal eosinophilic infiltration without full-thickness ulceration and a degenerate nematode within the mesenteric hematoma, supporting parasitic larval migration as the cause of mechanical mesenteric vascular rupture. Although definitive species identification was not possible, *A. vasorum* was strongly suspected based on clinical findings, positive antigen testing, and histopathology.

Unique Information:

This case highlights an atypical, life-threatening presentation of angiostrongylosis characterized by aberrant larval migration causing mesenteric vascular rupture and hemoperitoneum, requiring emergency surgical intervention. Parasitic larval migration should be considered in dogs presenting

with acute hemoabdomen, even in the absence of coagulopathy or typical clinical signs. This case was previously published in Veterinary Record Case Reports (2025;13:e70152).

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ACETAMINOPHEN INTOXICATION COMPLICATED BY SEVERE METHEMOGLOBINEMIA AND TRANSIENT CONSUMPTIVE COAGULOPATHY IN A CAT

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Case summary

A 1.5-year-old indoor cat was presented approximately 24 hours after confirmed ingestion of acetaminophen (paracetamol; 125 mg/kg). Facial edema, lethargy, and mucous membrane cyanosis were noted on admission, with pulse oximetry readings persistently in the mid-80% range. Venous blood gas analysis with co-oximetry demonstrated severe methemoglobinemia (60.8%) and hyperlactatemia (5.9 mmol/L), consistent with profound functional hypoxia from oxidative hemoglobin injury.

Complete blood count revealed hemoconcentration (HCT 56%), leukocytosis (27.6 × 10^3/µL), and thrombocytosis (549 × 10^3/µL). Serum biochemistry showed moderate elevations in alanine aminotransferase and alkaline phosphatase activities (676 U/L and 314 U/L, respectively), without hepatic synthetic function abnormalities. Intravenous N-acetylcysteine and oral S-adenosylmethionine–silybin were initiated; however, ongoing hypoxic deterioration prompted partial blood exchange transfusion, performed by replacing 10 mL/kg of phlebotomized blood with donated whole blood via a central jugular catheter.

Over the subsequent 24 hours, methemoglobinemia improved and marked clinical recovery was observed; however, spontaneous hemorrhage and anemia developed approximately 24 hours after presentation, including bleeding from venipuncture sites and hematuria. Coagulation testing revealed unmeasurable prolongation of prothrombin time and activated partial thromboplastin time with severe thrombocytopenia (26 × 10^3/µL). A coagulation panel demonstrated elevated D-dimer concentrations (1681 ng/mL), hypofibrinogenemia (73 mg/dL), and preserved antithrombin activity (90%), consistent with a consumptive coagulopathy, while liver enzyme activities and bilirubin concentration showed only minimal change and no progressive deterioration during the coagulopathy period.

Treatment with fresh whole blood (7.5 mL/kg) followed by multiple units of fresh frozen plasma (27.5 mL/kg in total) resulted in normalization of coagulation parameters within 36 hours, and the cat was subsequently discharged. At re-evaluation 48 hours later, severe anemia (PCV 13%), progressive hyperbilirubinemia (4.6 mg/dL), and marked Heinz body formation were identified, consistent with ongoing oxidative hemolysis. Oral carbocysteine and S-adenosylmethionine–silybin therapy was continued at home. Two weeks after presentation, the cat was clinically normal with resolution of laboratory abnormalities.

Clinical Relevance and Conclusion

This case highlights severe, clinically relevant consumptive coagulopathy during acetaminophen intoxication, hitherto unreported in cats, successfully managed with plasma transfusion. Early recognition of this potentially life-threatening complication is vital for timely intervention.

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CLINICAL MANIFESTATIONS AND MANAGEMENT OF ANDROCTONUS BICOLOR ENVENOMATION IN A DOG: A PREVIOUSLY UNDESCRIBED CLINICAL CASE

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Background: The black fat tailed scorpion (*Androctonus bicolor*), a member of the family Buthidae, is considered one of the most venomous native scorpion species in Israel. Its venom primarily affects voltage gated sodium channels, leading to autonomic overstimulation and a broad spectrum of cardiovascular, respiratory, and neuromuscular manifestations. The venom contains a unique cysteine rich peptide, Androcin, which induces akinesia and anxiety like behavior in rodents. In humans, envenomation by *A. bicolor,* neurological and autonomic signs are prominent. Reports of veterinary scorpion envenomation are scarce, and Canine *A. bicolor* envenomation remains undocumented in veterinary literature.

Case presentation: A 10-year-old male Golden Retriever was presented following an envenomation by *A. bicolor*. Within minutes, the dog developed labored breathing, hypersalivation, and collapse. The scorpion was identified by the Israel Nature and Parks Authority. On presentation, the dog was non ambulatory, quiet but responsive, with marked panting and tachypnea. A small puncture wound was observed over the right hind paw. Immediate treatment included analgesia, antihistamine administration, and intravenous isotonic crystalloids boluses. Initial bloodwork revealed a potassium concentration at the upper reference interval and increased alkaline phosphatase activity. Thoracic radiographs ruled out pulmonary congestion.The dog developed nystagmus, muscle spasms, and generalized hypertonicity with tachycardia of 168 beats per minute and systolic blood pressure of 190 mmHg. Administration of Midazolam followed by a single dose of Phenobarbital resulted in complete muscle relaxation and cessation of tremors. Treatment continued with supporative medications and antibiotics. Three days later the dog presented marked clinical improvement, however, two days following discharge, the dog was re-presented with tremors, inappetence, hematochezia, hyperthermia, mild tachycardia with occasional ventricular premature complexes, and labored breathing. Bloodwork revealed a markedly elevated C-reactive protein concentration and increased pancreatic lipase activity, consistent with systemic inflammation and mild pancreatitis. Supportive therapy was instituted, and clinical improvement was observed within 24 hours.

New and uinque information : This report describes the first documented case of *Androctonus bicolor* envenomation in a dog and highlights the prominent neurological and autonomic manifestations associated with this species. Despite severe initial clinical signs, early recognition and supportive care resulted in complete recovery.

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ACUTE SEIZURES SECONDARY TO CEREBRAL INFARCTION IN A DOG WITH POLYCYTHEMIA VERA: CONSIDERATIONS FOR THROMBOTIC RISK ASSESSMENT AND CYTOREDUCTIVE THERAPY

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Background:

Primary polycythemia vera (PV) is an uncommon myeloproliferative disorder in dogs associated with erythrocytosis, increased blood viscosity, and thrombotic complications. In emergency settings, PV may present with acute neurologic signs secondary to cerebrovascular events; however, guidance for thrombotic risk assessment and initial management is limited. While treatment has traditionally focused on hematocrit reduction via therapeutic phlebotomy, human PV literature suggests that erythrocyte indices such as mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH), reflecting iron-restricted erythropoiesis, may provide additional insight into thrombotic risk. This report describes a dog with PV presenting for acute seizures and explores how erythrocyte indices informed emergency management.

Case Presentation:

A 10-year-old spayed female Welsh Corgi presented on an emergency basis for acute-onset generalized seizures. Diagnostic evaluation revealed severe absolute erythrocytosis (hematocrit 83.9%) accompanied by microcytosis and reduced MCH. Relative erythrocytosis and secondary erythropoietin-driven causes were excluded through clinical assessment, laboratory testing, and advanced imaging. Brain magnetic resonance imaging identified a chronic ischemic infarction of the left caudate nucleus, consistent with a thrombotic cerebrovascular event, leading to a diagnosis of primary PV complicated by cerebral infarction.

Initial emergency management with therapeutic phlebotomy resulted in partial hematocrit reduction; however, seizures remained refractory and transient neurologic deterioration was observed, raising concern that viscosity reduction alone was insufficient for thrombotic risk control. Based on the presence of microcytosis and low MCH, considered indicative of iron-restricted erythropoiesis and a prothrombotic phenotype, cytoreductive therapy with hydroxyurea was initiated. Hydroxyurea administration was associated with rapid hematologic improvement, normalization of MCV and MCH, and complete resolution of seizure activity. Gastrointestinal adverse effects necessitated drug discontinuation; however, hematocrit suppression persisted for approximately eight weeks. No further seizures occurred during follow-up with adjunctive antiplatelet therapy.

New/Unique Information:

This case highlights the clinical relevance of erythrocyte indices, including MCV and MCH, as accessible indicators of thrombotic risk in dogs with PV presenting for acute seizures. Furthermore, it suggests that in emergency settings where thrombotic cerebrovascular events are suspected, early cytoreductive therapy with hydroxyurea may offer advantages over phlebotomy alone by modulating a broader prothrombotic cellular milieu rather than focusing solely on viscosity reduction

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FIRST REPORT OF HYENA (HYAENA HYAENA) BITE INJURIES IN 2 DOGS: AN UNRECOGNIZED PREDATOR TRAUMA

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Background:

Hyenas (*Hyaenidae*) are specialized apex predators possessing a robust cranial structure and a powerful masticatory apparatus capable of generating immense bite forces sufficient to crush cortical bone. Although often perceived as obligate scavengers, hyenas are highly efficient hunters. While hyena attacks on humans are documented in sub-Saharan Africa, literature describing similar traumatic injury patterns in domestic dogs is currently lacking. This report details the clinical presentation and multidisciplinary management of two canine patients following predatory hyena attacks.

Case Presentation:

Case 1: A 1-year-old intact male mixed-breed dog was presented following a "bite-and-drag" attack. Physical examination and CT imaging revealed catastrophic facial trauma, including right-sided traumatic ocular proptosis with corneal perforation and retinal detachment. Osseous trauma included complex comminuted fractures of the rostral and left mandible with symphyseal separation, and displaced fragments within the right nasal bone and frontal sinus. Esophagoscopy further identified severe esophagitis. Surgical intervention involved intermandibular wiring with bone-regenerative implants for mandibular stabilization, dental extractions, right-eye enucleation, and PEG-tube placement for nutritional support.

Case 2: A 1-year-old intact female Border-Collie sustained severe craniofacial injuries, including maxillary fractures, upper-lip degloving, and right-sided proptosis following hyena attack while walking off-leash. CT diagnostics revealed a temporal bone fracture associated with intracranial hemorrhage and pneumocephalus, alongside right orbital fractures penetrating the cranial vault. Management required a complex, multistage neurosurgical approach. Orbital exenteration was performed to expose the orbital floor and allow access to the cranial vault. To protect the brain parenchyma oxidized regenerated cellulose and a dural regeneration matrix were secured with

topical tissue adhesive to establish a hermetic seal between the cranial cavity and overlying soft tissues.

Both patients received rabies prophylaxis and intensive wound care. Despite the severity of the initial trauma, both dogs achieved full functional recovery.

New/Unique Information:

These reports provide the first documented evidence of hyena-inflicted trauma in domestic dogs. The observed injury patterns characterized by high-energy, crushing craniofacial and intracranial fractures parallel those described in human victims. These cases underscore the life-threatening nature of hyena attacks and highlight the necessity of aggressive, multidisciplinary management integrating advanced imaging, surgical reconstruction, and intensive-care to optimize clinical outcomes.

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HEMOPERITONEUM SECONDARY TO TORSION OF THE MIDDLE AND LEFT LATERAL HEPATIC LOBES IN A PUPPY

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Background:

Hepatic lobe torsion (HLB) is an uncommon cause of acute abdominal emergency in small animals, characterized by the twisting of a liver lobe around its vascular pedicle, leading to vascular compromise, congestion, and potential necrosis. The left lateral lobe is reported as the most frequently affected site in canine HLT, but torsion can involve other lobes. Clinical signs are often nonspecific. The cause is unknown, but it has been associated with ligamentous and anatomical abnormalities, trauma, previous gastric surgery, and hepatic neoplasia. Surgical resection (lobectomy) of the affected lobe is the mainstay of treatment, and prompt intervention is associated with generally favorable outcomes in animals that survive the acute phase. Although most documented cases involve adult dogs and cats, HLT should also be considered in pediatric patients presenting with acute hemoperitoneum.

Case presentation:

A 5-month-old intact puppy was presented to our referral center in a state of shock following a 12-hour history of acute vomiting, anorexia, and lethargy. Physical examination on admission revealed a depressed mental status, sinus tachycardia (128 bpm), pale mucous membranes, weak pulses, and hypotension (mean arterial pressure 52 mmHg). Complete bloodwork demonstrated regenerative anemia 26% (37.5–61.7%), thrombocytopenia 135 k/µL (148–484 k/µL), hypoproteinemia 4.7 g/dL (4.8–7.2 g/dL), hypoglobulinemia 2.2 g/dL (2.3–3.8 g/dL), and a marked increase in alanine aminotransferase activity 924 U/L (8–75 U/L). All other parameters, including coagulation times, were within reference ranges. Abdominal fluid analysis was consistent with active hemorrhage. Abdominal computed tomography revealed marked enlargement of the left medial and lateral hepatic lobes, with diffuse hypoattenuation and absence of contrast enhancement, findings consistent with hepatic lobe torsion. Emergency lobectomy of the affected lobes was performed. Histopathological examination confirmed hemorrhagic hepatic infarction. After 48 hours of intensive care, clinical recovery was favorable.

New/Unique information:

This report highlights hepatic lobe torsion as a rare but clinically relevant differential diagnosis in pediatric canine patients. Although uncommon, it should be considered in young dogs presenting with acute abdomen and hemoperitoneum, as early recognition and prompt surgical intervention can result in a favorable outcome even in severely ill cases.

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SUCCESSFUL MANAGEMENT OF A YOUNG DOG WITH PNEUMONIA AND CARDIAC PULMONARY EDEMA WITH MECHANICAL VENTILATION

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Background:

Positive pressure ventilation (PPV) is an established therapy for acute cardiogenic pulmonary edema (ACPE), commonly reported in older dogs with myxomatous mitral valve disease. Its successful use in juvenile dogs with decompensated congenital heart disease is poorly described, and particularly in the presence of concurrent pulmonary disease.

Case presentation:

A seven-month-old female Hollandse Herdshond was presented in lateral recumbency with acute dyspnea. Physical examination revealed tachypnea (100 breaths/min), cyanotic mucous membranes, and a continuous grade VI/VI cardiac murmur. Thoracic radiographs demonstrated severe bilateral alveolar lung pattern consistent with pulmonary edema and cardiomegaly. Cardiac Point-of-Care sonography revealed severe left ventricular and biatrial dilatation, a persistent ductus arteriosus (PDA) with left-to-right shunting, and severe mitral regurgitation. Lung ultrasound identified multiple B-Lines and shred signs. Blood work showed hypercapnia (PvCO2 79.7 mmHg) and moderate systemic inflammation raising suspicion of concurrent pneumonia. Initial treatment included high-flow oxygen therapy (HFOT), diuresis with furosemide (2 mg/kg IV bolus followed by continuous rate infusion (CRI) at 1 mg/kg/h), and antimicrobial therapy with amoxicillin-clavulanic acid. Because of persistent severe hypoxemia under HFOT, mechanical ventilation with positive end-expiratory pressure was rapidly initiated. Anesthesia was maintained using CRIs of alfaxalone, midazolam, and fentanyl. Oxygenation improved progressively and after approximately 20 hours of PPV, the dog was successfully weaned, and HFOT was reintroduced. Follow-up thoracic radiographs showed regression of pulmonary edema; however, CRP had increased markedly (246 mg/L), supporting suspected pneumonia, and antimicrobial therapy was escalated by adding marbofloxacin. Three days later recurrent pulmonary edema required a change in diuretic strategy from furosemide to torasemide, resulting in rapid clinical improvement. Surgical ligation of the PDA was performed 25 days after presentation. Four months later, the dog remained clinically stable with no recurrence of pulmonary edema.

New/Unique Information:

This case shows how early escalation to invasive PPV can be feasible in juvenile dogs with ACPE secondary to congenital heart disease, even when oxygenation is compromised by suspected concurrent pneumonia. Failure of response to diuresis and non-invasive oxygen supplementation should prompt consideration of mixed pulmonary pathology and timely escalation of respiratory support.

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SUCCESSFUL RETRIEVAL OF A NASOPHARYNGEAL FOREIGN BODY UTILIZING A NOVEL NEEDLE-ASSISTED TRANS-VELAR APPROACH IN A DOG

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Background:

This report describes a novel approach to managing a nasopharyngeal foreign body in a canine patient.

Case presentation:

A two-year-old intact female German Shorthaired Pointer was presented with an acute onset of open-mouth breathing, head shaking, and pawing at the face. Physical examination using the ABCDE approach revealed an alert but anxious dog, with a partially obstructed upper airway, as evidenced by stertor, snorting, retching, and reverse sneezing. Increased inspiratory effort was evident, with stress-related tachycardia and a head-down posture.

Radiographic examination of the skull (LL/DV/VD views) revealed a radiopaque (mineral-density) foreign body (FB), measuring approximately 2.0x3.0cm, lodged within the lumen of the nasopharynx.

Considering the risk of FB dislodgement and subsequent aspiration into the trachea, the dog was placed under general anesthesia. Digital exploration confirmed the presence of the FB above the soft palate, protruding into the oropharynx, yet manual retrieval could not be successfully achieved. Retrograde nasopharyngoscopy using a flexible endoscope and retrograde hydropulsion via nasal catheter both failed to retrieve the FB.

A ventral rhinotomy was ultimately planned after considering an ad hoc alternative retrieval technique. Thus, the patient was positioned in left lateral recumbency, with the head extended and the mouth secured wide-open. The operator's left index finger was advanced into the oropharynx and positioned dorsal to the velum palatinum, touching the caudal aspect of the FB. Using the right hand, a 21G syringe needle (0.8x50mm) was gently advanced through the soft palate in a tangential direction and maintained in a rostrocaudal orientation at a 45° angle. The tip of the needle was directed toward the rostral aspect of the FB, located within the dorsal nasal meatus. The needle (acting as propulsor) and the index finger (serving as guide) were moved in tandem, ensuring retrograde advancement of the FB toward the oropharynx. The FB was pushed caudally, grasped, and subsequently removed through the oropharynx.

New/Unique Information:

The clinical relevance of this technique lies in its simplicity and minimal invasiveness, offering a viable alternative to more invasive surgical interventions, in primary care practices where advanced endoscopic equipment is unavailable, or when conventional methods prove ineffective.

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SUCCESSFUL MECHANICAL VENTILATION IN A DOG WITH SEVERE ANGIOSTRONGYLUS VASORUM INFECTION

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Background:

Dogs with *Angiostrongylus* vasorum infection can manifest various clinical signs, ranging from subclinical disease to severe respiratory failure. Respiratory distress is primarily due to verminous pneumonia resulting from parasite migration, subsequent hatching of the ova within the pulmonary capillaries, and penetration of alveolar and bronchial walls.

Case presentation:

A one-year-old male German Hunting Terrier was presented with mixed dyspnea after hunting. The dog had a two-month history of intermittent coughing.

At presentation, the dog was apathetic, showed signs of orthopnea with a respiratory rate of 80 breaths per minute, and enhanced lung sounds on auscultation. Thoracic radiographs revealed a diffuse alveolar lung pattern, a slender cardiac silhouette, and aerophagia. The AngioDetect® rapid assay (*IDEXX Laboratories*) was positive. Coagulation testing revealed no abnormalities.

Initially, the dog was supported with high-flow oxygen therapy (HFOT, 0.8–1 L/kg, 90–100% FiO_2). Eight hours after HFOT initiation, the respiratory rate-oxygenation index was 1 (SpO_2: 94%, FiO_2: 1, respiratory rate: 90 breaths/min), and the dog showed signs of respiratory fatigue. Mechanical ventilation using pressure-controlled ventilation was therefore initiated. Cytologic examination of tracheal secretions revealed mixed inflammation and *Angiostrongylus vasorum* larvae. Anesthesia was maintained using a combination protocol with continuous rate infusions (CRI) of butorphanol, dexmedetomidine, ketamine, midazolam, propofol, and rocuronium titrated to effect. Additional treatment included amoxicillin-clavulanic acid (20 mg/kg TID IV), pimobendan (0.15 mg/kg BID IV), enoxaparin-CRI (1.5 mg/kg/d), and supportive therapy. Antiparasitic therapy was initiated with fenbendazole (50 mg/kg PO) two hours after admission and changed to imidacloprid/moxidectin (Advocate®, spot-on) in combination with prednisolone (1 mg/kg TID IV) on the second day due to deterioration of the dog's condition.

After approximately 24 hours of mechanical ventilation, pulmonary function improved, and the dog was successfully weaned from the ventilator 30 hours after initiation. HFOT was reinstated and slowly tapered over the following day. Follow-up thoracic radiographs showed significant improvement of the lung pattern. The dog was discharged after five days in good clinical condition.

New information:

This case describes a severe manifestation of *Angiostrongylus vasorum* infection, requiring mechanical ventilation and lung recruitment. Following successful weaning, the short-term prognosis appears to be good.

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DIAGNOSTIC VALUE FROM CT TO MRI IN CANINE REFRACTORY SEIZURES: PRESUMPTIVE GLIOMA AND CLINICAL IMPLICATIONS IN AN AMERICAN BULLY

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Background:

Refractory seizures and status epilepticus are common neurological emergencies in dogs and require rapid stabilization and diagnostic planning. Magnetic resonance imaging (MRI) is the gold standard for identifying structural intracranial disease, but anesthetic risk may delay its use in unstable patients. Computed tomography (CT) can be used as an interim diagnostic tool. This case describes the diagnostic progression from CT to MRI in a dog with refractory seizures and progressive neurological deterioration.

Case presentation:

A 5-year-old intact male American Bully, previously managed for presumed idiopathic epilepsy with phenobarbital and potassium bromide, was evaluated by the orthopedic service for progressive hindlimb weakness. During re-examination, the dog developed acute neurological deterioration characterized by recumbency, vertical nystagmus, dilated pupils, and intermittent seizure activity. The patient was transferred for emergency stabilization, where status epilepticus was controlled using intravenous diazepam, propofol, ketamine, and levetiracetam. Mannitol and corticosteroids were administered to manage suspected cerebral edema. Due to respiratory compromise and anesthetic risk, CT was performed without anesthesia and revealed no intracranial mass or contrast enhancement, although inflammatory or neoplastic disease could not be excluded. Presumptive meningoencephalitis was diagnosed, and treatment with corticosteroids and antibiotics (marbofloxacin and amoxicillin–clavulanate) was initiated. Over the following three weeks, seizure frequency decreased and the dog regained the ability to stand, though mild weakness and a persistent downward head posture remained. Several days later, the dog developed head pressing and rapid neurological decline. Blood tests revealed a normal complete blood count, C-reactive protein, and systolic blood pressure. Echocardiography and thoracic radiography identified dilated cardiomyopathy (DCM, stage B1). MRI revealed a large, heterogeneously hyperintense intra-axial lesion within the right cerebral hemisphere causing marked mass effect and midline shift, consistent with a presumptive glioma. Cerebrospinal fluid collection was contraindicated due to increased intracranial pressure. Recovery from anesthesia was prolonged and required mechanical ventilation, followed by cardiopulmonary arrest, likely associated with brainstem involvement.

Unique Information:

This case emphasizes stabilization before advanced diagnostics, the use of CT when anesthesia is unsafe, and the superior sensitivity of MRI for intracranial disease, highlighting diagnostic challenges when inflammatory and neoplastic conditions present similarly in dogs with refractory seizures.

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ATRIAL FIBRILLATION ASSOCIATED WITH SUSPECTED MYOCARDIAL INJURY AFTER BLUNT THORACIC TRAUMA IN A GREYHOUND

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Background:

Atrial fibrillation (AF) is the most common supraventricular arrhythmia in dogs and is usually associated with structural heart disease. Myocardial contusion is a recognized consequence of blunt thoracic trauma and typically causes ventricular arrhythmias, but trauma-associated AF has rarely been described in dogs. This report describes AF with evidence of myocardial injury and systolic dysfunction after blunt thoracic trauma in a Greyhound and outlines its management and the role of ambulatory electrocardiography (ECG) in assessing rate control.

Case presentation:

A six-year-old Greyhound was presented to an emergency service after a road traffic accident. The dog showed dyspnea, tachyarrhythmia, thoracic contusions and a forelimb degloving injury. Point-of-care-ultrasound, focused-cardiac-ultrasound and radiographs confirmed tension pneumothorax with mild pleural effusion and suspected left ventricular systolic dysfunction. Thoracocentesis resolved the pneumothorax, and he was stabilized with oxygen, analgesia, and later amputation. ECG revealed AF with a rapid ventricular response rate, and serum cardiac troponin I (cTnI) was markedly increased at 1332 ng/L (reference <110 ng/L), consistent with myocardial injury secondary to blunt cardiac trauma. Transthoracic echocardiography confirmed left ventricular systolic dysfunction with mild dilation. A rate-control strategy with oral diltiazem was started, and digoxin was later added because of poorly controlled heart rate. Serial echocardiograms showed progressive improvement in systolic function and normalization of chamber dimensions. A 24-hour Holter recorded persistent AF with a slow ventricular response rate (mean HR 108 bpm), indicating adequate rate control at home. Thyroxine and taurine concentrations were within reference intervals, and no structural heart disease was identified. The dog remained clinically well on medical therapy at follow-up.

New/Unique Information:

This case illustrates AF after blunt thoracic trauma in a sighthound, with evidence of traumatic myocardial injury and transient systolic dysfunction. It also underlines the need to monitor systolic function after blunt thoracic trauma and the difficulty of interpreting systolic measurements when a tachyarrhythmia is present, as AF itself may contribute to systolic impairment. Trauma-associated AF has only rarely been reported in dogs, and this appears to be the first detailed description associating persistent AF with suspected myocardial contusion using contemporary diagnostics such as cTnI, echocardiography and ambulatory ECG.

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AGAINST THE ODDS: SURVIVAL FOLLOWING BLOODSTREAM CANDIDIASIS SECONDARY TO PERITONITIS IN A DOG

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Backround:

In human medicine, candida peritonitis and candidemia are recognized clinical entities associated with substantial mortality. Conversely, veterinary reports of systemic canine candidiasis remain exceptionally rare; the two previously documented cases resulted in fatal outcomes.

Case Presentation:

A 4-year-old neutered male Labrador Retriever was presented with a 4-day history of vomiting and diarrhea. Upon admission, the patient was hemodynamically stable but exhibited cranial abdominal pain and a characteristic “prayer” position.

CBC revealed leukocytosis, while the serum chemistry panel remained unremarkable. Abdominal ultrasonography identified marked colonic mural thickening with loss of normal wall layering, irregular hypoechoic margins, surrounded regional steatitis, and scant peritoneal effusion. Initial management for suspected acute colitis was unsuccessful. Five days post-admission, the patient developed septic peritonitis and underwent exploratory laparotomy. A perforated necrotic colonic segment was resected; histopathology later confirmed colonic infarction. Aberrant *Spirocerca Lupi* migration was suspected as the primary cause, and ivermectin therapy was initiated. Four Days post-resection the dog’s clinical status deteriorated. Cytologic evaluation of the peritoneal effusion revealed bacterial and fungal elements. A second laparotomy confirmed and corrected an anastomotic dehiscence. Peritoneal cultures from the initial surgery isolated multidrug-resistant *Escherichia coli* and a resistant *Enterococcus Faecium*, sensitive only to Amikacin and Chloramphenicol, respectively. Antimicrobial therapy was adjusted accordingly. Peritoneal cultures from the second surgery were unfortunately lost. Despite targeted treatment and the second surgical revision, the patient remained febrile, no microorganisms were seen on peritoneal fluid cytology. A blood culture obtained three days post-revision isolated *Candida glabrata*. Following initiation of fluconazole, pyrexia resolved within 24 hours. The patient received a one-month course of fluconazole, which was well-tolerated without evidence of hepatotoxicity. The dog achieved a full clinical recovery, and a follow-up blood culture performed after the discontinuation of antifungal therapy was negative.

New Information:

This case represents the first documented survival of a canine patient with candidemia. It highlights that *Candida* peritonitis and systemic candidiasis are critical potential sequelae following gastrointestinal perforation and septic peritonitis in dogs.

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RODENTICIDE-ASSOCIATED INTRACRANIAL HEMORRHAGE AND BRAIN HERNIATION IN DOGS: A CASE SERIES

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Background:

Intracranial hemorrhage secondary to rodenticide intoxication is infrequently documented in dogs, particularly when clinical signs of bleeding are minimal or absent, and neurological signs dominate the clinical presentation. This case series describes three dogs with rodenticide-associated intracranial hemorrhage leading to subsequent brain herniation. The aim of this case series is to raise awareness of rodenticide intoxication as a potential differential diagnosis for acute, non-traumatic intracranial hemorrhage in dogs.

Case presentations:

Three dogs were presented individually following suspected rodenticide ingestion. Two dogs presented with acute onset of severe neurological signs, including decerebrate rigidity. The third dog developed acute signs consistent with intracranial hypertension within hours of presentation. Two dogs showed bleeding from previous injection sites, while one dog had no visible signs of bleeding on presentation but had been evaluated for joint swelling prior to referral. Coagulation testing revealed severely prolonged prothrombin time and activated partial thromboplastin time in all dogs, raising suspicion of anticoagulant rodenticide intoxication, which was subsequently confirmed based on history. Vitamin K1 and supportive therapy were initiated promptly in all cases.

Magnetic resonance imaging (MRI) revealed intracranial hemorrhage with subtentorial brain herniation in all dogs. Additional foramen magnum herniation was present in two dogs. One dog was euthanized based on the severity of the MRI findings. The second dog underwent decompressive surgery but was euthanized intraoperatively due to extensive hemorrhage and cerebral necrosis. The third dog received intensive medical management aimed at reducing intracranial pressure but continued to deteriorate and was ultimately declared brain dead. Post-mortem MRI in this dog confirmed severe cerebellar herniation identical to the findings in the other dogs.

New or unique information provided:

Rodenticide intoxication may present with severe, rapidly progressive neurological dysfunction when hemorrhage is confined to the central nervous system and may therefore be overlooked as a differential diagnosis. Once intracranial hypertension and brainstem involvement are clinically evident, rodenticide-associated intracranial hemorrhage appears to carry a grave prognosis despite prompt diagnosis and medical or surgical intervention. Rodenticide intoxication should therefore be considered an essential differential in dogs presenting with acute neurological signs consistent with non-traumatic intracranial hemorrhage.

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PERITONEAL ABSCESS CAUSED BY MULTI-RESISTANT SALMONELLA SPP. IN AN 11-WEEK RAW-FED PUPPY

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Background:

It is well reported that dogs fed raw meat diets are vectors for human Salmonella infection and have themselves much higher chance of Salmonella (or other Gram negative bacteria) infection than dogs consuming non-raw diets.

A presented case is an extreme example of potential complications from raw meat feeding.

Case presentation:

An 11-week old male, American Bully (XXL), was presented following 2 weeks of intermittend diarrhea and pyrexia, with no response to symptomatic treatment. Abdominal ultrasound revealed a 10 cm diameter fluid-filled mass; this was removed at celiotomy and was found to be an abscess. No other abodimal pathology was identified. On bacterial culture, a multi-drug resisant Salmonella was isolated.

New/unique information:

Salmonella is not a species seen commonly in the GI tract of dogs, which suggests a link between the raw meat diet and the peritoneal abscess. It emphises the need for education of both veterinarians and owners on the potential risks from feeding a raw meat diet.

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ACUTE POLYRADICULONEURITIS WITH RESPIRATORY FAILURE IN A DOG FOLLOWING RECREATIONAL WATER EXPOSURE: SUCCESSFUL MANAGEMENT WITH PROLONGED VENTILATION, TRACHEOSTOMY, AND IVIG

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Background:

Acute Polyradiculoneuritis (APN) is an immune-mediated radiculomyelopathy that produces rapidly progressive lower motor neuron dysfunction and, in severe cases, respiratory paralysis. There is no proven disease-modifying treatment in dogs; human therapies (therapeutic plasma exchange, intravenous immunoglobulin [IVIG]) have been applied in veterinary patients. This case report aims to describe the management and successful outcome of a dog with APN coinciding with an outbreak of Guillain–Barré syndrome (GBS) in humans in the region, requiring prolonged mechanical ventilation, tracheostomy, adjunctive human IVIG, and intensive physiotherapy, and to highlight practical ventilatory and airway strategies for such patients.

Case presentation:

A previously healthy adult dog developed acute ascending flaccid tetraparesis within days of exposure to a contaminated water source and progressed to ventilatory failure. Critical hypercapnic respiratory failure necessitated immediate invasive mechanical ventilation. The patient was started on pressure-controlled ventilation (PCV) and subsequently transitioned to pressure-controlled synchronized intermittent mandatory ventilation (PC-SIMV) with added pressure support ventilation (PSV) to promote spontaneous breathing. After repeated failed weaning attempts, a temporary tracheostomy was placed on day 5 and human IVIG (total 1 g/kg in a cautious, slow infusion) was administered. Despite intensive nursing, the progress was complicated by tenacious tracheal secretions, ventilator-associated infection and catheter-related infection requiring antimicrobial escalation, frequent tracheostomy tube changes, nebulized and oral mucolytics, and management of hypoxemia with advanced ventilator modes (Airway Pressure Release Ventilation, APRV). Ventilatory weaning consisted of Continuous Positive Airway Pressure (CPAP) /PSV, the adjunctive intermittent use of high-flow nasal oxygen and a tracheostomy heat-moisture exchanger (HME) filter during spontaneous breathing. Neurological improvement began on day 8, with progressive recovery of swallow, cough, voluntary motor function and deep pain sensation; tracheostomy was removed on day 12, and the dog was discharged on day 19. Full functional recovery was achieved by day 32 after initiation of mechanical ventilation. A direct link to the human GBS outbreak could not be confirmed.

New/Unique Information:

This case report details successful prolonged invasive ventilation with a tracheostomy tube and ventilatory weaning in dogs, combined with human IVIG and intensive rehabilitation in a canine APN, outlining ventilator modes, airway-clearance strategies, complication management, and physiotherapy.

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PERITONEAL DIALYSIS AS RENAL REPLACEMENT THERAPY IN A DOG WITH SEVERE VIPER ENVENOMATION

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Background:

Canine envenomation caused by European Vipera species (*V. latastei*, *V. seoanei*, *V. aspis*) is associated with a wide spectrum of clinical presentations, ranging from mild local reactions to severe systemic complications, including acute kidney injury (AKI), hemolysis and coagulopathies, frequently requiring intensive care. Management is primarily supportive, and although antivenom is recommended, access is limited in several European countries and may be associated with substantial cost. Consequently, alternative supportive strategies play a central role in management. Renal replacement therapies are rarely incorporated into treatment algorithms for snakebite-associated AKI, and extracorporeal techniques are not universally accessible. Peritoneal dialysis (PD) is a widely available renal support modality, yet its role in viper envenomation–associated AKI remains poorly defined, having been reported only once in veterinary literature. The objective of this report is to describe the integration of PD into the intensive care management of a severe viper envenomation–associated AKI case in a dog.

Case presentation:

A 4-year-old neutered male Dachshund was referred with suspected viper envenomation, presenting with acute facial/ventral edema, vomiting, lethargy and rapidly progressive azotemia. On admission, the patient was oliguric, severely azotemic (creatinine >10 mg/dL), anemic and hyperbilirubinemic, consistent with systemic envenomation and multiorgan dysfunction. Despite intensive care, the patient progressed to anuria, failed to respond to furosemide and developed clinically relevant fluid overload. Given the anuric AKI, refractoriness to medical management, and small body size limiting extracorporeal techniques, PD was initiated. It was performed for seven consecutive days, with 5 to 10 exchange cycles per day. Following initiation of PD, advanced ICU therapies, including blood products and parenteral nutrition, were safely administered. Progressive improvement in renal parameters, acid–base/electrolyte balance, and clinical condition was observed. The patient survived the acute phase and was discharged with stable chronic kidney disease.

New/Unique Information:

This represents the first report describing prolonged and intensive PD for viper envenomation–associated AKI in Europe. It highlights PD as a feasible and adaptable renal support strategy in anuric,

volume-overloaded patients refractory to medical therapy, particularly when antivenom or sustained extracorporeal renal replacement therapies are unavailable or impractical in the ICU setting.

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WHEN ONE PATHOGEN IS NOT ENOUGH: POXVIRUS AND CO-INFECTIONS IN A STRAY KITTEN

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Background:

Cowpox virus (CPXV) (genus O*rthopoxvirus*) causes dermatitis and occasionally pneumonia in cats. Due to frequent misdiagnosis and zoonotic risk, CPXV infection should be considered in cats with pneumonia.

Case Presentation:

A two-month-old female domestic shorthair was presented with respiratory and dermatological symptoms. Five days prior, a pruritic skin lesion on one forelimb and paw pad was noted. Subsequently, lesions appeared on one ear and lip, accompanied by diarrhea. Initial treatment by the referring veterinarian included amoxicillin-clavulanic acid SC (12.5mg/kg/day), meloxicam PO (0.1mg/kg/day) and topical selamectin (15mg/cat). Despite treatment, the kitten developed severe dyspnea. Physical examination showed a body condition score of 3/9, pale, dry mucous membranes, ocular discharge, and ulcerative, roundish crusting skin lesions on fore- and hindlimbs, nostril, and left pinna. Pulmonary auscultation revealed bilaterally harsh sounds with wheezes and crackles. Differential diagnoses included trauma, feline upper respiratory disease complex, toxoplasmosis, lungworm and CPXV infection. Thoracic radiographs showed a severe diffuse interstitial-alveolar pattern. Lung-ultrasonography showed multiple B-lines and shred-signs. Blood gas analysis and chemistry revealed mixed acidosis (pH:7.18, lactate:11.2mmol/L, $cHCO_3$:18.3mmol/L, pCO_2:49.48mmHg), severe leukocytosis (26.9x10^9/L) (neutrophilia:15.84x10^9/L; eosinophilia:3x10^9/L), moderate thrombocytosis (869x10^9/L), mild normocytic-normochromic anemia (23.5%), severe hyperglobulinemia (52.4g/L), and a mild increase in serum-amyloid A (5mg/mL). Skin cytology indicated severe purulent dermatitis. FIV-/FeLV-Snap test was negative. Treatment included crystalloid fluids, amoxicillin-clavulanic acid IV (20mg/kg TID), buprenorphine IV (0.01mg/kg TID), inhalation therapy (TID), and oxygen supplementation. PCR of respiratory swabs was positive for *Mycoplasma* spp., and *Toxoplasma* IgG was elevated (1:512), consistent with maternal antibodies.Clinical deterioration necessitated euthanasia. Post-mortem broncho-alveolar lavage indicated purulent-eosinophilic inflammation. Necropsy revealed severe granulomatous-eosinophilic pneumonia with intralesional nematode larvae and eggs (*Aelurostrongylus abstrusus*), severe desquamation of alveolar macrophages and pyogranulomatous lymphadenitis of the tracheobronchial lymph nodes. The skin had a moderate multifocal proliferative and ulcerative dermatitis with intracytoplasmic eosinophilic inclusion bodies. Pan-Pox PCR and subsequent sequencing confirmed CPXV infection in skin, tonsils and tracheobronchial lymph nodes, while Feline

Herpesvirus-1 PCR was negative. The cat also had moderate intestinal infestation with tape- and roundworms.

New/Unique Information:

Multiple infections may predispose stray kittens to systemic CPXV infection, indicating a role of immunosuppression and highlighting the importance of isolation measures.

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COMPLEX CANINE POLYTRAUMA FOLLOWING MISSILE FRAGMENT INJURY: A CASE REPORT

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Introduction:

Armed conflicts may result in complex polytrauma, including high-energy blast injuries and ballistic fragmentation. Managing such cases in veterinary medicine is exceptionally challenging due to the severity of injuries, systemic shock, and the necessity for prolonged, multidisciplinary intensive care. This report describes the clinical presentation, surgical management, and successful functional recovery of a dog sustaining catastrophic polytrauma following a missile strike.

Case Presentation:

A 9-year-old 30kg intact male mixed-breed dog was presented in hemorrhagic shock after being struck by missile fragments in a residential garden. Initial stabilization involved aggressive fluid resuscitation, multimodal analgesia, oxygen therapy, and broad-spectrum antibiotics. Diagnostic imaging and clinical evaluation revealed severe trauma to all four limbs:

Right forelimb: Open ulnar and radial fractures with associated vascular injury and active hemorrhage.

Left forelimb: Catastrophic comminuted fracture with extensive bone fragmentation and significant soft tissue loss.

Right hindlimb: Penetrating wound traversing the crus with vascular trauma and a deep laceration of the fifth digit.

Left hindlimb: Distal crus wound with an associated tarsal fracture and concurrent rupture of the common calcaneal tendon.

Advanced diagnostics revealed systemic inflammation and marked elevations in muscle enzymes. During hospitalization, the patient developed septic peritonitis secondary to shrapnel-induced gastrointestinal perforation, necessitating jejunal resection and anastomosis; liver lacerations were also identified. During hospitalization, the patient also developed recurrent urinary tract infections. Brainstem Auditory Evoked Response (BAER) testing subsequently confirmed bilateral deafness resulting from the blast overpressure.

Despite multiple salvage attempts, progressive tissue necrosis and loss of neurovascular integrity necessitated amputation of the left forelimb. Management included serial debridement, intensive wound care, and nutritional support via an esophagostomy tube. Rehabilitation focused on early physiotherapy and assisted mobility using a harness and cart. The patient adapted rapidly to three-

legged ambulation, eventually achieving complete wound healing and a return to a high quality of life.

Conclusions:

This case underscores the unique challenges of managing high-energy fragmentation trauma. Success in such catastrophic cases requires a multidisciplinary approach, combining aggressive surgical intervention with intensive nursing care and persistent rehabilitation. The recovery demonstrates that favorable long-term outcomes are possible in war-related trauma through comprehensive stabilization and specialized supportive care.

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SUCCESSFUL TREATMENT OF A DELAYED HEMOLYTIC XENOTRANSFUSION REACTION LEADING TO A SEVERE INFLAMMATORY RESPONSE

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Background:

Hyperthyroidism is the most common feline endocrinopathy. Treatment can include thioureylene medication administration but adverse effects of these drugs include blood dyscrasias, occurring in 4% of treated cats.

Due to a lack of commercial availability of feline packed red cells in the UK, canine xenotransfusions may be given to anemic cats in emergent situations. Acute and delayed hemolytic transfusion reactions (DHTRs) to xenotransfused cells have been described, though adverse systemic effects have not been reported.

Case presentation:

A recently diagnosed hyperthyroid thirteen-year-old female neutered domestic shorthair cat developed severe anemia (PCV of 9% and TS 65 g/l) and thrombocytopenia ($7x10^9$/L) three weeks after initiation of thiamazole therapy. The patient was administered a 20ml/kg xenotransfusion of canine packed red blood cells and was started on immunosuppressive corticosteroid therapy. After initial clinical improvement, the cat became systemically unwell 72-96 hours after the xenotransfusion with a marked pyrexia (41.2°C), obtundation, tachypnoea, hypotension (systolic blood pressure 60mmHg) and hyperbilirubinemia (373mmol/l (RI 5-60mmol/l). The patient had mild anemia (PCV 20% and TS 60g/dl) and a leukocytosis ($45.20x10^9$/l).

The patient was successfully managed with bolus isotonic crystalloid fluid therapy (2x 5ml/kg), nor-epinephrine (titrated up to 0.5µg/kg/minute), hydrocortisone infusion (0.25mg/kg/hour), feline fresh frozen plasma (30ml) and intensive nursing care. Immunosuppressive corticosteroids were continued, the anemia and thrombocytopenia improved and the patient was discharged, without requiring a feline red cell transfusion, 6 days later. The patient was subsequently managed with a commercial iodine-restricted diet prior to successful radioactive iodine treatment and remains clinically well.

New/unique information:

The patient met three of the four recognised criteria for systemic inflammatory response syndrome (SIRS), presumed due to a delayed hemolytic transfusion reaction to the xenotransfusion. Whilst fatal reactions to repeated xenotransfusions have been reported, the literature reports only mild transfusion reactions after a single xenotransfusion. The potential to mistake such a severe reaction for other differential diagnoses including sepsis is present. This case highlights that, whilst features of SIRS can occur with a DHTR, with prompt recognition and treatment, these cats can be successfully managed.

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SAVED BY THE ESOPHAGUS: A FELINE TRACHEAL EMERGENCY

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Background:

Tracheal injuries in cats can result from trauma, intubation, or foreign body removal. While a few case reports describe post-intubation tracheal injuries in cats, reports of complete tracheal lumen obliteration with temporary esophageal interposition are not described.

Case Description:

A two-year-old neutered male European Shorthair cat, Oscar, was referred to the emergency department for respiratory distress and stertor, by another hospital where it was anesthetized for removal of a linear gastrointestinal foreign body.

On presentation, the cat was sedated and the evaluation of the oral cavity and thoracic and abdominal point-of-care ultrasonography revealed no abnormalities explaining the respiratory signs. Emergency endoscopy was therefore performed to assess tracheal patency and revealed a tracheal stricture in the cervical and cranial thoracic trachea.

The cat was managed in an oxygen cage with continuous sedation using dexmedetomidine (2 µg/kg/h). Thoracic and cervical radiographs obtained the following day revealed pneumomediastinum without pneumoderma, but no evident signs of tracheal rupture. During the second night of hospitalization, the cat developed severe, diffuse pneumoderma and anisocoria, without overt respiratory distress.

A total-body computed tomography (CT) scan revealed complete obliteration of the tracheal lumen at the level of the cranial thoracic trachea secondary to a tracheal wall discontinuity. A concurrent esophageal wall defect could not be completely excluded. A subsequent endoscopic examination confirmed that the structure protruding into the tracheal lumen was consistent with the esophagus, which showed no evidence of laceration. Monolateral laryngeal paralysis was also noted before intubation.

Intraoperatively longitudinal tracheal defect was identified (temporarily sealed by esophageal interposition between the tracheal margins) and sutured. Postoperative recovery was uneventful, and the cat was discharged two days after surgery. Follow-up endoscopy six days later showed resolution of the tracheal lesion, with persistent laryngeal paralysis. Four months later, laryngeal endoscopy confirmed complete resolution of the paralysis.

Key Learning Points:

Tracheal injuries, although rare in cats, may occur following intubation. Pneumomediastinum, pneumoderma, and pneumoperitoneum should raise suspicion of tracheal wall disruption and prompt advanced imaging. Esophageal interposition may temporarily mask the severity of tracheal

injury. Early diagnosis, surgical management, and follow-up endoscopy are essential for successful outcomes.

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USE OF A LABETALOL CONTINUOUS RATE INFUSION IN THE MANAGEMENT OF HYPERTENSIVE EMERGENCY IN A FELINE PATIENT

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Background:

Hypertensive emergencies are uncommon presentations in veterinary medicine and evidence particularly for feline patients is limited. Previously described treatments include hydralazine, amlodipine, fenoldopam, however no previous literature has described the use of a labetalol infusion in feline patients for hypertensive crises.

Case presentation:

A 13-year old female neutered domestic shorthair was referred to the Emergency and Critical Care department with severe hypertension and deteriorating neurological status. On presentation, the patient's systolic blood pressure was 235mmHg. Neurological assessment revealed stupor, with a modified Glasgow Coma Score of 6/18. There was evidence of retinal detachment and point of care bloodwork demonstrated a moderate metabolic alkalosis. Labetalol therapy was chosen given the patient's emergent status, inability to tolerate oral medication and absence of other available parenteral therapies. A loading dose of 0.25mg/kg was given intravenously, followed by a continuous infusion at an initial rate of 25µg/kg/min. The patient's systolic blood pressure was monitored closely and a 5% decrease in blood pressure was achieved within the first hour, followed by a 15% decrease over the following six hours. This was in line with published consensus guidelines. The ongoing infusion rate was titrated to effect and discontinued after 54 hours, with introduction of oral amlodipine after 20 hours of labetalol infusion. The patient developed hypokalaemia 12 hours after admission. Intravenous potassium supplementation was initiated and subsequently weaned off once the patient was eating voluntarily and she remained normokalemic. No other adverse events were reported during or following the infusion.

Further tests led to a diagnosis of hyperthyroidism and primary hyperaldosteronism in this patient, for which specific therapies (methimazole, spironolactone) were also instituted. The patient's systolic blood pressure normalised, her retinal detachment resolved and she was discharged after four days. Follow up at six months found the patient to be doing well.

New/unique information

This abstract describes the successful use of labetalol for management of hypertensive emergency in a cat. Further studies are required to enable comparison of the efficacy and safety of labetalol versus alternative therapies across a variety of underlying aetiologies.

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USE OF VETRESQ HEMOPERFUSION DEVICE WITH HEMODIALYSIS IN THE MANAGEMENT OF SEVERE HYPERBILIRUBINEMIA IN A DOG WITH LEPTOSPIROSIS INFECTION

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Background:

To describe the use of adsorption hemoperfusion with the VetResQ hemoperfusion (VRQH) device and intermittent hemodialysis (HD) to treat severe hyperbilirubinemia secondary to leptospirosis infection in a dog.

Case presentation:

A 7-year-old female spayed Dachshund was presented for leptospirosis infection. The dog presented with generalized icterus, anuria, subcutaneous edema (suspect fluid overload), severe azotemia, and severe hepatopathy with hyperbilirubinemia. She had two HD treatments after which her anuria resolved and her azotemia improved. Her hyperbilirubinemia continued to worsen. In-series VRQH and HD was subsequently performed, with serial pre- and post-filter bilirubin measurements used to assess changes over time. A total of 1.33L/kg of blood (17 blood volumes) was processed over 240 minutes, resulting in a 57.5% decrease in serum total bilirubin levels. The greatest reduction occurred during the first hour of treatment. The previous intermittent hemodialysis with a high flux dialyzer decreased the total bilirubin by only 25%. The dog developed pulmonary hemorrhage shortly after VRQH and HD treatment, subsequently resulting in cardiopulmonary arrest and death. No further follow-up was possible and no post-mortem examination was performed.

New / Unique Information:

This is the first clinical report demonstrating the efficacy of the VetResQ hemoperfusion device and hemodialysis, using pre- and post-filter comparisons over time, for the treatment of hyperbilirubinemia in veterinary medicine.

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Poster Abstract – Student Grant Case Report

MECHANICAL VENTILATION AND PERITONEAL DIALYSIS IN A DACHSHUND FOLLOWING ACUTE TOAD POISONING.

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Background:

Toad (*Bufo spp.*) intoxication is a common veterinary emergency in Europe, particularly in dogs, given the widespread distribution of these amphibians. The severity of clinical signs correlate with the amount of venom absorbed, ranging from mild signs—such as hypersalivation, mucosal irritation or dyspnoea—to severe, life-threatening manifestations. The venom primarily consists of bufagenins and bufotoxins; these compounds act similarly to digitalis glycosides by inhibiting the Na+/K+-ATPase pump. Consequently, they can induce cardiac arrhythmias, electrolyte imbalances, and systemic compromise. This report describes the clinical progression of a fatal case of rapid-onset MODS complicated by severe arrhythmias, coagulopathy and acute kidney injury.

Case presentation:

A 5-year-old intact male Dachshund presented comatose (Modified Glasgow Coma Scale 3/18), hypothermic (36.4ºC) and bradycardic (64 bpm) with miosis after contact with a large toad. Pulse oximetry revealed a peripheral oxygen saturation of 96% and non-invasive blood pressure showed a sustained hypotension. Venous blood gas revealed a severe mixed acid-base disturbance with acidemia (pH 7.21), hyperkalemia (5.9 mmol/L), hyperlactatemia (8 mmol/L), and azotemia (creatinine 4.2 mg/dL). ECG monitoring initially identified second-degree AV blocks, which progressed to a sustained accelerated idioventricular rhythm (AIVR). Diagnosis was severe bufotoxin intoxication. Multimodal treatment included oxygen, atropine, bicarbonate, adrenaline and intravenous lipid emulsion (ILE) for stabilization and decontamination. Due to respiratory failure and neurological decline, the patient required mechanical ventilation. Persistent oligoanuric AKI (grade V), was managed with peritoneal dialysis. Despite transient neurological improvement and extubation after 15 hours, the patient deteriorated 4 hours later with hematochezia, petechiae, pigmenturia, and significantly prolonged clotting times confirming the development of disseminated intravascular coagulation, necessitating fresh frozen plasma transfusion. However, the patient developed pulmonary edema and bradycardia non-responsive to atropine, leading to cardiac arrest.

Key learning points:

1. Clinical signs of *Bufo spp*. intoxication can vary dramatically, ranging from mild to severe life-threating disease, depending on the amount of toxin absorbed.

2. Early and aggressive decontamination is critical; ILE might help neutralizing circulating lipophilic bufotoxins.

3. Despite the use of advanced life support (ventilation, dialysis), prognosis for rapid-onset MODS remains poor due to the combination of refractory coagulopathy and cardiovascular collapse.

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